THE STATE OF SOUTH CAROLINA

In The Supreme Court

APPEAL FROM GREENVILLE COUNTY
Court of Common Pleas

G. Edward Welmaker, Circuit Court Judge

Appellate Case No. 2013-001945

Charles Christopher Williams, Appellant,

v.

The State of South Carolina, Respondent.

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STATE OF SOUTH CAROLINA

COUNTY OF GREENVILLE

IN THE COURT OF COMMON PLEAS
2010-CP-39-0840

CHARLES CHRISTOPHER WILLIAMS,

Petitioner,

-vs-

THE STATE OF SOUTH CAROLINA,

Respondent.

APPENDIX OF FAS PUBLICATIONS

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Fetal Alcohol Syndrome and Fetal Alcohol Spectrum Disorders: A brief history

BY KENNETH LYONS JONES, M.D.
AND ANN P. STREISSGUTH, PH.D.

A similar pattern of altered growth and morphogenesis, referred to as Fetal Alcohol Syndrome (FAS), was reported in 1973 in eleven unrelated children all of whom were born to chronic alcoholic women who continued to drink heavily throughout pregnancy (Jones, Smith, Ulleland, & Streissguth, 1973; Jones & Smith, 1973). Soon after those initial reports, evidence became available indicating that an association between heavy maternal alcohol consumption and serious problems in the offspring was not a new observation.

For example, in response to outcries from social critics regarding the extent of alcohol consumption in 18th century London, a select committee of the British House of Commons was established in 1834 to investigate drunkenness, prior to the establishment in that same year of an Alcoholic Licensure Act (House of Commons, 1834). Evidence presented to that

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committee indicated that infants born to alcoholic women sometimes had "a starved, shriveled, and imperfect look." Thereafter, in 1899, Dr. William Sullivan investigated female alcoholics at the Liverpool Prison (four). He was able to document an increased frequency of early fetal death and early infant mortality in their offspring (Sullivan, 1899). Despite these worrisome indications that maternal alcoholism might lead to developmental anomalies in their offspring, no serious evaluation of the problem was undertaken and the medical community generally remained agnostic regarding this issue.

That was, in fact, the case in February 1973 when Dr. David Smith, a Professor of Pediatrics at the University of Washington, and his postdoctoral fellow (KLJ), were asked by Dr. Shirley Anderson, who directed the Pediatric Outpatient Clinic at the King County Hospital in Seattle, to come down from his office at the University of Washington to evaluate eight children, all of whom had been born to alcoholic mothers.

Those eight children were there that morning thanks to the intellectual curiosity and perseverance of Dr. Christie Ulleland, a pediatric resident at the University of Washington. One night five years earlier, Dr. Ulleland was informed by an Obstetric Resident that an alcoholic woman was in labor and that delivery was imminent. She raced to the library to find out everything she could about the effects of alcohol on fetal development and discovered that there was no information. Based on that lack of data, she set out to learn everything she could about the topic. From 1968 to 1969, she was able to find eleven babies who had been born to alcoholic women (Ulleland, Wenneberg, Igo, & Smith, 1970; Ulleland, 1972). Soon thereafter, Dr. Ulleland accepted an opportunity to join a pediatric practice in the Seattle area and she turned over the care of those children to Dr. Shirley Anderson.

Dr. Anderson invited eight of those eleven children to the Pediatric Outpatient Clinic at King County Hospital one morning for an evaluation by Dr. Smith. As one child after

another was examined, it became clear that four of the eight had a very specific pattern of malformation that included: microcephaly, short palpebral fissures, and a smooth philtrum. Later that day Dr. Smith's "unknown files," (which included hundreds of children with birth defects whom he had evaluated but had been unable to diagnose), were searched for children with the same three features. Two children were identified and when their mother's charts were evaluated it was discovered that both children had been born to alcoholic women.

Over the next week, two additional children were identified, one in Dayton, Ohio, where Dr. Smith was a visiting professor, and one at the Children's Orthopedic Hospital and Medical Center in Seattle. Over the subsequent two months, seven of the eight children underwent evaluations with one of us (APS), a psychologist at the University of Washington. In all, the children's social and motor performance was delayed. Fine motor dysfunction, including tremulousness, weak grasp, and/or poor hand eye coordination was present in the majority; most were delayed in gross motor performance. Their IQ and motor development index ranged from < 50 to 83.

Shortly after this first paper was published (Jones et al. 1973) three other patients were identified who were born to alcoholic mothers, including two who were identified as newborn infants (Jones & Smith, 1973). One of these died at five days of age and necropsy revealed serious dysmorphogenesis of the brain. Based on these observations, the term Fetal Alcohol Syndrome was first introduced.

Soon after the initial publications describing Fetal Alcohol Syndrome, a study published by Dr. Paul Lemoine of Nantes, France was identified. One of us (KLJ) wrote Dr. Lemoine and he wrote back saying that he had published a paper in 1968 in which he identified features very similar to what we had described in *Lancet* (Lemoine, Harousseau, Borteyru, & Menuet, 1968). "However," he wrote, "my French colleagues

did not believe me then and they do not believe me to this day."

It is important to recognize that Lemoine's colleagues in France were not the only naysayers. If something as common as alcohol, it was reasoned, had such a profound effect on fetal development, it would have been recognized years before as a human teratogen. Ironically, observations regarding the adverse effects of alcohol on fetal development had previously been made. Unfortunately, no one took those observations seriously. It took someone with immense creativity, intelligence, and energy, who trained himself to observe trifles on a physical examination, to make others recognize the importance of that observation. That man was David W. Smith, the father of the field of dysmorphology (Smith, 1970).

In 1974, the three of us (Drs. Smith, Jones, and Streissguth) flew to Washington D.C. to access a large national data base from the Collaborative Perinatal Project of the National Institute on Neurologic Diseases and Stroke. Begun in 1960, this was the most comprehensive study ever conducted on the causes of neurological impairment in children. Unfortunately, no one had imagined then that alcohol could be among the culprits, and no questions on maternal alcohol use or problems had been included in the protocol. However, in some instances, clinicians had noted in the medical records that a mother appeared to have alcohol problems when she came in for her prenatal care. These women became the focus for our study. Although there were only 23 clearly alcoholic mothers identified, the potential of the control group was enormous. Multidisciplinary teams of specialists had systematically examined the children of 55,000 women from 12 sites across the United States for the first seven years of their lives. For our study, the Perinatal Collaborative Project team matched the child of each of the 23 alcoholic mothers with the children of two control mothers from the pool of 55,000, matching on seven factors thought to affect child outcomes.

For our data gathering on this study we each had separate data abstracting tasks, and we did not discuss the data with each other as we abstracted. Without knowledge of child outcomes, Dr. Smith examined the maternal records of all mothers who had been flagged in the clinical notes as having signs of alcoholism. Without knowledge of the maternal history, Dr. Jones examined the 46 child pediatric records and Dr. Streissguth examined the comparable psychological records. In 1974, just one year after the first paper naming Fetal Alcohol Syndrome was published, we published the results of this case/comparison group study (Jones, Smith, Streissguth, & Myrianthopoulos, 1974). As the paper was able to adjust for the potentially confounding effects of socioeconomic background, maternal education, race, maternal age, parity, marital status, and regional affiliation, it became an important early document in clarifying that the fetal alcohol effects we described were not just a matter of poor upbringing.

Compared with nonalcoholic mothers from very similar backgrounds, the children of alcoholic mothers had much worse outcomes, including a perinatal mortality of 17% compared to 2% for the controls and a 32% rate of FAS compared to none for controls. In the survivors, borderline to moderate mental deficiency at 7 years of age was the most frequent problem, at 44% for children of alcoholic mothers vs. 8% for controls (Jones et al., 1974).

In the years since the initial delineation of FAS, it has become clear that prenatal exposure to alcohol may produce a broad spectrum of defects referred to now as Fetal Alcohol Spectrum Disorder (FASD). At the most severe end of the spectrum are children with the full-blown Fetal Alcohol Syndrome, the principal features of which include alterations of growth and performance as well as the characteristic facial features including short palpebral fissures, a smooth philtrum, and a thin vermillion border of the upper lip. It is now recognized, however, that the full spectrum includes children with prenatal alcohol exposure who have only some characteristic

physical features accompanied by neurobehavioral impairment (partial FAS or PFAS), as well as prenatally exposed children who may have no characteristic physical features but are neurobehaviorally compromised (Alcohol Related Neurobehavioral Disorders or ARND) (Stratton, Howe, & Battaglia, 1996). If the true prevalence of this disorder is to be fully appreciated, it is imperative that this broad spectrum of abnormalities associated with prenatal alcohol exposure be fully understood.

The prevalence of FAS is thought to be underestimated due to a number of factors: (a) the physical features of FAS are often subtle and extremely difficult to recognize in the newborn period, (b) there is a lack of clinical expertise among general pediatricians in diagnosing affected children, as well as a very limited number of expert diagnostic teams available for referral, and (c) the stigma associated with maternal alcohol use leads to both underreporting by mothers, and reluctance of clinicians to ask women about alcohol consumption during pregnancy. As a result, estimates gathered from usual sources for vital statistics on birth defects, such as birth certificates, have suggested that the prevalence of FAS is relatively low—approximately 1/10,000. More active ascertainment methods that involve casefinding, for example, among patients of specialty physicians, suggest that the prevalence of FAS is at least an order of magnitude more common, or 1 per 1,000 live births (Abel, 1998).

The situation with respect to underrecognition of FAS is compounded with respect to the full spectrum of FASD. There is no clear-cut alcohol-specific neurobehavioral phenotype for ARND, and in the absence of alcohol-related physical features, it is necessary to document significant prenatal alcohol exposure followed by neurobehavioral deficits in order to qualify for an ARND diagnosis. There is only one American study published well over a decade ago that attempted to systematically measure the prevalence of both FAS and ARND (Hanson, Streissguth, & Smith, 1978). This study, conducted

in Seattle, Washington, was part of a longitudinal prospective study in which 1500 mothers had been queried about alcohol use during pregnancy, and approximately 500 of their children were given several waves of examinations between day 1 and 7 years of age, measuring attention, neuromotor and mental function, and learning disabilities (Streissguth, 2007). On day 1 of their lives, a dysmorphologist, Dr. James Hanson, conducted blind examinations of two babies per morning (one alcohol exposed and one control), recording signs of FAS (Hanson et al., 1978). From these data, Sampson and colleagues later reported an incidence of FAS of at least 2.8/1,000 live births in 1975 and estimated the combined rate of FAS and ARND in Seattle to be at least 9.1/1,000 (Sampson et al., 1997). Although the term FASD had not yet appeared in 1997, this appears to be the first estimate of the prevalence of FASD. The rate of nearly one in every 100 live births confirms the perception of many health professionals that fetal alcohol exposure is a serious problem, and ranks FASD within the top two most common developmental disabilities of any type.

Relative to training general pediatricians in diagnosis of affected children, several guidelines for referral and diagnosis of FAS have been introduced. Beginning in 2003, an International Collaborative Initiative on the Fetal Alcohol Spectrum Disorders was funded in sites throughout the world by the National Institute of Alcohol Abuse and Alcoholism. This consortium was established to better understand the mechanisms, diagnostic approaches, prevention, treatment, and the full spectrum of effects in alcohol-exposed pregnancies. As part of that initiative, some sites have incorporated specialized training of pediatricians to increase the number of physicians who have expertise in the diagnosis of FAS.

In one of these sites, the Moscow Region of Russia, two dysmorphologists conducted a two-day training program for four pediatricians in the diagnosis of the physical features of fetal alcohol syndrome. Dysmorphologists and pediatricians

worked in teams to examine children, demonstrate techniques, and validate that pediatricians could identify physical features of children in 41 boarding schools and orphanages. Those children diagnosed with fetal alcohol syndrome or partial fetal alcohol syndrome (PFAS) by the pediatricians were then evaluated by the dysmorphologists. Accuracy of the diagnosis of FAS and PFAS was assessed. A total of 110 children were examined by both pediatricians and dysmorphologists. Of these, 79 were identified with FAS by the pediatricians; in 66 (83.5%) of these children, the diagnosis was confirmed by the dysmorphologists. Among 31 children who were classified as PFAS by the pediatricians, 21 (67.7%) were confirmed with either FAS or PFAS by the dysmorphologists. It was concluded that after a relatively short training session, pediatricians were reasonably accurate in diagnosing fetal alcohol syndrome on the basis of physical features and in recognizing most of the selected specific features associated with the disorder (Jones et al., 2006).

Although some countries such as Sweden and Canada, which have health care for all, have begun to identify and accommodate the needs of mothers with alcohol problems and their children, this has not occurred in the United States. On the other hand, the United States has been a leader in raising awareness of the potential dangers associated with drinking alcohol during pregnancy. For example, the Surgeon General's warning against alcohol use during pregnancy specifically states that women who are pregnant or who may become pregnant should abstain from alcohol (Surgeon General, 1981). Furthermore, the Alcoholic Beverage Labeling Act is a federal law that requires that the labeling of alcoholic beverages sold carry a government warning against alcohol use during pregnancy (Alcohol Beverage Labeling Act, 1988). France may have been the next country to follow suit, beginning around 2006. On the other hand, restrictions on public advertising of alcohol beverages in print, radio and television have not, to our knowledge, been successful in any country, and medical schools have not introduced courses on

FASD in the United States or in any other country. Hopefully, the papers in this issue of *Journal of Psychiatry and Law* will enlighten the legal professions about the vast implications of FASD for their clientele.

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Fetal Alcohol Syndrome:



Guidelines for Referral and Diagnosis

National Center on Birth Defects and Developmental Disabilities
Centers for Disease Control and Prevention
Department of Health and Human Services

in coordination with

National Task Force on
Fetal Alcohol Syndrome and Fetal Alcohol Effect

CDC

American Academy of Pediatrics

March of Dimes

N·O FAS

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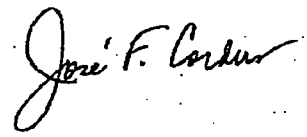
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Foreword

The National Center on Birth Defects and Developmental Disabilities at the Centers for Disease Control and Prevention, in collaboration with the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect, is pleased to present *Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis.* This document represents the deliberations of clinicians, researchers, parents, and representatives of governmental and non-governmental organizations, whose main goals were to increase the identification of individuals with fetal alcohol syndrome (FAS) using uniform criteria, and to improve the delivery of appropriate services to those individuals and their families. These new guidelines will help achieve those goals by educating medical and allied health professionals about FAS.

In 2003, we, in the FAS research and practice communities, celebrated the 30th anniversary of the first reports describing fetal alcohol syndrome. Since that time we have learned a great deal about this preventable condition. We now recognize that FAS represents the tip of the iceberg and that there is a continuum of outcomes associated with prenatal exposure to alcohol. These guidelines were undertaken, in part, as an effort to facilitate further identification, understanding, and study of all conditions resulting from prenatal exposure to alcohol. They build on previous work and incorporate important scientific and clinical knowledge that has been obtained in recent years. CDC is pleased to provide continuing support for the expansion and refinement of scientific descriptions for FAS and other disorders related to prenatal exposure to alcohol through its ongoing work with the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect and the federal Interagency Coordinating Committee on Fetal Alcohol Syndrome (ICCFAS).

Preventing all adverse outcomes associated with prenatal alcohol exposure remains a primary goal of CDC, as well as the entire U.S. Department of Health and Human Services. CDC is committed to working with other federal agencies, organizations in the private sector, relevant partners, and the public to achieve this goal. Similarly, CDC is committed to enhanced early identification of individuals with FAS and related disorders to ensure their access to appropriate services. These latest guidelines for referral and diagnosis are an important step towards that goal. Together we will ensure all persons with FAS and related disorders develop optimally and reach their full potential.



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Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis

EXECUTIVE SUMMARY

As part of the fiscal year 2002 appropriations funding legislation, the U.S. Congress mandated that the Centers for Disease Control and Prevention (CDC), acting through the National Center on Birth Defects and Developmental Disabilities (NCBDDD) Fetal Alcohol Syndrome (FAS) Prevention Team and in coordination with the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect (NTFFAS/FAE), other federally funded FAS programs, and appropriate non-governmental organizations, would:

- Develop guidelines for the diagnosis of FAS and other negative birth outcomes resulting from prenatal exposure to alcohol,
- Incorporate these guidelines into curricula for medical and allied health students and practitioners, and seek to have them fully recognized by professional organizations and accrediting boards, and
- Disseminate curricula to and provide training for medical and allied health students and practitioners regarding these guidelines.

Through the coordinated efforts of CDC, the NTFFAS/FAE, and a scientific working group (SWG) of experts in FAS research, diagnosis, and treatment, the following diagnostic criteria were developed over a 2-year period:

Facial dysmorphia

Based on racial norms, individual exhibits all three characteristic facial features:

- Smooth philtrum (University of Washington Lip-Philtrum Guide rank 4 or 5)
- Thin vermillion border (University of Washington Lip-Philtrum Guide rank 4 or 5)
- Small palpebral fissures (at or below 10th percentile)

Growth problems

Confirmed prenatal or postnatal height or weight, or both, at or below the 10th percentile, documented at any one point in time (adjusted for age, sex, gestational age, and race or ethnicity).

Central Nervous System Abnormalities

I. Structural

1) Head circumference (OFC) at or below the 10th percentile adjusted for age and sex.
2) Clinically significant brain abnormalities observable through imaging.

II. Neurological
Neurological problems not due to a postnatal insult or fever, or other soft neurological signs outside normal limits.

III. Functional
Performance substantially below that expected for an individual's age, schooling, or circumstances, as evidenced by:

1. Global cognitive or intellectual deficits representing multiple domains of deficit (or significant developmental delay in younger children) with performance below the 3rd percentile (2 standard deviations below the mean for standardized testing)

or

2. Functional deficits below the 16th percentile (1 standard deviation below the mean for standardized testing) in at least three of the following domains:

a) cognitive or developmental deficits or discrepancies
b) executive functioning deficits
c) motor functioning delays
d) problems with attention or hyperactivity
e) social skills
f) other, such as sensory problems, pragmatic language problems, memory deficits, etc.

Maternal Alcohol Exposure
I. Confirmed prenatal alcohol exposure
II. Unknown prenatal alcohol exposure

Criteria for FAS Diagnosis
Requires all three of the following findings:
1. Documentation of all three facial abnormalities (smooth philtrum, thin vermillion border, and small palpebral fissures);
2. Documentation of growth deficits
3. Documentation of CNS abnormality

A primary goal of these guidelines is to provide standard diagnostic criteria for FAS so that consistency in the diagnosis can be established for clinicians, scientists, and service providers. The guidelines are based on state-of-the-art scientific research, clinical expertise, and family input regarding the physical and neuropsychological features of FAS. The SWG sought to harmonize these guidelines with other diagnostic systems currently in use in this country and others (e.g., Canada). The SWG strove to provide a balance between conservative and overly inclusive diagnostic systems. Differential diagnosis from other genetic, teratological, and behavioral disorders was emphasized.

In addition to diagnostic guidelines, guidance about medical, educational, social, and family services appropriate for individuals with FAS and their families are reviewed. Services that are applicable to all individuals with FAS and their families, as well as age-specific services, are included. Such services focus on increasing parent and professional knowledge of FAS, characteristics of the disor-

der, differences between FAS and other disorders, and appropriate techniques for parenting or educating affected individuals.

Prevention of FAS and related disorders is of tremendous public health importance. A large amount of research in recent years has enabled researchers and service providers to develop programs that are effective and targeted to specific populations for reducing the risk of an alcohol-exposed pregnancy, which prevents FAS. This research is reviewed herein and recommendations for identifying and intervening with women at risk for an alcohol-exposed pregnancy are provided.

Finally, these guidelines are not intended to be an endpoint in the discussion of diagnosing FAS. There is a great need to acquire science-based information that will facilitate diagnostic criteria for additional related disorders, such as Alcohol Related Neurodevelopmental Disorder (ARND). These guidelines conclude with a call for further research and continuous refinement of the diagnostic criteria for FAS and related conditions so that affected individuals and their families can receive important services that enable them to achieve healthy lives and reach their full potential.

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Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis

Substantial empirical and clinical scientific evidence has shown that prenatal exposure to alcohol causes damage to the developing fetus. Such exposure is commonly cited as the leading preventable cause of birth defects and developmental disabilities (1-3). Children* exposed to alcohol during fetal development can suffer multiple effects. While the number and severity of negative effects can range from subtle to serious, the negative consequences are lifelong. The effects of prenatal exposure to alcohol and basic diagnostic features of fetal alcohol syndrome (FAS) were first described in the United States (U.S.) medical literature 30 years ago (4-8). In 1981, the U.S. Surgeon General issued a public health advisory warning that alcohol use during pregnancy could cause birth defects (9). Further, mandated labeling of alcohol products was established in 1989 (10). Despite the known adverse effects of prenatal exposure to alcohol, many children who experience these adverse effects do not receive proper diagnosis due to the absence of current diagnostic guidelines. These current guidelines, which were federally mandated of the Centers for Disease Control and Prevention (CDC) in the U.S. Department of Health and Human Services (DHHS) 2002 Appropriations Bill, seek to update and refine diagnostic and referral criteria in light of the scientific and clinical advances in the understanding of this disorder during the past 30 years.

These guidelines are organized into several sections. Background information and a history of the development of these guidelines are presented. Next, revised and refined diagnostic and referral criteria for FAS are described, including the empirical and clinical evidence that support each criterion. Comparison of these guidelines with other diagnostic methods currently in use is provided. Because diagnosis is not the endpoint for most clinicians who see children with FAS, a discussion of the essential services for affected individuals is included. Likewise, prevention of FAS by reducing the number of alcohol-exposed pregnancies is inherent in dealing with the disorder. Therefore, a discussion focused on identifying and intervening with women at risk for an alcohol-exposed pregnancy is provided. Finally, a discussion of future needs and efforts related to FAS and other prenatal alcohol-related disorders conclude this report.

BACKGROUND

Prevalence. Studies by CDC have reported FAS prevalence rates from 0.2 to 1.5 cases per 1,000 births across various populations (11-14). Other studies reflecting a variety of ascertainment methodologies have produced estimates ranging from 0.5 to 2.0 cases per 1,000 live births (15-16). Such rates are comparable with or above other common developmental disabilities such as Down syndrome or Spina Bifida (17). Using the CDC FAS estimates, among the approximately 4 million infants born each year, an estimated 1,000 to 6,000 will be born with FAS. Studies of particularly vulnerable populations yield prevalence estimates that far exceed those of other common disabilities. Disadvantaged groups, Native Americans, and other minorities have been documented to have

* Although referral and diagnosis for FAS can be made throughout the lifespan, the majority of individuals are referred and diagnosed in childhood. Thus, the terms "child" or "children" as used in these guidelines are not intended to preclude referral, assessment, and diagnosis of older individuals.

rates as high as three to five FAS affected children per 1,000 children (18-20). Available data also suggest that poverty is strongly associated with women's alcohol use before and during pregnancy, leading to an excess of children with FAS in impoverished groups (21-22).

The magnitude of the problem is even greater when the risk of FAS is considered by looking at the rate of alcohol-exposed pregnancies. In 1999, over half of all U.S. women of childbearing age reported alcohol consumption in the past month (23). The large majority of these women drank only occasionally, but 15% could have been classified as moderate or heavy drinkers (24-25). During that same period, 13% of women reported consuming five or more drinks on one occasion (binge drinking) in the past month (26). Given that nearly half of all U.S. pregnancies are unintended, and that millions of fertile women are sexually active while not using adequate contraception, an estimated 2% of women could be at risk for an alcohol-exposed pregnancy annually (27). More recently, higher rates have been found among subgroups of women, such as those treated for alcohol and drug problems, and women who have been incarcerated (28). Alcohol-related risk factors include drinking during pregnancy, pattern of alcohol use, alcohol dependence, use of multiple substances, having had a previous alcohol-exposed pregnancy, and having a partner or family member who drinks heavily (29-31). Women who receive little or no prenatal care, are unemployed, are socially transient, have lost children to foster or adoptive care because of neglect, abuse, or abandonment are more likely to have high alcohol use patterns that could affect a pregnancy (22,32). National survey data indicate that, while the percentage of women who abstain from alcohol use during pregnancy has increased slightly in recent years, 13% of women continue to use alcohol during pregnancy (26). Among pregnant women, approximately three percent report binge drinking (i.e., five or more drinks on any one occasion) or frequent drinking (i.e., seven or more drinks per week or five or more drinks on any one occasion) (1,33-34). Clearly, current prevalence rates of affected individuals and alcohol-exposed pregnancies indicate that the magnitude of the problem of FAS is a significant public health concern. However, because of the challenges of establishing accurate and timely prevalence information, the magnitude could be even greater than current data indicate.

Challenges in determining accurate prevalence. Despite the progress made over the past several decades to accurately establish and monitor the prevalence of FAS, the full magnitude of the problem is still not known. Primary care providers and others who care for children do not routinely or consistently identify individuals with FAS, which hinders efforts to account for these children in routine birth defects and developmental disabilities monitoring programs. Studies using multiple data sources (e.g., birth certificates, clinical charts, and medical records) show wide variations in identification of FAS cases depending on the population being surveyed (15,34). Four major factors lead to widespread failure to recognize FAS in primary pediatric care settings resulting in underestimates of the prevalence and impact of FAS (36-38):

- No specific and uniformly accepted diagnostic criteria have been available. The four broad areas of clinical features that constitute the diagnosis of FAS have remained essentially the same since first described in 1973: selected facial malformations, growth retardation, Central Nervous System (CNS) abnormalities, and maternal alcohol consumption during pregnancy. These four areas were reaffirmed in a 1996 report by the Institute of Medicine (IOM; 39-40). However, these broad areas of diagnostic criteria are not sufficiently specific to ensure diagnostic accuracy, consistency, and reliability . For example, clinicians do not have guidance about how many facial features must be present or the timing and severity of growth retardation needed to constitute

FAS diagnostic criteria. Thus, health providers are hampered in their efforts to screen and identify children with FAS.

- **FAS diagnosis is based on clinical examination of features, but not all children with FAS look or act the same.** Because each of the symptoms has a broad range of differential diagnoses, it is easy for a clinician to miss or misdiagnose FAS. Previous guidelines, including those included in the 1996 IOM report, did not account for children of different racial and ethnic groups or individuals of different ages . In addition, symptoms such as growth impairment, cognitive impairment, and learning disabilities can have a range of causes. Some of these causes or disorders have higher visibility and recognition than FAS, leading to misdiagnosis (or at least failure to include FAS in the total diagnosis). For instance, physicians are aware of the high prevalence of Attention Deficit, Hyperactivity Disorders (ADHD), but might not link attention problems to FAS. Without clear diagnostic criteria and instruction on their use, providers will continue to under-identify and under-diagnose FAS.

- **Lack of knowledge and misconceptions among primary care providers.** Many professionals believe that FAS can only occur if the mother is an alcoholic. Few know about the full range or progressive nature of the neurobehavioral symptoms that result from prenatal exposure to alcohol. Some incorrectly believe that FAS only occurs among low-income families or in Native American or other racial and ethnic minority groups (21). Better information on the impact of FAS among all populations and dissemination of race or ethnic variations in the diagnostic criteria can help clinicians understand the risk of prenatal alcohol exposure across populations. Knowledge about subpopulation variations in facial characteristics as well as growth curves for infants by gestational age also are important considerations (41).

- **Lack of diagnostic criteria to distinguish FAS from other alcohol-related conditions.** Creating and using diagnostic guidelines for FAS is a starting point for better defining the continuum of conditions related to prenatal alcohol exposure (40). FAS is a severe outcome of prenatal alcohol exposure. Other outcomes also occur and can result in major deficits. Terms such as Fetal Alcohol Effect (FAE), Alcohol-related Birth Defect (ARBD), and Alcohol-Related Neurodevelopmental Disorder (ARND) have been used to describe a spectrum of conditions related to prenatal alcohol exposure.

These four challenges indicate that what is urgently needed to advance the field of FAS diagnosis are current diagnostic guidelines based on empirical evidence as well as clinical experience. Such guidelines should be based on up-to-date scientific evidence and current clinical practices. Further, such guidelines would allow public health and service professionals to better determine the impact of FAS, and deliver needed services to affected children.

Congressional mandate. As part of the fiscal year 2002 appropriations funding legislation, Congress mandated that CDC, acting through the National Center on Birth Defects and Developmental Disabilities (NCBDDD) and in coordination with the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect (NTFFAS/FAE), other federally funded FAS programs, and appropriate nongovernmental organizations, would:

- Develop guidelines for the diagnosis of FAS and other negative birth outcomes resulting from prenatal exposure to alcohol;

- Incorporate these guidelines into curricula for medical and allied health students and practitioners, and seek to have them fully recognized by professional organizations and accrediting boards; and
- Disseminate curricula to and provide training for medical and allied health students and practitioners regarding these guidelines.

Limit in scope. The mandate to CDC indicates that, in addition to guidelines for FAS, guidelines should be developed for other negative birth outcomes resulting from prenatal exposure to alcohol. However, it was subsequently determined through discussions with all interested stakeholders that the best course was to first develop guidelines for the full FAS diagnosis (see following). Then, in subsequent efforts, these guidelines could be expanded or refined to include other alcohol-related disorders. This approach was determined to provide the most timely and scientifically grounded guidelines at this time. However, this decision does not curtail ongoing efforts to define conditions beyond FAS or develop diagnostic guidelines for those conditions.

Note on terminology. Many terms are used to describe the continuum of effects that result from prenatal exposure to alcohol, including: Fetal Alcohol Effect, Alcohol-related Birth Defects, and Alcohol-Related Neurodevelopmental Disorder. A more recent term that has been introduced is Fetal Alcohol Spectrum Disorders (FASD). In April 2004, several federal agencies [(National Institutes of Health (NIH), CDC, & Substance Abuse and Mental Health Services Administration (SAMHSA)] along with experts in the field were convened at a summit sponsored by the National Organization on FAS (NOFAS) to develop a consensus definition of FASD. The resulting definition, adopted in these guidelines, is:

> *Fetal Alcohol Spectrum Disorders (FASD) is an umbrella term describing the range of effects that can occur in an individual whose mother drank alcohol during pregnancy. These effects may include physical, mental, behavioral, and/or learning disabilities with possible lifelong implications. The term FASD is not intended for use as a clinical diagnosis.*

APPROACH AND METHODS FOR DEVELOPMENT OF GUIDELINES

To meet its Congressional mandate, CDC convened an internal working group, led by staff from the FAS Prevention Team of NCBDDD, to conduct preplanning meetings to determine the best methods for development of all aspects of the guidelines: (a) general framework for referral and diagnosis, (b) development of guidelines for physical features (dysmorphia and growth) as well as exposure, and (c) development of guidance to clinicians concerning potential CNS abnormalities. Each of these aspects involved review of the literature, as well as discussions with consultants, clinicians, researchers, and parents of affected children. A description of general and specific methods of development of each aspect follows.

General review of literature. CDC staff identified various reports and documents to be used as the scientific basis for diagnostic guidelines. The science base for this work included, but was not limited to:

- Published scientific, peer-reviewed, literature on physical and neurodevelopmental effects of prenatal exposure to alcohol;
- The report of the IOM Committee to study FAS (40);
- Results from the work of the NTFFAS/FAE (42) ;
- Criteria from standard, widely used dysmorphology and neurodevelopmental textbooks or guides (40,43-44);
- Research on measuring the FAS facial phenotype (45-47);
- Reports on systems that operationally interpret the 1996 IOM criteria(48-50);
- Experience in developing a surveillance case definition for the Fetal Alcohol Syndrome Surveillance Network (FASSNET) (51);
- Ongoing state surveillance and research data, particularly work of Alaska (19,35,52), Colorado (53), New Jersey, Nevada, Washington, and the Four State FAS Consortium (consisting of North Dakota, Montana, Minnesota, and South Dakota);
- The American Academy of Pediatrics (AAP) August 2000 statements and recommendations on FAS and other effects related to maternal alcohol use (54); Position of the American Academy of Family Physicians, which refers to the AAP statement; and
- Canadian National Committee's efforts concerning standardization of guidelines for screening, diagnosis, and surveillance of FAS.

Framework. Before developing the specifics of the diagnostic and referral guidelines, CDC staff determined that it would be helpful to conceptualize the entire diagnostic and referral process encountered by affected individuals and their families. Such a framework also would assist professionals in understanding their specific role in the referral and diagnostic process, as well as guide them with next steps for each case. The framework was reviewed by health care policy consultants as well as members of the NTFFAS/FAE. Revisions were made based on their suggestions. This framework is presented in detail later in these guidelines.

Creation of the Scientific Working Group. The first step in development of the guidelines was to convene an internal CDC working group. The group consisted of members of the NCBDDD FAS prevention team, geneticists, developmental pediatricians, epidemiologists, and psychologists, as well as other allied health professionals. This internal working group developed a list of potential external experts who could be convened to develop the actual guidelines. The large external panel of experts was designated as a scientific advisory panel. From this panel, a subset of experts formed a Scientific Working Group (SWG) that delineated the specifics of the diagnostic criteria.

The external SWG convened by CDC included researchers, clinicians in general and specialty medicine, representatives from academic centers and state health agencies, as well as consumer representatives from the National Organization on FAS (NOFAS) and The Arc of the United States. The scientific advisory panel met in Atlanta, Georgia, on July 12, 2002, to begin deliberations on the proposed guidelines. At that meeting, four subgroups were created: FAS Referral and Diagnosis; ARND issues; Essential Services for Children with FAS/ARND; and Identifying and Intervening with Women at Risk for an Alcohol-Exposed Pregnancy. The subgroups met and began deliberations related to the guidelines in their respective topic areas.

A subsequent meeting of the SWG occurred on September 20, 2002, in conjunction with an NTFFAS/FAE meeting, also in Atlanta. This offered the opportunity for information sharing and feedback on progress made thus far from a range of stakeholders represented on the NTFFAS/FAE

(e.g., parents, providers, and researchers). The FAS Referral and Diagnostic subgroup and the Essential Services subgroup met to further deliberate on their recommendations. The recommendations from these two subgroups were then presented to the NTFFAS/FAE for review and input. The component of the criteria for FAS screening and diagnosis that presented the most difficulty to the FAS Referral and Diagnosis subgroup was the central nervous system (CNS)/neurobehavioral component. This group felt that members of the ARND subgroup were most qualified to develop that particular component of the criteria.

The ARND subgroup drew important information from a poll of the experts that was conducted to identify the CNS/neurobehavioral domains most affected by prenatal alcohol exposure (further description follows). Results of this poll were incorporated into the FAS Referral and Diagnosis Guidelines within the CNS/neurobehavioral component. The third meeting of the FAS Referral and Diagnosis subgroup was teleconferenced on March 11, 2003, and draft guidelines were reviewed and revised. At that time and based on review of the available scientific evidence concerning diagnosis of ARND, the scope of the guidelines was limited to FAS, with future efforts to be devoted to other prenatal alcohol-related disorders. This revised version of the diagnostic criteria was presented to and approved by the NTFFAS/FAE on March 13, 2003 with recommendations. Consensus among members of the external SWG and the NTFFAS/FAE was used to finalize each criterion of the guidelines for dysmorphology, growth, and prenatal exposure to alcohol. At the December 8 and 9, 2003, meeting of the NTFFAS/FAE, further discussions were held regarding the CNS/neurobehavioral criteria and subsequent revisions were made. Finalization of the FAS criteria was reached during a teleconference of the NTFFAS/FAE on May 13, 2004.

Highlights of deliberations of SWG: Physical criteria. The SWG concluded that a strict definition of FAS should be established first, not including diagnostic terms such as fetal alcohol effect (FAE), alcohol-related neurodevelopment disorder (ARND), and alcohol-related birth defect (ARBD) at this time because of insufficient scientific evidence on which to base diagnostic criteria for these related conditions. The SWG urged that CDC diagnostic guidelines for FAS use objective, quantitative measures to improve accuracy and reproducibility and apply specific case definitions guided by evidence-based knowledge and new technologies. The SWG also recommended use of racial and ethnic norms for facial features, head circumference (occipitofrontal circumference, OFC), and other key features when available.

During its deliberations, the SWG acknowledged the need to keep recommendations for the diagnosis of FAS relevant to practitioners working in clinical settings. Also, the SWG acknowledged differences between screening, diagnosis, surveillance, and research activities and the separate definitional needs of each of these activities. Effective tools and practical strategies for primary care settings were considered. The SWG encouraged CDC to call for action and collaboration among obstetricians, pediatricians, family practice physicians, and others providing primary care and screening services to children. The SWG also discussed the need for more data regarding the range of essential services for children who are diagnosed positive for FAS and stressed the importance of a multidisciplinary plan for care that links to the child's community healthcare provider or medical home.

Central nervous system abnormality criteria. Because the scientific evidence and professional consensus on CNS criteria are not yet at the level of specificity equal to that available for physical features, a different approach was used in developing CNS criteria. Further, it was decided that rather than

creating strict criteria for specific CNS domains and level of severity needed, more general guidelines should be described. Such general guidelines should assist the clinician in identifying areas of deficit most likely to be found with individuals who have FAS, as well as individuals with FAS who have less common types of deficits. This approach was considered optimal because a number of structures of the brain are affected versus a single, isolated structure. This generalized nature of damage from prenatal alcohol exposure can result in a wide array of neurodevelopmental outcomes.

To develop such neurodevelopmental guidelines for referral and diagnosis for FAS, the ARND/CNS subgroup polled clinicians and researchers who have extensive knowledge and experience with individuals who have FAS or other related diagnoses. These experts were individuals who specialize in neurobehavioral issues, have extensive research and clinical experience making the FAS/ARND diagnosis, and have contact with families and children with FAS. They were queried to find out what behavioral domains they encountered most frequently or were most essential for making an FAS/ARND diagnosis. Twenty-two clinicians were contacted and their responses were synthesized. The clinicians were asked to identify five areas of deficit they considered most important for diagnosis of FAS or related disorders. In addition, the clinicians were also asked to identify three to five specific behaviors that could be used as examples of each of the five areas of deficit.

The resulting guideline of neurodevelopmental features associated with FAS are presented as being as inclusive as possible, while understanding that certain areas of neurodevelopmental functions are more vulnerable to prenatal exposure to alcohol. Each domain is presented so as to include exemplars from direct observation or parent report that can be documented through standardized testing.

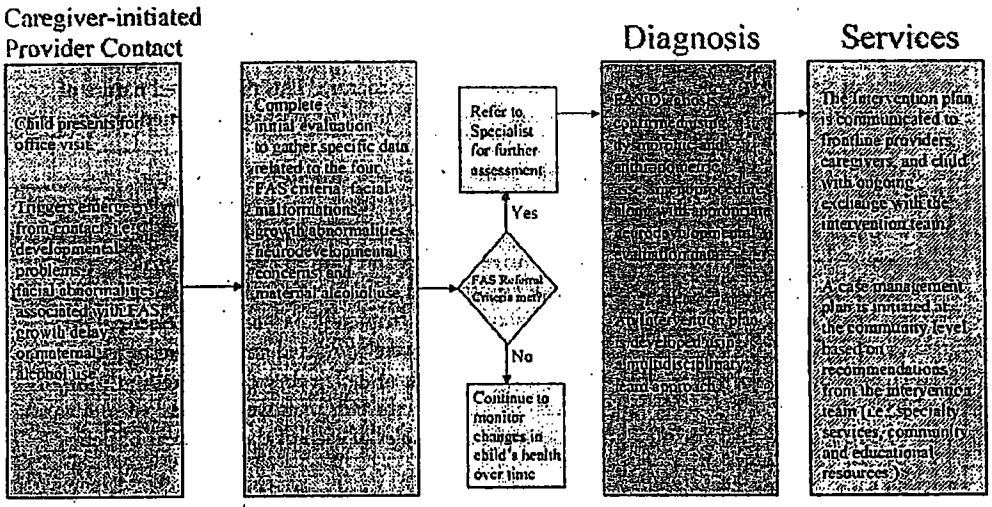
Medical diagnostic criteria are generally evaluated in two ways: (1) the criteria must be reliable and; (2) the criteria must be as valid as possible. The criteria that appear in these diagnostic guidelines meet both of these requirements. In developing these diagnostic guidelines, the SWG also considered the feasibility of applying the criteria in primary care practice and related settings where children are seen.

DIAGNOSTIC AND REFERRAL FRAMEWORK

The framework in Figure I was developed to help guide the discussions of the SWG as they deliberated on the guidelines for referral and diagnosis of FAS. The framework was developed to provide an overview of the entire identification, referral, diagnosis, and treatment process. This overview guided the SWG in identifying key points that needed to be addressed to develop specific guidelines. The framework reflects CDC's recommendation that developmental screening be implemented to improve children's health and help them reach their full potential. A discussion of the major points of the framework follows.

Initial identification. Initial recognition that a child or older individual has a potential problem can come from many sources. Often, parents notice differences between a child and his or her siblings. School systems, including Head Start and daycare staff, interact with a large number of children and often recognize when someone is having difficulty. Social service professionals, such as WIC clinic staff, social workers, and foster care agencies frequently recognize children and individuals having difficulty and needing evaluation. And finally, healthcare providers (particularly pediatricians) often are the first to screen for and detect problems; or obstetricians, who might be aware of

FIGURE 1. Framework for FAS Diagnosis and Services



a maternal substance abuse problem, might refer a newborn. Recognition of many of the problems associated with FAS is exactly the type of condition the "well child" visits to the doctor's office are meant to identify. It is assumed that triggers, such as facial abnormalities, growth delay, developmental problems, or maternal alcohol use, will emerge from the contact. Recognition of a potential problem should lead the provider, regardless of specific profession, to facilitate getting the person and his or her family to the appropriate next step.

Referral. The referral process is initiated at the point a clinician starts to have suspicions of an alcohol-related disorder for a child. This process is facilitated by thorough knowledge of the physical and neurodevelopmental domains affected in individuals with FAS, as well as characteristics that could trigger a referral. Examples of triggers are presented later, in the Referral section of these guidelines. In making a referral for a complete diagnostic evaluation for FAS, it is helpful for the referring provider to gather and document specific data related to the FAS criteria. These data will assist the provider in making the decision to diagnose the child or to refer the child to a multidisciplinary evaluation team for a confirmed diagnosis. In addition, these data could be forwarded to the multidisciplinary evaluation team to guide the diagnostic process. A complete review of systems, noting features consistent with FAS, would be most productive.

Diagnosis. At this stage, the child would be presented to a multidisciplinary team who would engage in a more thorough assessment of the child using FAS diagnostic procedures to evaluate dysmorphia and growth parameters, as well as obtain appropriate neurodevelopmental evaluation data. Once a diagnosis is made, an intervention plan would be developed using a multidisciplinary team approach. A variety of specialists could contribute to the multidisciplinary team, including dysmorphologists, developmental pediatricians, psychiatrists, psychologists, social workers, and educational specialists. Other clinicians, such as pediatricians and family practitioners, also might make the FAS diagnosis, with appropriate training in use of these guidelines. In many rural and less populated regions, these clinicians must make the diagnosis for many types of birth defects and developmental

disabilities. Many of these evaluation services are available within the community setting, for example school systems could provide neurocognitive assessments.

DIAGNOSTIC CRITERIA

Dysmorphia. Human congenital malformations are referred to as dysmorphic features or dysmorphia (55). Dysmorphia occurs when normal morphogenesis is interrupted, creating a particular feature which is shaped, sized, or positioned outside the normal range of development. Alcohol is a teratogen that results in dysmorphia through interference with nerve cell development and functioning, alterations in the ability of cells to grow and survive, increased formation of cell-damaging free radicals, altered pathways of biochemical signals within cells, and altered expression of certain genes and genetic information. In short, alcohol has been shown to interfere with fetal nerve cell development and function in a variety of ways (56-57).

In first describing the dysmorphic features of FAS, Jones and colleagues focused on short palpebral fissure, maxillary hypoplasia (with prognathism), and the presence of epicanthal folds that were observed for a majority of the children described. However, other features also were noted for some patients, including altered palmar fexional crease patterns (i.e., hockeystick crease), cardiac anomalies, joint disability, overlapping fingers, ear anomalies, hemangiomas, ptosis, hypoplastic nails, and pectus deformities (4,5). Over the next 30 years, additional features described included: microcephaly, short nose, smooth philtrum with thin vermillion border, cleft lip, micrognathia, protruding auricles, short or webbed neck, vertebra and rib anomalies, short metacarpal bones, menigomyelocele, hydrocephalus, and hypoplastic labia majora (43).

Despite the heterogeneity of expression for dysmorphic features related to prenatal exposure to alcohol, core facial dysmorphia have emerged through human and animal studies. Experimental studies with a mouse model and primates indicate that the facial dysmorphia observed for individuals with FAS are the result of disturbances of cellular migration during organogenesis along the midline of the face (58). Using anthropomorphic measurements of all facial features, clinical researchers have confirmed the midline feature abnormalities (59). Studies of clinic-referred samples also support these features as discriminant for FAS (60-61). Based on these scientific findings and the extensive clinical experience of the SWG, the following facial dysmorphic features were determined to meet the dysmorphia criteria essential for FAS (based on racial norms):

- Smooth philtrum (measured as 4 or 5 on Lip-Philtrum Guide*)
- Thin vermillion border (measured as 4 or 5 on Lip-Philtrum Guide) (i.e., upper lip)
- Small palpebral fissures (measured as ≤10th percentile according to age and racial norms)

The individual must exhibit all three characteristic facial features; however, additional features also can be present. For example, maxillary hypoplasia is often noted for individuals with FAS as well as those associated features described previously. Cross-sectional and longitudinal studies indicate that many features can change with age or development. After puberty, the characteristic facial features associated with FAS can become more difficult to detect (62). However, recent findings indicate that these three key features remain for the majority of individuals with FAS (47,50).

* University of Washington Lip-Philtrum Guide (49-50).

Use of these three cardinal features (smooth philtrum, thin vermillion border, and small palpebral fissures) to assess whether an individual's dysmorphia is consistent with FAS, is compatible with the IOM report and other diagnostic systems currently in use. Specific criteria were chosen by the SWG to maximize inclusiveness of potential cases on this diagnostic parameter and, therefore, might differ somewhat from other systems currently in use. For example, the 1999 version of University of Washington 4-digit code system uses the same philtrum and vermillion border criteria (as noted by reference to its Lip-Philtrum Guide), but uses a cutoff of the 3rd percentile (2 or more standard deviations below the norm) for palpebral fissures , which is a more conservative cutoff (50). This more conservative approach results in fewer individuals meeting the dysmorphia criteria for an FAS diagnosis but also reduces potential false positives for the diagnosis. Other ways to assess dysmorphia for the FAS diagnosis include checklists or weighted checklists (63-65). Of those checklists reviewed by the SWG, all designated the philtrum, vermillion border, and palpebral fissures as the cardinal facial features of FAS (either by higher weighting or explicit notation). However, because of the cumulative nature of some such lists, an individual who has several of the associated features but not the cardinal features could still be given the FAS diagnosis. Thus, the checklists tend to be more inclusive than the current guidelines, with greater potential for false-positive diagnoses. Review of available diagnostic systems seems to indicate that the dysmorphic criteria agreed upon by the SWG provide a balance between conservative and overly inclusive diagnostic systems.

Differential diagnosis of dysmorphia. Individual dysmorphic features are not unique to any particular syndrome. Even rare defects or certain clusters of dysmorphic features can appear in a variety of syndromes. Therefore, a process of differential diagnosis is essential in making an accurate FAS diagnosis. Following, in Table 1 is a list of syndromes with dysmorphic features that overlap with the primary features of the FAS diagnosis (43). As can be seen from Table 1, none of the syndromes with single overlapping features (except for Toluene embryopathy) have the full constellation of small palpebral fissures, thin vermillion border, and smooth philtrum. However, there are some syndromes in which the constellation of features (primary, occasional features, or both) give a "gestalt" that is similar to the "gestalt" of FAS. These syndromes should be considered in particular when completing the differential diagnosis. Table 2 lists these syndromes, along with the overlapping and differentiating features.

Growth problems. Growth retardation, variably defined, has been documented consistently in individuals with FAS. However, these observations used a variety of parameters (e.g., height, weight, and head circumference), severity levels (below 25th percentile, below 10th percentile, or below 3rd or 2nd percentile), and timing of growth problems (current, birth or present at any point during life). The SWG reviewed available literature, clinical expertise, and practical issues to arrive at benchmarks for each of these three aspects of growth abnormalities.

The primary parameters of growth that need to be impaired to meet the growth retardation criteria of FAS are height, weight, head circumference, or a combination thereof. Anecdotally, a small number of children with FAS have been found to have disproportionate height in relation to weight. However, because multiple organic factors can lead to growth deficiencies (e.g.,. brain structure abnormalities leading to poor skeletal growth or disruption of endocrine function leading to poor weight gain), and because most children with FAS are symmetrical for height and weight (66), it was determined that deficiencies in *either* height or weight, but not height for weight, should be included as growth parameters that might be affected by FAS. Thus, children with growth retarda-

Table 1. Differential diagnosis of individual features associated with FAS

Feature	Syndromes
Smooth philtrum	Cornelia de Lange syndrome
	Floating-Harbor syndrome
	Geleophysic dysplasia
	Opitz syndrome
	Toluene embryopathy
Thin Vermillion border	Miller-Dieker (Lissencephaly) syndrome
	Fetal Valproate syndrome
	Geleophysic dysplasia
	Cornelia de Lange syndrome
	Toluene embryopathy
Small palpebral fissures	Campomelic dysplasia
	DiGeorge sequence
	Dubowitz syndrome
	Duplication 10q sequence
	Duplication 15q sequence
	FG syndrome
	Maternal phenylketonuria (PKU) fetal effects
	Oculodentodigital syndrome
	Opitz syndrome
	Trisomy 18 syndrome
	Williams syndrome
	Velocardiofacial syndrome
	Toluene embryopathy

Note: Features that discriminate these disorders from FAS can be found in Jones, 1997.

tion in height or weight would meet the growth retardation criteria for the FAS diagnosis. Consistent with poor brain development, it was determined that head circumference should be included as a CNS parameter rather than a growth parameter (67-68).

Severity of growth retardation has been defined for each parameter as at or below the 10th percentile or at or below the 3rd percentile by a majority of FAS studies. The primary issue for severity is inclusion or exclusion of children for the FAS diagnosis. Use of the 10th percentile would result in more false-positive FAS diagnoses; while use of the 3rd percentile would result in a greater number of false-negatives. For public health reasons of capturing the largest number of children who might need services, the 10th percentile was chosen by the SWG. As noted previously in the dysmorphia section, use of the 10th percentile strikes a balance among criteria used in other diagnostic systems. Again, the 1999 edition of the University of Washington 4-digit code takes a conservative approach, using the 3rd percentile as the cutoff. Checklist systems often do not specify a particular level of growth retardation and some do not specify which growth parameters should be considered (i.e., height, weight, or height relative to weight). This lack of specificity could lead to inconsistency in diagnostic method, which in turn, could lead to inconsistent application of the FAS diagnosis.

Table 2. Differential diagnosis of syndromes similar to FAS

Syndrome	Overlapping Features	Differentiating Features
Aarskog syndrome	Small nose with anteverted nares, broad philtrum, maxillary hypoplasia, and wide-spaced eyes	Rounded face, down-slant to palpebral fissures, widow's peak, crease below lower lip, incomplete out folding of upper helices, and dental eruption problems.
Williams syndrome	Short palpebral fissures, anteverted nares, long philtrum, depressed nasal bridge, and epicanthal folds	Wide mouth with full lips, stellate pattern of the iris, periorbital fullness, and connective tissue disorders.
Noonan's syndrome	Low nasal bridge, wide-spaced eyes, and epicanthal folds	Down-slant to palpebral fissures, keratoconus, wide mouth, and protruding upper lip
Dubowitz syndrome	Short palpebral fissures, wide spaced eyes, and epicanthal folds	Shallow supraorbital ridge with nasal bridge near the level of the forehead, and broad nasal tip
Brachmann-DeLange syndrome	Long philtrum, thin vermillion border, anteverted nares, and depressed nasal bridge	Single, bushy eyebrow extending across forehead, long eyelashes, downturned mouth, high arched palate, and short limbs (yielding short stature)
Toluene embryopathy	Short palpebral fissures, midface hypoplasia, smooth philtrum, and thin vermillion border	Micrognathia, large anterior fontanel, down-turned mouth corners, hair patterning abnormalities, bifrontal narrowing, and ear abnormalities
Fetal hydantoin syndrome (Fetal dilantin syndrome)	Wide-spaced eyes and depressed nasal bridge	Short nose with bowed upper lip
Fetal valproate syndrome	Epicanthal folds, anteverted nares, long philtrum with thin vermilion border, and wide-spaced eyes	High forehead, infraorbital crease or groove, and small mouth
Maternal PKU fetal effects	Epicanthal folds, short palpebral fissures, long underdeveloped philtrum, and thin vermillion border	Small upturned nose, round facies, and prominent glabella

The current guidelines provide consistent growth parameters that should be assessed, as well as a more inclusive level of the 10th percentile.

The primary issue that emerged in the discussion of timing of growth retardation was whether growth retardation needs to be present at the time of the diagnosis, or whether it could have occurred previously and been resolved. This is particularly important when including prenatal growth retardation or early growth problems due to failure to thrive. Because a great number of treatments exist for growth problems (e.g. feeding tubes or hormone therapy), the SWG adopted the criteria that any history of growth retardation, including prenatal growth deficiencies, should be allowed within the diagnostic criteria (61).

Thus, the growth retardation criteria adopted by the SWG are: confirmed prenatal or postnatal height, weight, or both at or below the 10th percentile documented at any one point in time (adjusted for age, sex, gestational age, and race or ethnicity). The committee noted that the examiner should make sure that the single point in time when the growth deficit was present does not correlate with a point in time when the child was nutritionally deprived.

Differential diagnosis of growth problems. Growth retardation and growth deficiencies occur in children, adolescents, and adults for a great many reasons. Some of the most obvious reasons have to do with insufficient nutrition. This could be a particular problem for infants with poor sucking responses who experience failure to thrive. In addition, several genetic disorders result in specific growth deficiencies (e.g., dwarfism). Prenatal growth retardation can be due to a variety of factors, including maternal smoking or other behaviors leading to hypoxia, poor maternal nutrition, or genetic disorders. Both environmental and genetic bases for growth retardation should be considered for differential diagnosis when considering the FAS diagnosis.

Central nervous system abnormalities (CNS). More than 2,000 scientific papers regarding the teratogenic effects of alcohol exposure for CNS have been published over the past 30 years (69-71). Studies of the impact of fetal exposure to alcohol show a range of short- and long-term cognitive and behavioral outcomes resulting from these CNS abnormalities. Complicating detection of these abnormalities is that FAS neurobehavioral presentation changes across the lifespan (4,61,72-79). Despite this developmental process, CNS deficits generally persist throughout the lifespan (80). Longitudinal studies have found that many adults affected by FAS have complex mental health disorders, are affected by the consequences of neurobehavioral deficits, and are unable to sustain independent living (81).Prenatal exposure to alcohol can result in an array of structural, functional, neurological problems, or a combination of these, as well as abnormalities of the CNS (40). To meet the FAS diagnostic criteria for CNS abnormality, structural, neurological, or functional deficits, or a combination thereof, must be documented. Note that it is also possible for an individual to present with more than one CNS structural, neurological, functional deficit or abnormality. Guidelines for each type of CNS abnormality follow.

I. Structural

1. Documented small or diminished overall head circumference (OFC at or below the10th percentile) adjusted for age and gender (including head circumference at birth; 4,68). For children who have overall growth deficiency (i.e., height and weight below the 10th percentile) to meet this criteria for CNS abnormality, the child's head circumference should be disproportionately small to his or her overall size (i.e., OFC at or below the 3rd percentile).

2. Clinically significant brain abnormalities observable through imaging techniques (e.g. reduction in size, or change in shape of the corpus callosum, cerebellum, or basal ganglia) as assessed by an appropriately trained professional (4,58,82-88).

   Note: An individual could meet the CNS abnormality criteria for the FAS diagnosis through a structural abnormality, yet not demonstrate detectable functional deficits.

II. Neurological

Documented evidence of neurological damage to the CNS. Neurological problems of CNS can include seizures not due to a postnatal insult or fever or other soft neurological signs outside normal limits (e.g., in coordination, visual motor difficulties, nystagmus, or difficulty with motor control; 89-92). As with head circumference, abnormal neurological findings can be most predictive of underlying CNS abnormality due to prenatal alcohol exposure, rather than later environmental factors, in younger children. The use of norm-referenced measures of neurological functioning is recommended.

III. Functional

Assessment findings that indicate deficits, problems, or abnormalities in functional skills of the CNS. Early brain damage is usually generalized rather than specific, with increased specificity of abnormalities revealed as development progresses. The functional abilities affected by prenatal exposure to alcohol vary greatly from person to person, depending on the amount of alcohol exposure, timing of exposure, and pattern of exposure (e.g., chronic exposure versus binge episodes). Despite this inherent variation in effects, several areas of significant functional vulnerability have been observed consistently by clinicians and clinical researchers with particular damage to corresponding structures reported (e.g., corpus callosum, cerebellum, or basal ganglia). Variability in exposure impact results in variability of structural, neurological, or functional deficits, or a combination thereof in affected individuals.

For functional deficits, it is generally accepted that multiple locations in the brain (and corresponding functional capability) are affected by prenatal exposure to alcohol. To address this issue, functional deficits that fulfill the CNS abnormality criteria can be met in two ways:

(1) Global cognitive deficit (e.g., decreased IQ)
or significant developmental delay in children too young for an IQ assessment;

OR

(2) Deficits in THREE or more specific functional domains

These two ways of meeting the criteria for a functional CNS abnormality were adopted because of the composite nature of cognitive/intellectual and developmental measures (93-94). Decreased performance on a standardized measure of cognition/intelligence or development assumes deficits in multiple domains. In the absence of such a measure, several specific domains need to be assessed individually to determine that multiple functional domains have been affected. The specific domains most often cited as areas of deficit or concern for individuals with FAS are described below, although other domains and abilities can be affected and this list is not exhaustive. It should

be noted that for each of the following specific domains described, other agents and environmental factors can produce deficits or outcomes similar to prenatal alcohol exposure, making care differential diagnosis essential. Finally, these descriptions are intended to be suggestive and examples of likely and possible problems a clinician might encounter then need to assess using psychometric instruments. The exemplars are not intended to be exhaustive, or to present a necessary list of behaviors to be used as a checklist without reliable and valid assessment.

a) Cognitive deficits or significant developmental discrepancies. It is important to note that global deficits or delays can leave the child scoring in the normal range of development, but below what would be expected for his or her environment and background (61,95-99). In addition to formal testing (either through records or current testing), behaviors that may be observed (or reported) in the clinical setting that suggest cognitive deficits or developmental delays that should be assessed by standardized testing include but are not limited to specific learning disabilities (especially math and/or visual-spatial deficits); uneven profile of cognitive skills; poor academic achievement; discrepancy between verbal and nonverbal skills; and slowed movements or reaction to people and stimuli (e.g., poor information processing). (75,100-103)

b) Executive functioning deficits. Executive functioning (EF) is defined as the ability to maintain an appropriate problem solving set for attainment of a future goal and that this ability includes the more specific skills of inhibition, planning, and mental representation (104). Behaviors that can be observed (or reported) in the clinical setting that might indicate an EF deficit that should be assessed by standardized testing include, but are not limited to poor organization, planning, or strategy use; concrete thinking; lack of inhibition; difficulty grasping cause and effect; inability to delay gratification; difficulty following multistep directions; difficulty changing strategies or thinking of things in a different way (i.e., perseveration); poor judgment; and inability to apply knowledge to new situations. (105-108)

c) Motor functioning delays or deficits. Both gross motor and fine motor skills can be impaired for individuals with FAS (109-111). Visual-motor/visual-spatial coordination is a particularly vulnerable area of functioning (99,112-113). Behaviors that can be seen (or reported) in the clinical setting that indicate motor problems that should be assessed by standardized testing include, but are not limited to delayed motor milestones; difficulty with writing or drawing; clumsiness; balance problems; tremors; and poor dexterity. For infants, a poor suck is often observed. (61,114-116)

d) Attention and hyperactivity problems. Attention problems are often noted for children with FAS, with many children receiving a diagnosis of attention-deficit hyperactivity disorder (ADHD; 117). Although such a diagnosis can be applied, some research has shown that the attention problems for children with FAS do not seem to fit the classic pattern of ADHD. Individuals with FAS tend to have difficulty with the encoding of information and flexibility (shifting) aspects of attention; whereas children with ADHD typically display problems with focus and sustaining attention (118-119). Individuals with FAS also can appear to display hyperactivity because their impulsivity may lead to increased activity levels. Behaviors that may be observed (or reported) in the clinical setting that suggest attention problems related to FAS that should be assessed by standardized testing include, but are not limited to described by adult as "busy"; inattentive; easily distracted; difficulty calming down; overly active; difficulty completing tasks; and/or trouble with transitions. Parents might report inconsistency in attention from day to day (e.g., "on"

days and "off" days). (79,120-125)

e) **Social skills problems.** The executive, attention, and developmental problems described previously often lead to clinically significant difficulty for people with FAS when interacting with peers and others. Because of the mental representation problems, individuals with FAS often have social perception or social communication problems that make it difficult for them to grasp the subtler aspects of human interactions (107,126-127). Consistent difficulty understanding the consequence of behavior or inappropriate behavior is frequently described for individuals with FAS (62,80). Behaviors that can be observed (or reported) in the clinical setting that indicate these types of social difficulties that should be assessed by standardized testing include, but are not limited to lack of stranger fear; often scape-goated; naiveté and gullibility; easily taken advantage of; inappropriate choice of friends; preferring younger friends; immaturity; superficial interactions; adaptive skills significantly below cognitive potential; inappropriate sexual behaviors; difficulty understanding the perspective of others; poor social cognition; and clinically significant inappropriate initiations or interactions. (128-130) It should be noted that standardized assessment of social problems can be quite difficult. Social functioning is a multifaceted domain that can require several areas of assessment.

f) **Other potential domains that can be affected.** In addition to these five most often cited problem areas, deficits and problems to be assessed by standardized testing can present in several other areas, including sensory problems (e.g., tactile defensiveness and oral sensitivity); pragmatic language problems (e.g., difficulty reading facial expression; poor ability to understand the perspectives of others); memory deficits (e.g., forgetting well-learned material, and needing many trials to remember); and difficulty responding appropriately to common parenting practices (e.g., not understanding cause-and-effect discipline). While abnormalities in these "other" areas have been reported for some individuals with FAS, expert consensus suggests deficits in these areas present at a lower frequency than do those in the other five specific domains (62).

THESE GUIDELINES STRONGLY RECOMMEND THAT FUNCTIONAL DOMAINS BE ASSESSED USING NORM-REFERENCED STANDARDIZED MEASURES. DOMAINS SHOULD BE ASSESSED BY APPROPRIATE PROFESSIONALS USING RELIABLE AND VALIDATED INSTRUMENTS.

Level of functional deficit. In these guidelines, global cognitive deficits and developmental delay, or deficits in three or more specific domains, are defined as performance substantially below that expected for an individual's age, schooling, or circumstances. Several statistical thresholds have been suggested to operationally define performance substantially below expected levels. Previous research indicates that approximately only one-quarter of individuals diagnosed with FAS perform at the most conservative level of below the 3rd percentile (2 standard deviations below the mean) on standardized measures (95). In keeping with this finding, and to adequately capture the full spectrum of effects, the SWG adopted two levels of functional deficits that would meet the criteria for a CNS abnormality: (1) for significant global cognitive deficit performance below the 3rd percentile (i.e., 2 standard deviations below the mean); and (2) for three or more specific domains performance below the 16th percentile (i.e., 1 standard deviation below the mean) on standardized measu res of individual domains. Thus, individuals scoring below the normal range on a global measure of IQ or development and individuals scoring in the below average range on standardized measures of three

specific functional domains would meet the criteria for functional CNS abnormality for diagnostic purposes.

Ideally, functional deficits should be established through appropriate standardized neuropsychological testing by a trained professional. The SWG recognized that such standardized testing might not be readily available in all diagnostic settings. Clinicians are strongly encouraged to supplement their observations by obtaining standardized testing through early intervention programs, public schools, and psychologists in private practice. The SWG emphasizes the need for psychometric testing when evaluating an individual for the FAS diagnosis, and use of clinical judgment alone could veer away from the goal of implementing standard diagnostic criteria for FAS. In addition, such testing will facilitate the development of individualized and appropriate treatment plans for diagnosed individuals. These guidelines are intended to provide information concerning the types of CNS abnormalities that might be observed, as well as the level of deficit that can be expected. In addition, they seek to support the need for quality assessments administered by trained professionals when establishing CNS abnormalities associated with the FAS diagnosis.

These guidelines for assessment of CNS abnormalities for making the FAS diagnosis is in harmony with, although not duplicative of, other diagnostic guidelines and systems. The 1999 version of the University of Washington 4-digit code and the guidelines developed by Health Canada requires performance below the 3rd percentile (2 standard deviations below the mean on standardized testing) in three separate domains in which global deficits count as one domain. Some health systems might find this approach useful in situations in which resources for standardized testing are readily available.

Mental health problems and lifelong consequence. Difficulty in any of the functional CNS areas described above can lead to maladaptive behavior and mental health problems with lifelong consequences. Commonly co-occurring mental health issues (excluding attention problems) reported by clinicians and cited in the scientific literature to date, include ***conduct disorders, oppositional defiant disorders, anxiety disorders, adjustment disorders, sleep disorders, and depression (81,95,131-134).*** Although attention problems can be classified as a mental health issue or psychiatric condition, in these guidelines they are treated as a primary deficit resulting from alcohol-related CNS damage, rather than a secondary mental health issue. There are considerable animal, human, and clinical studies that document attention deficits for many individuals with prenatal exposure to alcohol (117). In addition, decreased adaptive skills and increased problems with daily living abilities have been consistently documented, although further research is needed . Such problems include dependent living conditions, disrupted school experiences, poor employment records and encounters with law enforcement (including incarceration; 95). Although these mental health and very debilitating lifelong consequences should not be used for the purpose of diagnosis, it should be noted that they are very prevalent among individuals with FAS and are very likely to be the presenting conditions that should trigger a referral and comprehensive diagnostic evaluation.

Differential diagnosis of CNS abnormalities. Differential diagnosis of CNS abnormities involves not only ruling out other disorders but also specifying co-occurring disorders. The CNS deficits associated with FAS, in particular functional deficits, can be produced by many different factors in addition to prenatal alcohol exposure. It is important to determine that the observed functional deficits are not better explained by other causes. In addition to other organic syndromes that produce deficits in one or more of the previously cited domains (e.g., Williams syndrome and Down syn-

drome), significantly disrupted home environments or other external factors can produce functional deficits in multiple domains that overlap with the domains that are affected by FAS. In making the differential diagnosis of FAS by ruling out other syndromes, CNS abnormalities should be evaluated in conjunction with dysmorphia and laboratory findings. The more difficult differentiation is for CNS abnormalities resulting from environmental influences (e.g., abuse or neglect, disruptive homes, and lack of opportunities). ***To assist with differential diagnosis between FAS and environmental causes for CNS abnormalities it is important to obtain a complete and detailed history for the individual and his or her family.***

In addition to ruling out other causes for CNS abnormalities, a complete diagnosis should identify and specify other disorders that can co-exist with FAS (e.g., autism, conduct disorder, and oppositional defiant disorder). ***It is very important to note that a particular individual might have a conduct disorder in addition to FAS, but that not all persons with a conduct disorder have FAS and not all individuals with FAS will have a conduct disorder.*** Thus, organic causes, environmental contributions, and comorbidity should all be considered for both inclusive and exclusive purposes when evaluating someone for the FAS diagnosis (62,135). Finally, differential diagnosis for the CNS abnormalities within the FAS diagnosis is extremely difficult and should be conducted by professionals trained not only in the features of FAS, but also in the features of a broad array of birth defects and developmental disabilities so as to understand the distinguishing characteristics.

Maternal alcohol exposure. Documentation and confirmation of prenatal alcohol exposure can be extremely challenging. For birth mothers, admission of alcohol use during pregnancy can be very stigmatizing. The situation can be further complicated if the woman is still using alcohol, especially at high consumption rates. In this situation, information about alcohol use might need to be obtained from other reliable informants, such as a relative. However, the overwhelming situation encountered in the clinical setting is when a child or adult is being evaluated for FAS and little or no information about the index pregnancy is available. This frequently occurs for children in foster and adoptive homes. In this situation, every effort should be made to obtain the necessary information, but lack of confirmation of alcohol use during pregnancy should not preclude an FAS diagnosis if all other criteria are present. This would be considered "unknown prenatal alcohol exposure". In very rare instances, there will be confirmed absence of exposure. Documentation that the birth mother did not drink any amount of alcohol from conception through birth would indicate that the FAS diagnosis is not appropriate. This typically implies that the birth mother knew the date of conception (e.g., a planned pregnancy) and did not consume alcohol from that day forward, or she was prevented from drinking for some reason (e.g., incarceration). It must be noted that simple denials of alcohol use might or might not be credible and corroborating evidence should be obtained whenever possible. However, given the imprecise nature of exposure information, the following two qualifiers for prenatal alcohol exposure were suggested by the SWG:

I. **Confirmed prenatal alcohol exposure** requires documentation of the alcohol consumption patterns of the birth mother during the index pregnancy based on clinical observation; self-report; reports of heavy alcohol use during pregnancy by a reliable informant; medical records documenting positive blood alcohol levels, or alcohol treatment; or other social, legal, or medical problems related to drinking during the index pregnancy.

II. Unknown prenatal alcohol exposure indicates that there is neither a confirmed presence nor a confirmed absence of exposure. Examples include: the child is adopted and prenatal exposure(s) is unknown; the birth mother is an alcoholic, but confirmed evidence of exposure during pregnancy does not exist; and conflicting reports about exposure exist that cannot be reliably resolved.

CRITERIA FOR FAS DIAGNOSIS

A diagnosis of FAS requires the presence of all three of the following findings:

1. Documentation of all three facial abnormalities (smooth philtrum, thin vermillion border, and small palpebral fissures);

2. Documentation of growth deficits; and

3. Documentation of CNS abnormalities (structural, neurological or functional, or combination thereof).

It should be noted that confirmed prenatal alcohol use can strengthen the evidence for diagnosis, but is not necessary in the presence of all the previous findings. Confirmed absence of alcohol exposure would rule out the FAS diagnosis. The FAS diagnosis should be made only after ruling out other possible diagnoses associated with each criterion. A brief outline of the diagnostic criteria for FAS is presented in Table 3.

Changes in presentation of criteria across development. As would be expected for any congenital syndrome, presentation of the diagnostic features can change over development. With regards to facial features (small palpebral fissures, smooth philtrum, and thin vermillion border), it has generally been accepted that these features are most evident during infancy and the early preschool years. However, longitudinal data that specifically address this issue are not available currently. One also might expect growth parameters to change (and generally normalize) with development. This might especially be the case for children who receive nutritional or other interventions. Perhaps the criterion for which the most change over development is observed is CNS abnormalities. While structural abnormalities would remain consistent, neurological and functional deficits could resolve or change presentation at various stages of development. During the newborn and infancy periods, difficulty with arousal or behavior regulation might be observed. Slightly older infants could display delayed or abnormal motor skills. During late infancy approaching the preschool period, general cognitive developmental delay is generally reflected through delayed milestones, especially early language acquisition (note, however, that basic language skills of vocabulary and syntax generally are not areas of deficit for children with FAS). During the preschool period, attention problems and hyperactivity can emerge. In typically developing children, the late preschool through early school age (e.g., 4 through 7 years of age) is when many executive functioning and social perception skills are acquired. For example, simple planning or organization skills are learned, as well as understanding the physical and mental perspective of others. Preschoolers and school-aged children often do not fully acquire these skills. Throughout the school-age period, children with FAS can exhibit deficits or difficulty with any of the CNS domains listed in the diagnostic criteria. However, each child's pattern of strengths and weaknesses is likely to be very individualized. During adolescence and through adulthood, the pattern of deficits continues to be present and in addition, lifelong conse-

Table 3: Brief Outline of Diagnostic criteria for Fetal Alcohol Syndrome

Facial dysmorphia

Based on racial norms, individual exhibits all three characteristic facial features:

- Smooth philtrum (University of Washington Lip-Philtrum Guide rank 4 or 5)
- Thin vermillion border (University of Washington Lip-Philtrum Guide rank 4 or 5)
- Small palpebral fissures (at or below 10th percentile)

Growth problems

Confirmed prenatal or postnatal height or weight, or both, at or below the 10th percentile, documented at any one point in time (adjusted for age, sex, gestational age, and race or ethnicity).

Central Nervous System Abnormalities

I. Structural

1) Head circumference (OFC) at or below the 10th percentile adjusted for age and sex.
2) Clinically significant brain abnormalities observable through imaging.

II. Neurological

Neurological problems not due to a postnatal insult or fever, or other soft neurological signs outside normal limits.

III. Functional

Performance substantially below that expected for an individual's age, schooling, or circumstances, as evidenced by:

1. *Global cognitive or intellectual deficits representing multiple domains of deficit (or significant developmental delay in younger children) with performance below the 3rd percentile (2 standard deviations below the mean for standardized testing)*

   or

2. *Functional deficits below the 16th percentile (1 standard deviation below the mean for standardized testing) in at least three of the following domains:*
   a) cognitive or developmental deficits or discrepancies
   b) executive functioning deficits
   c) motor functioning delays
   d) problems with attention or hyperactivity
   e) social skills
   f) other, such as sensory problems, pragmatic language problems, memory deficits, etc.

Maternal Alcohol Exposure

I. Confirmed prenatal alcohol exposure
II. Unknown prenatal alcohol exposure

Criteria for FAS Diagnosis

Requires all three of the following findings:

1 Documentation of all three facial abnormalities (smooth philtrum, thin vermillion border, and small palpebral fissures);
2. Documentation of growth deficits
3. Documentation of CNS abnormality

quences of those deficits emerge, including mental health problems, inability to achieve independent living, and criminal activity (62,95,126-137).

Individuals who do not meet the full diagnostic criteria for FAS. The 1996 IOM report noted that most individuals with deficits resulting from prenatal exposure to alcohol do not express all of the features necessary to meet the criteria for an FAS diagnosis. This was confirmed in the comments of researchers, clinicians, and members of the NTFFAS/FAE during the deliberations of the SWG. Consensus has not been reached by either of these groups, or the scientific and clinical community at large regarding evidenced-based diagnostic criteria for any prenatal alcohol-related condition other than FAS. However, there is grave concern that individuals who present with the same neurodevelopmental deficits as individuals diagnosed with FAS, but who do not present with the full facial features or growth deficits, are not being afforded services because they are not given a diagnosis of FAS. Ongoing funding has been provided by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) to conduct research that might result in evidence-based diagnostic criteria for individuals with other conditions caused by prenatal alcohol use. Currently, CDC is using a collaborative database of neurodevelopmental data from five intervention studies to explore the nature of individuals who could be considered in the diagnostic category of ARND, as well as data from a prospective cohort study of 5-year-olds in Denmark. However, at this time, the only diagnostic category with scientific evidence to support clinical criteria is FAS. As future data and science are available, these guidelines can be refined and expanded to delineate other conditions resulting from prenatal alcohol exposure.

CONSIDERATIONS FOR A REFERRAL FOR AN FAS DIAGNOSTIC EVALUATION

Very often the front-line providers of services (medical, educational, or social) are faced with making the decision of whether or not to refer a child, individual, or family for a full FAS diagnostic evaluation. The SWG recognizes that this may be a difficult decision. For biological families, there may be social stigma associated with any evaluation concerning prenatal alcohol exposure. In other families, direct information about alcohol use during pregnancy may not be available or only suspected. Thus, the following guidelines were developed to provide assistance in making the referral decision, although, it is recognized that each case must be evaluated individually. Further, these guidelines were developed with the idea that when in doubt, it was preferable to refer for full evaluation by a multidisciplinary team with experience in evaluating prenatal alcohol exposure.

- For situations with known prenatal alcohol exposure: A child or individual should be referred for full FAS evaluation when there is confirmed significant prenatal alcohol use (i.e., 7 or more drinks per week or 3 or more drinks on multiple occasions, or both). If prenatal alcohol exposure in the high risk range is known in the absence of any other positive screening criteria, the primary healthcare provider should document this exposure and closely monitor the childs ongoing growth and development.

- For situations with unknown prenatal alcohol exposure: A child or individual should be referred for full FAS evaluation when:
  - there is any report of concern by a parent or caregiver (foster or adoptive parent) that his or her child has or might possibly have FAS.
  - all three facial features are present (smooth philtrum, thin vermillion border, and small palpebral fissures).

- one or more facial features are present in addition to growth deficits in height or weight, or both.
- one or more facial features are present, along with one or more CNS abnormalities.
- one or more facial features are present, along with growth deficits and one or more CNS abnormalities.

SERVICES APPROPRIATE FOR AFFECTED INDIVIDUALS AND THEIR FAMILIES

Diagnosis is never an endpoint for any individual with a developmental disability and his or her family. This is particularly true for individuals with FAS, their families, and their community. As described in the framework section, the FAS diagnosis and the diagnostic process (especially the neuropsychological assessment) are part of a continuum of care that identifies and facilitates appropriate health care, education, and community services. The learning and life skills affected by prenatal exposure vary greatly among individuals, depending on the amount of alcohol exposure and the timing and pattern of exposure, as well as each individual's current and past environment (138-139). As a result, the services needed for individuals with FAS and their families vary according to what parts of the brain have been affected, the age or level of maturation of the person, the health or functioning of the family, and the overall environment in which the person is living. Thus, service needs for any particular individual and his or her family can be quite individualistic (140).

Despite the required individualization in service needs, some general areas of service and specific services have been identified as helpful to people with FAS and their families (62). While the ideal circumstance are services and interventions that have been specifically developed for individuals with FAS and their effectiveness has been established through rigorous scientific evaluation, such programs are only now being researched and developed. Thus, most evidence for the benefit of services has been gleaned from research with other populations, clinical wisdom, and family experiences. These three sources have drawn heavily from information obtained concerning risk and protective factors that have been found through systematic research, using natural history methodology, to promote positive development or reduce the incidence of negative long-term consequences of FAS (i.e., reduce secondary conditions; 141). First, these factors will be reviewed. Then, services that are applicable to all individuals with FAS, regardless of life stage will be presented. Finally, essential services appropriate for individual life stages will be presented.

Risk and Protective factors. In a landmark study of secondary conditions for individuals with FAS, Dr. Ann Streissguth and her colleagues delineated not only the lifelong consequences of FAS, but also several basic factors that are protective or increase the risk of negative outcomes (95). This study identified many lifelong negative consequences of FAS, including disrupted school experiences, legal problems, incarceration, mental health problems, substance abuse problems, inappropriate sexual behavior, dependent living, and poor employment history. Importantly, these risks are not mutually exclusive. Any individual can (and most likely will) experience multiple risk factors that can have a cumulative effect (141). Moreover, these risks can be exacerbated by family and community expectations. Research has shown a relation between the number of risk factors encountered by an individual and poor outcomes. Reducing the number and severity of risk factors is an important step in providing services to individuals with FAS and their families. The disorder of FAS has often been described as a "hidden" disability because of the subtlety of the dysmorphia and good basic language skills (e.g., vocabulary and syntax) of many affected individuals. These factors lead to individuals with FAS being treated inappropriately because of caregivers' unrealistic

developmental expectations. FAS might be either not recognized or mislabeled as stubbornness or "bad" behavior by a caregiver, or others who encounter the affected individual (e.g., teachers, extended family, and friends). Families raising children with developmental disabilities typically report considerable parental stress related to this aspect, particularly for parents of children with FAS (62).

Several factors have been identified that can potentially reduce the odds of long-term negative outcomes in children with FAS (protective factors), including a stable and nurturing home environment during the school years, early diagnosis (before 6 years of age), absence of exposure to violence, few changes in caretaking placements, and eligibility for social and educational services. Interventions and services that maximize these protective factors while reducing risk factors will provide the best benefit to anyone with FAS and improve their chances for achieving their developmental potential (95,138).

General Needs. Helpful interventions should include those that stabilize home placement and improve parent-child interaction (138). One method for accomplishing this goal is to increase the understanding of the disorder by parents, teachers, law enforcement personnel, and other professionals who might become involved with the affected individual. Children with FAS often need unique parenting because of their difficulty with cause and effect reasoning and other executive functioning skills. Caregiver education should highlight and explain differences in the thought processes of children with FAS from typically developing children and children with other developmental disabilities. This would enable parent s to avoid potentially difficult situations (e.g., avoiding overly stimulating environments) and better manage problems when they do arise. Overall, a better functioning family that results from caregiver education promotes the stable, nurturing home that has been shown to be a positive protective factor for children with FAS (142).

Beyond the home environment, other professionals also need increased education and information concerning FAS (135). Parents can facilitate this understanding by learning to become advocates for their child. Such advocacy includes both linking families with needed community resources and making sure that the child receives maximum benefit from that service. Because the myriad of service systems is confusing and inconsistent across states, families must be educated about them at the local level. The world of social and educational services can be overwhelming, confusing, and inconsistent, and usually has a unique vocabulary that must be learned. Thus, it is important that along with a diagnosis, clinicians need to help caregivers in learning about available services, how to determine which services are appropriate for their child, and how to work productively with service providers (62).

Many prenatally exposed infants and children enter the foster or adoptive care system at an early age. A recent study estimated that the prevalence of children with FAS (or a related disorder) in the foster care system is 10 times that of the general population (143). However, while protective service agencies (PSAs) might have information about a child's prenatal history, staff members are generally not knowledgeable about FAS, do not understand the impact of the child's having FAS, or do not communicate the child's FAS status to other service systems. As a result, foster and adoptive families are most often not educated about the long-term effects and are unprepared to meet their child's needs. However, most PSAs require foster parents to take a specified number of educational courses annually. These courses should include education about the effects and developmental needs

of the child with FAS since the majority of foster parents will encounter at least one child with FAS or a related disorder during their time as a foster parent.

The assessment process is integral to a well-developed treatment plan. As has been emphasized in these guidelines, part of the diagnostic process is a comprehensive neuropsychological assessment, not only to establish CNS abnormities for the diagnosis, but also to develop the best treatment plan possible. Such a treatment plan minimizes risk factors for lifelong negative consequences and promotes protective factors that maximize developmental potential. Clinicians and s ervice providers must ensure that assessments include communication and social skills; emotional maturity; verbal and comprehension abilities; language usage; and, if appropriate, referral for medication assessments. Finally, it is the responsibility of the community at large to ensure that children with disabilities, including children with FAS, have access to and are assimilated into school, recreational, and social activities.

Age-specific services. Basic child development informs clinicians and service providers that the abilities of any one individual change dramatically at different stages of development. Intervention research informs providers that the most effective programs are those that are geared towards an individual's developmental level. There are specific "turning points" during which children demonstrate rapid and fundamental changes in their understanding of the world and in their problem-solving skills (e.g., development of object permanence or acquisition of formal operational thought). The services identified in the following sections include those for both the child and the family, grouped broadly by developmental stage. It is recognized that many of these services span beyond a single age category with considerable overlap, especially for the family.

Prenatal services. Significant development of all major organ systems occurs throughout gestation; thereby, making it imperative that women who drink during pregnancy be identified by the medical community as early as possible and be provided intervention services. Findings have indicated that children born to women who stop drinking at any point during their pregnancy have better outcomes than those who continue to drink throughout pregnancy (73). To ensure this happens, physicians, nurses, and other allied health professionals need to be trained to screen patients for possible alcohol use, to understand the detrimental effects of prenatal exposure to alcohol, to understand alcoholism as a treatable disorder, and to be familiar with treatment services. Many women who drink during pregnancy and their partners are not educated about FAS or the harmful effects of drinking during pregnancy. These prospective parents might not be prepared to address their shame or the needs of their children. Some women who experience an alcohol-exposed pregnancy might have been exposed themselves prenatally to alcohol. Therefore, treatment personnel need to be educated about both FAS and needed services to provide more appropriate treatment, as well as provide ongoing support and monitor these families closely.

Services for birth to 3 years of age. The first years of life are an important time for physical, cognitive, and emotional development. Decades of research have consistently shown the benefits of early intervention for children with developmental disabilities. Clinicians working with this age group need to familiarize themselves with the state systems that service this population. In particular, Part C of the Individuals with Disabilities Education Act (IDEA) provides early intervention for children 0 to 3 years of age . In many states, this program is administered through local health departments. A particularly appealing aspect of the Part C portion of IDEA is that FAS is considered a "presumptive eligibility" diagnosis. Presumptive diagnoses allow children "at risk" of later develop-

mental delay to be served without meeting particular eligibility criteria. That is, children who are at risk for later developmental problems can receive services, even if they test in the normal range or do not meet other eligibility criteria. This is very important for children with FAS because only about 25% score in the significantly developmentally delayed range (or the range of mental retardation for older children; 95). Referring children who have the FAS diagnosis as well as children who are exposed but do not meet the full diagnostic criteria for FAS, ensures that these children are monitored and, at appropriate ages, referred to appropriate ancillary services as needed. Many states maintain birth defects registries that track the ICD-9 code encompassing FAS (i.e., 760.71 Fetal Alcohol Syndrome) which also can facilitate such monitoring. All infants who are known to have been exposed prenatally included in these registries can be referred to the state's special child health services (note: each state will have its own name for this service) that are part of all states' maternal and child health systems. Special child health services programs provide case management and referral services for children with birth defects or developmental disabilities as well as facilitate evaluation for early intervention programs.

As noted previously, a stable and nurturing caregiving environment is a protective factor for children with FAS. Child development literature states that stable and nurturing environments promote secure attachments between infants and caregivers (144). Secure attachment facilitates emotional, social, and personality development in positive ways. Insecure or negative attachment can lead to inappropriate development in these areas, and at the most severe end of the continuum, the psychopathology of reactive attachment disorder (RAD). The time between birth and 3 years of age has been shown to be a particularly salient time for development of child and caregiver attachment. Disruption in the caregiving environment can lead to poor or negative attachment between infant and caregiver (145). Because many children with FAS are in the foster care system and experience multiple placements (due both to the nature of the system and the difficulty in parenting a child with FAS), they are at tremendous risk for negative attachment including RAD.

Services for children 3 to 6 years of age and school age. It is often during the toddler period that children with FAS will be identified and can be diagnosed. It is essential that states establish FAS diagnostic centers or ensure that their child evaluation centers have clinicians who are trained in the dysmorphic and other diagnostic criteria associated with prenatal exposure.

Following the families themselves, the educational system serves as the most constant service provider for individuals with FAS from early childhood through adolescence. Therefore, beginning with preschool programs and through secondary education, generalized essential services can be delineated. When a child reaches three years of age, early intervention services (IDEA Part C) end and families are referred to preschool handicapped programs (sometimes called special needs preschool) that are administered through IDEA Part B. Part B of IDEA differs from Part C in that there are no presumptive eligibility diagnoses. Eligibility for educational services under this program is related entirely to functional criteria and their relation to educability. As noted previously, this can make it difficult for children with FAS to qualify for these special education services because only a quarter of the children with FAS achieve standardized test scores in the range of significant developmental delay (or mental retardation), the usual eligibility threshold. However, within the IDEA framework, there are a few categories that allow children with FAS who score above the eligibility threshold to still qualify. A few children with FAS will qualify in the category of learning disabled (LD). This category is for children who demonstrate a statistically significant discrepancy (2 standard deviations or more) between overall IQ and one or more academic skills (e.g., math).

Another eligibility category that has been helpful in qualifying children with FAS is behavior disorder. However, this category should be used with care because children with FAS can learn negative behaviors from other children without receiving the benefits of a structured environment. Finally, the category of other health impaired (OHI) can be used at the discretion of the individual education plan (IEP) committee, which includes parents as well as school personnel. Services available through the school system go beyond classroom settings. Children can receive various therapies, including physical therapy (usually most appropriate for very young children), speech and language therapy, occupational therapy, or social skills training. These last two areas are particularly helpful to children with FAS because of the visual-motor deficits and problems in social interactions they encounter.

Training parents to be effective educational advocates is essential to maximize the benefits of their child's special education and to understand their child's rights. The purposes of the IEP are to insure access to appropriate habilitation and rehabilitation services (physical, occupational, speech, behavioral, mental health, and other related services) and to ensure that academic curricula are balanced with vocational training and skills of daily living (e.g., personal hygiene, money management, and family life education), when appropriate. In addition to training parents about the educational system, the preschool period, as well as elementary school years, are times when parents become more acutely aware of their child's limitations. Reinforcement and updating of information learned in early parent education settings will benefit both the child and the parents. Reviewing lessons learned will help parents adjust their expectations for the child's current functioning, as well as his or her future possibilities.

As noted previously, it is important that school staff be trained to recognize possible characteristics associated with FAS, as well as appropriate techniques for instructing students with FAS. Irrespective of whether alcohol-exposed children are in regular or handicapped preschool programs, educational methodologies need to be developed that best address their learning styles and that appropriate behavioral or mental health services are available and initiated.

Beyond services available through the educational system, families raising preschool and school-aged children continue to need services to promote positive family functioning. Such services might include behavior management training, family (or child) counseling, parenting workshops that focus on the unique aspects of parenting a child with FAS, or other types of continuing education. One service that becomes very important during these years is respite care. Such care allows a trained individual to stay with the affected child while caregivers or other family members take advantage of some time away from the child with FAS. Respite care has been shown to significantly reduce family stress and improve family functioning (146-147). Unfortunately, respite care, especially formalized and high-quality respite care, is not readily available in most communities. A clinician can help in this situation by working with the family to develop informal respite care situations, such as help from an extended family member working with the family and to provide the necessary education about FAS to such a respite care provider.

Services for adolescents. Adolescence, and even preadolescence, is one of the major turning points in the life of an individual with FAS. His or her body is changing, cognitive abilities are changing, peer groups are changing, and community expectations are changing. Because of the confusing nature of all these changes, adolescence often is the period when behavioral and mental health problems become more pronounced. Depression or anxiety, or both, can set in as the individual

struggles to cope with these changes. Increased opportunities to experience alcohol and/or drugs can lead to substance abuse problems. Families could become involved with juvenile or criminal justice systems. These are some of the most serious secondary conditions that research has identified for individuals with conditions associated with prenatal alcohol exposure (95).

It is often during adolescence that families experience high levels of stress and tension. As such, the need for individual counseling (for both child and parent), family counseling, and a strong support network becomes more crucial. However, this can be the exact stage at which agencies are reluctant to provide such services, especially if the FAS-related disability factor is not recognized. Because some amount of rebellion is expected during adolescence, the challenging behaviors of the teenager with FAS might be dismissed as transient. At the other end of the spectrum, the challenging behaviors of the adolescent with FAS can be so severe as to involve the criminal justice system.

Because adolescents will soon be leaving the safety and structure of the educational system, vocational and transitional services become essential during this stage. These services often represent a shift from academic skills and achievements to daily living skills, including employment skills. It is very important that these services be started in early adolescen ce, and not left until the individual is about to age-out of the educational system. In addition, beyond teaching the specific skills that go with a particular job, it might be necessary to explicitly teach those skills related to being a good employee (e.g., punctuality and minimized socializing). Most individuals will learn these skills through basic maturity and observational learning. Individuals with FAS often need explicit instruction as well as lifestyle supports (e.g., a job coach).

As for all adolescents, sexual behavior often becomes a critical issue during this stage. The boundaries for appropriate interaction with the opposite sex, the subtle nature of social cues, and impulse issues are difficult for any adolescent, but more so for the adolescent with FAS. Close supervision is the first line of defense during the adolescent years. However, such supervision often conflicts with the adolescent's growing desire for independence. This must be navigated with care to avoid alienating the adolescent. Also, it is probably best to be open and explicit with the teenager with FAS concerning the issues of contraception, sexually transmitted diseases, and sexual harassment. Failure to address these issues can have serious, and possibly life-threatening consequences for the affected individual, his or her family, and any children resulting from unintended pregnancies.

As mentioned previously, individuals with FAS are at high risk for involvement with the juvenile and criminal justice systems. Their lack of executive functioning skills (i.e., poor judgment), fluid language skills, and naïve social skills make them particularly vulnerable to participating in criminal activity. However, these same deficits demand that when they do encounter the justice system, their deficits should be taken into account during all aspects of justice proceedings (i.e., charges, process, punishment, and rehabilitation). As such the juvenile and criminal justice systems are major social systems in need of education regarding FAS. Special rehabilitation programs with staff that are trained to work with adolescents and young adults with FAS should be established. Such programs should be based on scientifically-based research findings that evaluate practicality as well as effectiveness.

Services for adults. In addition to all the services mentioned for the preceding age groups, adults with FAS often need support in every area of their lives. Everyday needs such as transportation issues, job assistance, housing assistance, medication reminders, money assistance, and support and

assistance when unpredicted issues arise should continue to be monitored and supported. Although not consistently available, clearly a system needs to be established that assists people with FAS in living as independently in the community as possible and includes support for housing, healthcare, and employment.

Because of federal, and often state legislation, it is very difficult for people with FAS to receive services from state developmental disability agencies, unless exposed individuals have met the eligibility criteria for services before 22 years of age. Eligibility criteria are generally based on levels of intelligence, as well as functional limitations in at least three areas associated with skills of daily living. As a result, many exposed individuals will not be eligible for services that often include an individual service plan (ISP), case management, residential and employment assistance, and possibly social security disability payments.

People with FAS might be eligible for federal assistance, such as, Medicaid, Supplemental Security Income (SSI), and Section 8 Housing subsidies, not because of their disabilities but because of their low income status. However, obtaining these services can be difficult. For example, most states have long waiting lists for Section 8 housing because of both the high demand and great need often leading to a shortage of rental units. Eligibility criteria for these services, even when based on income, should include a priority category for persons with FAS. Unfortunately, many of these housing options do not offer the support and structure that adults with FAS often need. Housing remains one of the main issues in supporting individuals with FAS for which there are no appropriate services developed or identified.

IDENTIFYING AND INTERVENING WITH WOMEN AT RISK FOR AN ALCOHOL-EXPOSED PREGNANCY

Despite ongoing efforts to inform childbearing-aged women of the risk associated with alcohol use during pregnancy, surveys conducted by the CDC and SAMHSA indicate that 9% to 12% of pregnant women in the United States report consuming alcohol and that approximately 3% report drinking at levels than have been consistently associated with adverse effects on the fetus (148-149). Also of concern are the high proportions of non pregnant childbearing-aged women whose drinking patterns exceed safe levels as defined by public health agencies (150-151). Currently more that half of all women of childbearing age (18 through 44 years of age) report alcohol use, and one in eight report binge drinking in the past month. Many of these women are sexually active and are not taking effective measures to prevent pregnancy. These women are at risk for an alcohol-exposed pregnancy in that they could have an unrecognized pregnancy and continue drinking early in pregnancy at levels that are harmful to the fetus.

One public health strategy for preventing alcohol-exposed pregnancies is to identify characteristics of women at greatest risk of having a child affected by prenatal alcohol exposure and implement prevention programs in subpopulations with higher proportions of these identified risk factors. Over the past 20 years, concerted efforts have been made to identify factors among childbearing-aged women associated with harmful patterns of alcohol consumption. One extensive review of studies reporting characteristics of women giving birth to a child with FAS found that low socioeconomic status (SES), African-American and American-Indian/Alaska-Native ethnicity, and being a smoker were characteristics commonly found among women in this group (152). Additional studies using cross-sectional survey data and special populations have extended our understanding of char-

acteristics associated with childbearing-aged women at high risk for having an alcohol-exposed pregnancy based on current drinking patterns. Factors associated with risk include being a smoker, having a low SES, being unmarried, having a history of previous or current illicit drug use, having a history of physical or sexual abuse, having psychological stress, and having mental health disorders (153-156). In an attempt to identify community-based settings that have higher proportions of preconceptional women at increased risk for an alcohol-exposed pregnancy, CDC sponsored an epidemiological study of special populations that included: women in alcohol and drug treatment centers, a large urban jail, publicly funded primary care clinics, a university-based gynecology clinic in a large urban hospital, and a cohort of women responding to a newspaper solicitation (27). Being at risk for an alcohol-exposed pregnancy correlated significantly with being (or having ever been) a smoker, having a history of inpatient treatment for drugs or alcohol, having a history of inpatient mental health treatment, having multiple sex partners, and having experienced recent physical abuse.

Primary prevention of alcohol-exposed pregnancies requires the accurate identification of women who are drinking at thresholds that have been associated with adverse pregnancy and infant outcomes before pregnancy occurs. Dietary guidelines from the U.S. Department of Health and Human Services recommend that women drink no more than one drink per day to avoid increased risk for adverse health conditions and that women who are pregnant, planning a pregnancy, or at risk of pregnancy abstain from alcohol use altogether. Research findings support these recommendations with evidence of increased risk for birth defects, spontaneous abortions, and deficits in neurocognitive development and growth at levels of seven or fewer drinks per week in some studies (157-162).

Of further concern is the pattern in which alcohol is consumed. Heavy episodic drinking, as in binge drinking, can result in increased severity of the teratogenic exposure effects because of the higher peak blood alcohol levels achieved in this pattern of consumption as opposed to lower level daily consumption (40,163). Historically, a binge episode has been considered to be five or more drinks on any one occasion, but evidence of the presence of gender effects in alcohol metabolism and higher morbidity and mortality among women than men with similar consumption patterns has prompted recommendations for defining a binge episode for women as four or more drinks on any one occasion or on any one day. Current recommendations on clinical thresholds published by the NIAAA are that women drinking more than seven drinks per week or more than three drinks on any given day in the past month be further assessed for risk of developing alcohol-related problems. As stated earlier, pregnant women are advised to abstain from alcohol use. This is a long-standing federal advisory and one supported by major professional societies as well (9,164).

Measurement of current alcohol consumption can be enhanced through the use of reliable screening and assessment methods. A number of screening instruments have been developed that offer the practitioner options for clinical assessment of childbearing-aged women. When selecting a screening tool for routine implementation, healthcare professionals should consider factors such as the goals of the screening process, the target population, and the ease of administration. All women of childbearing age should be screened for alcohol use, including women who are pregnant or nursing, women who are planning a pregnancy, and women who are sexually active and not using contraception (such as teens and college-aged women).

Methods and Considerations for Establishing Reliable Estimates of Alcohol Use. Measures most commonly used in alcohol assessment include items that address quantity, frequency, and pattern (variability) of drinking. Quantity-frequency measures (QF) inquire about average or typical consumption patterns. The simplest measures assess the amount of drinking on average drinking days (Q), and the average number of days on which alcohol is consumed (F). QF measures can be used to estimate a woman's average number of drinks per day or the amount of absolute alcohol consumed per day (AA score). To assess for binge drinking, some investigators have recommended that screening questions should include measures of maximum quantity consumption and frequency of maximum quantity consumption (165). *Helping Patients with Alcohol Problems: A Practioner's Guide,* developed by the NIAAA, (166) recommends quantity-frequency and maximum quantity questions as the primary screening test. These questions have been shown to have relatively high sensitivity and specificity, are easy to use, and can be incorporated into a health practice with minimal cost and effort (167).

Additional considerations for alcohol screening include measurement of the types of alcoholic beverages the woman consumes. The development of new ways of marketing wine and beer, including higher alcohol concentration malt liquors and beer in 20- to 45-ounce containers, has increased the need to provide women who drink with more specific standards to estimate their consumption. Studies show that reliance on standard drink measurements when assessing drinking practices of heavier drinkers or those consuming higher alcohol content beverages can result in considerable underestimation of alcohol consumption (168-169). Moreover, beverage-specific questions have been shown to be more accurate than grouped beverage questions (170).

One of the most reliable assessment tools for gathering accurate self-reported alcohol use is the timeline follow back (TLFB) measure (171). This method of alcohol use assessment typically asks respondents to think back over the past 90 days and report daily drinking amounts during that period of time. Respondents are given a calendar and asked to identify special events that occurred during that time period, such as parties, birthdays, or holidays that might serve to trigger recall of drinking occasions. A major strength of the TLFB measure is its ability to capture both average daily drinking and sporadic drinking that might entail drinking at higher levels of consumption. Because of the time requirements necessary for gathering information using the TLFB approach, it is not easy to use in primary care settings but it has been used in clinical research to establish the reliability and validity of other briefer screening and assessment tools (172).

Screening Tools. Several brief questionnaires have been developed over time to screen for problematic alcohol use in adults in diverse populations and settings. Screening tools currently in use include the CAGE, AUDIT, T-ACE, TWEAK, MAST, S-MAST, NET, RAPS4, and RAPS4-QF (173). The CAGE (174)is a four-item alcohol use measure designed to detect alcoholism and is one of the most commonly used screening instruments for men and women. The four questions ask the respondent: (C) Have you ever felt you should CUT down on your drinking? (A) Have people ever ANNOYED you by criticizing your drinking? (G) Have you ever felt bad or GUILTY about your drinking? and (E) Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover (EYE OPENER)? Each item is scored as a 0 (no) or 1 (yes) and scores of 2 or greater are considered clinically significant.

The AUDIT is a 10-item self-report questionnaire developed by the world health organization (WHO) for identifying individuals whose alcohol consumption has become hazardous or harmful

to their health (175). The items on the AUDIT are constructed to measure frequency of alcohol consumption, dependence symptoms, and the personal and social consequences of drinking. The first three questions of the AUDIT address the quantity, frequency, and maximum amount of alcohol consumed. The remainder of the questionnaire has two items from the CAGE, (feelings of guilt about drinking and the need for a drink first thing in the morning after a heavy drinking session—eye-opener) in addition to questions on the frequency of being unable to stop drinking once drinking starts, frequency of being unable to do what is normally expected because of alcohol use, frequency of memory loss of the previous night due to drinking, frequency of injury to self or others as a result of drinking, and frequency of others expressing concern over the individual's drinking (a relative, friend, or doctor). A score of eight or more is considered significant for high-risk consumption. The measure has good psychometric properties and has an advantage over other screening tools in that it measures not only experienced consequences of drinking, but also whether an individual is currently drinking at levels likely to eventually result in problems (176). The instrument has been used and validated in cross-cultural populations (177). One review of 38 studies on screening for alcohol problems in women and men in primary care settings found the AUDIT was more effective in identifying individuals with at-risk, hazardous, or harmful drinking patterns, while the CAGE proved superior in detecting alcohol abuse and dependence (178). Another study of women who were receiving outpatient care through the Veterans' Administration found that the AUDIT-C (comprised of the first three items of the AUDIT on quantity, frequency, and maximum drinks per drinking occasion) proved to be more sensitive than the full 10-item AUDIT (179). Such findings suggest that when time of administration is a consideration, the shorter AUDIT-C might be used as the screener of choice.

Two screening tools that have been specifically developed and used with pregnant women are the T-ACE and the TWEAK (180-183). The T-ACE has four questions that take less than a minute to ask. The questions are: (T) TOLERANCE, how many drinks does it take to make you feel high? (A) Have people ANNOYED you by criticizing your drinking? (C) Have you ever felt you ought to CUT DOWN on your drinking? (E) EYE OPENER, Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover? One point is given for each affirmative answer to the A, C, E questions, two points when a pregnant woman reports a tolerance of more than two drinks to feel high. A positive screen is a score of two or more points. The T-ACE has been shown to be an efficient screen for risk drinking in pregnant women and out-performs medical staff assessment alone (184). Its brevity (four-items) is an important asset and increases its utility for clinical practice.

Like the T-ACE, the TWEAK attempts to elicit information on the following: (T) TOLERANCE for alcohol; (W) WORRY or concern by family or friends about drinking behavior; (E) EYE OPENER, the need to have a drink in the morning; (A) "blackouts" or AMNESIA while drinking; and (K) the self-perception of the need to CUT DOWN on alcohol use. Scores range from zero to seven. The tolerance and worry questions each contribute two points and the other three questions contribute one point each. Any endorsement of the worry question is scored a two. On the tolerance question, if three or more drinks are needed to feel high, the question is scored as a two. Other versions of the tolerance question ask: How many drinks does it take before the alcohol makes you fall asleep or pass out? Or, if you never drink till you pass out, what is the largest number of drinks you have or can hold? These questions are scored as a two if the woman answers five or more drinks. Using these questions, however, results in lower sensitivity and specificity. A total score of three or more on the TWEAK is suggestive of harmful drinking pat terns (185).

Studies assessing the relative effectiveness of various screening tools have yielded different findings depending on the populations studied. Bradley et al. (186) summarized data from 13 published, peer-reviewed articles that contained information on the performance of alcohol screening questionnaires with non pregnant women and with obstetric patients during the periconceptional period. The results revealed that, for non pregnant women, the CAGE had low sensitivity in predominantly White female populations, but was a reasonable choice for identifying past year or lifetime alcohol dependence in predominantly African American female populations. Overall, the five-item TWEAK appeared to be the optimal screening questionnaire for identifying heavy drinking or alcohol abuse and dependence in racially mixed populations of non pregnant and pregnant women.

Some clinicians have suggested that the use of screeners best suited for identifying high-risk heavy drinkers might not be optimal for identifying pregnant women with irregular or lighter patterns of consumption that might still pose a risk for fetal development. Using confidential reporting procedures, one study examined the effectiveness of the TWEAK for assessing any report of drinking following pregnancy recognition in a group of low-income pregnant women participating in WIC (187). With a cut point score of two or greater, the specificity of the TWEAK was high for all ethnic groups studied; however sensitivity, while high for White non-Hispanic women, was low for African-American non-Hispanic and Hispanic women. Because any endorsement of alcohol use was sufficient for classification as a drinker in this sample, low sensitivity on the TWEAK might have been due to the fact that women were drinking at levels that were too low to result in alcohol-related problems but, were still high enough to be detrimental to the developing fetus. From a practical standpoint, the high specificity of the TWEAK supports its utility in busy clinic settings. High specificity suggests the TWEAK is effective in screening women who are not high risk, and as shown in the study cited previously, women who report not drinking at all. Nevertheless, the fact that the TWEAK had variable sensitivity for women from different ethnic backgrounds suggests that additional methods of screening should be employed in order to increase the detection of women who might need intervention.

Drinking among adolescents and college students has long been recognized as a significant problem with far-reaching public health implications (188-189). Although the most problematic drinking in adolescence has been documented among males when compared to females, alcohol use in females has been associated with decreased use of contraception (increasing the likelihood of an unintended pregnancy), increased sexual assault, and more sexually transmitted diseases. Because of these significant negative health consequences, the American Medical Association Guidelines for Adolescent Preventive Services recommend screening of adolescents for alcohol and other drug use as part of routine medical care (190). To effectively screen adolescents, the ideal screening tool must be developmentally appropriate and practical for use in busy medical offices or clinics. Although the CAGE and the AUDIT are relatively brief, their developmental appropriateness is questionable. Indeed, current research suggests that the CAGE is not appropriate for screening adolescents and that a much lower cut point of two (rather than the eight recommended for adults) on the AUDIT is optimal for identifying alcohol use problems in this population (191).

Several measures have been developed specifically for use with adolescents, such as the Rutgers Alcohol Problem Index (192) and the College Alcohol Problem Scale (193); however, these tools might not be practical for universal screening. One brief screening device, the CRAFFT, developed for adolescents, is simple to score, inquires about alcohol and drug use, and was found to have good psychometric properties in a sample of predominately female youths 14 through 18 years of age

(194). CRAFFT is an acronym for the first letters of key words in the test's six questions: (C) Have you ever ridden in a CAR driven by someone (including yourself) who was high or had been using alcohol or drugs? (R) Do you ever use alcohol or drugs to RELAX, feel better about yourself, or fit in? (A) Do you ever use alcohol or drugs while you are by yourself, ALONE? (F) Do you ever FORGET things you did while using alcohol or drugs? (F) Does your family or FRIENDS ever tell you that you should cut down on your drinking or drug use? (T) Have you ever gotten into TROUBLE while you were using alcohol or drugs? Each question on the CRAFFT is given a score of one and a cut point of two provides moderate sensitivity (.70) and excellent specificity (.94) for identifying alcohol use disorders in adolescents. The authors CRAFFT recommend that any positive answer on this measure be followed by further assessment of quantity, frequency, and pattern of use to increase sensitivity and to guide decisions about the need for intervention.

A number of studies have shown a strong association between alcohol intoxication and trauma (195). The Trauma Questionnaire was developed to screen patients in an unobtrusive way without asking them directly about their alcohol use (196). The questionnaire consists of four questions asking about injuries in the last five years (fractures or dislocations of bones or joints, traffic accidents, head injury, or injury during a fight or assault). The questionnaire has been shown to have high sensitivity and specificity for identifying high-risk alcohol use and to be acceptable to respondents and physicians.

Computer-Assisted Interviews and Laboratory Screening Measures. The method of delivery of questions about alcohol use has been shown to influence truthful reporting. Clinical interviews might not be as effective in eliciting truthful responses from women because practitioners are not always comfortable asking these questions in a face-to-face interview. Moreover, women could underestimate their alcohol use because of reluctance to discuss this potentially sensitive subject. For this reason, self-administered questionnaires might improve the validity of self-report.

The Audio Computerized Self-Report Interview has been successfully used in prenatal clinics serving disadvantaged low-literacy minority women (197). Questions asked by a recorded voice through earphones are drawn from the TWEAK with quantity and frequency questions pertaining to three months before and during pregnancy are included. Acceptability studies have revealed that patients liked this method of screening.

Laboratory screening measures offer promise for obtaining objective evidence of problem drinking (198-199). The most common biomarkers are gamma glutamyltransferase (GGT) and carbohydrate-deficient transferring (CDT). Fatty acid ethyl esters synthase (FAEE) can be found in the hair of alcohol using women. This biomarker could hold promise for screening for alcohol use in pregnant women, although the dynamics of enzyme expression appear to be complex and changes occur only at high alcohol doses. Low sensitivity in non alcoholic women and the high cost of laboratory analysis make these measures less feasible for use in more universal screening.

Brief Intervention. Brief intervention (BI) has been shown to be a low-cost, effective treatment alternative for alcohol problems that uses time-limited, self-help, and preventative strategies to promote reductions in alcohol use in nondependent individuals, and in the case of dependent people, to facilitate their referral to specialized treatment programs (200-203). Overall, BI for alcohol problems has been shown to be more effective than no intervention and often as effective as more extensive intervention (204). Effective prevention programs frequently employ a multicomponent

approach combining cognitive-behavioral techniques with norms clarification, education, and motivational enhancement interventions. BI is usually restricted to fewer than four sessions and is typically performed in a treatment setting that is not specific for alcoholism. It is often performed by personnel who are not specialists in the treatment of alcohol use and abuse, and is provided to individuals at varying levels of risk for negative consequences because of drinking, rather than those who are considered alcohol dependent (205).

Six elements characterize the key ingredients of standardized brief intervention summarized by the acronym FRAMES (206). These are: Feedback of personal risk, Responsibility for personal control, Advice to change, Menu of ways to reduce or stop drinking, Empathetic counseling style, and Self-efficacy or optimism about cutting down or stopping drinking. The intervention also involves establishing a drinking goal in the form of a signed contract and follow up of progress with ongoing support. Studies reveal that supportive, nonjudgmental techniques in which trained personnel counsel women can lead to decreased alcohol consumption during pregnancy. The most effective intervention approaches avoid the use of moral or volitional injunctions and instead focus on reduction of alcohol use without criticism or provocation of guilt (205). Effective interviewers have been found to have a thorough knowledge of the intervention technique, an optimistic attitude about change, a compassionate style, genuineness and respect for clients, an ability to avoid arguments that evoke patient defensiveness, and comfort discussing alcohol problems (207-208).

Motivational Interviewing. A technique often used in brief interventions is motivational interviewing (MI). MI uses an empathic , client-centered counseling approach to increase readiness for change by resolving ambivalence about behavior change (207). The process explores the client's ambivalence in an atmosphere of acceptance, warmth, and positive regard. Although the session is structured and to the point, direct persuasion and coercion are avoided. The goal is to enhance the discrepancy between the reasons for changing versus staying the same. More than 24 studies of MI have found beneficial effects in decreasing problem drinking and other health-related problem behaviors (209).

Manualized Brief Intervention. Although the findings of the previously cited studies provide the foundation for intervention and prevention efforts, the usefulness of MI in a busy clinic or medical practice might be limited in that it requires additional training and clinical skills development. Recently, standardized manualized BI techniques to reduce alcohol consumption have been developed. One such intervention is Project CHOICES (Changing High-risk alcohol use and Increasing Contraception Effectiveness Study), which is funded by CDC. Project CHOICES is an example of a brief intervention using motivational interviewing techniques aimed at preventing alcohol-exposed pregnancies among high-risk women in various community settings (210). This project focused on both alcohol risk reduction and pregnancy postponement until alcohol use was decreased in a group of non pregnant women who were of childbearing age, fertile, sexually active, and using ineffective or no contraception. Phase I results revealed that at six-month follow up, 68.5% of the women were no longer at risk of having an alcohol-exposed pregnancy, 12.6% had reduced their drinking only, 23.1% reported using effective contraception only, and 32.9% reported doing both. A Phase II randomized controlled trial is currently underway testing the efficacy of this approach and is slated for completion in fall 2004. Another recent CDC-funded study still underway is Project Balance, which uses a briefer adaptation of Project CHOICES and is aimed at female college students, encouraging them to abstain from alcohol or to use contraception if they drink.

Four randomized controlled studies, funded by NIAAA, serve as further examples of the utility and effectiveness of this approach. These studies included women of different socioeconomic and cultural backgrounds and were conducted in doctors' offices and community settings. Two of the studies found that manualized BI was successful in decreasing alcohol use during subsequent pregnancies in high-risk women, thus preventing possible negative developmental sequelae in their offspring (211). The other two studies, one working with high-risk, White, middle class pregnant women in physicians' offices and one working in WIC community clinics with primarily low risk, low-income Hispanic clients, also found manualized BI to be a promising approach (212-214). In the WIC study, better infant outcomes were reported, including longer birth lengths and a lower infant mortality rate. Significantly, in both studies, the control conditions that included an assessment of alcohol consumption and simple advice to stop or cut down on drinking were almost as effective as the manualized BI itself. The success of these projects in reducing alcohol consumption in both experimental and control groups was postulated to be due to the desire of pregnant women to have healthy pregnancies and to the time and attention that interventionists provided for women in both groups.

Computerized Brief Intervention One recently developed study is investigating the use of a computerized method of a BI that incorporates the use of vessel size and normative education that allows a woman to evaluate her own consumption levels (169). Once a woman has been educated about her consumption levels, she then participates in a standardized BI. This approach is promising in that it incorporates many recommended aspects of screening, educating, and assisting women in recognizing that they might have a drinking problem, and then providing a brief intervention. The automated computerized assessment is designed for use in prenatal clinics and is currently being tested at a large HMO through an NIAAA grant. Although the effectiveness of this intervention has yet to be validated, it offers a potentially useful method that could be expanded nationally.

Improving Use of Screening and Brief Intervention Technology by Clinicians. Research devoted to finding ways to encourage clinicians to use brief interventions indicates that routine educational approaches might not be effective. Effective strategies include (1) conducting educational programs at the intervention site; (2) using specific step -by-step, evidence-based clinical protocols; (3) using skills-based role playing; (4) holding peer group discussions; and (5) using a credible expert trainer or educator. Brevity, repetition, and reinforcement of recommended practices are also key program elements (215). In an effort to enhance physician uptake of current screening and intervention approaches for preventing alcohol-exposed pregnancies, the NIAAA and the Office of Research on Minority Health recently collaborated in the development of a guide for primary care providers for screening pregnant and non pregnant women on selected health behaviors, including alcohol use, and recommendations for appropriate advice depending on the level of alcohol use and consequences (166). More recently, CDC funded four regional training centers on FAS to provide education and training to medical and allied health professionals and students about identification and diagnosis of children affected by prenatal alcohol exposure and effective approaches for intervening with and preventing these conditions. More information about these and other resources can be found by accessing the websites provided at the end of this section.

Universal screening for alcohol use should be conducted among all women of childbearing age. Screening can be done in both physicians' offices and in community health settings. Simple screening techniques that include measures of quantity, frequency, and heavy episodic drinking, as well as

behavioral manifestations of risk drinking, have proven to be most beneficial. For non pregnant women, the AUDIT, TWEAK, and CAGE have been found to be useful screening tools depending on the population characteristics of the community as described in the studies reviewed earlier. However, studies on gender-specific modifications of these screening tools have been recommended because women report increased psychosocial problems at lower levels of consumption than men. Based on some empirical evidence, reduced cut points for women have been recommended; including a cut point of 4 or more for the AUDIT, 2 or more for the TWEAK, and 1 or more for the CAGE (186). For pregnant women, the T-ACE and the TWEAK are the recommended screening tools of choice. The CRAFFT shows promise as an alcohol and other drug screener for female adolescents. Findings support better identification when screening instruments are used versus clinical assessment alone. Brief interventions administered by physicians and allied health professionals in medical and non medical settings are effective in bringing about reductions in drinking.

Women who are pregnant, planning a pregnancy, or at risk of pregnancy should be advised not to drink, as no safe threshold of alcohol use during pregnancy has been established. Non pregnant childbearing-aged women should be advised to drink no more than seven drinks per week and no more than three drinks on any one occasion.

Both BI approaches and MI techniques have been found to be efficacious with pregnant and non pregnant women in promoting reduction in alcohol consumption. Furthermore, some evidence suggests pregnant women are motivated to stop drinking even if the intervention includes only an assessment of alcohol use with simple advice to stop or reduce drinking. Research also indicates that interventions are effective with pregnant women who are light as well as high-risk drinkers. Moreover, preconceptional counseling of women of childbearing age who are at risk for an alcohol-exposed pregnancy and who are not using effective contraception has been demonstrated as a promising method of prevention as well as computerized versions of standardized methods of intervention. This year the U.S. Preventive Services Task Force released recommendations calling for use of the screening and behavioral counseling interventions for adults in primary care setting, including pregnant women (216-217). These evidence-based recommendations mirror the content of these guidelines with respect to the specific types of screening tools recommended and the components of effective brief interventions to be used. The recommendations of the U.S. Preventive Services Task Force, which is supported by the federal Agency for Healthcare Research and Quality, provide further support for the need for widespread public health implementations of these prevention technologies to reduce the harmful consequences of alcohol misuse, including FAS and other prenatal alcohol-related conditions. Information about reported and other federally sponsored studies in FAS and prenatal alcohol screening and intervention can be obtained at the following websites: www.nih.gov; www.cdc.gov; www.samhsa.gov; and www.preventiveservices.ahrq.gov.

SUMMARY AND FUTURE STEPS

In 2002, CDC was congressionally mandated to develop diagnostic guidelines for FAS and other prenatal alcohol-related disorders and integrate them into medical and allied health education.

With input from a SWG composed of clinicians, experts, and families, and from the NTFFAS/FAE, scientific and clinical evidence was reviewed to develop guidelines that offer a balance between conservative and overly inclusive definitions of FAS. Criteria for conditions not meeting the clinical definition of FAS (e.g., ARND) were not established because scientific evidence is insufficient at this time. Clinical and scientific research on FAS and those conditions resulting from prenatal alcohol exposure that do not meet the criteria for an FAS diagnosis is currently underway. These findings and advances will contribute to further refinement of the FAS criteria, and could potentially delineate additional diagnostic categories and criteria for conditions other than FAS. The development of these FAS guidelines is a continuous process. Efforts to develop and refine other diagnostic categories to identify FAS and related conditions need to continue.

During this guidelines development process, several key issues emerged that deserve mention.

1. More information on the neurodevelopmental effects of prenatal exposure to alcohol is needed. Particular emphasis should be placed on finding the unique aspects of FAS that will help differentiate it from other birth defects or developmental disabilities, or both.

2. Efforts to improve the clinical assessment tools (e.g., facial and growth measures) used to diagnose FAS should continue, particularly in terms of racial and ethnic variations and age.

3. All children should be screened for the possibility of an FAS diagnosis. As physicians and other allied health professionals become educated about this disorder, screening for FAS should become routine.

4. Better communication between obstetricians, gynecologists, and pediatricians is needed to improve documentation on prenatal alcohol use. This would help with the diagnosis of prenatal alcohol exposure in the child and could help identify women at risk for future alcohol-exposed pregnancies.

5. Service agencies must provide a way to qualify children with FAS and related disorders who do not meet their traditional eligibility requirements.

6. Further research and resources are needed to identify and treat women at risk for an alcohol-exposed pregnancy.

7. Awareness, both in the public and professional arenas, about the dangers of drinking alcohol during pregnancy and about FAS and how the condition affects children and their families is essential. A key avenue to avoiding FAS is active promotion of programs to increase awareness of the dangers of drinking alcohol during pregnancy and promotion of prevention activities that increase understanding of the risks of alcohol as well as the risks for an alcohol-exposed pregnancy.

Over 30 years ago, researchers first described FAS . Much has been learned about the disorder since that time, as is reflected in these guidelines. However, there is still much more to learn about the entire spectrum of effects from prenatal alcohol exposure. Future work will address these gaps. To reduce FAS and other prenatal alcohol-related conditions, the key is prevention. Federal, state, and local agencies; clinicians and researchers; educational and social service professionals; and families

need to work together to educate women of childbearing age and communities across the country about the risks of drinking alcohol during pregnancy.

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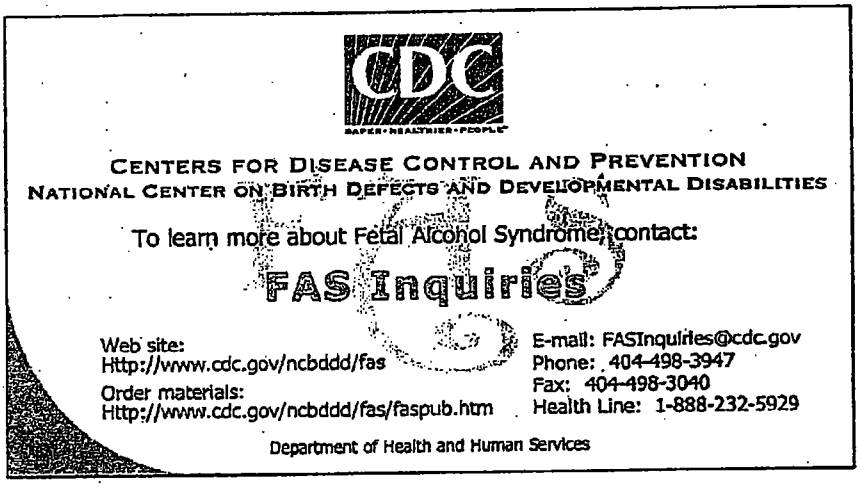
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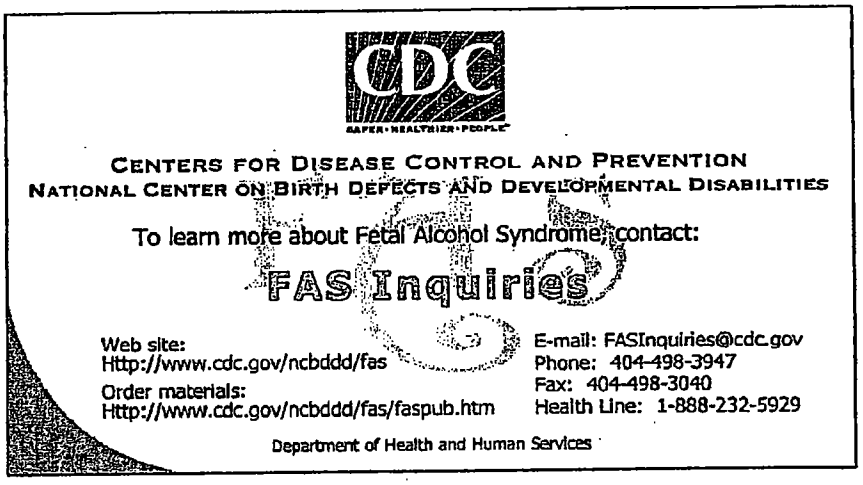
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Notes

Notes


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NATIONAL CENTER ON BIRTH DEFECTS AND DEVELOPMENTAL DISABILITIES
To learn more about Fetal Alcohol Syndrome, contact:
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South Carolina Fetal Alcohol Spectrum Disorders (FASD) Strategic Plan, State Fiscal Years 2011-2013

DAODAS MISSION

The mission of DAODAS is to ensure the provision of quality services to prevent or reduce the negative consequences of substance abuse and addictions.

BACKGROUND

Just over a decade ago, DAODAS organized the South Carolina Fetal Alcohol Syndrome Resource Network (SC FASRN), a multi-agency network with a mission to "to provide information to the citizens and health and human service professionals in South Carolina to help reduce the exposure of infants to alcohol, tobacco, and other drugs during pregnancy." A secondary purpose of this organization was to assist individuals working with substance-exposed children by providing practical tips, networking opportunities, and resource information.

The SC FASRN met quarterly in the state capital (Columbia, S.C.). Subcommittees were utilized for planning activities related to the observance of FAS Awareness Week and an annual FASRN Conference. In addition, the SC FASRN monitored new developments in research related to Alcohol-Related Birth Defects, as well as followed legislative efforts affecting this area.

One of the first activities of the SC FASRN was to organize regional trainings-of-trainers in Columbia, Greenville, Florence, Charleston, and Rock Hill in the spring and summer of 1998. The purpose of these trainings was to provide 120 professionals from around the state with up-to-date information on Fetal Alcohol Syndrome. The attendees were asked to provide at least two trainings in their communities on FAS.

In January 2002, DAODAS revised and reissued a very effective and popular four-color brochure, "Important Facts to Remember: A Special Delivery Should Be Handled With Care," which featured images of 15 appealing babies on its cover. The brochure encourages women who are pregnant or trying to get pregnant, as well those who are breast-feeding, to abstain from all types of alcohol, as well as to avoid the use of tobacco products and illicit drugs and to follow doctor's orders with regard to prescription and over-the-counter medications.

Since the early part of this decade, DAODAS has been reorganized and downsized (from a high of 125 positions in 2003 to its current staff size of 33 positions), and its state funding has been cut as part of efforts to balance the state budget. (From July 2008 to date, the DAODAS budget has been cut by 55%.). However, DAODAS was able to continue to observe FAS Awareness Week and to hold an annual FASD conference.

One salutary effect of the downsizing, reorganization, and budget cuts was increased cross-discipline teamwork at DAODAS. After successfully managing major SAMHSA-funded grant projects that involved most of its staff, the department's FASD points of contact mobilized other DAODAS staff, and together they embarked on the revitalization of the FASD initiative, this time with an emphasis on parental involvement. Renamed as the SC FASD Collaborative, the rejuvenated group produced this Strategic Plan with the assistance of a technical expert provided by the FASD Center for Excellence and has held trainings delivered by national FASD researchers.

INTRODUCTION

One of the most preventable yet tragic consequences of substance abuse is Fetal Alcohol Spectrum Disorders (FASD), which may result from the consumption of alcohol during pregnancy. "FASD" refers to a spectrum of conditions that include Fetal Alcohol Syndrome (FAS), Fetal Alcohol Effects (FAE), Alcohol-Related Neurodevelopmental Disorder (ARND), and Alcohol-Related Birth Defects (ARBD).

Every time a pregnant woman has a drink, her unborn child has one too. Alcohol, like carbon monoxide from cigarettes, passes easily through the placenta from the mother's bloodstream into her baby's blood and puts her fetus at risk of having an FASD. Resulting impairments may include, but are not limited to, intellectual disability; learning disabilities; attention deficits; hyperactivity; and problems with impulse control, language, memory, and social skills. Although skilled intervention services can help individuals with an FASD improve their quality of life, there is no effective treatment or cure.

Despite the risks associated with alcohol use during pregnancy, Centers for Disease Control and Prevention (CDC) statistics show that from 1991 to 2005, 12.2% of pregnant women reported some level of alcohol use in the past 30 days, and 2% reported binge drinking. In 2002, among women who might become pregnant (i.e., women who were not using birth control), 54.9% reported alcohol use in the past 30 days, and 12% reported binge drinking *(CDC, 2004)*.

FASD is a serious national problem. It is the leading known cause of intellectual disability. The prevalence of FAS in the United States is estimated to be at least 10 per 1,000, or 1% of all births. Based on estimated rates of FASD per live births, FASD affects nearly 40,000 newborns each year. The cost to the nation of FAS alone may be up to $6 billion each year, and for one individual with FAS, the lifetime cost is at least $2 million.

Individuals (especially youth) with FASD are at high risk of engaging in criminal activity. They face many challenges that make them vulnerable, such as being easily influenced by peer pressure; lacking impulse control; not understanding cause and effect; not learning from mistakes; making poor decisions; having memory problems; and having difficulty understanding future consequences. Researchers at the University of Washington estimate that 35% of individuals with an FASD have been in jail or prison, and more than half of the individuals diagnosed with an FASD have been in trouble with the law.

FASD is not as widely known as other negative consequence of substance use. It *is* preventable by eliminating alcohol consumption during pregnancy, however, and continuing research has indicated that some intervention methods help alleviate its effects. It is indeed time to mobilize our efforts to increase public awareness of FASD, to prevent alcohol use during pregnancy, and to provide effective interventions to individuals with an FASD.

GOALS & STRATEGIES

Goal 1 (Prevention)

Decrease alcohol-exposed pregnancies.

STRATEGIES: PREVENTION	SFY 2011	SFY 2012	SFY 2013
1.1. Partner with organizations that reach priority populations to implement evidence-based programs to eliminate alcohol use among women who are or may become pregnant or to prevent pregnancy among women who drink alcohol.	X	X	X
1.2. Advocate for policies that support access to reliable and affordable contraception and evidence-based sex education.		X	X

Goal 2 (Intervention)

Increase the developmental progress of children who have an FASD so they can function to the best of their abilities at home, in school, and in their communities.

STRATEGIES: INTERVENTION	SFY 2011	SFY 2012	SFY 2013
2.1. Partner with the State Department of Education to educate school professionals (e.g., principals, teachers, school nurses, counselors) about the effects of FASD and how to identify and effectively work with students who have an FASD.		X	X
2.2. Partner with Greenwood Genetic Center to deliver education and training about FASD to school systems and other youth-serving organizations.	X	X	X
2.3. Link partners and other relevant organizations to technical assistance resources (e.g., Substance Abuse and Mental Health Services Administration, Centers for Disease Control and Prevention, National Organization on Fetal Alcohol Syndrome) for FASD screening, diagnostic evaluation, and interventions for individuals with an FASD and their birth, adoptive, and foster families.	X	X	X

Goal 3 (Knowledge and Awareness)

Broaden the knowledge and awareness of FASD in the public and the professional community.

STRATEGIES: KNOWLEDGE AND AWARENESS	SFY 2011	SFY 2012	SFY 2013
3.1. Disseminate FASD prevention information through public and private organizations, mass media, and other channels.	X	X	X
3. 2. Advocate for inclusion of information about FASD with pregnancy-related products.	X	X	X
3.3. Recruit and train health professionals, parents of children with an FASD, and other knowledgeable individuals to inform priority populations about FASD prevention and/or intervention.	X	X	X
3.4. Link partners and other relevant organizations to accurate, current federal, state and local resources on FASD prevention and FASD screening, diagnostic evaluation, and interventions for individuals with an FASD.	X	X	X
3.5. Advocate with colleges and universities to integrate FASD education into relevant degree programs (e.g., health, behavioral and human services, education, criminology).			X

Goal 4 (Mobilization)

Develop resources, collaboration, policies, and data/evaluation capacity to effectively address FASD statewide.

STRATEGIES: MOBILIZATION	SFY 2011	SFY 2012	SFY 2013
4.1. Expand the SC FASD Collaborative.	X	X	X
4.2. Establish an effective and efficient organizational structure for the SC FASD Collaborative that facilitates collaboration toward goals.	X	X	X
4.3. Advocate for the appointment of a State FASD Coordinator for South Carolina.		X	
4.4. Establish an FASD point of contact/liaison in every collaborating organization.	X		
4.5. Collaborate with the Governor's Council on Substance Abuse Prevention and Treatment and the Joint Council on Children and Adolescents to address FASD.	X		
4.6. Advocate for policies that support FASD prevention and intervention goals.		X	X
4.7. Advocate for integrating FASD into the strategic plans of partner organizations.	X	X	X
4.8. Obtain public and/or private funding for FASD initiatives in South Carolina.		X	X
4.9. Identify existing FASD-related state and local data and data gaps for planning and evaluation purposes.	X	X	X
4.10. Transition the three-year DAODAS FASD strategic plan to a five-year statewide collaborative FASD strategic plan.			X

ACTION PLAN SFY 2010 (July 1, 2010 - June 30, 2011)

Goal 1 (Prevention)

Decrease alcohol-exposed pregnancies. *(DAODAS Goal Champions: Brenda Powell, Crystal Gordon; Contributors: Elizabeth Fore, Jonathan P. Scaccia)*

Strategy 1.1: Partner with organizations that reach priority populations to implement evidence-based programs to eliminate alcohol use among women who are or may become pregnant or to prevent pregnancy among women who drink alcohol.

2010-2011 Activities	Activity Lead
1.1.1: Identify populations at high risk of alcohol-exposed pregnancies.	Jonathan P. Scaccia, University of South Carolina
1.1.2: Identify evidence-based programs appropriate for high-risk populations.	Jonathan P. Scaccia, University of South Carolina
1.1.3: Identify organizations that reach high-risk populations to explore opportunities for collaboration on the provision of evidence-based FASD prevention programs.	Jonathan P. Scaccia, University of South Carolina

Strategy 1.2: Advocate for policies that support access to reliable and affordable contraception and evidence-based sex education. *(No activities planned for 2010-2011.)*

Goal 2 (Intervention)

Increase the developmental progress of children who have an FASD so they can function to the best of their abilities at home, in school, and in their communities. *(DAODAS Goal Champions: Hannah Bonsu, Susie Williams-Manning; Contributors: Kris Rife, Kennard DuBose, Cathy Mathles, Robin Simmons)*

Strategy 2.1: Partner with the State Department of Education to educate school professionals (e.g., principals, teachers, school nurses, counselors) about the effects of FASD and how to identify and effectively work with students who have an FASD. *(No activities planned for 2010-2011.)*

Strategy 2.2: Partner with Greenwood Genetic Center to deliver education and training about FASD to school systems and other youth-serving organizations.

2010-2011 Activities	Activity Lead
2.2.1: Contact the Greenwood Genetic Center about making a presentation on FASD to the S.C. Department of Education's Special Education Fall Training (tentatively scheduled for September 23-24, 2010).	Hannah Bonsu, DAODAS, and Robin Simmons, Department of Education
2.2.2: Invite Dr. Roger Zoorob to present on FASD during the staff psychiatrist meeting of the Departments of Mental Health and Juvenile Justice.	Hannah Bonsu, DAODAS, and Kennard DuBose, Department of Mental Health

Strategy 2.3: Link partners and other relevant organizations to technical assistance resources (e.g., Substance Abuse and Mental Health Services Administration, Centers for Disease Control and Prevention, National Organization on Fetal Alcohol Syndrome) for FASD screening, diagnostic evaluation, and interventions for individuals with an FASD and their birth, adoptive, and foster families.

2010-2011 Activities	Activity Lead
2.3.1: Identify other youth-serving entities to be recruited to the SC FASD Collaborative.	Kris Rife, SC FASD Collaborative

Goal 3 (Knowledge and Awareness)

Broaden the knowledge and awareness of FASD in the public and the professional community. *(DAODAS Goal Champions: Frankie Long, Patricia Colclough; Contributors: Cookie Grant, Lachelle Frederick, Jimmy Mount)*

Strategy 3.1: Disseminate FASD prevention information through public and private organizations, mass media, and other channels.

2010-2011 Activities	Activity Lead
3.1.1: Engage different agency heads to submit op-eds on a quarterly basis.	Frankie Long, DAODAS
3.1.2: Obtain information about upcoming conferences that reach large segments of priority populations.	Lachelle Frederick, DAODAS
3.1.3: Send FASD information packets to schools and other agencies (e.g., parks, gymnasiums) for dissemination in those settings.	Lachelle Frederick, DAODAS
3.1.4: Obtain information about free advertising available on television, in newspapers, and on the radio.	Jimmy Mount, DAODAS

Strategy 3.2: Advocate for inclusion of information about FASD with pregnancy-related products. *(No activities planned for 2010-2011.)*

Strategy 3.3: Recruit and train health professionals, parents of children with an FASD, and other knowledgeable individuals to inform priority populations about FASD prevention and/or intervention.

2010-2011 Activity	Activity Lead
3.3.1: Plan a training activity around September 9, 2010 (FASD Awareness Day).	Hannah Bonsu, DAODAS

Strategy 3.4: Link partners and other relevant organizations to accurate, current federal, state and local resources on FASD prevention and FASD screening, diagnostic evaluation, and interventions for individuals with an FASD.

2010-2011 Activities	Activity Lead
3.4.1: Include a link to www.FASDCenter.com and to the Greenwood Genetic Center web site.	Jimmy Mount, DAODAS
4.4.2: Recruit individuals who are willing to provide a specified number of FASD trainings.	Frankie Long, DAODAS
4.4.3: Provide a training of trainers.	Frankie Long, DAODAS

Strategy 3.5: Advocate with colleges and universities to integrate FASD education into relevant degree programs (e.g., health, behavioral and human services, education, criminology). *(No activities planned for 2010-2011.)*

Goal 4 (Mobilization)

Develop resources, collaboration, policies, and data/evaluation capacity to effectively address FASD statewide. *(DAODAS Goal Champion: Carl Kraeff; Contributors: Joyce Dilleshaw, Martha Critchley, Kris Rife, Dan Walker)*

Strategy 4.1: Expand the SC FASD Collaborative.

2010-2011 Activities	Activity Lead
4.1.1: Identify and recruit important community and government partners.	Carl Kraeff, DAODAS, and Joyce Dilleshaw, Cornerstone
4.1.2: Identify and recruit individuals and family members affected by FASD.	Kris Rife, SC FASD Collaborative
4.1.3: Use existing social networks (e.g., Autism Society, EASlink) to identify and invite additional members.	Martha Critchley, LRADAC

Strategy 4.2: Establish an effective and efficient organizational structure for the SC FASD Collaborative that facilitates collaboration toward goals.

2010-2011 Activities	Activity Lead
4.2.1: Convene a meeting of the DAODAS FASD team, the SC FASD Collaborative leadership, and other interested persons to design an infrastructure for plan implementation (e.g., leadership, interest groups for each goal, membership, communications, data/evaluation).	Carl Kraeff, DAODAS
4.2.2: Put the infrastructure in place.	Carl Kraeff, DAODAS
4.2.3: Conduct a six-month assessment of effectiveness and efficiency and make infrastructure adjustments as needed.	Carl Kraeff, DAODAS

Strategy 4.3: Advocate for the appointment of a State FASD Coordinator for South Carolina. *(No activities planned for 2010-2011.)*

Strategy 4.4: Establish an FASD point of contact/liaison in every collaborating organization.

2010-2011 Activity	Activity Lead
4.4.1: Ask each collaborating organization to name a point of contact/liaison who is committed to addressing FASD in South Carolina.	Carl Kraeff, DAODAS

Strategy 4.5: Collaborate with the Governor's Council on Substance Abuse Prevention and Treatment and the Joint Council on Children and Adolescents to address FASD.

2010-2011 Activity	Activity Lead
4.5.1: Ensure SC FASD Collaborative representation on the councils.	Frankie Long, DAODAS

Strategy 4.6: Advocate for policies that support FASD prevention and intervention goals. *(No activities planned for 2010-2011.)*

Strategy 4.7: Advocate for integrating FASD into the strategic plans of partner organizations.

2010-2011 Activity	Activity Lead
4.7.1: Collect source documents.	Carl Kraeff, DAODAS

Strategy 4.8: Obtain public and/or private funding for FASD initiatives in South Carolina. *(No activities planned for 2010-2011.)*

Strategy 4.9: Identify existing FASD-related state and local data and data gaps for planning and evaluation purposes.

2010-2011 Activities	Activity Lead
4.9.1: Identify existing FASD-related state and local data and data gaps.	Dan Walker, DAODAS
4.9.2: Establish a data and evaluation workgroup.	Dan Walker, DAODAS

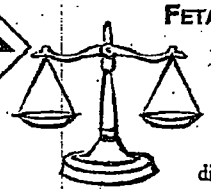
Strategy 4.10: Transition the three-year DAODAS FASD strategic plan to a five-year statewide collaborative FASD strategic plan. *(No activities planned for 2010-2011.)*

Fetal Alcohol Spectrum Disorders and the Criminal Justice System

There was a part of me that was angry, but I also knew that the police department and the justice system were uninformed about how vulnerable and easily swayed people [with an FASD] are.

—Mother whose son with an FASD was wrongly convicted of a crime

WHAT YOU NEED TO KNOW



Fetal Alcohol Spectrum Disorders

FASD is an umbrella term describing the range of effects that can occur in an individual prenatally exposed to alcohol. These effects may include physical, mental, behavioral, and/or learning disabilities with possible lifelong implications. FASD is not a clinical diagnosis. It refers to conditions such as fetal alcohol syndrome (FAS), alcohol-related neurodevelopmental disorder (ARND), and alcohol-related birth defects (ARBD).

Reasons People With an FASD Get in Trouble With the Law

Studies show that people with an FASD have specific types of brain damage that may cause them to get involved in criminal activity. These individuals show:

- Lack of impulse control and trouble thinking of future consequences of current behavior
- Difficulty planning, connecting cause and effect, empathizing, taking responsibility, delaying gratification, or making good judgments
- Tendency toward explosive episodes
- Vulnerability to peer pressure (e.g., may commit a crime to please their friends).

Persons with an FASD may break the law without intending to do so. For example, they may touch people when it is unwanted and think they are just being friendly. They may take things that do not belong to them because they like them.

People can take advantage of individuals with an FASD. They may talk them into committing crimes. Females with an FASD may be involved with destructive men for food, shelter, attention, or drugs.[3] These relationships put them at risk for arrest.

Number of People in the Criminal Justice System With an FASD

It is difficult to know how many people in the criminal justice system have an FASD. Data are limited, and populations vary by State. In addition, few systems screen for FASD or conduct a full diagnostic assessment. Researchers at the University of Washington estimate that 35 percent of individuals with an FASD have been in jail or prison at some point. They also estimate that more than half the people with an FASD have been in trouble with the law.[1]

The number of people with an FASD in the criminal justice system is assumed to be high. In the United States, approximately 3 million people are in jail or prison. Based on estimates of FASD in the general population, as many as 28,036 inmates could have an FASD.[2]

Issues Related to FASD in the Criminal Justice System

Laws vary by State and case law is binding only in the State or circuit where the case was decided. Only Supreme Court cases are binding nationally. However, several general issues can arise for attorneys and judges dealing with persons with an FASD:

- Competency to stand trial, which is the ability to understand the charges, participate in a trial, and assist in one's own defense. Persons with an FASD may not understand the charges against them. They may find criminal proceedings confusing. They may have problems with time management and come to court late or not at all. Several cases address competency and FASD.[4–6]
- Validity of expert testimony regarding diagnosis. Questions arise about the types of exams that are sufficient to determine a diagnosis of an FASD. For example, what if maternal alcohol use during pregnancy is unknown?[7,8]
- Diminished capacity. Capacity refers to the ability to understand right and wrong and to understand the

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likely outcome at the time of the act. Some crimes require evidence of intent for the person to be found guilty. Defense lawyers may argue that persons with an FASD cannot form the intent to commit crimes because they cannot foresee the likely outcome.[9]

- Effect of FASD on sentencing. Lawyers have appealed the death penalty by arguing that FASD was not introduced as evidence to support a lesser sentence.[10–12]
- Ability to testify. Persons with an FASD are highly suggestible and may not be able to give accurate testimony. They are prone to making false confessions.[13]
- Recidivism. Offenses do not appear to get worse, such as from auto theft to robbery. However, persons with an FASD tend to repeat crimes of opportunity, such as shoplifting. Their thought process seems to be, "I want. I take."[14]

Ways the Criminal Justice System Can Address the Needs of Persons With an FASD

Because of their disabilities, persons with an FASD may repeat the same mistakes many times. Thus, support to improve funtioning might be more appropriate than rehabilitation. This approach focuses on education, job training, and family support, rather than punishment. Medication may also help. In some cases, adults with an FASD who had multiple jail stints for petty, impulsive acts avoided jail when given appropriate medical treatment.[3]

Understanding how persons with an FASD respond to certain situations can help. Due to sensory issues, they can become overwhelmed by bright lights, causing them to panic and run from the police or resist arrest. Because they are eager to please, many unknowingly waive their rights by signing forms that they do not understand. In addition, they may consent to being searched or take responsibility for the crimes of others to win favor.

Sentencing is also an issue. Some persons with an FASD respond well to the intense structure and rules of prison. Others are vulnerable to attack, exploitation, and manipulation by other inmates. Some do not understand prison rules and break them. Because corrections officers may not understand FASD, they may punish inmates with an FASD for failing to follow directions. It is critical to offer training on FASD to all corrections staff so they can learn strategies to respond to inmates with an FASD.

Once on probation, persons with an FASD may have trouble meeting probation requirements. They can have problems managing time, recalling appointments, and making plans. Therefore, they may need a greater level of supervision. A relative or support person may need to be assigned to follow up on probation requirements. Highly structured probation that includes supervised living, life skills education, and drug and alcohol treatment can be very effective.[15] In fact, supervision can help prevent crime. Many clients with an FASD can remain crime free with intense supervision.[14]

Other effective alternatives to prison include halfway houses, group home treatment centers, or electronic monitoring at home. In such cases, emphasis must be placed on creating a well-structured environment with predictable rules and consequences. In these settings, persons with an FASD can continue to participate in the community but their behavior will be more closely monitored.[16]

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Stop and think. If you're pregnant, don't drink.

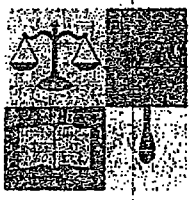
For more information, visit fasdcenter.samhsa.gov or call 866-STOPFAS.

01/06

National Organization on Fetal Alcohol Syndrome

Helping children & families by fighting the leading known cause of mental retardation & birth defects

FASD: What the Justice System Should Know



Alcohol abuse and pregnancy are common among women in the criminal justice system.

- An estimated 70 to 85 percent of inmates need substance abuse treatment.
- Approximately one in four women is either pregnant or postpartum when she enters prison.

The Justice System can help to prevent Fetal Alcohol Spectrum Disorders (FASD) among the incarcerated population by offering educational workshops on FASD and addiction counseling for women inmates.

Behavioral impairments due to FASD make affected individuals more likely to get in trouble with the law

- Sixty-one percent of adolescents and 58% of adults with FASD have been in legal trouble.
- Thirty-five percent of those with FASD over the age of 12 had been incarcerated at some point in their lives.

Many individuals with FASD will never socially mature beyond the level of 6 year-old child.

Other factors that may place persons with FASD at risk for involvement with the criminal justice system include:

- Difficulties in impulse control;
- Intellectual deficits;
- Poor judgment skills; and
- A history of abuse and/or neglect.



Problems individuals with FASD may encounter when dealing with police include:

- Being persuaded by the police (even inadvertently) to admit to crimes which they did not commit;
- Taking responsibility for crimes committed by others in order to win the favor of more sophisticated companions or to please the police;
- Consenting to searches of themselves or their possessions in circumstances in which non-disabled sophisticated individuals would not;
- Panicking during encounters with the police, running away or resisting arrest;
- Saying that they understand their legal rights when in fact they do not; and
- Making potentially incriminating statements about how serious any misconduct may have been.

The Justice System can help FASD-affected individuals by:

- Educating judges, lawyers and parole officers about the characteristics and behaviors of persons with FASD;
- Establishing screening, analysis, and treatment procedures for those with FASD who enter the juvenile justice or adult criminal justice system;
- Establishing/utilizing alternative sentencing programs for persons with FASD who have committed non-violent offenses; and
- Offering referral information for the children of incarcerated women who may have been prenatally exposed to alcohol.

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Consensus Statement on

Recognizing Alcohol-Related Neurodevelopmental Disorder (ARND) in Primary Health Care of Children

A Conference Organized by the Interagency Coordinating Committee on Fetal Alcohol Spectrum Disorders (ICCFASD)

Oct. 31–Nov. 2, 2011, Rockville, MD

Introduction

The nondiagnostic umbrella term "fetal alcohol spectrum disorders (FASD)" is now used to characterize the full range of damage from prenatal alcohol exposure, varying from mild to severe and encompassing a broad array of physical defects and cognitive, behavioral, emotional, and adaptive functioning deficits. FASD includes diagnoses such as fetal alcohol syndrome (FAS), partial FAS (pFAS), ARND, and alcohol-related birth defects (ARBD), which are congenital anomalies including malformations and dysplasias of the cardiac, skeletal, renal, ocular, auditory, and other systems.

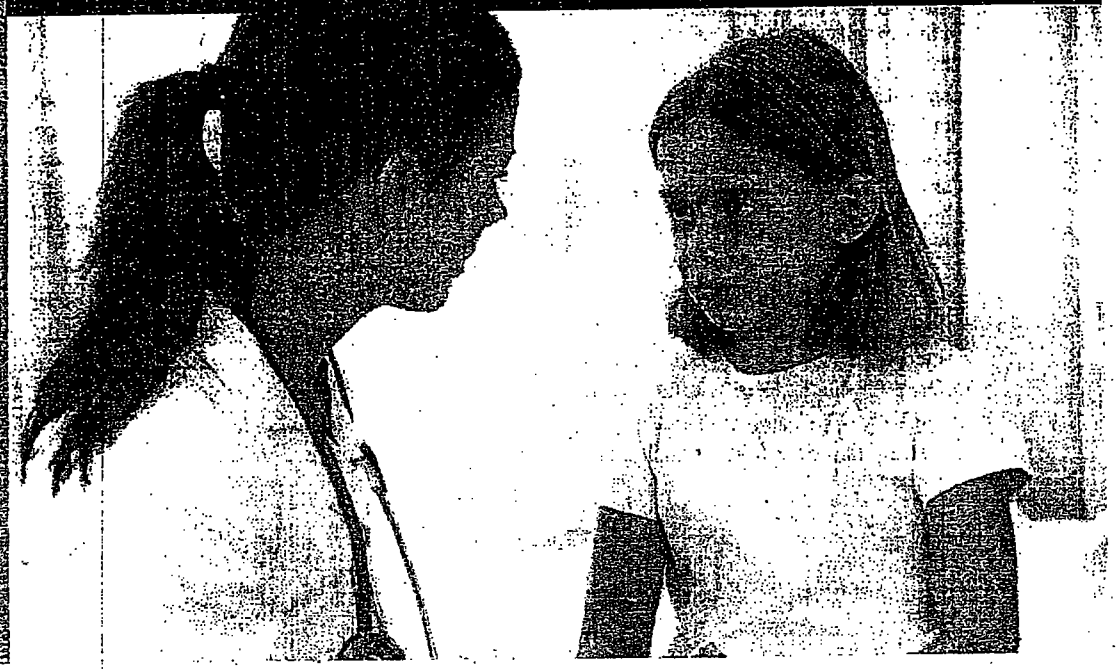
The negative effects of prenatal alcohol exposure on the developing brain and the resulting neurological and/or cognitive, behavioral, emotional, and adaptive functioning deficits are seen in individuals with FAS, pFAS, and ARND. Significant alcohol exposure early in prenatal development often results in growth retardation and facial anomalies. These physical characteristics have been useful tools for diagnosing FAS and pFAS. Identifying persons who do not have the physical characteristics of FAS but do have neurodevelopmental disorders induced by prenatal alcohol exposure has proven to be much more challenging, with broad implications. Current prevalence estimates for FAS range from 0.5 to 7 cases per 1,000 live births in the United States, and the prevalence of FAS and ARND combined is thought to be three times that of FAS alone.

In 2004, the National Center on Birth Defects and Developmental Disabilities of the Centers for Disease Control and Prevention and the National Task Force on FAS and Fetal Alcohol Effect issued *Guidelines for Referral and Diagnosis of FAS*. Evidence for recommending screening and referral for diagnosis of ARND was considered insufficient at that time. In the past 7 years, a large body of research evidence has been published on further characterization and differentiation of the cognitive, behavioral, emotional, and adaptive functioning deficits

CONSENSUS STATEMENT

Recognizing Alcohol-Related Neurodevelopmental Disorder (ARND) in Primary Health Care of Children

October 31–November 2, 2011 = Rockville, MD 20852



associated with prenatal alcohol exposure. Based on this evidence and identification of the principal issues with researchers and clinicians, ICCFASD determined that the time had come to reassess whether sufficient evidence now existed to recommend screening and/or referral for diagnosis of ARND in primary health care of children. ICCFASD then proceeded to convene a conference during which a multidisciplinary panel would respond to the principal issues, with the objective of arriving at a statement that would advance an understanding of the issue and that would be useful to health care professionals. This document summarizes the outcome of the conference.

Process

In late 2011, ICCFASD assembled a broad-based, independent panel of knowledgeable and unbiased critical thinkers to hear and evaluate evidence presented by experts in the field of FASD. The goal of the conference was to arrive at recommendations and future directions on whether to encourage screening and diagnosis (or referral for diagnosis) of ARND in primary health care of children.

The consensus statement that follows was prepared by the panel, which included health care professionals, biomedical researchers, academics, educators, and child advocate/policy/legal representatives. It is based on (1) relevant published studies assembled by the scientific committee of the conference, (2) presentations of data from the peer-reviewed scientific literature by experts working in areas relevant to the conference questions, (3) questions and comments from conference attendees during open discussion periods, and (4) closed deliberations by the panel. This statement is an independent report of the panel and is not a policy statement of ICCFASD or the National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health; the Centers for Disease Control and Prevention; or the American Academy of Pediatrics, which cosponsored the conference.

Conference Questions

The panel used the evidence presented to them by the experts in the field to develop answers, in the form of a consensus statement, to the following questions:

1. What is ARND; how can it be diagnosed (classical, current diagnostic schemes, in practice today)?
   Part A: Evidence of Central Nervous System Developmental Abnormalities
   Part B: Evidence of a Complex Pattern of Behavior and Cognitive Abnormalities
2. Can ARND be differentiated from other disorders?
3. What prenatal alcohol exposure evidence is necessary for an ARND diagnosis?
4. What signs/symptoms will be useful as screening criteria?
5. What are the treatment needs for those diagnosed with ARND?

Preamble

Children, adults, and families who live with disabilities related to prenatal alcohol exposure (PAE) face extraordinary challenges daily. The work of clinicians and researchers who have worked to understand and improve outcomes on their behalf must be commended. Together, this community has pioneered medical, educational, social, and scientific initiatives, all in pursuit of improving the quality of life for those affected and their families and of reducing the public health burden resulting from PAE.

PAE can cause significant neurodevelopmental and behavioral disorders as well as adaptive and self-regulatory impairments that can have lifelong consequences. Early diagnosis and intervention may help to reduce the long-term challenges potentially facing individuals with PAE. Therefore, primary health care clinicians serving children[1] should be alert to evidence of any maternal use of alcohol during pregnancy in order to provide timely evaluations and appropriate interventions for affected children and help prevent PAE during future pregnancies.

Question 1: What is ARND; how can it be diagnosed (classical, current diagnostic schemes, in practice today)?

Alcohol-related neurodevelopmental disorder (ARND) refers to a complex range of disabilities in neurodevelopment and behavior, adaptive skills, and self-regulation in the presence of confirmed PAE. ARND is one of the fetal alcohol spectrum disorders that also include fetal alcohol syndrome (FAS), which is additionally characterized by distinct facial features and growth retardation.

The term ARND was used in a 1996 report developed under the auspices of the Institute of Medicine (IOM)[2] to recognize the existence of neurodevelopmental disorders associated with confirmed PAE. Specifically, individuals with ARND do not present with the FAS facial phenotype (reduced palpebral fissure length, smooth philtrum, and thin upper vermillion border), but may present with structural and/or functional central nervous system (CNS) abnormalities, and may or may not present with growth deficiencies or decreased cranial size at birth. Acknowledging some degree of uncertainty that PAE caused the presenting adverse effects in any particular individual, the 1996 IOM report on FAS defined ARND as CNS neurodevelopmental abnormality evidenced by decreased cranial size at birth, or structural brain

[1] Primary health care clinicians for children include pediatricians, pediatric nurse practitioners, family medicine physicians, family nurse practitioners, pediatric and family physician assistants, and certain nurses in public health clinics or school-based health clinics.

[2] Stratton K, Howe C, Battaglia F. (Eds.) *Fetal alcohol syndrome: Diagnosis, epidemiology, prevention, and treatment.* Washington, DC: National Academies Press, 1996. Available online at: http://www.nap.edu/openbook.php?isbn=0309052920.

abnormalities, or neurological hard or soft signs in the presence of a pattern of confirmed excessive maternal prenatal alcohol use. Alternatively, ARND could be defined by evidence of a complex pattern of behavioral and cognitive abnormalities that are inconsistent with developmental level and cannot otherwise be explained by the genetic contribution of the biological parents, nor by impairments in brain maturation conferred by adverse environmental factors. Alternative descriptors have emerged, including "neurodevelopmental disorder/alcohol exposed" and "static encephalopathy/alcohol exposed."[3] In the ensuing years, animal research and human studies have helped families, clinicians, and researchers develop a deeper understanding of the relationship between PAE and neurodevelopmental manifestations subsequently evident in children, adolescents, and adults.

Part A: Evidence of CNS Developmental Abnormalities

The brain is susceptible to the neurotoxic effects of alcohol at all stages of gestation. Based on extensive, mutually reinforcing animal and clinical research, there appear to be patterns of significant structural and functional changes in the CNS attributable to PAE. Basic research suggests that numerous processes of neuronal development and functioning can be affected by PAE. Animal studies demonstrate that the timing, dose, and frequency of PAE differentially harm specific neuronal structures and brain circuits. Brain imaging and cognitive and behavioral studies have substantiated similar structural and functional alterations in humans.

Part B: Evidence of a Complex Pattern of Behavior and Cognitive Abnormalities

There is clear and compelling evidence from animal studies that PAE negatively affects behavior, cognition, motor function, self-regulation and adaptive function, executive function, activity, and mood in a complex way. Children with PAE frequently exhibit behavioral and emotional problems such as inattention, hyperactivity, anxiety, and mood dysregulation. These problems may emerge early in life and continue to significantly impair an individual's functioning in numerous domains throughout the lifespan. Identified problems may be primary to PAE, be primary to a comorbid condition, or result from the contribution of and interaction among a number of factors, including interactions with the environment. Research with children suggests that PAE can be associated with general cognitive impairments and specific impairments in the following areas: information processing, attention, executive function, language, memory/learning, social cognition, number processing, and sensorimotor function. One or more behavioral and cognitive phenotypes specific to PAE have been elusive. Identification of specific phenotypes is confounded by variability of exposure (dose, duration, and timing) and potential interactions among other factors, which may include qualities of the prenatal and postnatal environments, genetics, and exposure to other toxic substances.

[3] Currently, there are several commonly accepted diagnostic schemes and interpretations of the diagnostic guidelines presented in the 1996 IOM report on FAS (see Resources for Further Information and Application at the end of this statement for commonly accepted diagnostic schemes providing guidelines on diagnosing ARND). This panel is not advocating any particular system for the diagnosis of ARND or the use of these alternate descriptors.

Question 2: Can ARND be differentiated from other disorders?

Alcohol is a known teratogen and is strongly associated with a range of neurodevelopmental and behavioral disorders that may affect numerous domains of functioning across the lifespan. Emerging evidence from animal and human studies suggests that there is a constellation of symptoms attributable to PAE that may include (1) neurocognitive impairments, (2) self-regulatory challenges, and (3) impairments in adaptive functioning. However, differentiating ARND from other complex neurodevelopmental disorders can be challenging due to limited available studies attempting to distinguish ARND phenotype(s) from other disorders.

Even when a history of PAE is available, diagnosing ARND and distinguishing it from other complex developmental disorders requires prudent clinical judgment and consideration of other potential causes. In the future, we anticipate that clinicians will be assisted in making this diagnosis through advances in the identification of biomarkers sensitive to the detection of significant PAE and the development of tests that are both sensitive to and specific for alcohol-induced neurobehavioral disorders. We recommend additional rigorous scientific investigation to further refine understanding of the cognitive, behavioral, neurologic, and psychiatric clinical profiles attributable to PAE, as well as of the patterns of development evidenced by individuals with PAE. We further recommend that investigations of other complex developmental disorders include inquiry about PAE to identify the contribution of PAE to the phenotypes of other developmental disorders.

Question 3: What prenatal alcohol exposure evidence is necessary for an ARND diagnosis?

An ARND diagnosis requires confirmed, significant PAE. Determination of alcohol exposure can be based on maternal self-report; the report of a spouse, partner, relative, or friend who observed the birth mother drinking alcohol during the index pregnancy; and/or documentation in medical or other records about maternal alcohol use during the index pregnancy.

Data from animal studies across multiple species confirm that, at the highest levels of alcohol exposure in the first trimester, facial abnormalities and brain maldevelopment occur in concert. However, alterations in brain development that subsequently affect behavior can occur with a range of alcohol dosages throughout gestation, even when the face and brain appear to be structurally normal. Variable patterns of maternal drinking, including binge drinking resulting in significant peak levels or sustained drinking resulting in significant cumulative exposures, may lead to differential fetal outcomes. In addition, evidence suggests that variability in both maternal and fetal characteristics affects the potential for alcohol-induced alterations in brain

development. Thus, because there is no known safe threshold for PAE, it is not currently possible to define a safe limit of alcohol consumption during pregnancy.[4]

Question 4: What signs/symptoms will be useful as screening criteria?

The U.S. Surgeon General recommends regular screening of every woman of childbearing age for alcohol use. Screening should be conducted by adult primary health care clinicians and obstetric caregivers to protect the health of women and any subsequent offspring. For children, pediatric primary health care clinicians should obtain medical records about PAE and other potential risks from the birth mother's obstetric caregiver. For children who are not living with their birth parents, clinicians should obtain any available records that may provide information about PAE or other relevant family history. Clinicians should query families in a nonjudgmental way about all risks to a child's development, including maternal alcohol use prior to and during pregnancy. We recommend that clinicians be trained regarding the most effective ways to ask about alcohol use to ensure that this practice is adopted as routine.[5] Regular screening of parental alcohol use should continue as part of the process of child health supervision and developmental surveillance.

If PAE is confirmed, primary care clinicians should be alert for signs and symptoms that can occur during the child's development. Primary care clinicians should complete a comprehensive history for any child at risk for ARND that includes questions about developmental milestones, school functioning, peer and family relationships, adaptive and self-help skills, and specific areas of impairment, and they also should conduct a physical and neurological examination. A concern identified in any of these areas warrants a referral for a complete evaluation and followup. Absence of a concern should result in continued developmental surveillance of the child, as problems related to PAE may emerge during maturation, particularly during adolescence and young adulthood when latent ARND as well as other comorbidities commonly arise.

Question 5: What are the treatment needs for those diagnosed with ARND?

Given that the manifestations of PAE are heterogeneous, vary across development, and can be lifelong, treatment plans need to be multimodal and specific to the strengths and weaknesses of the affected individual and family across the lifespan. Treatment begins with support of the affected individual and family and education on the manifestations of ARND, risks for other

[4] The 1996 IOM report on FAS describes necessary or required confirmed alcohol exposure as "a pattern of excessive intake characterized by substantial, regular intake or heavy episodic drinking. Evidence of this pattern may include frequent episodes of intoxication, development of tolerance or withdrawal, social problems related to drinking, legal problems related to drinking, engaging in physically hazardous behavior while drinking, or alcohol-related medical problems such as hepatic disease." New data are accumulating to suggest that ARND can occur at lower levels of alcohol exposure than indicated in the 1996 IOM report.

[5] See Resources for Further Information and Application at the end of this statement for a list of commonly used screening tools for parental alcohol use.

problems, and available treatments. Treatments should draw on evidence-based practices as they pertain to an individual's specific needs. Treatment should be implemented flexibly, but with fidelity, in addressing the specific developmental strengths and weaknesses of the individual and family. Modifications of evidence-based treatments should only be considered when those treatments have been implemented with integrity over an adequate period of time and have failed to yield improvement.

Some interventions for ARND have targeted common manifestations of PAE, including problems with mathematics, attention, self-regulation, adaptive functioning and problem-solving, social impairment, and working memory. Other interventions have focused on individual and group-based skills development or training of caregivers in behavior management. Large, well-controlled behavioral intervention studies specific to ARND are needed. First, studies are needed to determine whether currently available evidence-based interventions for problems common to children without PAE yield similar benefits in children with ARND. Second, when evidence indicates a reduced or inadequate benefit, modifications of existing interventions or development of entirely new interventions need to be completed and evaluated. In addition, these research objectives also may be accomplished by assessing for PAE those participants with other mental health disorders and developmental disabilities in treatment outcome studies. Finally, these studies should target children across the developmental spectrum, with special attention directed at the periods of challenging transitions from childhood to adolescence and from adolescence to adulthood.

Some medications have been shown to effectively treat emotional and behavioral problems in children. Although there is an impression that many of these medications may be less effective in children with PAE or may lead to an atypical response, the literature is sparse and inconclusive. High-quality randomized controlled trials of medication treatments are needed for individuals across the lifespan in order to identify which medications work best for the specific problems affecting individuals with ARND.

Many of the problems experienced by children with PAE manifest as academic impairments or other problems at school. Given the limited number of educational interventions specific to children with ARND, clinicians may need to draw on evidence-based educational interventions for other disorders. For any school-based educational, medical, or mental health intervention, it is particularly important to engage educators, school health and mental health professionals, and other staff in assessing, planning, and implementing the intervention with high fidelity to enhance the effectiveness of the specific intervention plan.

Problems in self-regulation and social and adaptive functioning can manifest as early as infancy and continue throughout the lifespan. For young children, it is important to educate caregivers and early intervention providers about the manifestations of ARND as well as the usefulness of specific intervention strategies for this age group. In addition, individuals with ARND can have poor decision-making skills, placing them at risk for a range of behavioral problems that may

lead to contact with school-based professionals and other community service providers who may be unaware of how ARND can affect a child's functioning. It is therefore important to help affected individuals and families become self-advocates and to broadly educate the public and relevant professionals about behavior problems associated with ARND.

In conclusion, children and youth with PAE have a Special Health Care Need[6] and should have ongoing developmental and behavioral surveillance by their primary health care clinician in a "medical home."[7] This surveillance should continue throughout their lifespan to assess ongoing treatment and referral needs.

[6] As adopted in 1998 by the American Academy of Pediatrics (AAP), children with Special Health Care Needs are those who have or are at increased risk for a chronic physical, developmental, behavioral, or emotional condition and who also require health and related services of a type or amount beyond that required by children generally. This definition originally was proposed in McPherson M, Arango P, Fox HB. A new definition of children with special health care needs. *Pediatrics*. 1998;102:137–40.

[7] The AAP defines a "medical home" as one in which the care of infants, children, and adolescents is delivered or directed by well-trained physicians who provide primary care and help to manage and facilitate essentially all aspects of pediatric care. The physician should be known to the child and family and should be able to develop a partnership of mutual responsibility and trust with them. Ideally, care is accessible, continuous, comprehensive, family centered, coordinated, compassionate, and culturally effective. The Affordable Care Act of 2010 endorses the medical home model throughout the lifespan. For more information, see http://aappolicy.aappublications.org/cgi/content/full/pediatrics;110/1/184.

Resources for Further Information and Application

Commonly Accepted Diagnostic Schemes Providing Guidelines on Diagnosing ARND:

Astley SJ. *Diagnostic guide for fetal alcohol spectrum disorders: The 4-digit diagnostic code. 3rd ed.* Seattle, WA: University of Washington Publication Services, 2004. Available online at: *http://depts.washington.edu/fasdpn/pdfs/guide2004.pdf.*

Chudley AE, Conry J, Cook JL, Loock C, Rosales T, LeBlanc N. Fetal alcohol spectrum disorder: Canadian guidelines for diagnosis. *Can. Med. Assoc. J.* 172(5 Suppl.):S1–S21, 2005. Available online at: *http://www.cmaj.ca/content/172/5_suppl/S1.full.*

Hoyme HE, May PA, Kalberg WO, Kodituwakku P, Gossage JP, Trujillo PM, et al. A practical clinical approach to diagnosis of fetal alcohol spectrum disorders: Clarification of the 1996 Institute of Medicine criteria. *Pediatrics.* 115(1), 39–47, 2005. Available online at: *http://pediatrics.aappublications.org/content/115/1/39.full.pdf+html.*

Stratton K, Howe C, Battaglia F. (Eds.) *Fetal alcohol syndrome: Diagnosis, epidemiology, prevention, and treatment.* Washington, DC: National Academies Press, 1996. Available online at: *http://www.nap.edu/openbook.php?isbn=0309052920.*

Commonly Used Screening Tools for Screening Men and Women for Alcohol Use:

National Institute on Alcohol Abuse and Alcoholism. *Helping patients who drink too much: A clinician's guide: Updated 2005 edition.* Bethesda, MD: National Institutes of Health. Publication No. 07-3769, 2007. Available online at: *http://www.niaaa.nih.gov/Publications/EducationTrainingMaterials/Pages/guide.aspx.* (Related resources, most of which are available online only, include professional support resources, manuals, forms, and a slide show. An online training is approved for continuing medical education/continuing education credit.)

Other Alcohol Screening Instruments Recommended for Use With Women:

Barry KL, Caetano R, Chang G, DeJoseph MC, Miller LA, O'Connor MJ, et al., National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect. *Reducing alcohol-exposed pregnancies: A report of the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect.* Atlanta, GA: Centers for Disease Control and Prevention, March 2009. Available online at: *http://www.cdc.gov/ncbddd/fasd/documents/redalcohpreg.pdf.*

Acknowledgments

Conference Moderator **Tom Donaldson,** Chief Executive Officer and President, National Organization on Fetal Alcohol Syndrome, Washington, DC, maintained the smooth conduct of the meeting, ensuing ample opportunity for speaker presentations and scheduled discussions.

Expert Chair **Claire D. Coles, Ph.D.,** Professor, Departments of Psychiatry and Behavioral Sciences and Pediatrics, Emory University School of Medicine; Director, Fetal Alcohol and Drug Exposure Center, Marcus Autism Center, Atlanta, GA, led the assembled experts in presenting the available scientific and clinical evidence on ARND to the panel during public sessions.

Panel Members

Chairperson **Joseph F. Hagan, Jr., M.D., FAAP,** Pediatrics Clinical Professor, University of Vermont College of Medicine and the Vermont Children's Hospital, Burlington, VT; Co-Editor of *Bright Futures Guidelines for Health Supervision of Infants, Children and Adolescents, Third Edition;* and Chair, American Academy of Pediatrics Committee on Psychosocial Aspects of Child and Family Health, led a distinguished panel of specialists knowledgeable about developmental disorders to craft a consensus statement with practical recommendations based on these questions.

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Joyce Maring, Ed.D., PT, Associate Professor, Director of Physical Therapy Program and Interim Chair, Physical Therapy and Health Care Sciences, The George Washington University School of Medicine and Health Sciences, Washington, DC

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Speakers and Topics

Overview of Topic

Alcohol-Related Neurodevelopmental Disorder (ARND): Clinical and Empirical Evidence for an Independent Effect on Behavior
Claire D. Coles, Ph.D.; Professor, Department of Psychiatry and Behavioral Science and Pediatrics, Emory University School of Medicine; Director, Fetal Alcohol Program, Marcus Autism Center

Question 1: What is ARND and how is it diagnosed (classical, current schemes, in practice today)?

Part A: Evidence of Central Nervous System Neurodevelopmental Abnormalities

The Role of a Neurological Exam in the Evaluation of FASD
Sterling K. Clarren, M.D., FAAP, Chief Executive Officer and Scientific Director, Canada Northwest Research Network; Clinical Professor, Pediatrics, University of Washington, Seattle, and University of British Columbia, Vancouver, British Columbia, Canada

Biobehavioral Markers of ARND
Sandra W. Jacobson, Ph.D. [presenter], and Joseph L. Jacobson, Ph.D., Professors, Department of Psychiatry and Behavioral Neuroscience, Wayne State University School of Medicine; Honorary Professors, Departments of Human Biology and Psychiatry, University of Cape Town Faculty of Health Sciences, South Africa

The Brain in Children With FASD
Elizabeth R. Sowell, Ph.D., Professor, Department of Pediatrics, University of Southern California/Children's Hospital Los Angeles, California; Director, Developmental Cognitive Neuroimaging Laboratory, University of California, Los Angeles

Animal Models of FASD: Pathologies Inform Behavior
Susan Smith, Ph.D., Professor, Department of Nutritional Sciences, University of Wisconsin, Madison

Part B: Evidence of a Complex Pattern of Behavior and Cognitive Abnormalities

Neurocognitive Profile of Children With ARND
P.W. Kodituwakku, Ph.D., Clinical Neuropsychologist and Associate Professor, Center for Development and Disability, University of New Mexico School of Medicine, Albuquerque

Socioemotional and Mental Health Issues in Individuals Prenatally Exposed to Alcohol
Mary J. O'Connor, Ph.D., ABPP, Adjunct Professor, David Geffen School of Medicine, University of California, Los Angeles (UCLA) and Training Director, UCLA Tarjan Center for Excellence in Disabilities Education, Research, and Service

ARND Symptoms of Dysregulation and Poor Adaptive Functioning
Julie A. Kable, Ph.D., Assistant Professor, Department of Pediatrics, Emory University School of Medicine, Atlanta, GA; Assistant Director, Fetal Alcohol and Drug Exposure Center, Marcus Autism Center, Atlanta, GA

Animal Models of FASD: Focus on Behavior
Joanne Weinberg, Ph.D., Professor and Distinguished University Scholar, Department of Cellular and Physiological Sciences, University of British Columbia, Vancouver, British Columbia, Canada

Question 2: How can ARND be differentiated from other disorders?

The Role of Genetic Investigations in the Assessment of Children at Risk for FASD
Albert Chudley, M.D., FRCPC, FCCMG, Professor, University of Manitoba, Winnipeg, Canada; Medical Director, Winnipeg Regional Health Authority Program in Genetics and Metabolism, Canada

Differential Diagnosis of ARND: Other Toxic Exposures
Joseph L. Jacobson, Ph.D. [presenter], and Sandra W. Jacobson, Ph.D., Professors, Department of Psychiatry and Behavioral Neurosciences, Wayne State University School of Medicine, Detroit, MI; and Honorary Professors, Departments of Human Biology and Psychiatry, University of Cape Town Faculty of Health Sciences, South Africa

Ecological Factors: Influence of Diagnostic Criteria for ARND
Ira J. Chasnoff, M.D., President, Children's Research Triangle and Professor, Clinical Pediatrics at the University of Illinois College of Medicine, Chicago

Specificity of the Neurobehavioral Profile of ARND: Comparisons With Attention Deficit Hyperactivity Disorder (ADHD)

Jeffrey R. Wozniak, Ph.D., Associate Professor, Division of Child and Adolescent Psychiatry, University of Minnesota, Minneapolis [Presenter], and Sarah Mattson, Ph.D., Professor, Department of Psychology, San Diego State University and Associate Adjunct Professor, Department of Psychiatry, University of California, San Diego

ARND: Mechanisms of Phenotype Expression and Comorbidity
Larry Burd, Ph.D., Professor, Department of Pediatrics, University of North Dakota School of Medicine; Director, North Dakota Fetal Alcohol Syndrome Center and FAS Clinic, Grand Forks

Question 3: What prenatal alcohol exposure evidence is necessary for an ARND diagnosis?

Models of FASD/ARND: What Moderate Ethanol Exposure Paradigms Suggest About Fetal Alcohol Effects and Fetal Alcohol Exposure
Daniel Savage, Ph.D., Regents' Professor and Chair, Department of Neurosciences, University of New Mexico School of Medicine, Albuquerque; Director, New Mexico Developmental Alcohol Research Center, Albuquerque

What Evidence Is Necessary for an ARND Diagnosis? What Do We Know About the Effects of Low and Moderate Levels of Prenatal Alcohol Exposure?
Nancy L. Day, Ph.D., Professor, Western Psychiatric Institute and Clinic, University of Pittsburgh School of Medicine, Pennsylvania

What Prenatal Alcohol Exposure Is Necessary for an "ARND" Diagnosis?
Susan Astley, Ph.D., Professor, Center on Human Development and Disability, University of Washington, Seattle; Director, Washington State Fetal Alcohol Syndrome Diagnostic and Prevention Network

Population Issues and Moderators of Risk for FASD
Philip A. May, Ph.D., Research Professor, Gillings School of Global Public Health, University of North Carolina, Chapel Hill; Adjunct Professor Of Pediatrics, Sanford School of Medicine, University of South Dakota, Sioux Falls; Professor Emeritus, Center on Alcoholism, Substance Abuse, and Addictions, University of New Mexico, Albuquerque; and Extraordinary Professor, Faculty of Health Sciences, University of Stellenbosch, South Africa

Question 4: What signs and symptoms will be useful as screening criteria?

Screening for ARND in the Context of Developmental Delay and Other Red Flags: Perspectives From Primary Care and Subspecialty Practice
Christine Loock, M.D., FRCPC, DABP, Associate Professor and Developmental and Social Pediatrician, University of British Columbia, Vancouver, British Columbia, Canada

Collaboration With Schools To Screen for ARND
Molly N. Millians, D.Ed., Special Educator Evaluator, Fetal Alcohol and Drug Exposure Clinic, Marcus Autism Center, Atlanta, GA

The Minnesota Experience: Establishing Systems of Care for Fetal Alcohol Spectrum Disorders (FASD)—Screening, Referrals, Diagnosis, and Interventions
Mary Jo Spencer, R.N., CPNP, M.P.H., FASD Clinical Consultant, Minnesota Organization on Fetal Alcohol Syndrome; Pediatric Nurse Practitioner, University of Minnesota Physicians, Department of Pediatrics and University of Minnesota/St. Joseph's Home for Children Clinic Collaboration, Minneapolis, MN

Question 5: What are the treatment needs for those diagnosed with ARND?

What Are the Treatment Needs of Individuals With ARND and Their Families? General Overview
Heather Carmichael Olson, Ph.D., Faculty, Department of Psychiatry and Behavioral Sciences, University of Washington School of Medicine; Fetal Alcohol Syndrome Diagnostic and Prevention Network, Seattle Children's Hospital Child Psychiatry Outpatient Clinic, Seattle Children's Research Institute, Families Moving Forward Program

Early Intervention for Fetal Alcohol Spectrum Disorders
Blair Paley, Ph.D., Clinical Professor, Department of Psychiatry and Biobehavioral Sciences, Semel Institute for Neuroscience and Human Behavior, David Geffen School, University of California, Los Angeles

Empirically Validated Treatment Approaches for School-Age Children With FASD
Joanne F. Rovet, Ph.D., Professor, University of Toronto, Ontario, Canada; Senior Scientist, Neuroscience and Mental Health Program, Hospital for Sick Children, Toronto

Treatment Needs and Interventions for Adolescents With an FASD
Jacqueline Pei, Ph.D., R.Psych., Assistant Professor, Department of Educational Psychology, and Assistant Clinical Professor, Department of Pediatrics, University of Alberta, Edmonton, Alberta, Canada

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Disclosure Statement

All of the panel members who participated in this conference and contributed to the writing of this statement were identified as having no financial or scientific conflict of interest, and all of them signed forms attesting to this fact. Unlike the expert speakers who presented scientific data at the conference, the individuals invited to participate on the consensus panel were reviewed prior to selection to ensure that they were independent and unbiased, and not proponents of any advocacy position with regard to this topic.

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Interagency Coordinating Committee on Fetal Alcohol Spectrum Disorders (ICCFASD)

ICCFASD (formerly called the Interagency Coordinating Committee on Fetal Alcohol Syndrome) was created in October 1996, following a recommendation in the Institute of Medicine's *Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention, and Treatment* report that the National Institute on Alcohol Abuse and Alcoholism chair a broad Federal effort to coordinate activities associated with FAS and related health conditions. The mission of ICCFASD is to enhance and increase communication, cooperation, collaboration, and partnerships among disciplines and Federal agencies to address health, education, developmental disabilities, alcohol research, and social services and justice issues that are relevant to disorders related to prenatal alcohol exposure. (More information about ICCFASD, its mission, vision, membership, work groups, and past activities is available at *http://www.niaaa.nih.gov/AboutNIAAA/Interagency/Pages/default.aspx*.)

ICCFASD Executive Committee

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FASD Resources

Several organizations are involved in ongoing research, program evaluation, and advocacy for improved diagnosis, treatment, and public awareness of FASD, including ARND. A few of these organizations are:

Centers for Disease Control and Prevention, Fetal Alcohol Spectrum Disorders, at *http://www.cdc.gov/ncbddd/fasd/index.html*.

National Institute on Alcohol Abuse and Alcoholism, at *http://www.niaaa.nih.gov*.

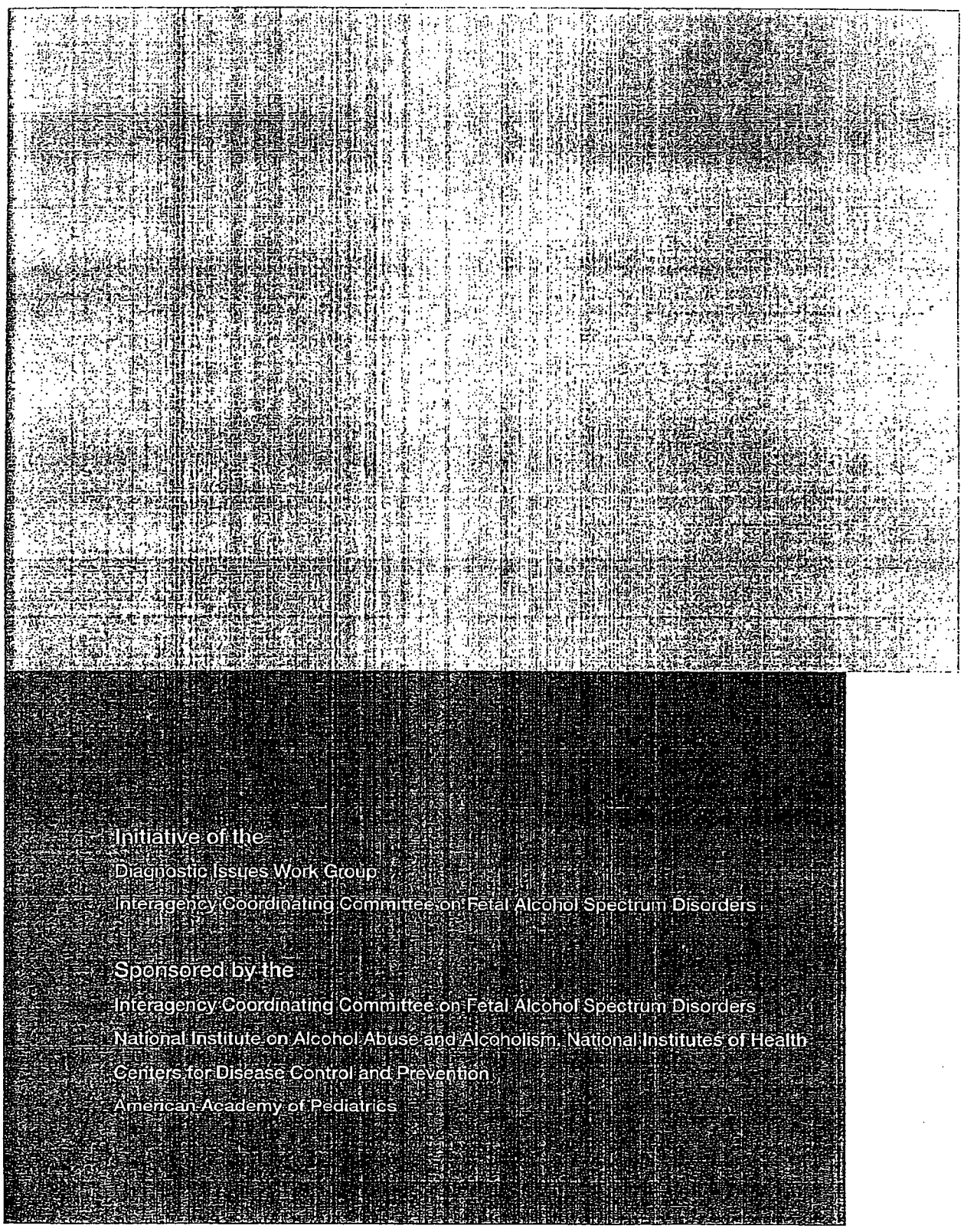
Substance Abuse and Mental Health Services Administration, FASD Center for Excellence, at *http://www.fasdcenter.samhsa.gov*.

American Academy of Pediatrics, at: *http://aappolicy.aappublications.org/index.dtl*.

Collaborative Initiative on Fetal Alcohol Spectrum Disorders, at *http://www.cifasd.org*.

Fetal Alcohol Spectrum Disorders Study Group, at *http://fasdsg.org*.

National Organization on Fetal Alcohol Syndrome, at *http://www.nofas.org*.


Initiative of the
Diagnostic Issues Work Group
Interagency Coordinating Committee on Fetal Alcohol Spectrum Disorders
Sponsored by the
Interagency Coordinating Committee on Fetal Alcohol Spectrum Disorders
National Institute on Alcohol Abuse and Alcoholism, National Institutes of Health
Centers for Disease Control and Prevention
American Academy of Pediatrics

RECOGNITION OF THE FETAL ALCOHOL SYNDROME IN EARLY INFANCY

KENNETH L. JONES DAVID W. SMITH

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Summary Historical reports indicate that the observation of an adverse effect on the fetus of chronic maternal alcoholism is not new. Three additional cases of the fetal alcohol syndrome have been recognised in two newborn infants and a 7-month-old baby. The immutable nature of the prenatal-onset growth deficiency was further confirmed. The first necropsy performed on a patient with fetal alcohol syndrome disclosed serious dysmorphogenesis of the brain, which may be responsible for some of the functional abnormalities and the joint malposition seen in this syndrome.

Introduction

A PATTERN of altered growth and morphogenesis has lately been described in eight offspring of chronic alcoholic mothers.[1] We call this disorder the "fetal alcohol syndrome". The purpose of this report is to draw attention to historical evidence which indicates that the observation of fetal malformation associated with maternal alcoholism is not new and to describe three additional cases of the fetal alcohol syndrome, with special reference to recognition of this syndrome in the newborn baby. The main abnormalities of these three new cases and the original eight are summarised in the accompanying table.

Historical Review

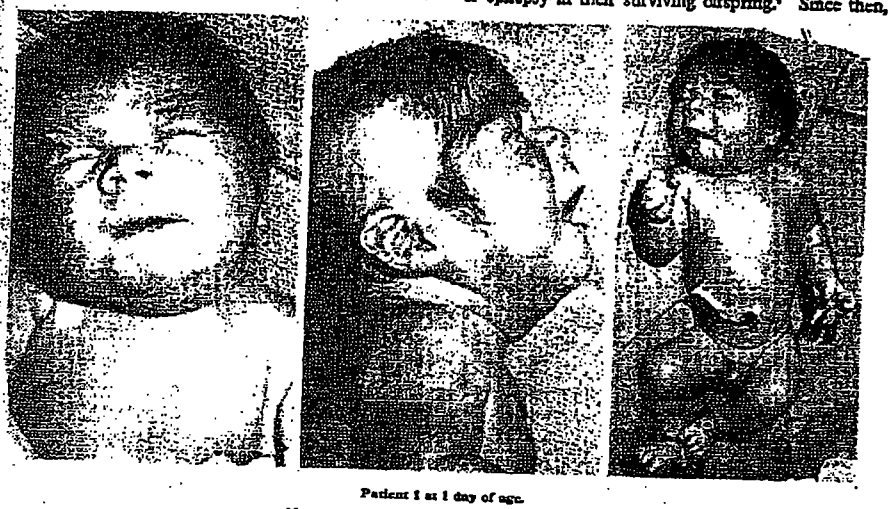
An association between maternal alcoholism and faulty development of the offspring is alluded to in early Greek and Roman mythology. In Carthage, the bridal couple was forbidden to drink wine on their wedding night in order that defective children might not be conceived.[2] In 1834 a report to the House of Commons by a select committee investigating drunkenness indicated that infants born to alcoholic mothers sometimes had "a starved, shrivelled and imperfect look".[3] In 1900, Sullivan reported increased abortion and stillbirth rates among chronic alcoholic women and an increased frequency of epilepsy in their surviving offspring.[4] Since then,

ABNORMALITIES IN THE THREE PATIENTS IN THE PRESENT STUDY PLUS THE PREVIOUS EIGHT PATIENTS WITH THE FETAL ALCOHOL SYNDROME

Abnormality	No. affected
Performance:	
Prenatal growth deficiency	11
Postnatal growth deficiency*	10
Developmental delay*	10
Craniofacies:	
Microcephaly	10
Short palpebral fissures	11
Epicanthal folds	4
Maxillary hypoplasia	7
Cleft palate	2
Micrognathia	3
Limbs:	
Joint anomalies	8
Altered palmar crease pattern	8
Other:	
Cardiac anomalies	7
Anomalous external genitalia	4
Capillary hemangiomata	4
Fine-motor function	9

The number of patients with each abnormality includes only those patients for whom a definite decision could be made. Joint anomalies consist of limitation of motion at elbow, interphalangeal and metacarpal-phalangeal joints, and/or hip dislocation. Fine-motor dysfunction consists of tremulousness, weak grasp, and/or poor eye/hand coordination.

* Not assessed in patient 2, who died 5 days after birth.



Patient 1 at 1 day of age.
Note short palpebral fissures and hirsutism.

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sporadic clinical reports have appeared suggesting an association between maternal alcoholism and serious abnormalities in the offspring.[1-4]

The effects of ethanol on early morphogenesis in laboratory animals, reviewed by St. Sandor of Rumania,[5] are variable. St. Sandor demonstrated ethanol-induced dysmorphogenesis in chick as well as albino-rat embryos.[5-11] In the developing chick, deformed brain vesicles and spinal cord, abnormal development of somites, and retardation of general growth and stage of morphogenesis were noted.[9-10] Extrapolating from these animal studies, he warned in 1968 and again in 1971 that there is "a serious danger signal of prenatal risk of ethanol intoxication during early pregnancy in humans".[11]

Case-reports

Patient 1

A newborn American Indian male (see accompanying figure) was ascertained because his 30-year-old mother was a chronic alcoholic. His mother had been an alcoholic for six years before his birth, during this time she also had three first-trimester spontaneous abortions. Her seven other children, all born before she became chronically alcoholic, are living in foster homes and are reported to be of average stature and intelligence. She had cirrhosis and nutritional anæmia, had experienced delirium tremens, and had been admitted to hospital twice with upper-gastrointestinal-tract bleeding secondary to alcoholic gastritis. She drank about two quarts of red wine daily throughout this pregnancy, and her nutritional intake seemed inadequate by history. At the end of the first trimester, she was admitted to hospital with diphtheria and treated with benzathine penicillin, diphtheria antitoxin, and erythromycin. Maternal weight-gain during pregnancy totalled 15 lb. Shortly after delivery, her nutritional status was assessed. Iron deficiency was indicated by a packed-cell volume of 25%, a serum-iron of 52 µg. per 100 ml., a total iron-binding capacity of 500 µg. per 100 ml., and a percentage iron saturation of 10%. Other studies of nutritional status were normal, including serum vitamin A, vitamin C, folic acid, and total protein and albumin. Delivery was from a breech presentation after a 38-week gestation, during which there was limited fetal activity. Endotracheal intubation was performed shortly after birth, because of a 1-minute Apgar score of 1 that rose to 6 after 5 minutes. The attending physician noted "alcohol on his breath". Birth-weight was 2020 g. (50th percentile for 34 weeks' gestation), birth length was 43 cm. (50th percentile for 32·5 weeks' gestation), and head circumference was 29 cm. (below the 3rd percentile).

There was pronounced hirsutism, especially over the forehead. The eyes were small and the palpebral fissures measured 1·1 cm. on the right and 1·2 cm. on the left eye. A grade 2 out of 6 systolic murmur was thought to represent a ventricular septal defect. There was a left congenital hip dislocation and bilateral simian creases. The immediate neonatal period was complicated by the following problems: mild respiratory distress lasting 5 days and requiring 40% ambient oxygen concentration, transient hypoglycæmia in the first 24 hours, and unexplained hypocalcæmia and hyperbilirubinæmia in the second 24 hours. Tremulousness, noted soon after birth, was initially thought to be secondary to alcoholic withdrawal but did not respond to sedation with phenobarbitone and was still present at 4 weeks of age. The infant also had a weak suck. He was discharged to a foster home. Despite an intake of 140 calories per kg., most by nasogastric tube, he gained only 410 g. in the first 4 weeks, at which time his length was 43 cm. and head circumference 31 cm.

Patient 2

A newborn American Indian female was ascertained because her 40-year-old mother had been a chronic alcoholic for an unknown time. Although no complications of alcoholism had been recorded, the mother had a blood-alcohol level of 157 mg. per 100 ml. during an afternoon clinic visit at which she was not obviously drunk. Maternal weight-gain during pregnancy was 11 lb. Delivery at 32 weeks' gestation was from vertex presentation. Apgar score was 5 and rose to 8 at 5 minutes. Birth-weight was 1300 g. (50th percentile for 30 weeks' gestation), birth length was 38·5 cm. (50th percentile for 29 weeks' gestation), and head circumference was 27 cm. She was unusually hirsute, especially over the forehead. She had obvious microphthalmia. There was a cleft of the soft palate. The superior helices of both ears were incompletely developed. The following joint anomalies were present: overlapping of the third fingers over the second fingers; clinodactyly of the left fifth finger; and camptodactyly of the right third finger, with absence of the distal interphalangeal crease. There was a harsh systolic murmur along the left sternal border. The vagina was bisceptate. There were only 2 vessels in the umbilical cord. Cyanosis developed at 5 hours of age and multiple apnœic episodes culminated in death at 5 days of age. Necropsy revealed a membranous ventricular septal defect and areas of focal pulmonary atelectasis. The brain weighed only 140 g. Histological examination disclosed extensive developmental anomalies, including aberration of neuronal migration resulting in multiple heterotopias. The anterior superior gyri were fused through infiltration by leptomeningeal hamartomata of glial and neuronal cells. The cerebral cortex was incompletely developed, as shown by relative agyria (lissencephalia) and large lateral ventricles, and there was agenesis of the corpus callosum.

Patient 3

A 7-month-old American Indian female was retrospectively ascertained because her pattern of malformation was that of the fetal alcohol syndrome. Her mother, who was deemed to be a severe alcoholic, was treated in hospital before pregnancy for complications of chronic alcoholism and again 3 weeks post partum for severe alcoholic neuropathy. Gestational timing is unknown. Birth-weight was 964 g. When evaluated at 7 months of age, she weighed only 2260 g. and was 46 cm. long (both measurements are at the 50th percentile for 35 weeks' gestation). She had short palpebral fissures. There was a cleft of the soft palate. A grade 3 out of 6 systolic murmur was thought to be secondary to a ventricular septal defect. The hips abducted poorly, and there was limitation of complete extension at both elbows. The labia majora were hypoplastic. There was a 1×1 cm. capillary hæmangioma on the back. She was hypertonic and exhibited increased motor activity when disturbed, but otherwise there was little spontaneous activity. At 7 months her developmental age was estimated to be at a 2–3 month level.

Her maternal step-brother was evaluated elsewhere by Dr John Opitz and was also judged to have the fetal alcohol syndrome. He was the product of a 32-week pregnancy complicated by maternal gastrointestinal hæmorrhage at 26 weeks' gestation due to chronic severe alcoholism. Delivery was by cæsarean section because of transverse lie. Birth-weight was 1530 g. At 7 weeks of age he was 44 cm. long, weighed 2693 g., and had a head circumference of 29 cm. There was a submucous cleft palate. He had a left simian crease. There was a grade 3 out of 6 systolic murmur. Prominent glabellar

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and occipital capillary hæmangiomata were present. Neurological examination revealed decorticate rigidity, hyperacusis, and myoclonic jerk-like seizures.

Chromosome studies were carried out on all three patients evaluated by us and disclosed no abnormality.

Discussion

The pattern of altered growth and morphogenesis in the two newborn babies we describe is strikingly similar to that of the previously reported children with the fetal alcohol syndrome. Pertinent additional findings include the following: both newborn infants had serious problems of respiratory adaptation, and one of them had problems with biochemical adaptation, as shown by hypoglycæmia, hypocalcæmia, and hyperbilirubinæmia. The observation of mild microphthalmia in both newborn babies tends to accord with the suggestion that the consistently short palpebral fissures in this syndrome are secondary to reduced ocular growth.

All three patients were judged to have a cardiac anomaly, further emphasising the frequency of this defect in the fetal alcohol syndrome. Two of them had a cleft soft palate, a new observation in this disorder.

The findings in the brain of patient 2, the first case of the fetal alcohol syndrome on whom a necropsy was performed, are of special relevance. There was serious disorientation of both neuronal and glial elements as well as incomplete development of the brain which must have started before 80 days' gestation, judging from the absence of the corpus callosum. Some of the functional and structural abnormalities in this syndrome may relate to the types of aberration in brain morphogenesis observed in this patient. These secondary features include microcephaly, developmental delay, and fine-motor dysfunction, which showed itself in early infancy by tremulousness. Some of the joint anomalies could be related to neurological impairment of the fetus, including reduced movement.

The most profound degree of prenatal-onset growth deficiency yet noted in the fetal alcohol syndrome occurred in patient 3. The findings in patient 3, who at 7 months of age was still below normal newborn size and showed no growth response to high-caloric feedings, provide further evidence of the immutable nature of the adverse prenatal effect on growth-rate in this disorder. The findings in the two newborn infants accorded with the previous observation that prenatal growth in length is more severely affected than weight gain.

The mother of patient 1 provided us with the first opportunity to study the nutritional status of a chronically alcoholic woman at the time of birth of her affected child, and of investigating the suggestion that a secondary nutritional deficiency could be the cause of the syndrome. Serious iron deficiency was the only abnormality detected. Since neither this degree of growth deficiency nor this pattern of malformation have to our knowledge been noted in offspring of iron-deficient anæmic women who were non-alcoholic, it seems unlikely that iron-deficiency anæmia is the cause of this disorder.

The risk of the initial diagnosis being wrong is high. For example, the 18 trisomy syndrome and Cornelia de Lang syndrome were seriously considered by the physicians who initially evaluated these patients. An incorrect diagnosis could lead to inappropriate advice about the risk of malformation in future children. Risk of fetal alcohol syndrome in future children is potentially high, as indicated by patient 3, unless maternal alcoholism is controlled.

Additional studies are needed to determine the incidence of the fetal alcohol syndrome in offspring of alcoholic mothers. Preliminary data from the collaborative study of the National Institute of Neurologic Disease and Stroke indicate a 32% incidence of this condition in offspring of women whose chronic alcoholism was ascertained during their pregnancy. The results of that study will form the basis of a future report.

We thank Dr John Opitz for calling to our attention patient 3, whom we had previously evaluated; Dr Frederick Harvey who performed the neuropathological evaluation of patient 2; Mrs Mary Ann Harvey and Dr Gordon Green, Dr K. M. Laurance, and Dr Jan Langman for making us aware of pertinent historical data; Mrs Lyle Harrah, research librarian; Mrs Christine Hansen for secretarial assistance; and Mr Bradley Gong for the photography. This study was supported by grants from the Maternal and Child Health Services, the National Institutes of Health, and the National Foundation-March of Dimes.

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PERSON-TO-PERSON SPREAD OF SALMONELLA: A PROBLEM IN HOSPITALS?

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Summary 8 patients with acute diarrhœa were admitted to hospital and appropriate stool-barrier techniques were not used to avoid secondary spread. These patients later proved to have salmonellosis. Their surroundings were extensively contaminated, exposing 265 patients and staff to infected fæces. No case of symptomatic or asymptomatic salmonellosis was found as a result of person-to-person spread. It is concluded that person-to-person spread of salmonella in hospital is difficult to accomplish without an intermediary common vehicle.

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Understanding the Occurrence of Secondary Disabilities in Clients with Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE)

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1 Executive Summary

The purpose of this research project was:

- To examine the types and magnitude of secondary disabilities that are associated with Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE), and
- To assess the risk and protective factors that might alter the rates of occurrence of secondary disabilities.

For the purposes of this study, primary disabilities are defined as those that reflect the CNS dysfunctions inherent in the FAS or FAE diagnosis. Secondary Disabilities are those that a client is not born with, and that could presumably be ameliorated through better understanding and appropriate interventions.

Primary Disabilities associated with FAS/FAE were examined in 473 clients who ranged in age from 3 to 51 years. Those with FAS (n= 178) had an average IQ of 79, average reading, spelling, and arithmetic standard scores of 78, 75, and 70, respectively, and an average Adaptive Behavior standard score of 61. Those with FAE (n= 295) had an average IQ of 90, average reading, spelling, and arithmetic standard scores of 84, 81, and 76, respectively, and an average Adaptive Behavior score of 67. (For both IQ and Adaptive Behavior, a score of 100 is normal.)

Secondary Disabilities were examined with the Life History Interview (LHI), which was developed for this study. Risk and protective factors were assessed primarily from the LHI. The LHI was administered to all available caretakers/informants of 415 clients with FAS/FAE, who ranged in age from 6 to 51 years with a median of 14.2 years. Six main secondary disabilities were studied.

- *Mental Health Problems* (MHP) was by far the most prevalent secondary disability, experienced by over 90% of the full sample.
- *Disrupted School Experience* (DSE) (defined as having been suspended or expelled from school or having dropped out of school) was experienced by 60% of the clients (12 and over).
- *Trouble With the Law* (TWL) (defined as ever having been in trouble with authorities, charged, or convicted of a crime) was experienced by 60% of the clients (12 and over).
- *Confinement* (CNF) (including inpatient treatment for mental health problems or alcohol/drug problems, or ever having been incarcerated for a crime) was experienced by about 50% of the clients (12 and over).
- *Inappropriate Sexual Behavior* (ISB) was noted for about 50% of the clients (12 and over).
- *Alcohol/Drug Problems* (ADP) was noted for about 30% of the clients (12 and over).

In an effort to determine how many clients became self-sufficient as adults, two additional secondary disabilities were evaluated for the 90 clients who were at least 21 years old (median age 26 years):

- *Dependent Living* (DPL) (operationally defined in text) characterized about 80% of the sample (21 and over).
- *Problems With Employment* (PWE) (operationally defined in text) characterized about 80% of the sample (21 and over).

Only seven of the 90 adults in this sample live independently and without employment problems (according to these definitions).

Males have higher rates of Disrupted School Experience, Trouble With the Law, and Confinement than do

females; otherwise, rates of Secondary Disabilities are nearly equal across the sexes. Clients 12 years and older have a higher rate of all Secondary Disabilities except Mental Health Problems than younger clients. Compared to clients with FAS, those with FAE have a *higher* rate of all Secondary Disabilities, except Mental Health Problems.

Adults with FAE have as high a rate of Dependent Living as do those with FAS, but a somewhat *lower* rate of Problems With Employment, which may reflect their higher IQ level.

A low IQ score (70 and below) is a protective factor for Disrupted School Experience, Trouble With the Law, Confinement, and Alcohol and Drug Problems. IQ level has little relationship to Mental Health Problems or Inappropriate Sexual Behavior, but low IQ is obviously a risk factor for Dependent Living and Problems With Employment.

A diagnosis before 6 years of age is a strong protective factor for all Secondary Disabilities except Mental Health Problems.

A set of 21 possible risk and protective factors was examined through an analysis of odds ratio plots across the first six secondary disabilities (Table 6.1 and Figure 6.1). For items that involve continuous scores, 1, 4, 5, and 8 below, the sample was divided at the median, which yields the classification appearing in the text below (for these items only). Eight factors emerged that are almost universally protective in terms of secondary disabilities. In order of their strength as "universal" protective factors, they are:

1. Living in a stable and nurturant home for over 72% of life;
2. Being diagnosed before the age of 6 years;
3. Never having experienced violence against oneself;
4. Staying in each living situation for an average of more than 2.8 years;
5. Experiencing a good quality home (10 or more of 12 good qualities) from age 8 to 12 years;
6. Having applied for and been found eligible for DDD (Division of Developmental Disabilities) services;
7. Having a diagnosis of FAS (rather than FAE);
8. Having basic needs met for at least 13% of life.

In addition to being "universal" or "specific," we also categorize risk and protective factors as either "intrinsic" (i.e. attributes of the client or measures of the clients' putative brain damage) or "extrinsic" (i.e. environmental). The following pattern of relationships between specific secondary disabilities and risk and protective factors was found through analysis of odds ratio plots:

Odds of Mental Health Problems are reduced primarily by the universal protective factors.

Odds of Disrupted School Experience are reduced primarily by the universal protective factors.

The rate of Trouble With the Law is related to all the universal protective factors, most notably: DDD eligibility for services.

Confinement also is related to the universal protective factors, especially: living in a stable and nurturant environment, and being diagnosed prior to age 6.

Odds of Inappropriate Sexual Behavior are reduced by all universal protective factors.

Alcohol and Drug Problems have one specific protective factor in addition to universal protective factors, namely: having lived with an alcohol abuser less than the median for the group, which was 30% of life.

Odds of Dependent Living are increased over fourfold for clients who had an IQ score of 70 or below, an Adaptive Behavior score below 65, or an IQ/Adaptive Behavior Discrepancy score of over 15 points. Other strong intrinsic risk factors for Dependent Living are: a high FABS score, a Performance Scale IQ minus Verbal Scale IQ score of more than 15 points, and being male. An extrinsic factor that is protective against Dependent Living is having a diagnosis before 6 years of age. (Home quality and stability,

basic needs met, living with alcohol or drug abusers, or having FAS versus FAE were not associated with Dependent Living as an adult.)

Odds of Problems with Employment are increased more than two to four fold by an IQ score of 70 or below, an Adaptive Behavior Score below 65, an IQ/Adaptive Behavior Discrepancy score of over 15 points, and being FAS rather than FAE. Some universal factors are also protective against problems with employment, namely, an early diagnosis, longer time in a stable and nurturant home, longer duration in each household, and not being a victim of violence.

We note that:

- The correlations reported may or may not be causative. They nonetheless suggest courses of action that may be beneficial both to these clients, and ultimately to society.
- Many environmental influences that appear beneficial for clients with FAS/FAE are, of course, good for all people—all the more reason that society should safeguard them for people born with a birth defect, particularly a "hidden" birth defect like FAS/FAE.
- Seven of the eight universal protective factors are extrinsic and presumably could be modified by society.
- Some of the risk factors are intrinsic, indicating that subgroups of high risk individuals could be detected with special screening techniques.
- Efforts to intervene with alcohol-affected children should proceed simultaneously with efforts to prevent future children from being born with FAS and FAE.

Highlights of Findings

1. Across the full age spectrum of 415 individuals with FAS/FAE, Mental Health Problems characterize 94%, followed by Inappropriate Sexual Behavior (45%), Disrupted School Experience (43%), and Trouble with the Law (42%) (Figure 7.1).

2. As anticipated, the most protective environmental factors *against* secondary disabilities are: living in a stable and nurturant home of good quality, not having frequent changes of household, and not being a victim of violence (Figure 6.1).

3. Three intrinsic characteristics are associated with a *higher* level of secondary disabilities: (1) having FAE rather than FAS; (2) having a higher (rather than a lower) score on the Fetal Alcohol Behavior Scale; and (3) having an IQ above 70 rather than below (Figure 6.1). Special attention should focus on clients with these "risky" characteristics in order to prevent secondary disabilities.

4. The 90 adults studied (21 years and over) had an 83% rate of living dependently and a 79% rate of problems with employment (Figure 7.1). Only 10 to 13% of the clients were classified as DDD eligible. The most important environmental factor protecting against these two secondary disabilities is an early diagnosis, suggesting that families and communities may have provided special help and opportunities for those clients identified early in life as FAS/FAE.

   Our search for intrinsic client characteristics that might identify a subgroup among those with FAS/FAE who, despite a higher IQ, are unexpectedly unable to achieve independence and satisfactory employment produced one interesting measure deserving further study. This is the "IQ minus Adaptive Behavior Discrepancy Score", which may hold promise for identifying a subgroup of clients with FAS/FAE for whom early job skills and basic living skills might be especially fruitful.

5. An early diagnosis is a strong universal protective factor for all secondary disabilities, yet only 11% of these individuals with FAS/FAE were diagnosed by age 6 (Figure 6.1). Every effort should be made to provide an early diagnosis for every child with FAS and FAE.

6. Applying for and receiving eligibility for services from the state's Division of Developmental Disabilities

(DDD) is also a strong protective factor for most secondary disabilities. The services provided by DDD would appear to be both useful and necessary for many clients with FAS/FAE who do not now qualify.

7. Violence against individuals with FAS/FAE occurred at an alarming rate: 72% had experienced physical or sexual abuse, or domestic violence. Being a victim of violence was a strong risk factor for Inappropriate Sexual Behavior, increasing the odds fourfold. Children and adults disabled by FAS/FAE must have better protection and their families may need special training and guidance about Inappropriate Sexual Behavior.

8. Thirty females with FAS/FAE had given birth to a child. Of these, 57% no longer had the child in their care; 40% were drinking during pregnancy; 17% had children diagnosed FAS or FAE; and an additional 13% had children who were suspected by the informants of having FAS/FAE. Special advocacy services for these high risk mothers who themselves have FAS/FAE and special attention to their birth control needs and child care needs should be a top priority.

Recommendations Deriving from the Findings

1. Develop statewide networks of local FAS/FAE Diagnostic Clinics coordinated with local community service providers, to facilitate early diagnosis. This could reduce disrupted schooling, trouble with the law, inappropriate sexual behavior, confinement, alcohol and drug problems, dependent living, and problems with employment, because the organic problems of the child will be recognized from an early age.

2. Develop a coordinated system of parent and citizen education centers, and a system for ongoing in-service training programs for all relevant service providers. These should focus on strategies for improving the quality of life, increasing the duration of stay in each placement, and providing the appropriate life skills and job skills training. This could decrease all secondary disabilities.

3. Develop a state inter-agency network specifically on FAS/FAE including representatives from key state and private agencies and parents groups, to identify an "FAS/FAE Coordinator" within each agency, and to develop methods for detecting, diagnosing, and serving this population. Multiple state agencies, including mental health, schools and special education, the juvenile and criminal justice system, alcohol and drug abuse treatment, and the health department must be involved in this effort. No single agency can be responsive to all the broad-based needs of people with FAS/FAE and their families.

4. Fund research on methods to quantify the Central Nervous System impairments associated with FAS/FAE in order to develop clinically-useful diagnostic tools for the neurobehavioral effects of prenatal alcohol exposure. This should facilitate diagnosis of alcohol-affected individuals without the facial dysmorphology and growth deficiency, and permit a speedier response to their service needs before the onset of secondary disabilities.

5. Develop and test new methods (such as those identified in this report) that could be utilized for modifying the eligibility criteria for services from the Division of Developmental Disabilities. Enhancing eligibility for case management, job coaching, and supervised housing should reduce the level of costly secondary disabilities among individuals with FAS/FAE who are unable to live and work independently, but are now unable to get appropriate services because they may not have an IQ below 70 or a full FAS diagnosis.

6. Fund a model long-term residential/job training program for youth and adults with FAS/FAE, and then implement this statewide and nationwide.

7. Mandate the full disclosure of medical/mental health/background history when placing a child in foster or adoptive placements, and provide education and training on FAS/FAE and appropriate support services to families raising such children, including biological families.

8. Prevent future children from being born with FAS/FAE: (1) Expand alcohol/drug inpatient treatment programs for women and their children; (2) Fund a state-wide network of Birth to 3 Advocacy programs

for working with the highest risk mothers abusing alcohol/drugs during pregnancy; and (3) Provide advocacy and free long-term birth control options to women with FAS/FAE.

Conclusions

1. People with FAS and FAE have an unacceptable level of secondary disabilities that severely impairs their quality of life and is extremely costly to society.
2. The low level of societal protection and support given to people with FAS and FAE and their families is unacceptable and further compromises their lives. They should be given an appropriate level of societal protection and support. To do this, their primary disabilities must be better understood by families, service providers, and by society at large.
3. The permanent, organic brain damage of people with FAS and FAE is often "hidden" in that it often does not conform to current system guidelines for providing services, such as: a low IQ score, a debilitating physical handicap, serious mental illness, or even an FAS face (and diagnosis).
4. By understanding the devastating secondary disabilities that characterize most individuals with FAS/FAE, and by understanding the intrinsic and extrinsic risk and protective factors that exacerbate or ameliorate these disabilities, we should be able to improve the quality of life for people with FAS and FAE and their families, and to reduce costs to society.
5. The magnitude and cost of these secondary disabilities is huge—when calculated against the estimated figure of 1 to 3 FAS per 1000 births (NIAAA, 1987) and several fold this figure for FAE.

2 Introduction

It is fitting that the International Conference on Overcoming and Preventing Secondary Disabilities in Fetal Alcohol Syndrome and Fetal Alcohol Effects is being held at the University of Washington School of Medicine, September 4-6, 1996. This University has been an important site for fetal alcohol studies for the past 23 years.

In 1973, Kenneth Lyons Jones and David W. Smith, dysmorphologists at the University of Washington Medical School, Department of Pediatrics, identified a "similar pattern of craniofacial, limb, and cardiovascular defects associated with prenatal onset growth deficiency and development" in eight unrelated children of three ethnic groups, all born to chronic alcoholic mothers (Jones, Smith, Ulleland, & Streissguth, 1973). The distinctive pattern of malformations indicated that the damage was of prenatal origin. In a second report, three more infants were identified, and the first necropsy on such a patient "disclosed serious dysmorphogenesis of the brain," which the authors thought might be responsible for some of the functional abnormalities and joint malpositions seen in the syndrome. The naming of this syndrome as Fetal Alcohol Syndrome (FAS) put the emphasis squarely on the presumed etiology and brought international attention to this phenomenon (Jones & Smith, 1973). Among the letters that came to David Smith, was one from Paul Lemoine of Nantes, France, who had published a large study of children born to alcoholic mothers who had similar features and behaviors. Lemoine and colleagues had published their findings in 1968 in a local pediatric journal, but the article had not been translated into English, nor had it appeared in the international medical literature.

From the start, both groups of investigators were fascinated with the unusual behaviors of these children who looked alike (although they were not related) and behaved in a hyperactive and unfocused manner. Through utilization of records from the National Perinatal Collaborative Project (NPCP), Jones and colleagues (1974) were able to demonstrate that 44% of the children of chronic alcoholic mothers identified during pregnancy had "borderline to moderate mental retardation" (defined as an IQ of 79 or below) when examined at 7 years of age. Thirty two percent had enough abnormal features from the physical examination alone to suggest the Fetal Alcohol Syndrome. In a carefully matched comparison group selected from over 50,000 other women and children in the NPCP study, fewer than 10% of the other children had IQ scores below 79, and none had the physical features of FAS.

Clarren and colleagues (1978) presented additional evidence of Central Nervous System (CNS) damage in patients with FAS: "neuropathologic findings on four human neonates exposed to large amounts of ethanol at frequent intervals during gestation...All four brains showed similar malformations stemming from errors in migration of neuronal and glial elements." Two had hydrocephalus and only two of the four were diagnosed as having the Fetal Alcohol Syndrome from external criteria. They concluded: "...the brain alterations may be the only distinct abnormality produced by in utero ethanol exposure." Since 1978 children who had some but not all the features of FAS have been referred to as having Fetal Alcohol Effects (FAE) (Hanson et al., 1978) or Possible Fetal Alcohol Effects (PFAE) (Clarren & Smith, 1978). Recently there have been suggestions that the terminology should be reconsidered (Aase et al., 1995; IOM, 1996).

Within 4 years of the naming of Fetal Alcohol Syndrome, experimental studies of laboratory animals were published demonstrating that alcohol is teratogenic and can produce malformations from prenatal exposure (e.g., Chernoff, 1977; Randall et al., 1977). By 1978, 245 clients with Fetal Alcohol Syndrome had been reported in the medical literature, and FAS was described as the "most frequent known teratogenic cause of mental deficiency in the western world" (Clarren & Smith, 1978). It also was recognized by this time that prenatal alcohol exposure produces a whole spectrum of effects, of which FAS is the most clearly definable.

By the mid 1980's, it was apparent that the physical features of FAS were less unique and characteristic after the onset of adolescence. Clients with this disorder whom we had watched grow up in Seattle had far more life problems than would be expected solely on the basis of their mental retardation or delayed development. Furthermore, as more clients were evaluated, it was clear that there were many children with the full features of FAS who did not technically qualify as mentally retarded, and in many instances, had trouble obtaining appropriate services (Streissguth et al., 1985). It also was clear that many children without the physical manifestations of FAS were also born to alcohol abusing mothers, and that these too had cognitive and adaptive behavior problems similar to children with FAS (Streissguth et al., 1991a).

Efforts to prevent FAS began almost as soon as alcohol was clearly identified as a teratogen, and several public policy decisions were made to enhance efforts to prevent the prenatal effects of alcohol. The Surgeon General of the U.S. (1981) recommended that women not drink alcoholic beverages during pregnancy or when planning a pregnancy. Congress passed legislation in 1989 to mandate warning labels on all alcohol beverage containers sold in the U.S. that included a warning against drinking alcohol during pregnancy.

Understanding the long-term impacts of FAS and FAE on society has taken more time. Understanding the life problems that characterize people with FAS and FAE as they grow and mature is essential to: developing effective interventions, estimating the costs to society, and mandating appropriate public policies.

FAS and FAE have been recognized for the past 20 years as major known causes of developmental disability. Yet, it is only in the past 10 years that the lifelong implications of these disabilities have been recognized. Follow-up studies in four countries have demonstrated the continuing adverse effects of prenatal alcohol exposure into adolescence and adulthood (Aronson, Olegard et al., 1987; Lemoine & Lemoine, 1992; Majewski, 1993; Spohr et al., 1993; 1994; Steinhausen et al., 1993; 1994; Streissguth et al., 1985; 1991).

In 1992, in recognition of this problem, the Centers for Disease Control and Prevention (CDC) through the National Center for Environmental Health, Disabilities Prevention Program, issued a request for proposals. The present research project was undertaken in response to that request. The aim of this project has been to build a prevention information base fundamental to the amelioration of secondary disabilities in clients with FAS and FAE.

For the purposes of this study, primary disabilities are defined as those that reflect the CNS dysfunctions inherent in the FAS or FAE diagnosis. Secondary Disabilities are those that a client is not born with, and that could presumably be ameliorated through better understanding and appropriate interventions.

The study has three main goals:

1. To document the occurrence and range of secondary disabilities that are associated with FAS and FAE.
2. To determine the risk factors associated with these secondary disabilities in order to make recommendations for preventive strategies.
3. To develop a brief Fetal Alcohol Behavior Scale (FABS) so that state/community agencies may identify clients with probable FAS/FAE who may be in need of special services to prevent additional secondary disabilities.

This document is the Final Report of that project.

3 Methods

3.1 Diagnostic Criteria

Fetal Alcohol Syndrome (FAS) is diagnosed when three primary characteristics occur together: growth deficiency, a characteristic pattern of abnormalities primarily observable in the face, and some manifestations of Central Nervous System (CNS) dysfunction. The definition of CNS criteria used here is in keeping with that originally used by Clarren and Smith (1978) and is *not* wholly consistent with the modification suggested by the recent IOM (1996) report for diagnosing FAS. Over the years, a small number of children had been diagnosed PFAS (possible or probable FAS). This term was applied to borderline cases: either the CNS and facial features were classic but the growth was marginal, or the CNS and growth deficiency were classic and the face was "almost" classic. For this report, PFAS and FAS were combined. Fetal Alcohol Effects (FAE) and PFAE (possible or probable FAE) are terms that have been used clinically to apply to individuals who manifest some, but not all of the characteristics of FAS, but were exposed prenatally to significant levels of alcohol. The terms FAE and PFAE, as they have been used by Seattle dysmorphologists since 1974, *are* consistent with the new diagnostic category of ARND (Alcohol Related Neurodevelopmental Disabilities) suggested by the IOM (1996). For this report, FAE and PFAE were combined.

In the present study, a diagnosis of FAS was assigned to clients who had: (1) a clear history of prenatal alcohol exposure; (2) dysmorphic features (primarily observed in the face, such as short palpebral fissures, a pattern of flattened midface, smooth and/or long philtrum, and thin upper lip); (3) growth retardation for height and/or weight below the 10th percentile; and (4) CNS dysfunction (as manifested by microcephaly, developmental delay, hyperactivity, attention and/or memory deficits, learning difficulties, intellectual deficits, motor problems, neurologic signs, and/or seizures). A diagnosis of FAE or PFAE was attributed to those who had a clear history of prenatal alcohol exposure and CNS dysfunction, but did not manifest all of the physical features of FAS. All diagnostic evaluations were performed by physicians trained in dysmorphology and genetics.

3.2 Ascertainment

The 661 clients in this study represent a gradually accrued group that began with the first patients diagnosed FAS in 1973 by Jones and Smith and ended with those who came to the University of Washington FAS Diagnostic Clinic between 1993 and 1995. The clients were largely ascertained through clinical referral across a 22-year period, and diagnosed by a small homogeneous group of dysmorphologists who were trained by David W. Smith at the University of Washington Dysmorphology Unit. The sample includes, but is not limited to all those available from the following published studies: 11 clients from the two 1973 Lancet papers describing FAS (Jones et al., 1973; Jones & Smith, 1973); 20 and 17 clients respectively, from the first two FAS follow-up studies (Streissguth, Herman, & Smith, 1978a and b); 8 clients from the 10-year follow-up study of the first 11 clients diagnosed with FAS (Streissguth et al., 1985); Northwest sample only (n=31) of the first FAS follow-up study of adolescents and adults (Streissguth et al., 1991a); 40 clients in the test-retest IQ study of adolescents and adults (Streissguth, Randels, & Smith, 1991b); and 24 clients participating in the FAS genotype study (Faustman et al., 1992). The published groups are overlapping, but data for each client are counted only once in the report that follows.

Ascertainment of the 661 clients was through clinical referral for an FAS diagnostic evaluation or through participation in ongoing FAS research projects conducted by the Fetal Alcohol and Drug Unit. Sterling Clarren was the primary diagnostician, diagnosing two-thirds of the clients; David W. Smith, Kenneth L. Jones, and Smith's other dysmorphology fellows diagnosed 7% of the sample. Twenty four percent of the sample were diagnosed during or before 1990, 15% were diagnosed 1991-1992, and 61% were diagnosed in 1993 or later at the University of Washington FAS Diagnostic Clinic, directed by Sterling Clarren. The clients were primarily from the Pacific Northwest.

The University of Washington FAS Diagnostic Clinic was established with primary funding from CDC in January 1993. Patients are referred to the FAS clinic by physicians, parents, teachers, caseworkers and other concerned caregivers. Patients are typically referred for cognitive and behavioral problems, and must have a confirmed or suspected history of prenatal alcohol exposure. Only a small number of patients are referred to the clinic based on there being a history

of prenatal alcohol exposure, without major concern for behavioral or cognitive delays. The clinic meets one day per week and can accommodate 4 or 5 patients per day. As many more clients apply than can be seen, priority is given to those whose biologic mothers are living and known to be in the area, in keeping with the goals of the CDC grant that provides basic funding for the FAS clinic (Clarren & Astley, 1997).

3.3 Recruitment

Patients were referred from the FAS Diagnostic Clinic for our Secondary Disabilities Study. Those clients who met diagnostic criteria and who, along with their caregivers, consented to participate were enrolled in the study. The study, which was approved by the University of Washington Human Subjects Review Committee, was explained to the families by our client advocate. Additionally, a Confidentiality Certificate was obtained from the Public Health Service to further protect the clients and their families. Three kinds of consents were obtained: (1) for participation in the study, (2) for photographs, and (3) for release of information from schools, hospitals, etc. Clients in our other FAS research projects who met the diagnostic criteria were also notified about the new study and asked to participate. Caregivers were contacted over a four-year period to gather and update information on past and current client status, Secondary Disabilities, and Risk and Protective Factors. Reports of psychological evaluations conducted for research purposes were provided upon request to the clients or their caregivers. In case of a crisis call, clients were referred to appropriate community agencies and professional local services.

3.4 Samples

This final report utilizes two overlapping samples from the 661 clients in this study, one sample for describing the Primary Disabilities (n=473) and one for describing the Secondary Disabilities (LHI Sample, n=415—see Table 3.1). All clients were diagnosed either FAS, FAE, PFAS, PFAE, or ARND as described above. Clients from southwest Indian reservations who were included in earlier reports (Streissguth et al., 1988 Manual; Streissguth et al., 1991a; LaDue, Streissguth, & Randels, 1992) are not included in the current report because their recruitment and follow-up were not comparable to those clients in the Northwest. The Primary Disabilities sample includes clients from three years of age and up; the Secondary Disabilities sample includes those 6 years and older. Although the two samples are not entirely nested, the referral source, the diagnosing physician, and the age and date of diagnosis were comparable for the two samples.

Table 3.1 Secondary Disabilities Sample (LHI): enrollment and interview status

Interview status	(n)
Enrolled patients	661
too young (< 6 years old)	82
SW reservation clients	64
enrolled too late/data not processed in time	9
family could not be found	52
caretaker refused interview	22
caretaker not available for interview	16
invalid interview	1
Total valid interviews conducted	415

The 91 eligible clients for whom LHI's were not obtained do not differ from the 415 for whom LHI's were obtained in terms of alcohol-related diagnosis, age at diagnosis, frequency of diagnosis prior to 1980, ethnicity, or sex. The loss of these 91 does not seem to present a bias in extent of Primary Disability in that they do not differ significantly from the 415 valid interviews in: IQ (as measured either by mean IQ score or by number of IQ scores at or below 70); achievement (WRAT reading, spelling, arithmetic scaled scores); or adaptive behavior (VABS: Communications, Daily Living Skills, Socialization, and Adaptive Behavior Composite). The interviewed sample appears to be fairly representative of the full set of eligible clients accumulated in this database.

3.5 Primary Disabilities Sample (IQ): Tests Administered

Clients were administered an age-appropriate Wechsler IQ Test: Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R); Wechsler Intelligence Scale for Children-Revised (WISC-R); and the Wechsler Adult Intelligence Scale-Revised (WAIS-R) (Wechsler, 1967, 1974, 1981). The Wide Range Achievement Test-Revised (WRAT-R) (Jastak & Wilkinson, 1984) was also administered individually to each client, and the Vineland Adaptive Behavior Scale (VABS) (Sparrow et al., 1984) was administered to a caretaker or person who knew the client well, usually at the time the IQ and achievement tests were administered. Testing was carried out primarily at the Fetal Alcohol and Drug Unit (FADU) or in clients' homes or schools.

Every effort was made to schedule testing at a time and location convenient for the client and caregiver, including evenings and road trips to the client's school or home. If clients were unable to be seen for psychological testing, scores from previous IQ and achievement evaluations were used for analysis when available. Reports of psychological evaluations were systematically requested from the schools. All applicable scores were entered into the database and used in analysis. The clients were not asked to come in for psychological evaluations if they had been administered IQ tests within the past year and achievement tests within the past 6 months.

The following psychological test scores were used in the present analyses: From the WPPSI, WISC-R, and WAIS-R: the full scale IQ score, the Verbal Scale IQ score (VIQ), the Performance Scale IQ score (PIQ), and the 11 Subtest Scores were used. WISC-III IQ scores, corrected for comparability with the WISC-R, were used for 11 clients who either could not be seen at our lab for testing or who had been tested at their schools on the WISC-III within the prior year, so were not eligible for retesting. From the WRAT-R: the Standard Scores (SS) for Reading, Spelling, and Arithmetic were used. From the VABS: the Standard Scores from the Adaptive Behavior Composite (ABC) and the Standard Scores for Socialization, Communication, and Daily Living Skills were used.

3.6 Secondary Disabilities Sample: Life History Interview (LHI)

The Life History Interview (LHI) was developed in the third year of the project to evaluate clients of any age and any degree of disability in order to attain maximum coverage of the sample. The focus of the LHI was on the kinds of Secondary Disabilities and Risk and Protective Factors that characterize these clients. The interview was administered by telephone to caretakers/informants of the clients.

The LHI grew out of our experience with deriving secondary disability data from the clinical database that has been accruing during the many years we have been following these clients. Some preliminary secondary disabilities data, coded directly from our clinical records, were presented previously (Streissguth, Kopera-Frye, & Barr, 1994). Higher rates of Secondary Disabilities were obtained from the LHI than from abstracting our clinical database.

The LHI is a comprehensive structured evaluation of ten major areas of possible long-term functional covariates or consequences characteristic of clients diagnosed with FAS/FAE: (1) household and family environment; (2) independent living and financial management; (3) education; (4) employment; (5) physical abuse, sexual abuse and domestic violence; (6) physical, social and sexual development; (7) behavior management and mental health issues; (8) alcohol and drug use; (9) legal status and criminal justice involvement; and (10) companionship and parenting. These areas of concern were explored in terms of past and current client status, secondary disabilities, and possible risk and protective factors.

Interviewers phoned ahead of time to schedule a convenient time for the interviewee and asked to interview the person who knew the client best. Adoptive mothers were the most frequent informants, 33%; followed by biologic mothers, 17%; foster mothers, 12%; stepmothers, 6%; fathers (all types), 7%; legal guardians, 7%; grandmothers, 4%; other relatives, 4%; caseworkers, 4%; spouses or partners, 1%; residential caretakers, 1%; and by others, 2%. A small proportion of the clients, 2%, had no one available who knew them well, so were interviewed themselves for the LHI. Self-report data were only used when they appeared valid in terms of existing data in the clinical database. A total of 415 valid interviews were obtained and coded during a 4-month interval in 1995.

The 37-page LHI contains over 450 separate questions organized to provide the interviewer with clear visual guides for accurate coding. Most questions required the interviewer to code a choice according to a prespecified list of responses. Other questions permitted open-ended responses, which were written in verbatim and coded later. Validity ratings by the interviewer follow each section of the LHI. A section was coded "valid" when the responses appeared to be genuine and focused on the questions being posed. A section was coded "questionable" if the informant did not know the client well, seemed guarded, seemed confused and/or contradicted himself/herself, seemed to be biased, seemed hostile, did not understand or speak English very well, or seemed mentally handicapped. All sections with questionable validity codes were reviewed prior to data analysis.

Seven interviewers were trained in administration and coding procedures by the project director, who was regularly available to address queries. Each completed interview was reviewed by the team of interviewers, and coding consensus was reached for any items under contention. Finally, all coded interviews were reviewed by the project director before they were submitted for data entry.

The LHI took, on average, 70 minutes to administer (range, 18 minutes to 3½ hours). Interviews took longer to administer when they involved older clients or those with a greater number of Secondary Disabilities. Interview length was not related to sex, race, or fetal alcohol-related diagnosis of the client.

Most caregivers felt that the LHI covered major areas of the client's functioning, in terms of prevalent problems, possible environmental buffers, and assessment of service needs. Many caregivers stated they felt that the data generated from this interview, once disseminated to the service agencies and community advocates, would help them provide better care for their children with FAS/FAE.

3.7 Quantifying Data Across the Life Span

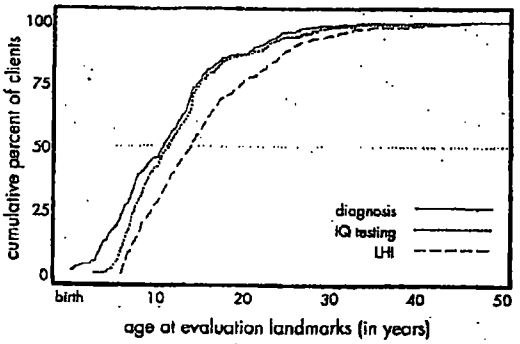
Data presented in the following chapters were collected across the life span of each client. A given client may have been diagnosed FAS (or FAE) at age 3 years, given an IQ test at age 5 years, and had a caretaker provide information for the LHI when the client was 16 years old. In addition, questions on the LHI assess the onset for many outcomes (age of onset of alcohol/drug problems, age of onset of problems in school). Thus, a given client may contribute data to any of several age groups depending on the topic at hand.

Figure 3.1 presents the cumulative age distributions for ages at which three key evaluation landmarks were made: diagnosis, IQ testing, and LHI. One at a time, follow the dotted horizontal line (at 50%, or median) to each of the three curves and drop a vertical line to the "age" axis. It is seen that the median ages at diagnosis, IQ testing, and LHI are approximately 10, 11, and 14 years, respectively. The dashed line for LHI is to the right of the other lines indicating that this was generally the most recent data collected.

The eight Secondary Disabilities are defined by any history of the secondary disability since birth. Thus, a comment that 59% of clients 21 years and older were reported as having a history of Disrupted School Experience does not mean that problems in school happened after age 21 years; the disruptions may have occurred at much younger ages. Ages at interview correspond to age covered by the history of secondary disability as follows:

- History of secondary disability for clients 21-51 years old at interview covers ages: birth-age at LHI (birth to 51 years)
- History of secondary disability for clients 12-20 years old at interview covers ages: birth-age at LHI (birth to 20 years)
- History of secondary disability for clients 6-11 years old at interview covers ages: birth-age at LHI (birth to 11 years).

3.1 Client ages at evaluation landmarks (LHI sample, n=415)



3.8 Strengths and Limitations of Study Design and Analysis

The strengths of this study design are: It attempts complete coverage of a large and established roster of clients, recruited in a similar manner and examined in a uniform setting. There is no apparent bias in data collection or cooperation. The clients span a broad spectrum of ages, socio-economic backgrounds, and rearing conditions. The instrument (the Life History Interview—LHI), in the hands of a skilled interviewer, seems robust over a huge range of informants and across the full age range of clients, 6 through 51 years, providing extensive coverage of life experience. The LHI has a rich and redundant coding scheme covering many different kinds of behaviors and difficulties.

The limitations of the study design are: It does not involve a representative sample of any defined population. It contains no measures of dose except that all clients were born to mothers who abused alcohol. The diagnostic categorization, although carried out by a small number of dysmorphologists with the same training, is subject to temporal changes in interpretation and referral patterns. Effective utilization of the LHI requires gifted interviewers, trained by those with extensive experience with FAS across the lifespan, and the prior establishment of trust and rapport with families. Nevertheless, the LHI data are only as valid as the knowledge and capabilities of the informants. Obviously there was no random assignment of clients, caretakers, or interventions.

The strengths of the data analysis are: It represents an effective distillation of the vicissitudes of hundreds of lives into a small number of major channels of secondary disability. The sample is large enough to subset informatively in many different and interesting ways. Statistical analysis easily translates into English sentences. Findings are strong and plausible in respect of aspects of the client and also of the client's environment. Many risk/protective factors common to most secondary disabilities have been detected. The statistics are quite simple; the graphics are powerful. The findings lead easily to policy implications and recommendations.

The limitations of the data analysis are: There is no real possibility of assessing causation. The secondary disabilities are intercorrelated as are the risk/protective factors. There is no apparent concern for hypothesis-testing of the academic flavor. The absence of intervention research on this population precludes assessment of the causal role of interventions.

4 Client Characteristics

Two samples are described in this report: The Primary Disabilities Sample (determined by the 473 clients on which a valid IQ test was obtained) and the Secondary Disabilities Sample (determined by the 415 clients on whom a Life History Interview (LHI) was obtained). The two samples are comparable demographically with respect to sex, ethnicity, and diagnosis. Each sample is approximately one-third FAS and two-thirds FAE. The Secondary Disabilities Sample excludes clients below age 6, but includes more clients who are over 21 years.

The two samples have 378 clients in common. Thirty-seven individuals have an LHI but no IQ exam, and 95 have an IQ exam but no LHI. As IQ data from past examinations on our unit had to be used for old clients who could not be brought in for examination, 73 of the 473 IQ scores are evaluations conducted when the clients were 3-6 years of age.

Table 4.1 presents demographic characteristics of the Secondary Disabilities Sample.

Table 4.1. Demographic Characteristics of the Secondary Disabilities (LHI) Sample (N=415).

Demographic Characteristics	N	(%)
SEX		
Male	236	(57%)
Female	179	(43%)
ETHNICITY		
White	248	(60%)
Native American	103	(25%)
Black	30	(7%)
Hispanic	27	(6%)
Asian	5	(1%)
Other	2	(<1%)
PATIENT AGE AT TIME OF INTERVIEW		
6-11 Years	162	(39%)
12-20 Years	163	(39%)
Over 21 Years	90	(22%)

The 415 clients in the Secondary Disabilities Sample ranged in age from 6 to 51 years at the time the interview was administered to their caretakers or informants. Overall, their median age was 14.2 years. The data in this report are divided into three age groups as shown below. Table 4.2 shows the breakdown of these age groups by sex and diagnosis; Table 4.3, by age at diagnosis and year of diagnosis.

Table 4.2 The Secondary Disabilities (LHI) Sample (n=415) by sex, diagnosis, and age at interview

	6-11 years		12-20 years		21-51 years		Total	
Median age	8.7 years		15.8 years		25.7 years		14.2 years	
(n)=	(162)		(163)		(90)		(415)	
Diagnosis	Male	Female	Male	Female	Male	Female	Male	Female
FAS	24	22	34	22	26	27	84	71
FAE	67	49	68	39	17	20	152	108
Total	91	71	102	61	43	47	236	179

Table 4.3. The Secondary Disabilities (LHI) Sample by age of diagnosis, year of diagnosis, and age at interview

Age at LHI:	(n)	6-11 years N=162	12-20 years N=163	21-51 years N=90
Age at Diagnosis:				
Birth to 5	(94)	65 (40%)	12 (7%)	17 (19%)
6-11	(145)	97 (60%)	44 (27%)	4 (4%)
12-20	(130)	0 (0%)	107 (66%)	23 (26%)
21 and over	(46)	0 (0%)	0 (0%)	46 (51%)
Year of Diagnosis:				
1973-1975	(18)	0 (0%)	3 (2%)	15 (17%)
1976-1980	(15)	0 (0%)	5 (3%)	10 (11%)
1981-1985	(18)	2 (1%)	5 (3%)	11 (12%)
1986-1990	(49)	15 (9%)	22 (13%)	12 (13%)
1991-1992	(62)	25 (16%)	27 (17%)	10 (11%)
1993-1995	(253)	120 (74%)	101 (62%)	32 (36%)

Table 4.4 shows the current living situation of these 415 clients by age at interview. About half of these clients lived with biologic or adoptive parents at the time the interview was conducted.

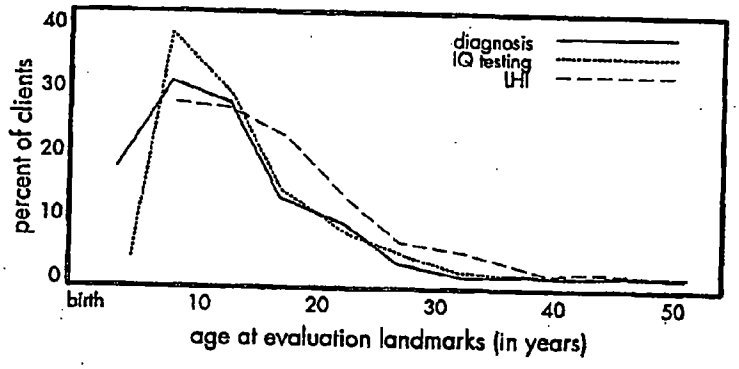
Table 4.4. Current Living Situation of 415 Clients with FAS/FAE at time of interview

Living Situation		Client age at interview: 6–11 years		12–20 years		21 years and over	
	TOTAL	FAS	FAE	FAS	FAE	FAS	FAE
Biological Parents	9	2	1	1	3	1	1
Biological Mother	51	4	13	6	19	2	7
Biological Father	34	3	14	3	10	3	1
Adoptive Relatives	10	1	4	1	4	0	0
Non-Adoptive Relatives	22	9	4	3	3	0	3
Adoptive Parents	94	12	39	16	20	5	2
Foster Parents	55	8	23	9	12	3	0
Legal Guardian	25	7	11	2	4	1	0
Friends	7	0	0	1	5	0	1
Spouse only	5	0	0	0	0	3	2
Spouse and Children	3	0	0	0	0	2	1
Partner Only	9	0	0	1	3	2	3
Partner and Children	3	0	0	0	1	2	0
Single Parent with Children	5	0	0	0	0	2	3
Live-in Work	1	0	0	0	1	0	0
Armed Forces	2	0	0	1	0	1	0
Alone	19	0	0	0	0	10	9
With or Near Attendant	1	0	0	0	0	1	0
Rent Subsidy Program	1	0	0	0	0	1	0
Assisted Living	2	0	0	0	0	2	0
Shared Living	6	0	0	0	2	3	1
Therapeutic Foster Home	8	0	2	0	5	1	0
Group Home	6	0	2	0	1	3	0
Adult Family Home	4	0	0	1	1	1	1
Residence School	1	0	0	1	0	0	0
Residence Treatment	8	0	2	3	1	1	1
Residence Corrections	6	0	0	1	4	1	0
Juvenile Corrections	4	0	0	3	1	0	0
Unstable or Homeless	8	0	0	2	4	2	0
Don't Know	6	0	1	1	3	0	1

The general level of intellectual functioning for the clients in the LHI sample is in the "borderline" range, their median IQ is 86. The group's average reading and spelling levels are consistent with their intellectual level: median WRAT-R Reading SS, 82; median Spelling SS, 80. But Arithmetic is lower (median WRAT-R Arithmetic SS, 75). The median for the Vineland Adaptive Behavior Scales (VABS) composite score is 62. The averages for the three summary subdomain scores in Communication, Daily Living Skills, and Socialization are 67, 69, and 66 respectively.

Figure 4.1 shows three relevant ages of events for the LHI sample: the ages at which the clients were diagnosed; the ages at which the IQ test used in this report was administered; and the ages of the clients when the interview was conducted with their caretakers.

4.1 Percent of clients getting diagnosed, tested and the LHI within age ranges, n=415



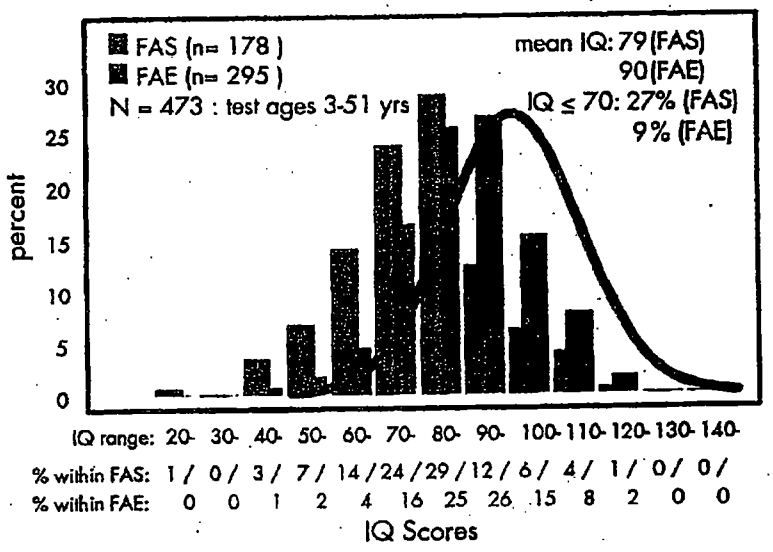
5 Primary Disabilities

General intelligence, mastery of reading, spelling, arithmetic, and general level of adaptive functioning are considered here as measures of "primary disabilities," representing the CNS manifestations of FAS, the birth defect with which these clients were born. Most of these clients have been raised in environments in which they had the opportunities to learn basic academic skills and adaptive behaviors. Rather than trying to "adjust" such data for adverse early environmental circumstances, we evaluate the effects of such circumstances in the section on risk and protective factors.

IQ data were available on 473 clients in the Primary Disabilities Sample: 178 have a diagnosis of FAS and 295 a diagnosis of FAE. Clients with FAS have a mean IQ score of 79, with a range from 29 to 120. Clients with FAE have a mean IQ of 90, with a range from 42 to 142. Twenty-seven percent of the clients with FAS and 9% of the clients with FAE have an IQ of 70 or below.

Figure 5.1 depicts the frequency distribution of the two sets of IQ scores superimposed on the normative bell curve of IQ. Both the FAS sample and the FAE sample deviate to the low side of the standard IQ distribution. The 11-point IQ discrepancy between clients with the full FAS and those with FAE replicates one we have previously reported (Streissguth et al., 1991a).

5.1 IQ distributions in the Primary Disabilities Sample: FAS and FAE



The Wechsler IQ tests contain 11 subtests, which are often interpreted clinically as a profile. We "centered" those subtest profiles by subtracting the client's mean subtest score from each of the 11 raw scores, to derive a set of 11 centered scores varying about zero as shown in Figure 5.2.

5.2 Profile of IQ subtests: FAS and FAE

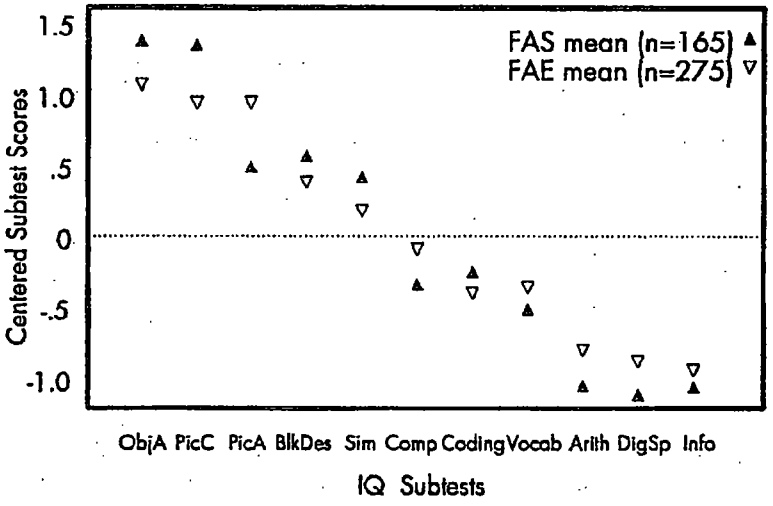


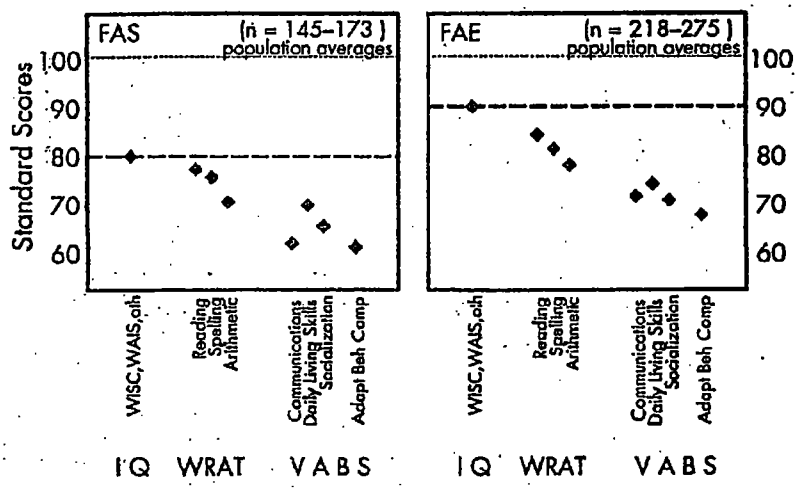
Figure 5.2 shows the group means of the "centered" subtest scores for clients with FAS versus FAE. Three subtests on which the FAS/FAE clients do poorly relative to their other subtest scores are Arithmetic, Digit Span and Information. This is a similar pattern to that seen in learning disabled children whose arithmetic, coding or vocabulary, information, and digit span are often the lowest subtests (Vargo, Grosser, & Spafford, 1995). Two subtests on which the FAS/FAE clients do best relative to their other subtest scores are Object Assembly and Picture Completion. This specific pattern of subtest strengths and weaknesses is similar to that associated with prenatal alcohol exposure in the 500 subjects in the Seattle Longitudinal Prospective Study on Alcohol and Pregnancy (Streissguth et al., 1993, Figure 7.4, page 159).

A "strength of profile" score was calculated for each client which reflects the degree to which an individual's subtest profile parallels the group subtest profile shown in Figure 5.2. This "strength of profile" score appears as item 18 on Table 6.1 of the most important Risk and Protective Factors. A high score equals high congruence, a low score, little congruence. This pattern profile covariance is computed as a weighted sum of 11 centered subtest scores. The weight for each score is the sample mean for that score. The "strength of profile" score is not correlated with IQ or age.

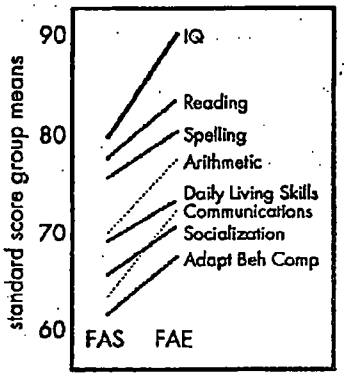
We compared academic achievement to what might be anticipated from overall IQ level. Figure 5.3 shows that clients with FAS are functioning close to their IQ level for Reading and Spelling, but are two-thirds of a standard deviation low in Arithmetic. For FAE, average Arithmetic scores are nearly a full standard deviation lower than would be expected from IQ levels; the other two achievement scores are relatively lower as well. These data replicate the findings of the previous study restricted to adolescents and adults (Streissguth et al., 1991a).

Figure 5.3 shows that both groups of clients have low adaptive behavior scores relative to their respective IQ levels. The relative deficit in these scores, in fact, is considerably greater than shortfalls in achievement, regardless of diagnosis. Figure 5.4 clarifies all these comparisons in a different graphic style.

5.3 IQ, WRAT, VABS: FAS and FAE



5.4 Relationship of achievement and adaptive behavior to IQ, by diagnosis



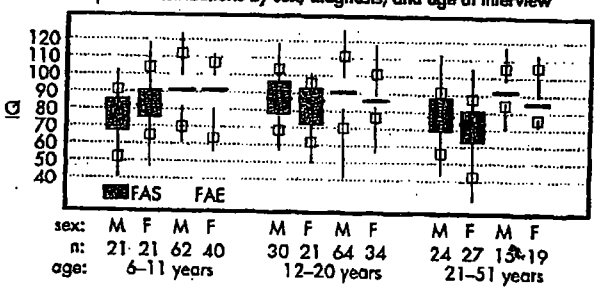
In Figure 5.4, each mean score for the FAS group has been connected by line to the corresponding mean score for the FAE group. The darkest line (at the top) is IQ, which is higher for both FAS and FAE clients than are the Achievement and Adaptive Behavior scores for the same group of clients.

The lines for Arithmetic and Communications are approximately parallel to the IQ line. This indicates that although Arithmetic and Communications are lower than IQ, the discrepancy is about the same for FAS clients as it is for clients with FAE.

The five darker lines (Reading, Spelling, Daily Living Skills, Socialization, and the Adaptive Behavior Composite) are relatively flatter. This indicates that although all 5 mean scores are lower than IQ within their respective samples, the discrepancy appears to be greater for FAE clients than for FAS clients.

Figure 5.5 presents data from only the Secondary Disabilities (LHI) Sample on which IQ was available (n= 378). This figure shows that the IQ scores for the clients at each LHI age grouping were comparable for subgroups defined by sex and diagnosis. The distribution of IQ scores is presented with a boxplot for each of the 12 groups: age (3) by sex (2) by diagnosis (2).

5.5 LHI Sample: IQ distributions by sex, diagnosis, and age at interview



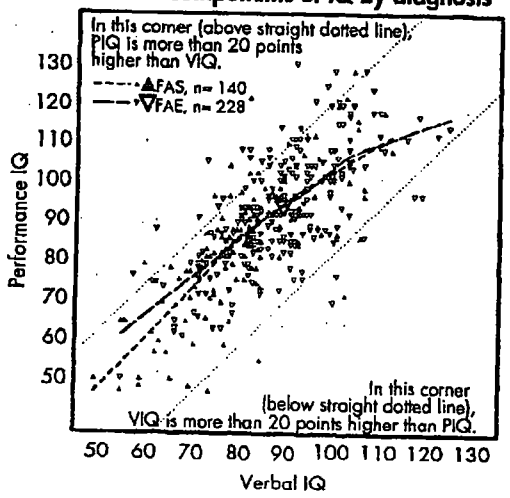
The box in a boxplot spans the middle half of the data from 25th to 75th percentiles; the dark horizontal line within a box indicates the median IQ score; the whiskers extending vertically from each box reach to the lowest and highest IQ; the open boxes on lower and upper whiskers indicate the 10th and 90th IQ percentiles respectively.

Figure 5.6 describes the discrepancies between PIQ (Performance IQ) scores and VIQ (Verbal IQ) scores, and how these discrepancies for the FAS/FAE clients differ from the normal population. Notice that more individuals fall outside the dotted line at the top of the figure compared to the number outside of the lower dotted line. This indicates that more FAS/FAE individuals have a PIQ greater than VIQ by 20 or more points, compared to the converse. Actually, 8% of the clients fall above the line compared to 2.4% below the line. According to Sattler (1988), only 5% of a population would be expected to have a PIQ greater than VIQ by this magnitude.

Notice also the two heavy dashed curves illustrating that for individuals with IQ's up to about 100, clients were more likely to deviate in the direction of PIQ greater than VIQ rather than the converse.

The large black triangle represents the mean scores of all individuals with FAS. Here we see that the mean PIQ is 84 while the mean VIQ is 79, for an average discrepancy of 5 points. The large white triangle represents the mean scores of all individuals with FAE. There is also a 5 point discrepancy between the mean PIQ of 93 and the mean VIQ of 88.

5.6 The two components of IQ by diagnosis



6 Risk and Protective Factors: Overview & Definitions

Our main goal in this research project was to study the prevalence in this sample of the deleterious life outcomes we have called Secondary Disabilities. These are examined in Chapters 7 through 17. Of course, risks of these outcomes are not the same for all clients. In this Chapter, we describe our rationale and methodology for quantifying these risks. Table 6.1 contains our final list of 21 Risk and Protective Factors and their definitions; Figure 6.1 depicts the relationship of these 21 Risk and Protective Factors to our six main Secondary Disabilities, and Figure 6.2 displays the 8 by 8 matrix of all eight Secondary Disabilities against each other. Of course, we cannot maintain that these relationships are causal or independent.

Definition of the Risk and Protective Factors

We considered two types of Risk and Protective Factors: Intrinsic and Extrinsic. *Intrinsic* Factors include attributes of the client that directly characterize his/her biology or extent of putative brain damage and include age, sex, diagnosis, IQ, IQ subtest profile, and ABC score. *Extrinsic* Factors represent environmental influences that have the potential of affecting life outcomes; these can be gleaned from the Life History Interview (LHI). Extrinsic Risk and Protective Factors were subjectively evolved from clinical experience, for example: fraction of life in a stable and nurturant home, living in non-alcoholic families, not being abused or neglected, receiving special help as needed. Some of the Risk and Protective Factors were available from the test and demographic data in our database, the rest were developed into questions on the LHI, dispersed appropriately within the various sections of the interview. Variables like "fraction of life" came from questions like: "During what ages did client live in a stable and nurturant household?" The quality of "nurturance" is subjective, but the fraction of life is intended as an explicit quantification. Typically the informant would think back over the history of living situations and list for the interviewer the inclusive periods during which the situation was judged "nurturant"; hence, the dates are probably fairly accurate. (Not all Risk and Protective Factors fit under intrinsic or extrinsic, e.g. the FABS is neither.)

After administering 415 LHI's, we developed a scoring system that included two types of scores. Some were median splits, in which the top half of the distribution was compared with the lower half on a given item, such as "fraction of life lived with alcohol and drug abusers"; others were simple thresholds—binary scores that applied only to a proportion of the subjects, such as "violence against client." For some scores like this, with broader coverage, separate components (domestic violence, physical abuse, and sexual abuse) were also examined individually as risk factors. A likely group of 33 such candidates was examined in relation to the Secondary Disabilities discussed in Chapter 7.

After examination of the data, we retained the set of 21 Risk and Protective Factors depicted in Table 6.1, which presents the definition of each, the type of score obtained, distributions for all dichotomies, and the source of the information from which the data derive. For items deriving from the LHI, the specific item numbers are documented in Table 6.1.

Table 6.1. Definitions and derivations of risk/protective factors as reported by the caretaker, physician and psychometrist with distribution for clients 12 years and over.

Definition	Type	*Derivation of binary protective/ risk factors**	%***	Source of info
1. Stable and nurturant household	E	Median split on fraction of life: P: 72 – 100% of life R: 0 – 71% of life	 49% 51%	LHI
2. Age of diagnosis of FAS/FAE	E	Age at diagnosis split at 6 years: P: under 6 years R: 6 years and over	 11% 89%	Dysmorphology Exam
3. Violence against client	E	Yes/no if ever in life patient experienced domestic violence, sexual abuse, and/or other physical abuse: P: no violence experienced R: violence was experienced	 28% 72%	LHI
4. Average number of years per living situation till age 18	E	Median split on number of years/HH: P: 2.8 – 18.0 yrs per household R: <2.8 yrs per household	 62% 38%	LHI
5. Quality of home environment during during later childhood (8–12 years)	E	Median split on a count of positive qualities of home: P: 10 – 12 good qualities R: 0 – 9 good qualities	 65% 35%	LHI
6. DDD eligibility status	E	Indicator of whether the family both applied for and was eligible for DDD: P: applied for and was eligible R: applied for, but was not eligible	 56% 44%	LHI
7. Living with alcohol/drug abusers	E	Median split on fraction of life the proband has lived with alcohol/drug abusers: P: 0 – 29% of life R: 30 – 100% of life	 51% 49%	LHI
8. Alcohol-related diagnosis	I	Indicator of FAS rather than FAE: P: FAS or PFAS R: FAE, PFAE, ARND	 43% 57%	General Information Form
9. Basic needs are not met	E	Median split on fraction of life that basic needs are not met: P: 0 – 12% of life R: 13 – 100% of life	 49% 51%	LHI
10. Fetal Alcohol Behavior Scale (FABS)		Median split on number of fetal alcohol behaviors according to caretaker: P: 0 – 20 of 36 behaviors endorsed R: 21 – 36 of 36 behaviors endorsed	 44% 56%	Personal Behavior Checklist
11. Intelligence Quotient (IQ)	I	Full Scale IQ P: IQ ≤ 70 R: IQ > 70	 16% 84%	FADU testing (occasionally, from school report)

Definition	Type	"Derivation of binary protective/ risk factors""	%"""	Source of info (LHI items)
Table Continues				
12. Driver's license attainment (≥16 years of age only)	E	Whether or not patient ≥16 years has has a driver's license:		LHI
		P: yes	31%	
		R: no	69%	
13. Sex	I	Sex:		General
		P: female	43%	Information
		R: male	57%	Form
14. VABS ABC Standard Score	I	Median split on the Adaptive Behavior Composite Standard Score		VABS Interview
		P: VABS ABCss ≥ 65	38%	(FADU)
		R: VABS ABCss < 65	62%	
15. Difference between IQ and ABCs	I	Comparison of IQ & VABS ABCss		Testing &
		P: IQ & ABCss are within 15 pts	32%	interviewing
		R: more than 15 pts different	68%	(FADU)
16. Has there always been at least one caretaker after age 18 years	E	Yes/No		LHI
		P: never after age 18 years	64%	
		R: sometimes—always	36%	
17. Difference between performance and verbal IQ	I	Performance IQ (PIQ) minus verbal IQ (VIQ)		FADU testing
		P: PIQ more than 15 points higher	17%	
		R: PIQ not more than 15 points higher	83%	
18. Similarity of IQ subtest profile to other FAS/FAE patients	I	Median split on the IQ subtest strength of profile score		FADU testing
		P: weak IQ profile (not similar)	49%	
		R: strong IQ profile (similar)	51%	
19. SSI eligibility status	E	Indicator of whether the patient both applied for and was eligible for SSI		LHI
		P: applied for, but was ineligible	28%	
		R: applied for, and was eligible	72%	
20. Age of Secondary Disability assessment	E	Status as adult (≥21 years)		from dates
		P: under 21 years old	64%	
		R: 21 years and older	36%	
21. Always 2 caretakers available before 18 yrs	E	yes/no		LHI
		P: not always 2 caretakers	57%	
		R: always 2 caretakers available	43%	

* E = Extrinsic; I = Intrinsic

"Item polarities are assigned according to the median plotted in Figure 6.1., which pertains *only* to the first six Secondary Disabilities. For Risk and Protective Factors for the last two Secondary Disabilities (Dependent Living and Problems with Employment) see Figures 14.2 and 15.2, respectively.

P=protective end; R=risk end

"""Percents apply only to clients 12 years and over.

Odds Ratios

Through most of this report, we will express effects of Risk and Protective Factors in terms of *odds ratios*. (Some readers may recognize this approach as a series of univariate logistic regressions.) An example will make the computation clear. Suppose we are interested in the role of low IQ (less than or equal to 70) as a predictor of some binary outcome, in this case, "Trouble with the Law," one of the summary Secondary Disability scores introduced in the next chapter. The subsample to which this outcome score seems most pertinent is the set of clients 12 and older. For this subsample, the 2 x 2 table is as follows:

	Trouble	
	no	yes
>70	72	122
IQ		
≤70	20	18

The odds of no Trouble for IQ>70 are 72:122. The odds for IQ≤70 are 20:18. The *odds ratio* for the effect of the IQ dichotomy on this outcome is thus (20/18)/(72/122), or 1.88, about a 2:1 *protective* effect. The clients of lower IQ have *relatively fewer* problems with the law; that is, they do better. For a different cut of "low IQ," at 85, the table is:

41	88
51	52

The odds ratio (51/52)/(41/88), or 2.10, stands for just about the same "protective effect." Ratios less than 1 indicate "risk" rather than protection. The method of multiple logistic regression would attempt to "adjust" all such odds ratios for associations among the separate factors. We have no reason to believe that any of the assumptions of these analyses are met, and so we do not pursue this refinement here.

For each Secondary Disability in the subsequent chapters, each risk or protective factor is associated with an odds ratio of this sort. For IQ ≤ 70, for instance, the ratios are: for Mental Health Problems, (2/36)/(9/187) or 1.15 (clearly a problem of small cell counts here); for Disrupted School Experience, (19/17)/(73/122), or 1.87; for Confinement, (22/16)/(95/100), or 1.45; for Inappropriate Sexual Behavior, (17/21)/(99/92), or 0.75 (a slight escalation of risk); for Alcohol/Drug Problems, (28/9)/(123/73), or 1.85.

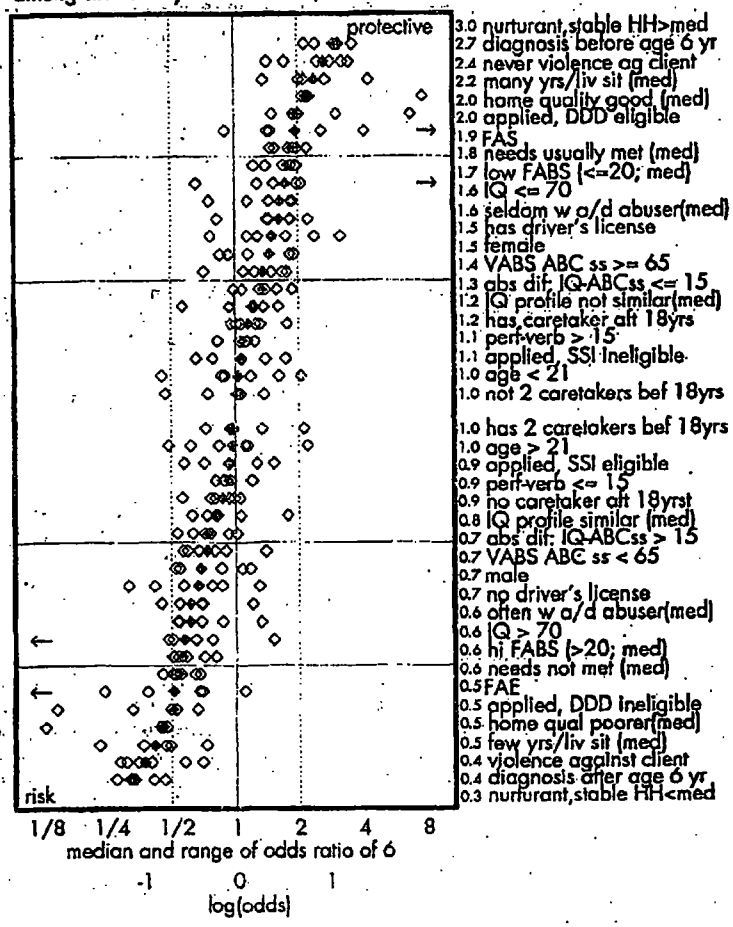
As a sorted list, these odds ratios are (.75, 1.15, 1.45, 1.85, 1.87, 1.88) with median (1.45 + 1.85)/2 = 1.65. Thus, "IQ ≤ 70" is plotted at height 1.65 in Figure 6.1 with the separate odds ratios around it. Each later chapter begins with a graph of similar information as a "profile" across all the risk or protective factors as they pertain to that outcome, one at a time.

The entire set of odds ratios for these 21 Risk and Protective Factors is presented in Figure 6.1. The horizontal scale in this figure is a log scale, so that reciprocals (e.g., 4 and 1/4) are at equal distances from the centerline in opposite directions. These mirrored points represent equivalent odds ratios of either risk (left) or protection (right). The "Protective" Factors, which lie in the top half of the page are the converse of the "Risk" Factors, which lie in the bottom half of the page. Each row on the figure represents a single Risk/Protective Factor. The one at the top of the page, "percent of life in a stable and nurturant household," is the strongest Protective Factor, on average, against the first six Secondary Disabilities described in Chapter 7. The median impact of this one Protective Factor on the six Secondary Disabilities is represented by a black diamond. Risk/Protective Factors that have the least impact (odds ratios closest to 1.0, whether risky or protective) on these six Secondary Disabilities appear at the midpoint of the page, at the end of the list of 21. Arrows indicate scores that could not be calculated for one of the diamonds because there was a zero in either the numerator or the denominator of the odds ratio. The vertical column of decimal numbers to the left of the Risk/Protective Factor names on Figure 6.1 is the median of the six odds ratios observed for that Factor.

The white diamonds on each row represent the relationships of that Risk/Protective Factor to each of the six Secondary Disabilities studied in Chapters 8 through 13. Each of these chapters has a graph with one set of diamonds, reflecting how the 21 Risk/Protective Factors are related to the Secondary Disability discussed in that chapter. For simplification, these graphs only depict the Protective side; one can imagine the Risk side to be the reflection. These separate graphs appear in each chapter as its second figure. Each white diamond found on a graph can be found at exactly the same spot in Figure 6.1. The ordering of Risk/Protective Factors have been retained across chapters. The Risk and Protective Factors are ordered by median odds ratio seen in Figure 6.1, from most protective at the top through most risky at the bottom.

In Figure 6.1, two types of Risk and Protective Factors can be detected. Recall that the vertical line at the center differentiates the Risk side of the distribution (at the left) from the Protective side (right). Any Factor for which all the open diamonds are to the same side of center line is a "*Universal*" Factor. The Universal Protective Factors are those that are consistently protective for each of the six Secondary Disabilities. Except as noted in Table 6.1, the sample for each Risk and Protective Factor was divided at the median, which is the number appearing below. These Universal Protective Factors include: living in a nurturant and stable home at least 72% of life; being diagnosed before the age of 6 years; never having experienced violence; getting to stay longer than an average of 2.8 years in each household; experiencing a good quality of home (10 of 12 "good" qualities); being FAS (rather than FAE); and having basic needs met at least 13% of life. Of course, many of these items overlap substantially among themselves; we are making no claim that any of these Risk or Protective Factors are independent of others. Other Risk/Protective Factors are called "Specific" because they are Protective Factors for some Secondary Disabilities yet may be Risk Factors for others.

6.1 Risk/Protective Factors across 6 Secondary Disabilities among clients 12 years and older, max n = 253

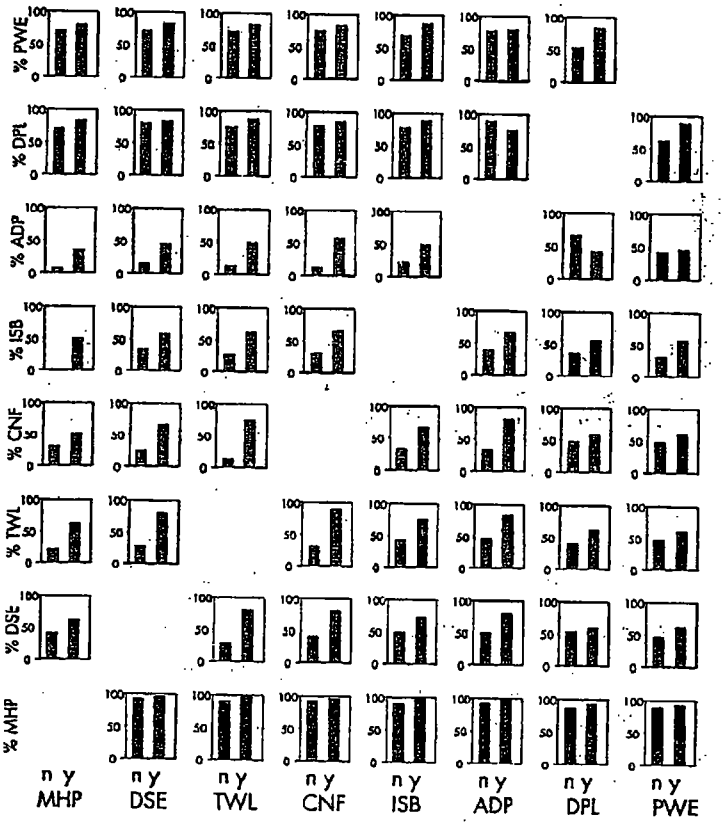


Odds ratios larger than 2 (or less than 0.5) indicate interesting protective effects (or risks). An odds ratio of about 2, such as seen for DDD eligibility, indicates that the chance of avoiding a Secondary Disability is about twice as high for a child who receives DDD services as for one who does not.

Two Secondary Disabilities pertaining only to adults are not included in Figure 6.1 due to the necessarily smaller sample size: Dependent Living and Problems with Employment. These could be assessed realistically only for clients who were 21 years of age and older. However, they were examined according to the same Risk and Protective Factors described in Table 6.1. These latter two Secondary Disabilities are described in Chapter 14 (Dependent Living) and Chapter 15 (Problems with Employment). The odds ratio graphs for these latter two Secondary Disabilities appear as the second graph in their respective chapter.

We also consider each of the eight Secondary Disabilities to be Risk and Protective Factors for any of the others. Figure 6.2 shows an 8 x 8 matrix of these binary 2 x 2 comparisons in graphical form. The only pair of Secondary Disabilities that are negatively associated—for which the prevalence of either is lower for the group coded "yes" for the other—is Alcohol and Drug Problems with Dependent Living.

6.2 Prevalences of each secondary disability controlling for the others one by one

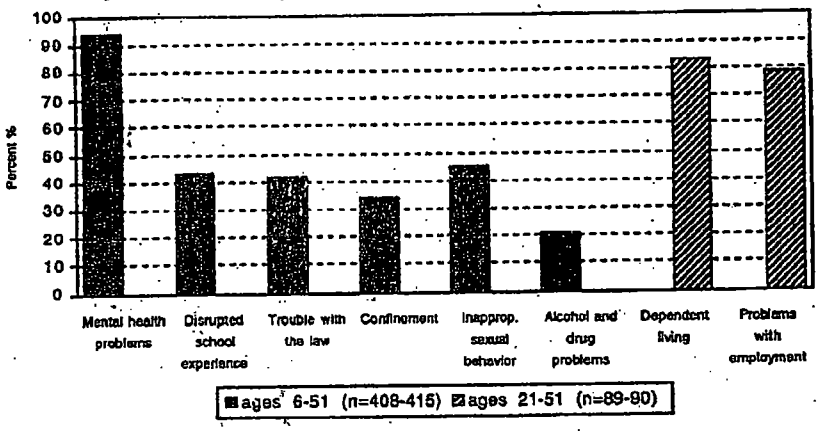


7 Secondary Disabilities: Definitions & Overview

Secondary disabilities are those that a client is not born with, and that could presumably be ameliorated (either fully or partially) through better understanding and appropriate interventions. The project examined altogether eight Secondary Disabilities. The first six pertain to clients of all ages and are easily quantifiable. The latter two, Dependent Living (DPL) and Problems with Employment (PWE), pertain only to adult clients (21 and over), restricting the sample size to 90 compared to 415 for the other Secondary Disabilities. DPL and PWE are by nature more subjective and required more complex and qualitative definitions. In the graphs that follow in this chapter, the two latter Secondary Disabilities are set apart from the others to draw attention to this difference. All data on Secondary Disabilities derive from the Life History Interview (LHI). This means that the accuracy of the information is dependent on the knowledge of the informant, which varied depending on the age and living circumstances of the client.

Figure 7.1 shows the prevalence of all eight Secondary Disabilities across the entire sample of 408-415 clients. The remaining figures in this chapter focus primarily on clients 12 years of age and older, the age at which most of these Secondary Disabilities were manifest.

7.1 Prevalence of Secondary Disabilities across the Life Span



Secondary Disabilities Definitions

Mental Health Problems (MHP) was coded "yes" if the client was described as (a) ever having any of a list of mental health problems, or (b) gone to a psychiatrist, psychotherapist, or counselor for mental health problems, or (c) ever having been a client in a psychiatric or a mental health hospital. (Chapter 8)

Disrupted School Experience (DSE) was coded "yes" if the client was described as ever being suspended or expelled from school, or as having dropped out of school. (Chapter 9)

Trouble With the Law (TWL) was coded "yes" if the client was described as having been charged or convicted or in trouble with authorities for any one or more of these main categories of criminal behavior: crimes against persons (excluding sex-related); crimes against property; possession or selling of illegal substances; sex crimes; driving violations; parole violations, skipping bail, or escape; or other crimes (primarily victimless, such as running away from home). (Chapter 10)

Confinement (CNF) was coded "yes" if the client was described as ever having been incarcerated for a crime or having received inpatient treatment for mental health problems or for alcohol or drug abuse problems. (Chapter 11)

Inappropriate Sexual Behavior (ISB) was coded "yes" if the client had ever been sentenced to sexual offenders treatment or had ever been reported as "repeatedly" having one or more problems with sexuality. (Chapter 12)

Alcohol and/or Drug Problems (ADP) was coded "yes" if the client was reported to have had alcohol abuse problems, drug abuse problems, and/or alcohol/drug treatment, whether inpatient or outpatient. (Chapter 13)

Dependent Living (DPL) was coded "yes" for clients *21 years old and over* and *not* independent. Independence requires *either:*

(a) —that the client can handle 12 daily living activities without help
or
(b) —that the client does not have another person organizing his or her life,

—does not live with a caregiver,

—needs help with at most 3 of the 12 critical daily activities, and

—can pay at least some of his or her own expenses. (Chapter 14)

Problems With Employment (PWE) was coded "yes" if a client is *21 years old and over* and *not* effectively employed. Effectively employed is defined as meeting *any* one of the following three criteria:

1. Having no other cash support except his/her own wages

2. Weekly earnings of $280 or more

3. Being currently employed 20 hours or more per week, *and*

—Having none or only mild job problems (that is, no repeated problems and a maximum of two "minimal problems;" minimal problem is a score of "1" on LHI questions that follow: trouble getting hired, trouble holding a job, being fired from a job, and losing a job without understanding why),
and
—Having had three or fewer jobs in the past two years. (Chapter 15)

Secondary Disabilities Overview

Figure 7.2 shows prevalences of the six major Secondary Disabilities by three age groups. Prevalence in adults does not exceed prevalence in adolescents except for Confinement and Alcohol and Drug Abuse.

7.2 History of Secondary Disabilities by three age groups at interview

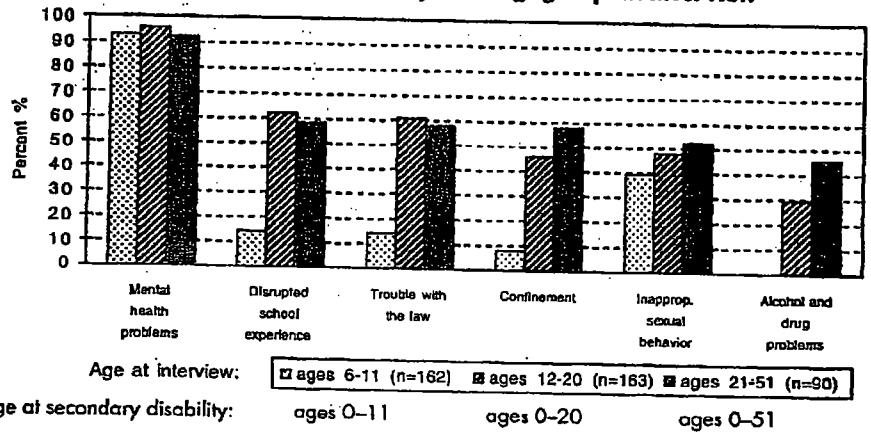
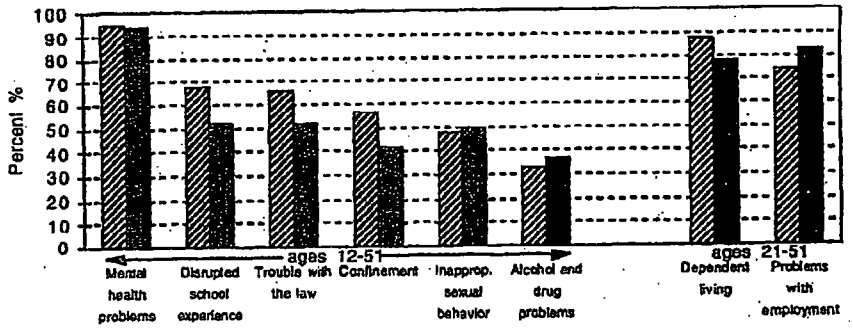


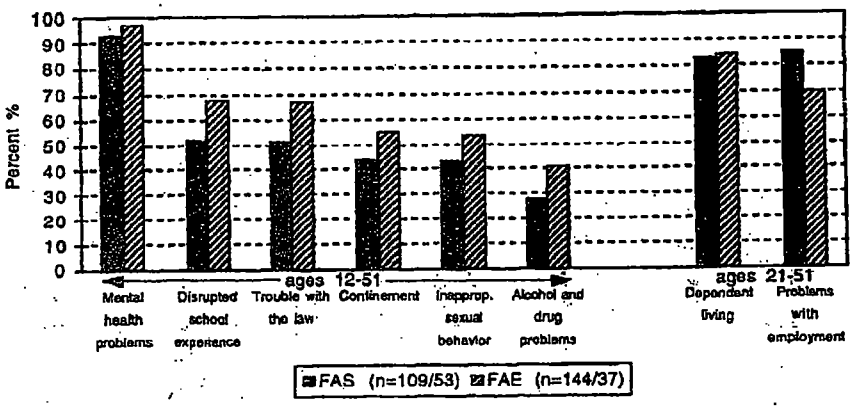
Figure 7.3 through 7.5 consider only those 253 clients 12 years and over. Figure 7.3 compares the prevalence of Secondary Disabilities for males and females. Males have higher rates of Disrupted School Experience, Trouble With the Law, and Confinement.

7.3 History of Secondary Disabilities among clients ≥ 12 years old by sex



There are mild but systematic differences between clients with FAS and those with FAE in terms of the prevalence of Secondary Disabilities (see Figure 7.4). In all matters except independence and employment, clients with FAE (although often thought to be more "mildly affected") have higher rates of the Secondary Disabilities.

7.4 History of Secondary Disabilities among clients ≥ 12 years old by diagnosis



32

Low IQ seems to have a similar pattern of "protective" effect as a diagnosis of FAS (with which it is formally tied through the CNS criterion of FAS). For the 38 clients technically classifiable as mentally retarded, there are lower rates of Alcohol and Drug Problems, Disrupted School Experience, Trouble With the Law, and Confinement. All of them are in dependent living situations, and 94% have employment problems (Figure 7.5). An IQ above 85 (average for this full sample) is clearly not "protective" and may actually be associated with additional legal troubles and additional confinements.

7.5 History of Secondary Disabilities among clients ≥ 12 years old by IQ level

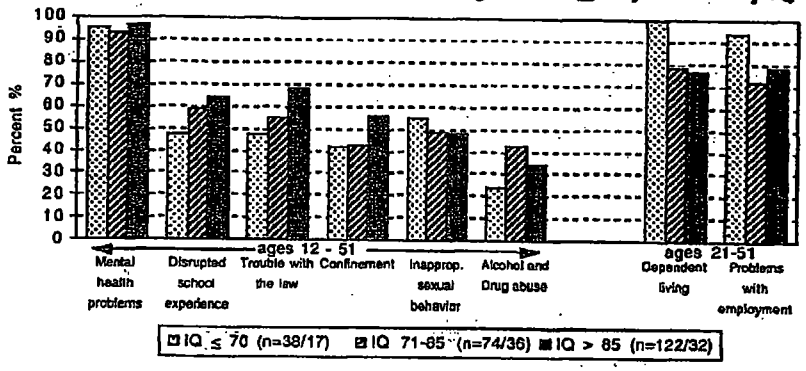
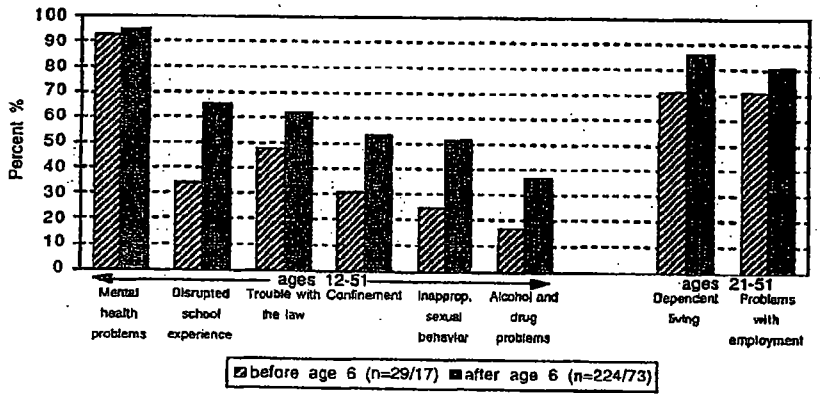


Figure 7.6 shows that a diagnosis before age 6 is related to lower prevalence of all secondary disabilities except perhaps Mental Health Problems.

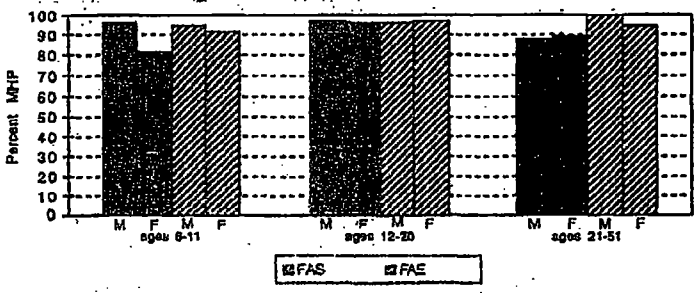
7.6 History of Secondary Disabilities among clients ≥ 12 years old by age at diagnosis



8 Mental Health Problems (MHP)

Mental Health Problems (MHP) is a binary summary of reported mental health concerns, problems, or treatment for clients of any age. It is coded 1 for those clients who: had one or more of a long list of possible mental health problems, had ever gone to a psychotherapist or a counselor for a mental health problem, or had ever been a client in a psychiatric or mental hospital. At 94% of the full Secondary Disabilities sample (Figure 8.1), Mental Health Problems is by far the most prevalent Secondary Disability, accounting for twice as many clients as Disrupted School Experience or Trouble with the Law, the next most frequent problems. Because of the overwhelming prevalence of Mental Health Problems, hardly any clients were, in fact, "protected."

8.1 History of Mental Health Problems (MHP) by sex, diagnosis and age at interview (n=414)



For those who were protected, Figure 8.2 shows the strongest protective factors against MHP: (1) DDD services eligibility, (2) below median FABS score, (3) higher than the median number of years per living situation, (4) living in a home of above median quality (during childhood ages 8-12), and (5) living in a stable and nurturant household above median proportion of life (72-100%). Because the base rate of Mental Health Problems in this sample is so close to 100%, these odds ratios are more subject to sampling error than the others in Figure 6.1.

8.2 Risk/Protective Factors for History of Mental Health Problems among clients 12 years and older at interview, max n = 253

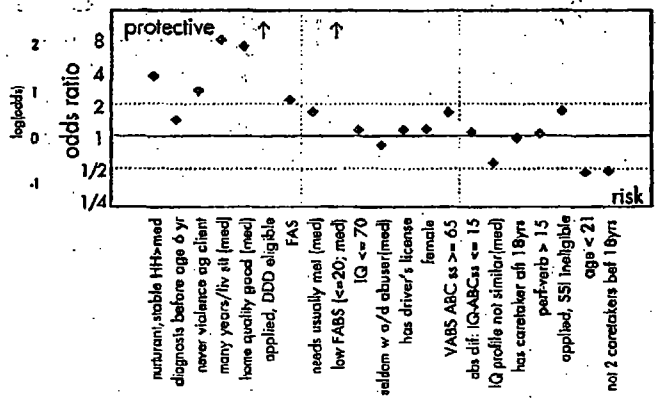


Figure 8.3 shows that most clients of any age with Mental Health Problems receive treatment for these problems.

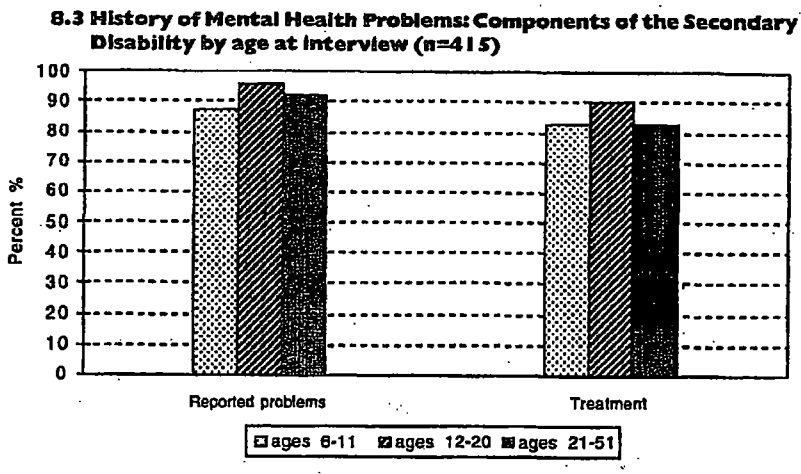


Figure 8.4 shows the rate of Mental Health Problems by age. The most frequent mental health problems for children and adolescents are attention deficit problems (61%), followed by depression and suicide threats. Over half the adults were reported to have depression problems, 43% have made suicide threats, and 23% have made suicide attempts; 29% of adults have psychotic symptoms.

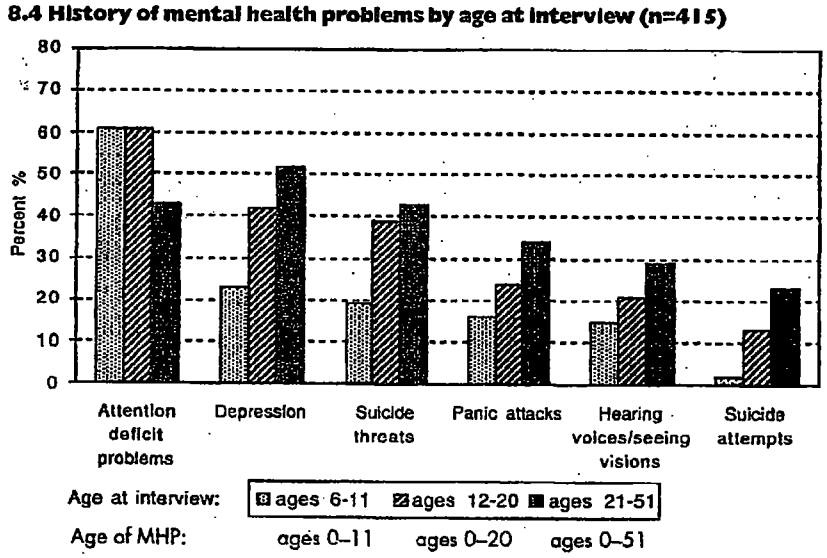
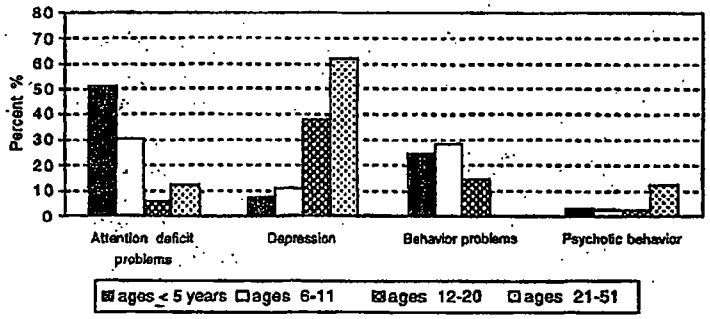


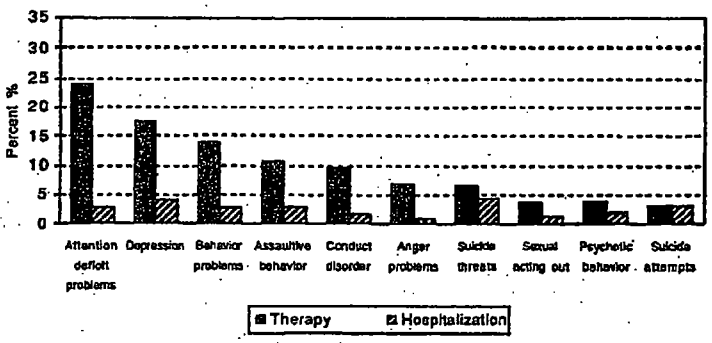
Figure 8.5 shows that among clients who were reported to have a mental health problem, the first sign of mental health problems with onset at age five or under, was attention deficit problems (51%), followed by behavior problems (25%) including conduct problems (6%) and assaultive behaviors (7%). By contrast, if the onset of mental health problems was after age 21, the most commonly reported Mental Health Problem was depression (62%), or psychotic behavior (12%).

8.5 First sign of a mental health problem by age of onset among clients with MHP (n=310)

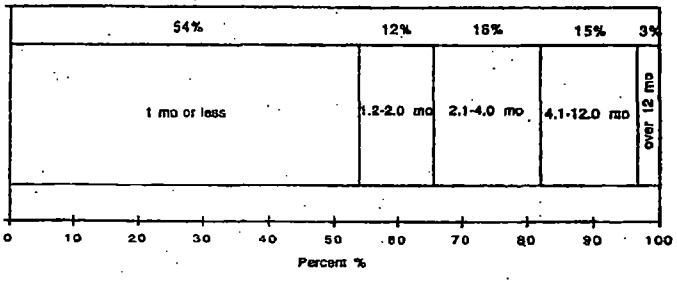


The most frequent reason reported for obtaining outpatient mental health treatment is attention problems, followed by depression. For inpatient treatment, the primary reasons are suicide threats and depression (Figure 8.6). One in three of all hospitalized clients have been hospitalized for over two months, including 3% for over one year (Figure 8.7).

8.6 Mental Health Problems: Prevalence of therapy and hospitalization by reason for treatment



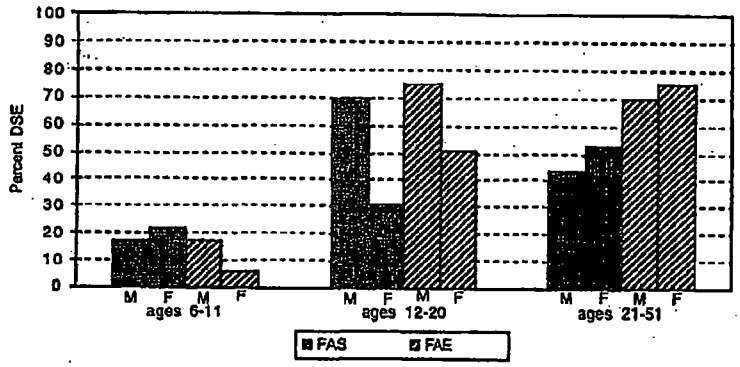
8.7 Length of stay in a mental health hospital (n=67)



9 Disrupted School Experience (DSE)

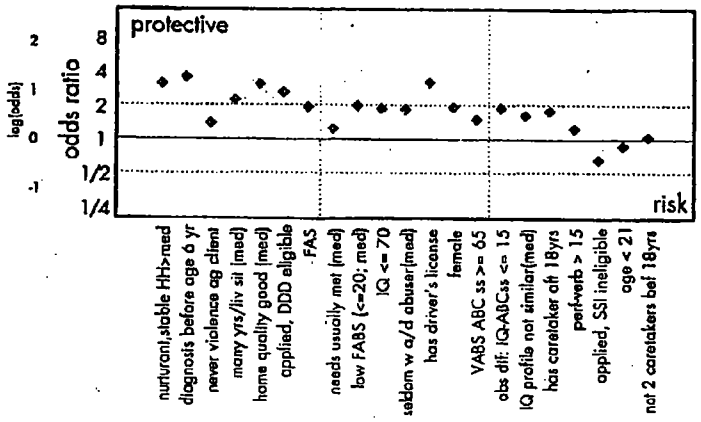
Disrupted School Experience (DSE) is a binary summary of reported disruptions of schooling for clients of any age. It is set to 1 for those clients who ever were suspended from school, expelled from school, or dropped out of school. Figure 9.1 shows that 60% of clients 12 years and older have had Disrupted School Experience.

9.1 History of Disrupted School Experience (DSE) by sex, diagnosis and age at interview (n=411)



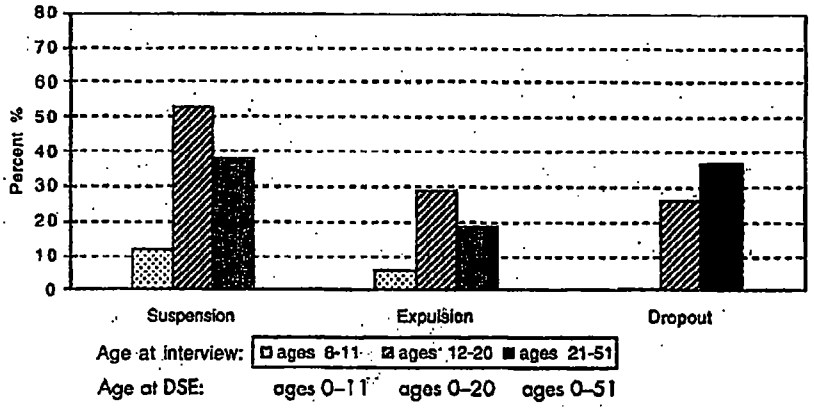
The important protective factors against DSE (items in Figure 9.2 that lower the odds of DSE) include: an early diagnosis, longer duration of living situations, longer duration in a stable and nurturant home, living in a good quality home, and having a driver's license. In addition, having applied for DDD (Division of Developmental Disabilities) services and found to be eligible was a specific protective factor against DSE.

9.2 Risk/Protective Factors for History of Disrupted School Experience among clients 12 years and older at interview, max n = 250



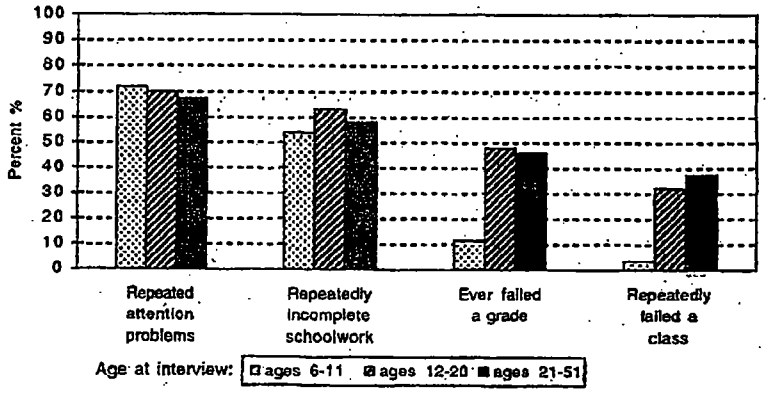
The rate of DSE is higher after childhood when one in two adolescents have been suspended, one in four expelled, and more than one in four have dropped out of school (Figure 9.3).

9.3 Disrupted School Experience: Components of the Secondary Disability by age at interview (n=411)

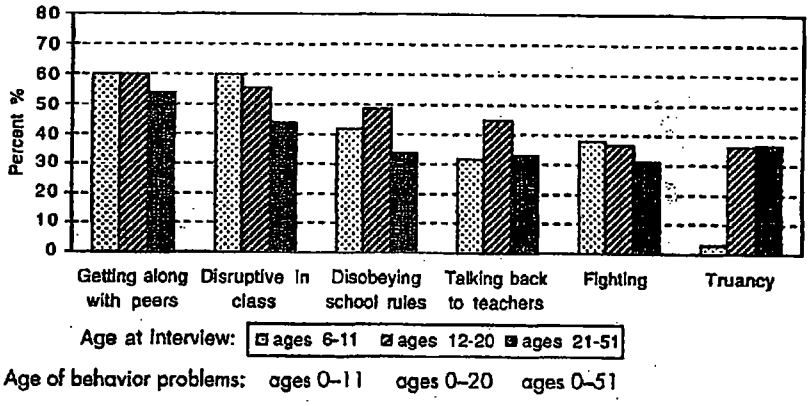


The most frequent learning problems (Figure 9.4) are attention problems (70%) and repeatedly incomplete school work (55-60%). Nearly half the adolescents and adults had failed a grade in school. The most frequent behavior problems ever experienced (Figure 9.5) were repeated problems in getting along with peers (≈60%) and repeatedly being disruptive in class (55-60%). Clients with DSE had about twice as many of these problems as compared to clients without DSE (Figure 9.6).

9.4 Learning problems by age at interview (n=395-407)

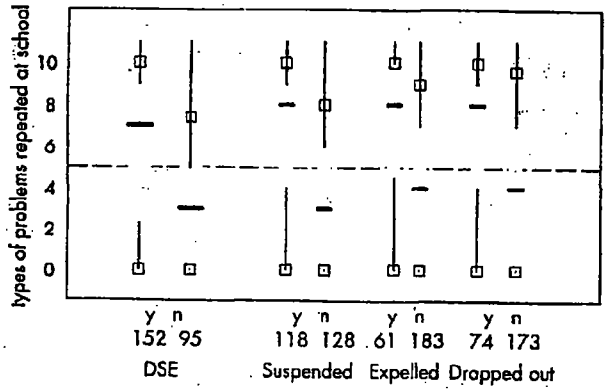


9.5 Repeated behavior problems by age at interview (n=403-408)



Figures 9.6 through 9.8 show that DSE is correlated with learning problems and behavior problems. Although rates of repeated poor attention are high whether or not students also experience DSE, rates of failing grades and repeatedly failing classes are much higher among those who also had DSE. Being suspended, expelled from school or dropping out is an unfortunate concomitant for students with problems doing the school work and/or have behaviors that are disruptive or aggressive. School truancy is a frequent behavior problem (48%). Clearly children who are not at school for any reason are unable to benefit from the structure and support that the school offers for the time they are away from school.

9.6 Distributions of number of problems observed repeatedly in school among patients 12 years and older



9.7 Learning problems by presence or absence of DSE (n= 235-244)

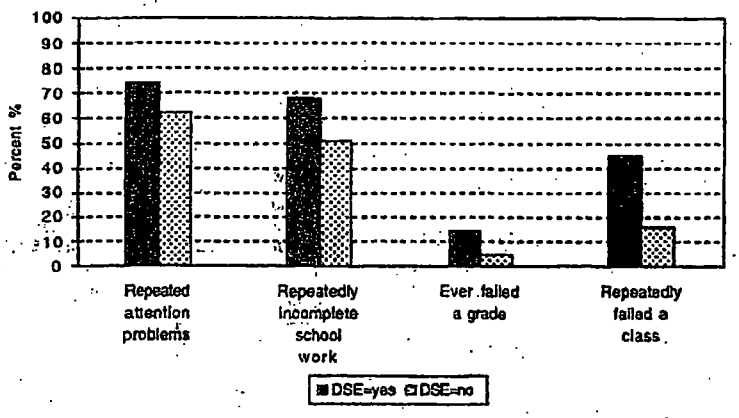


Figure 9.8 shows that all repeated behavior problems display higher rates among clients with DSE.

9.8 Repeated behavior problems by presence or absence of DSE (n= 240-245)

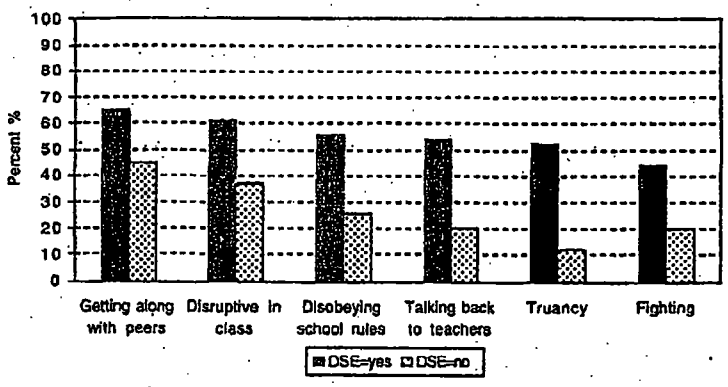
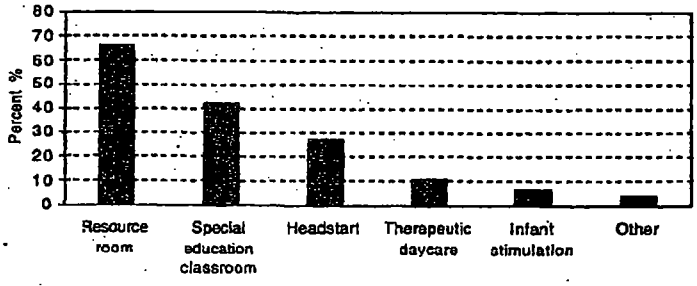


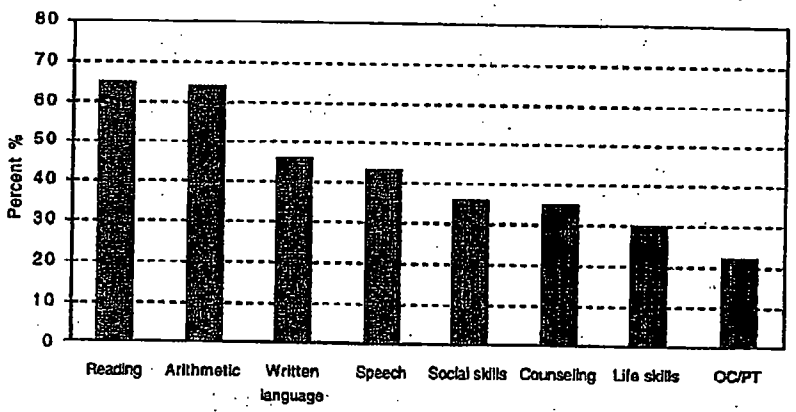
Figure 9.9 shows that 40% of the clients with FAS/FAE were known to have been in special education.

9.9 Types of early intervention programs attended (N=369-404)



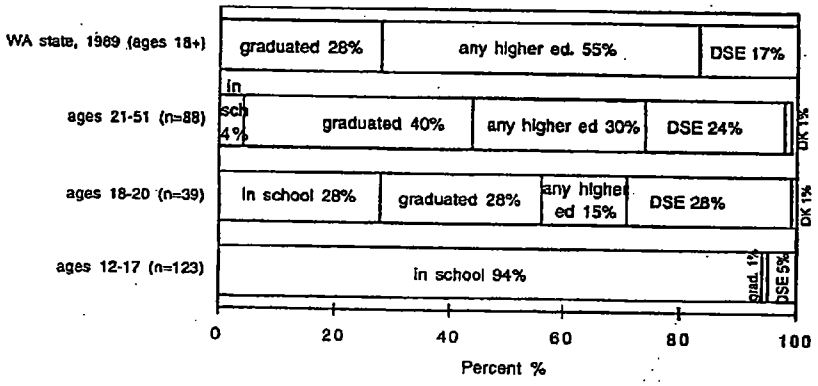
Where schools put emphasis on remediation is of interest, given that over 80% of clients over age 21 are in dependent living situations. It may be more effective for schools to focus more on practical daily living skills training. We found that 65% of the clients with FAS/FAE had received remedial help in Reading and Arithmetic, but fewer than one in three had received life skills training, and only one in five had received occupational or physical therapy (Figure 9.10).

9.10 Types of remedial interventions received at school (N=393-397)



Compared to Washington State statistics (Figure 9.11), clients with FAS/FAE graduate less frequently from high school than other students (counting all types of graduation including special education, GED, etc.). Even after age 21 years, only 30% have gone to any type of post-secondary school education. The category membership of a person in the LHI sample is determined hierarchically from left to right for Figure 9.11. Thus, a 16 year old who is both in school and has been suspended twice is coded as being among the 94% who are "in school." The 5% of 12-17 year olds graphed as DSE are those who are not still in school.

9.11 Educational status in full study sample and in WA state



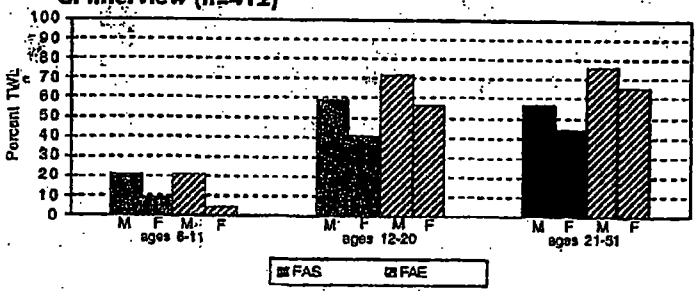
Notes:
"in school" means current attending *and* does not yet have a high school diploma. This includes a few clients who may be attending community college GED programs.
"graduated" means the client does have a high school diploma (any kind), but does not have any higher education experience.
"higher education" means the client *does* have a high school diploma and has received (or is receiving) some higher education.
"DSE" refers to clients who are not in school and never graduated, and are DSE positive.
To obtain fraction of clients who have a high school diploma (any type), add together "graduated" plus "higher education."

10 Trouble With the Law (TWL)

Trouble with the Law (TWL) is a binary summary of reported interactions with police, authorities, or the judicial system for clients of any age. It is set to 1 for those clients who ever were in trouble with the police or authorities, or were ever arrested for, charged with, or convicted of any of the seven types of criminal behavior delineated in Figure 10.3.

TWL is reported for 14% of 6-11 year olds, and higher rates in adolescence and adulthood (61% and 58%, respectively). Figure 10.1 shows the prevalence of TWL by sex, diagnosis, and age at interview.

10.1 History of Trouble With the Law (TWL) by sex, diagnosis and age at interview (n=412)



The most effective factor protecting against TWL is eligibility for DDD (Division of Developmental Disabilities) services (Figure 10.2). Of clients 12 years and over, 62 had applied for DDD (applying, by itself, is neither a risk nor a protective factor): 35 were found eligible and 27 were not. Of the 27 "not eligible," 21 (or 78%) were coded yes for TWL. Of the 35 found eligible for DDD services, 16 (46%) were coded yes for TWL. (No data on the relative timing of these events is available.) Whereas DDD provides services (case managers, job placement and coaching, and shelter), SSI provides only money; eligibility for SSI (Supplemental Security Income) was *not* a protective factor against TWL.

10.2 Risk/Protective Factors for History of Trouble with the Law among clients 12 years and older at interview, max n = 251

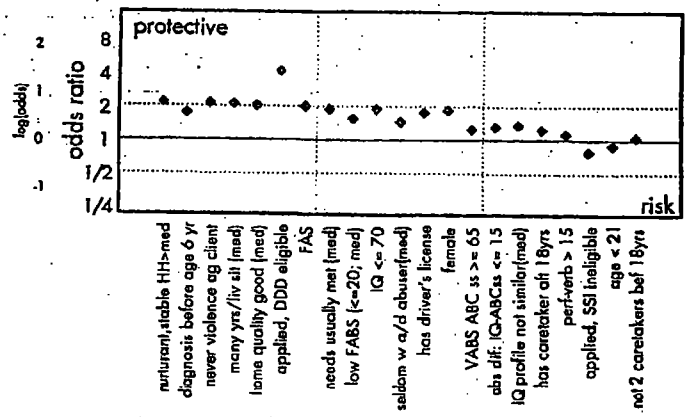


Figure 10.3 shows that crimes against persons are the most frequently reported in every age group. This category encompasses theft (including shoplifting), burglary, assault, and murder.

10.3 History of Trouble with the Law: Components of the Secondary Disability by age at interview (n=407-412)

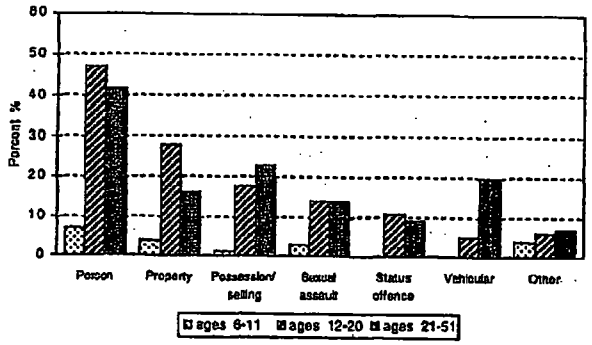
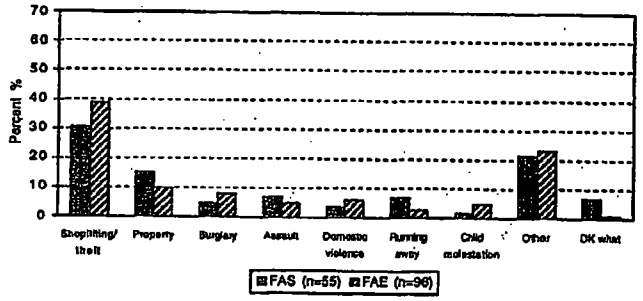


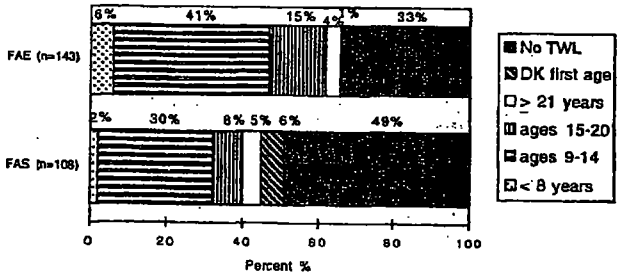
Figure 10.4 shows that shoplifting/theft is most often the first type of crime reported.

10.4 Nature of first trouble with the law among clients ≥ 12 years old with TWL by diagnosis (n=151)



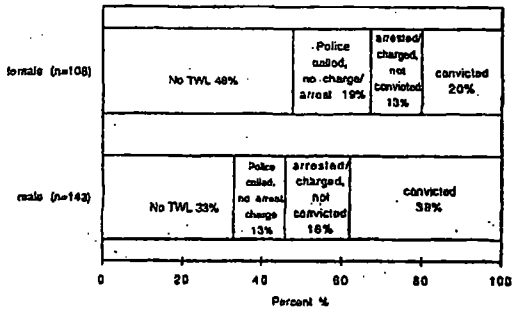
About one in three clients 12 years and older committed a first crime between 9 and 14 years of age. Very few clients had their first TWL after age 20 (Figure 10.5). The rate of TWL was mildly lower in the FAS subgroup, but distributions of first offenses for the two diagnoses were similar (Figure 10.4).

10.5 Age at first trouble with the law by diagnosis among clients≥ 12 years old at interview



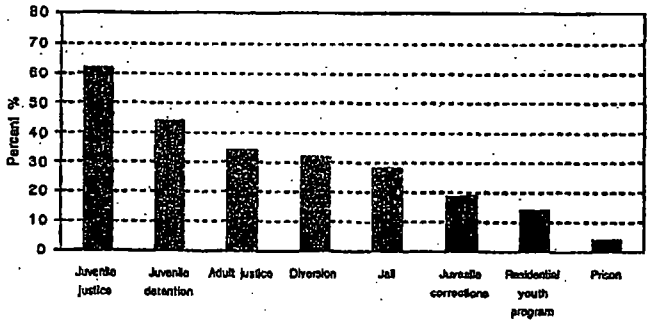
Females were less likely to be charged after the police were called, and less likely to be convicted than were males. (Figure 10.6).

10.6 Trouble with the law: arrests, charges, convictions by sex among clients ≥ 12 years old at interview

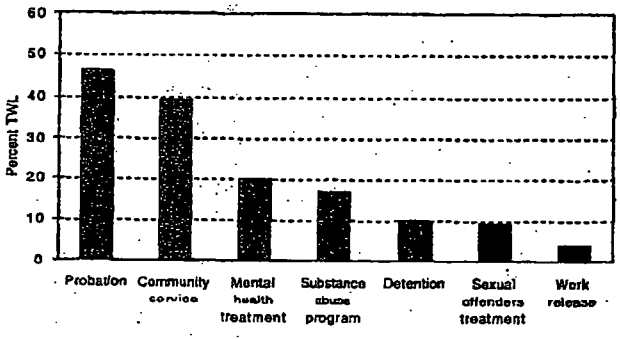


Figures 10.7 and 10.8 show that among clients who have had trouble with the law, the most prevalent types of sentencing are juvenile justice and juvenile detention, and the most prevalent sentencing alternatives are probation and community service.

10.7 Trouble with the law: type of sentence among clients ≥ 12 years old at interview with TWL (n=151)

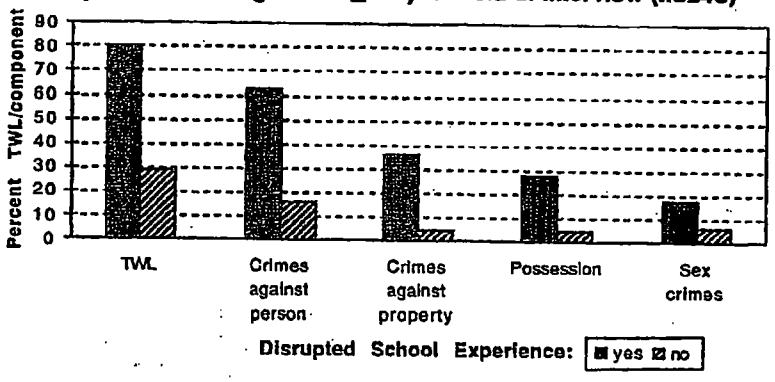


10.8 Trouble with the law: type of sentencing alternatives among clients ≥ 12 years old at interview with TWL (n=150)

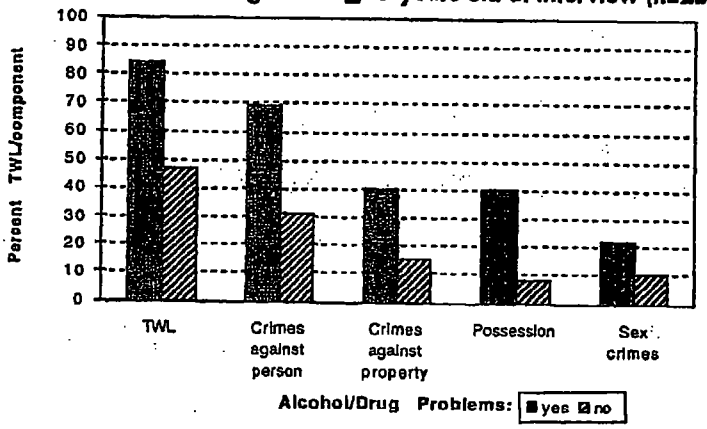


Figures 10.9 and 10.10 examine TWL in relation to two other Secondary Disabilities: Disrupted School Experience (DSE) and Alcohol and Drug Problems (ADP). Staying in school is related to a lower rate of TWL by a factor of 2½. Absence of Alcohol and Drug Problems (ADP) is also a factor against TWL, decreasing the rate by almost half.

10.9 History of TWL and its components by Disrupted School Experiences among clients ≥ 12 years old at interview (n=248)



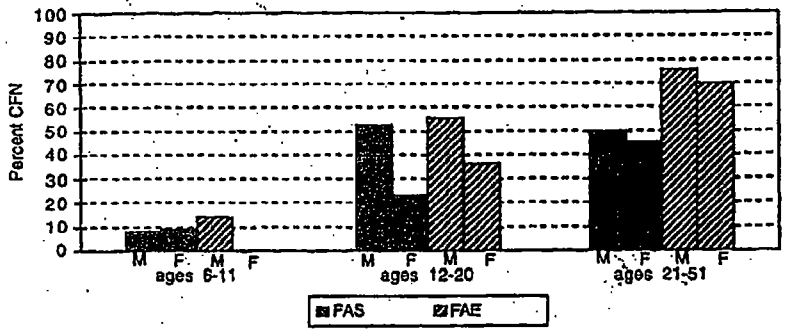
10.10 History of TWL and its components by Alcohol and Drug Problems among clients ≥ 12 years old at interview (n=250)



11 Confinement (CNF)

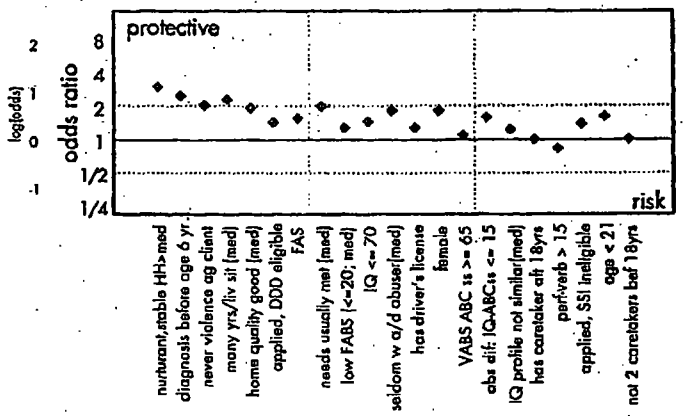
Confinement (CNF) is a binary score summarizing restrictions of personal freedom—the most serious consequence of any secondary disability. CNF is set to 1 for those clients who have ever been in a mental hospital (23% of the clients over 12 years of age), have ever been in an alcohol/drug inpatient treatment program (15%), or have ever been incarcerated for a crime (35%).

11.1 History of Confinement (CNF) by sex, diagnosis and age at interview (n=412)



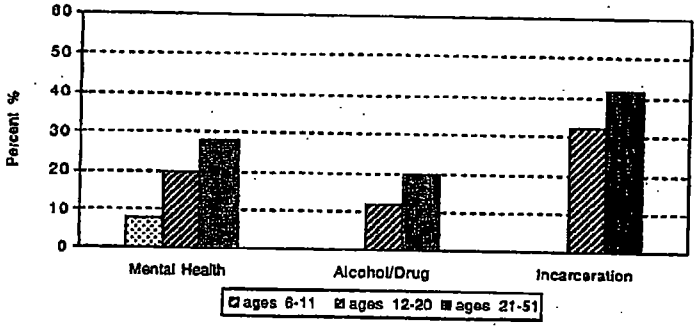
The protective factors against Confinement are the same as the "universal" set for the first six Secondary Disabilities as a group (notice how smooth the profile is in Figure 11.2). This score seems to extract the essence of the three most serious specific secondary disabilities that we have already examined. What protects the client against confinement is what protects him or her against the other secondary disabilities, namely, a stable and nurturant household, an early diagnosis, not being a victim of violence, relatively few distinct living situations, and the like.

11.2 Risk/Protective Factors for History of Confinement among clients 12 years and older at interview, max n = 251



Confinement was reported by caregivers of clients under 12 years of age at the time of interview only for mental health problems (Figure 11.3), but Figure 11.4 shows that age at first confinement was often younger than 12 years.

11.3 History of Confinement: Components of the Secondary Disability by age at interview (n=410-415)



11.4 Age of first confinement by type of confinement among clients ≥ 12 years old at the time of interview

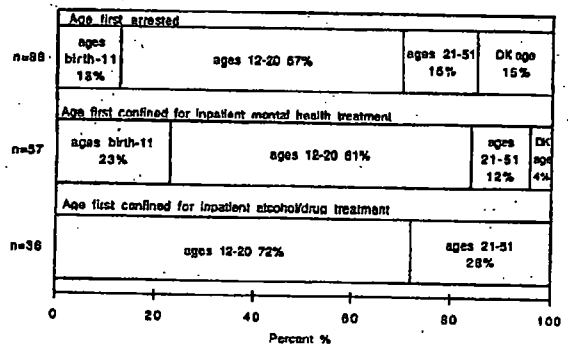
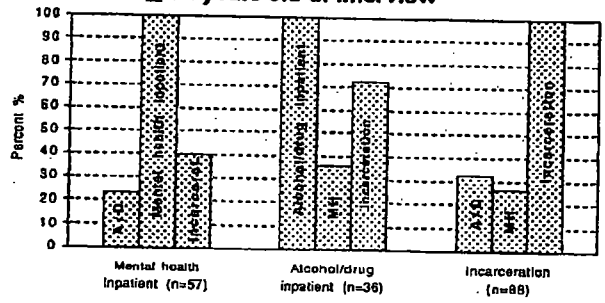


Figure 11.5 indicates that among the clients who were confined, many were confined for more than one reason. For example, clients who had been in mental health inpatient treatment, had also been in alcohol and drug inpatient treatment (22%) or had also been incarcerated (40%). Furthermore, 13 clients were reported to have experienced confinement of all three types.

11.5 Types of confinements in combination with each other among clients ≥ 12 years old at interview

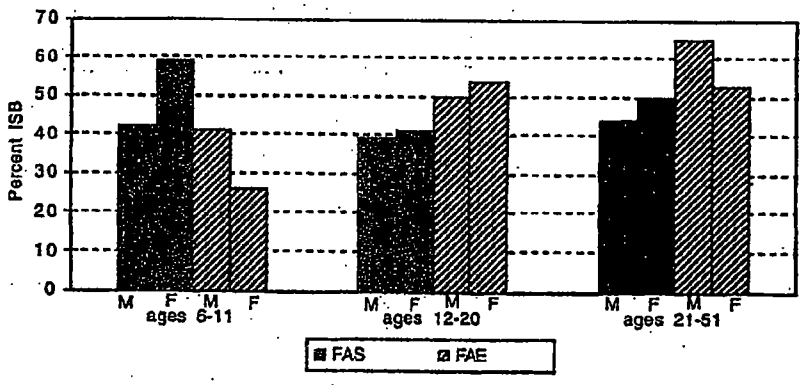


12 Inappropriate Sexual Behavior (ISB)

Inappropriate Sexual Behavior (ISB) is a binary score summarizing problems with sexual behavior among clients of any age. ISB is coded 1 for clients who were ever sentenced to a sexual offender's treatment program or who were reported to have "repeated" problems with one or more of the 10 inappropriate sexual behaviors identified in Figure 12.3.

Figure 12.1 shows prevalence of ISB by sex, age, and diagnosis. There do not seem to be important differences in rates of ISB by these three client characteristics.

12.1 History of Inappropriate Sexual Behaviors (ISB) by sex, diagnosis and age at interview (n=408)



The strongest risk factor against ISB is violence against client (Figure 12.2). Being a victim of violence increases the odds of sexually inappropriate behavior by fourfold. Inappropriate Sexual Behavior is the only one of the six main Secondary Disabilities (Figure 6.1) for which low IQ is not a protective factor. (As we will see in Chapters 14 and 15, IQ≤70 is also not protective for the two adult problems.) The reason that low IQ does not protect against ISB is that low IQ is a ***risk*** factor for three components of ISB, namely masturbating in public (odds ratio 6.5), inappropriate advances (odds ratio 2.8), and inappropriate touching (odds ratio 2.6).

12.2 Risk/Protective Factors for History of Inappropriate Sexual Behavior among clients 12 years and older at interview, max n = 247

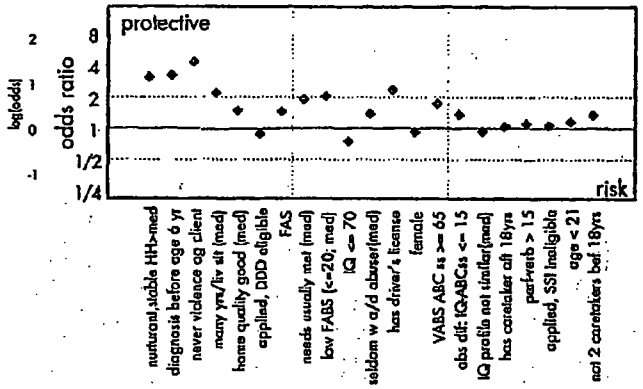
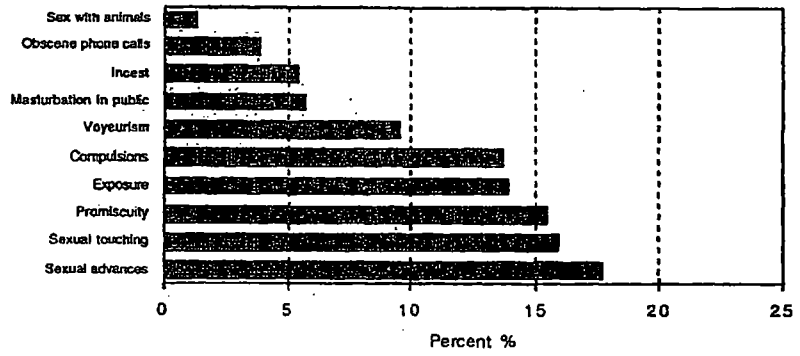


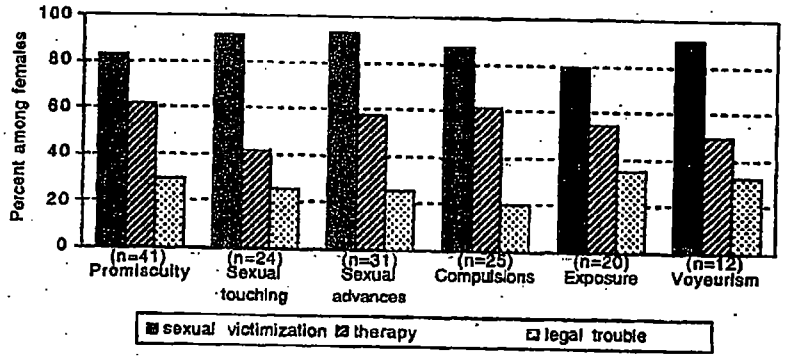
Figure 12.3 itemizes 10 inappropriate sexual behaviors and their prevalence in this sample. Repeated sexual advances toward others are the most prevalent, at 18% of the sample overall.

12.3 History of Inappropriate Sexual Behaviors reported as repeated behaviors (n=388-408)

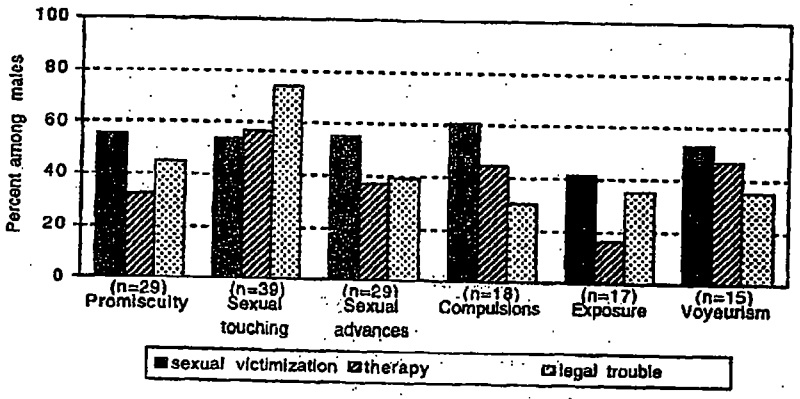


Figures 12.4 and 12.5 show that female clients who exhibited inappropriate sexual behaviors, were more likely to have experienced sexual victimization, and to receive therapy to treat their ISB. Whereas male clients who exhibited inappropriate sexual behaviors were more likely to get involved in legal trouble.

12.4 Prevalence of sexual victimization, therapy for ISB, and trouble with the law for sexual behavior by six common inappropriate sexual behaviors: Among females



12.5 Prevalence of sexual victimization, therapy for ISB, and trouble with the law for sexual behavior by six common inappropriate sexual behaviors: Among males

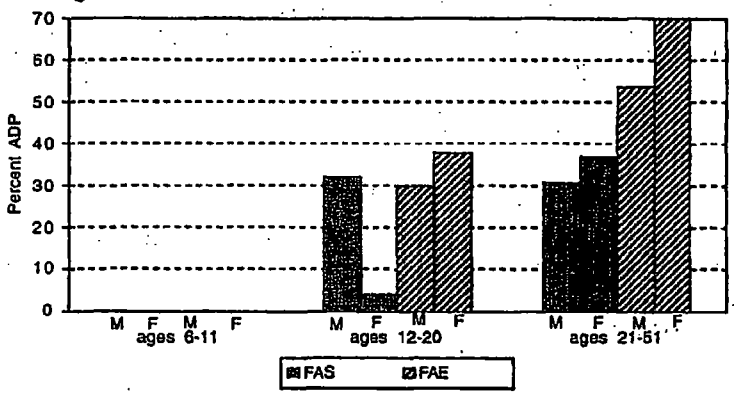


13 Alcohol and Drug Problems (ADP)

Alcohol and Drug Problems (ADP) is a binary score summarizing problems with alcohol or drug habits among clients of any age. ADP is coded 1 for clients who have ever had alcohol abuse problems, drug abuse problems, or have ever been in alcohol or drug abuse treatment (either outpatient or inpatient).

Figure 13.1 shows that report of ADP is not different for male versus female clients, but is related to age at interview and to diagnosis.

13.1 History of Alcohol/Drug Problems (ADP) by sex, diagnosis and age at interview



The obvious specific risk factor for ADP is living with alcohol abusers and drug users. Other important risk factors are from the "general" RPF set: late diagnosis of FAS/FAE, and violent behavior against clients (Figure 13.2).

13.2 Risk/Protective Factors for History of Alcohol/Drug Problems among clients 12 years and older at interview, max n = 252

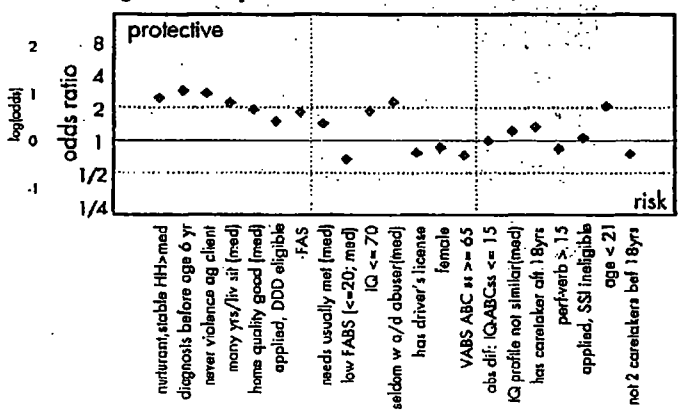
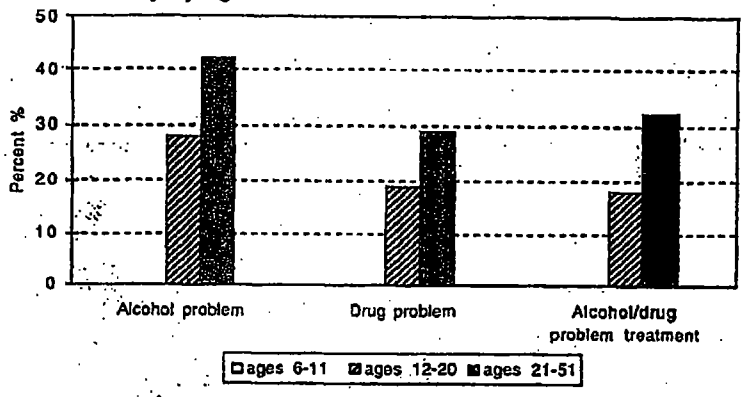


Figure 13.3 shows that among clients who were still younger than 12 years at interview, there was no Alcohol and Drug Problems reported. However, as will be seen in subsequent figures, problems often began at ages younger than 12 years among clients 12 years and older at the time of the interview (Figure 13.4). Overall, ADP-related rates among adolescents and adults were 33% for alcohol problems, 23% for street drug problems, and 23% for treatment. Overall ADP was noted for 35% of the clients 12 years and older.

13.3 Alcohol/Drug Problems: Components of the Secondary Disability by age at interview



The 57 clients with street drug abuse problems are almost entirely "nested" within the 81 clients with alcohol problems. The same is true for use of treatment programs. Among clients with both alcohol and street drug problems, alcohol abuse began on average about 2 years before the abuse of street drugs. Sixty-five percent of clients 12 years and older who had abused alcohol later abused street drugs. Also, among clients engaging in treatment for both alcohol and street drug abuse, the age at first treatment was the same for alcohol as for drug use, as if the multiple addictions were often needed to ensure action.

Possibly quicker treatment for alcohol abuse could lower the rate of later street drug abuse.

13.4 Age of onset of alcohol problems, by sex and diagnosis among clients with alcohol problems

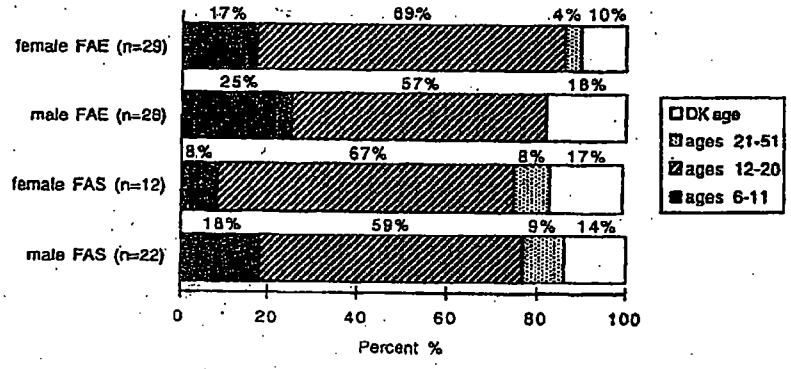
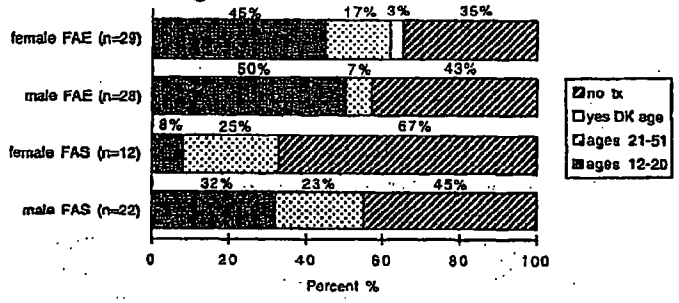


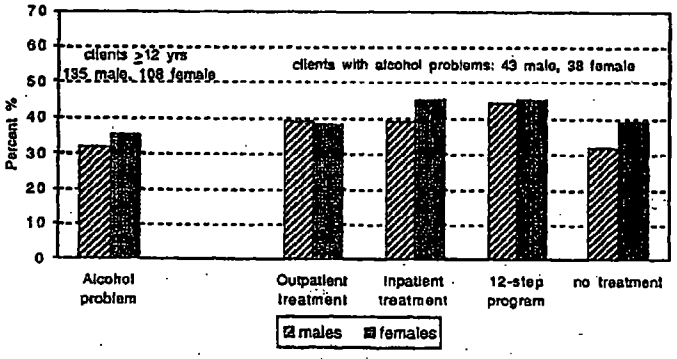
Figure 13.5 shows that 70% of clients who have been in treatment had their first alcohol treatment before age 21.

13.5 Age of first alcohol treatment among alcohol abusers, by sex and diagnosis

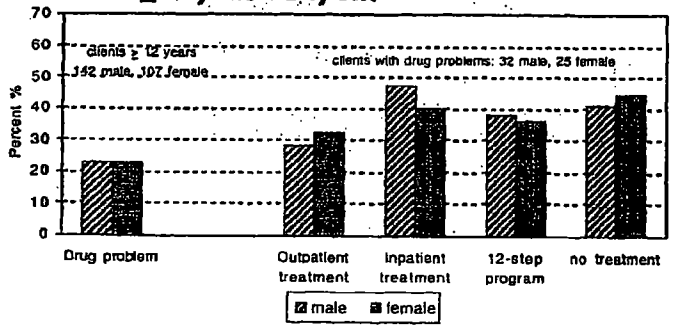


Figures 13.6 and 13.7 show the choices of treatment used by clients with alcohol abuse problems and drug abuse problems by sex of client. There is not much difference in use of the different treatment types by sex. There also does not seem to be an overall difference in rate of utilization of different treatment types. Inpatient treatment is as highly utilized as other types of treatment.

13.6 Alcohol problems and treatment type among clients ≥ 12 years old by sex

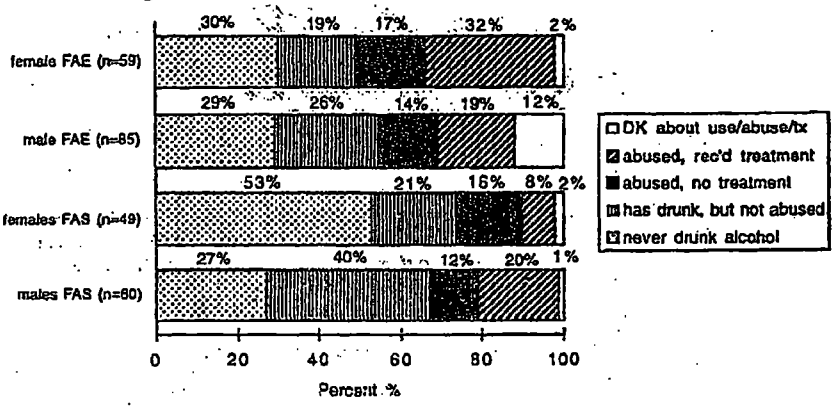


13.7 Illicit drug problems and treatment type among clients ≥ 12 years old by sex



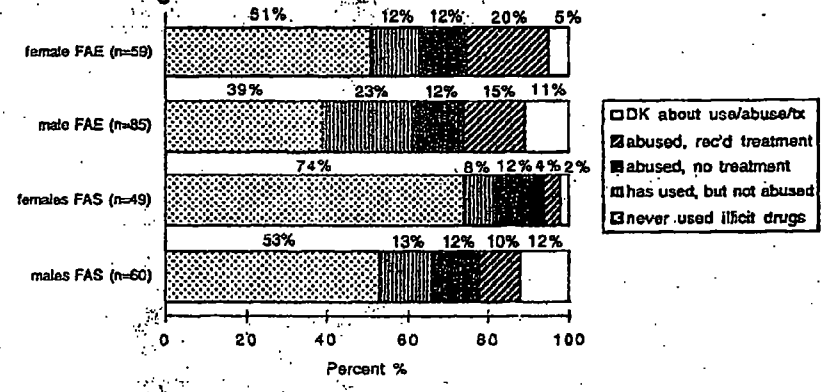
Figures 13.8 and 13.9 depict the four stages of alcohol and drug use respectively: "no use"/"using, but not abusing"/ "abusing, but no treatment yet"/"abusing and treatment received." Female FAS clients have the highest rate of abstinence. Among those who become alcohol abusers, they have the lowest rate of treatment (40% compared to 65% for alcohol abusers as a group). Male FAS clients have the highest rate of use. Among those who become alcohol abusers, they also have the highest rate of treatment.

13.8 Stages of alcohol use among clients ≥ 12 years old by sex and diagnosis



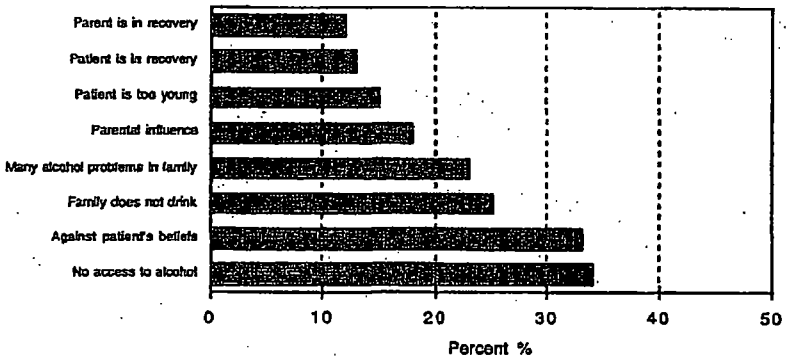
A very similar pattern is observed for street drug use (Figure 13.9). Female FAS clients have the highest rate of street drug avoidance, but among those who become drug abusers, they have the lowest rate of treatment (33% compared to 58% for all street drug abusers as a group). Male FAS clients have the highest rate of street drug use.

13.9 Stages of illicit drug use among clients ≥ 12 years old by sex and diagnosis



Many clients have avoided alcohol use completely. The reasons caretakers report for the clients abstaining from alcohol are as presented in Figure 13.10.

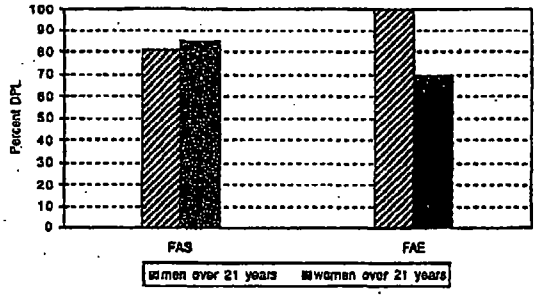
13.10 Reasons for abstinence among clients ≥ 12 years old reported not to consume alcohol at present (n=163)



14 Dependent Living (DPL) Over 21 Years

Dependent Living (DPL) could only reasonably be defined for the 90 people who are 21 years and older. Two sets of criteria were developed for independence; one could qualify as "independent" by either one. The first focuses on independence in 12 daily living activities (regardless of whether or not support people are available); the second focuses more on *not* having a support person organizing their lives, *not* living with a caregiver, and being able to handle at least 9 of the 12 daily living activities, including paying own expenses. No one was counted as an independent adult who does not manage his or her own money (See Figure 14.3 for this list of 12 daily activities). Figure 14.1 shows that 83% of adults live dependently.

14.1 Prevalence of Dependent Living (DPL), by sex and diagnosis



Only one of our "universal" RPF's is protective against DPL: diagnosis before age six. Several RPF's specific to DPL are apparent in Figure 14.2, including a VABS ABC score of 65 or above, IQ above 70, a VABS ABC being within 15 points of IQ, a low FABS score (below the median), and being female. The environmental RPF's include having a driver's license and not having (presumably, not needing) two caretakers.

Dependent living and problems with employment each characterized approximately 80% of the adults with FAS/FAE; 70% had both. Only 8% (7 clients) had neither.

14.2 Risk/Protective Factors for Dependent Living among clients 21 years and older at interview, max n = 89

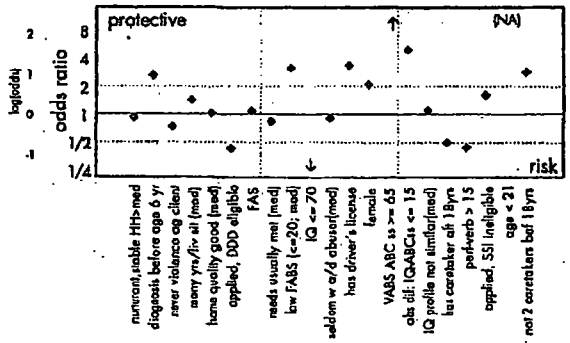


Figure 14.3 lists 12 daily activities that people sometimes or frequently need help with. The daily tasks requiring help by the largest numbers of clients 21 years and older include managing money and making decisions.

14.3 Daily life activities among clients ≥ 21 years old (n=90)

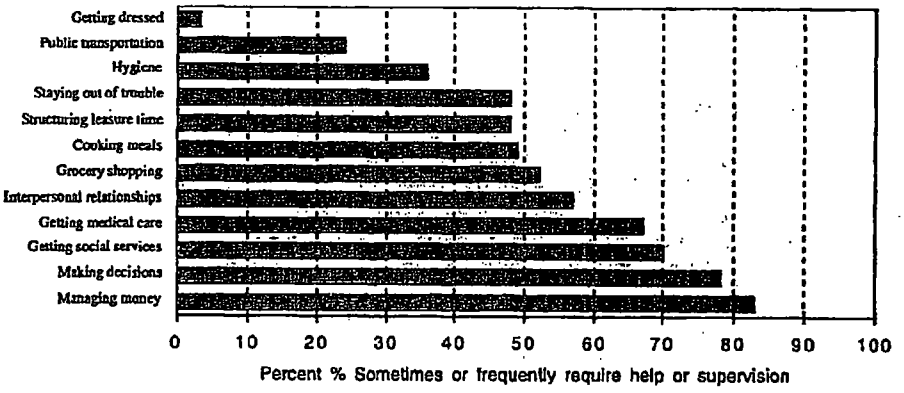


Figure 14.4 shows that female FAE clients are most likely to be able to do all 12 daily activities without help, but all 20 are over 21 years old, and only 5 (25%) of them are reported to be capable of doing these tasks.

14.4 Number of daily activities among clients ≥ 21 years old, by sex and diagnosis

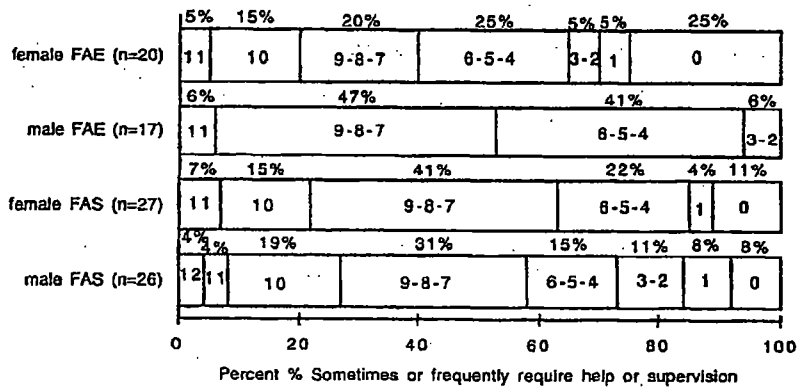
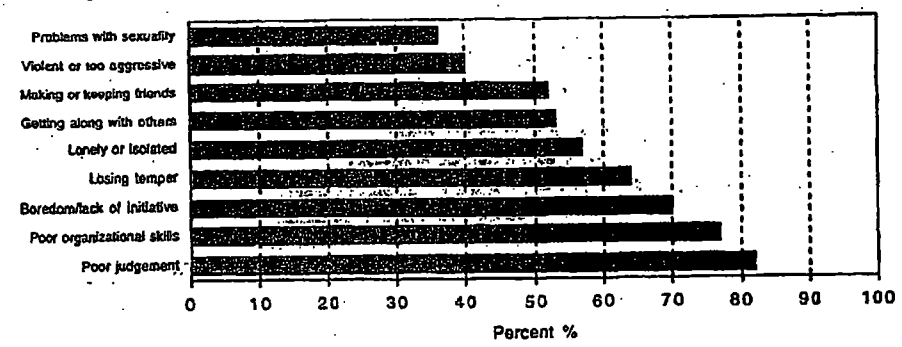


Figure 14.5 shows that even as adults, people with FAS and FAE experience social problems which significantly compromise their daily functioning. Hence, it is not surprising that over 80% of these adults have difficulty living independently and need intensive help and supervision with managing their daily activities.

14.5 Current caregiver reports of social problems "sometimes" or "frequently" experienced among clients ≥ 21 years old (n=76-88)



15 Problems With Employment (PWE)

Problems With Employment (PWE) were endemic and variable in this group of clients. We ended up defining a group who were *relatively* problem-free. The clients in this group have little in common except that they are all employed. Beyond that, we have combined three more specific groups, overlapping, but not nested, defined as follows:

Group 1. No financial support except own earnings.

Group 2. Earns $280 a week or more.

Group 3. This category was added based on the characteristics of several specific individuals who seemed to be effectively employed but didn't meet any of the preceding criteria. These were people who were not in a sheltered workshop, were working at least half time, and had no more than 3 jobs in the past 2 years. In addition, from the list of problems under Group 1, they had no repeated problems and only 1 or 2 problems of any kind.

PWE is defined by all clients 21 years and older who are *not* relatively problem-free. Figure 15.1 shows that 79% of clients 21 years and older have PWE.

15.1 History of Problems With Employment (PWE) among clients ≥ 21 years old, by sex and diagnosis (n=90)

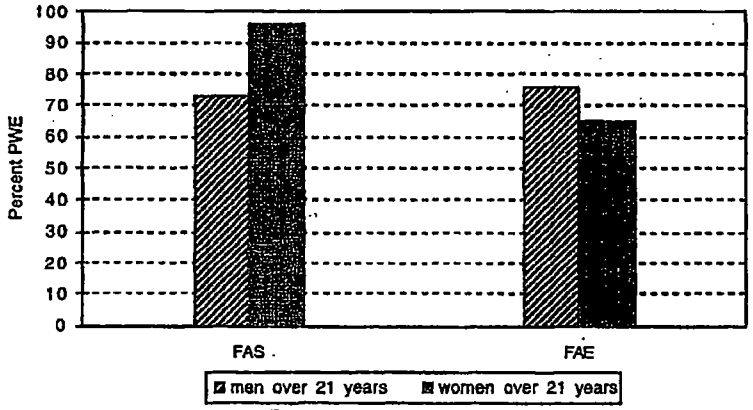


Figure 15.2 shows that a VABS Adaptive Behavior Composite higher than the median is a protective factor for successful employment. There are also two clear intrinsic risk factors against successful employment: being FAS (as opposed to FAE) and having a low IQ. No person with FAS or FAE who had an IQ 70 or below met our criteria for successful employment, even though low IQ was not explicit in the definition. In addition, one specific protective factor emerged (having a driver's license), and some of the "universal" set of protective factors emerged: more than the median number of years per living situation followed closely by never experiencing violence, an early diagnosis, and living in a nurturant and stable home for over 72% of life.

15.2 Risk/Protective Factors for Problems With Employment among clients 21 years and older at interview, max n = 90

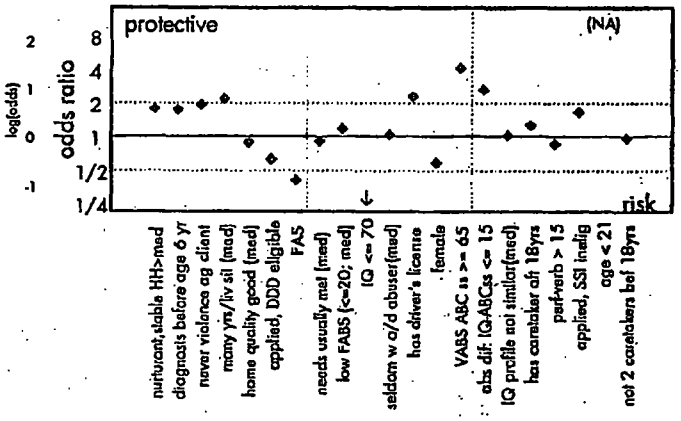


Figure 15.3 shows that holding a job is a bigger problem than getting a job.

15.3 Problems acquiring and maintaining employment by clients ≥ 21 years old (n=78-83)

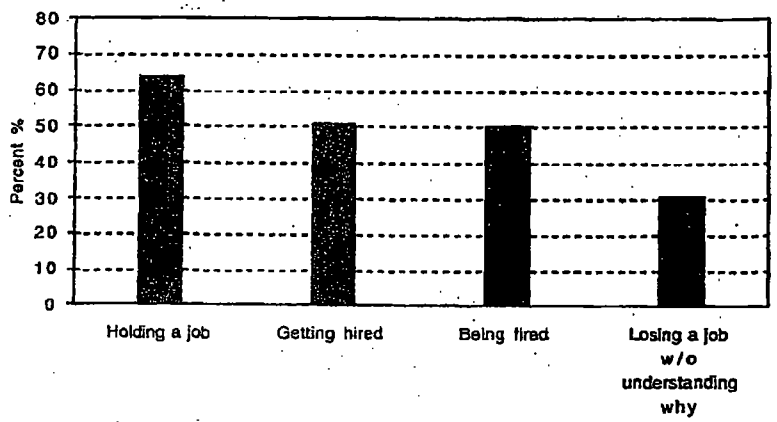
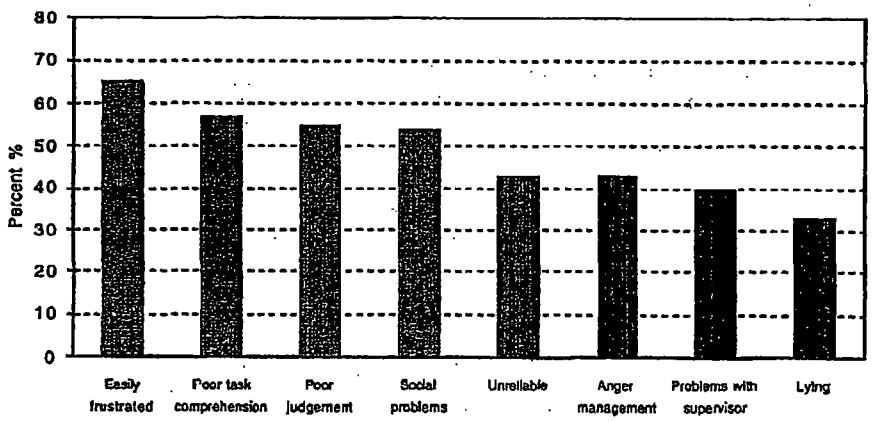


Figure 15.4 itemizes many problems that clients have on the job. At least one, "poor task comprehension" (57%), may reflect poor job placement. Educating personnel directors and job placement services staff to special characteristics of clients with FAS or FAE may be mutually beneficial both to the clients and employers.

15.4 Type of employment problems experienced while on a job (n=80-83)



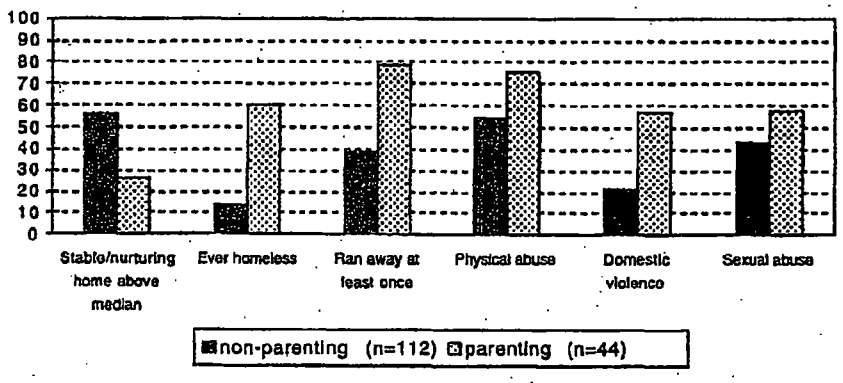
16 Problems with Parenting (PWP)

Of the 253 clients who were over 12 years old at the time of the LHI, 44 (17%) had become parents; 30 females and 14 males (28% of females 12 years and over and 10% of males). For females, the mean age of the first pregnancy was 18 years, for males, the mean age of first fathering of a child was 20 years. The youngest age at which a client with FAS or FAE had a child was 13 years. The maximum number of children born to a mother was four (Only children known to the respondent were reported on the LHI, as clients were not interviewed themselves).

In order to compare clients who became parents with those who did not, a subset of "non-parenting clients" was selected from the database, based on age of client at interview. There was little difference in the average IQ of the two groups. Average IQ was 84 for parents, ranging from borderline mental retardation (IQ 70) to above average (IQ 117). Parents had a slightly higher VABS Adaptive Behavior score than non-parents (66 versus 60).

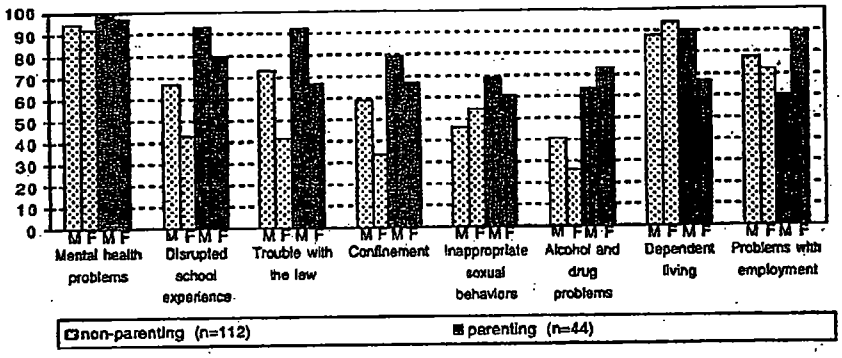
Compared to the non-parenting clients, clients who became parents had a higher rate of history of living in unstable homes. They were much more likely to have been homeless, more likely to have run away from home, and experienced over twice the rate of domestic violence (Figure 16.1).

16.1 Prevalence of risk and protective factors by parenting status



Parenting clients had more Disrupted School Experience, more Trouble with the Law, more Confinement, and more Alcohol and Drug Problems, compared to non-parenting clients (Figure 16.2). They were over two times more likely to have dropped out of school (66% versus 26%).

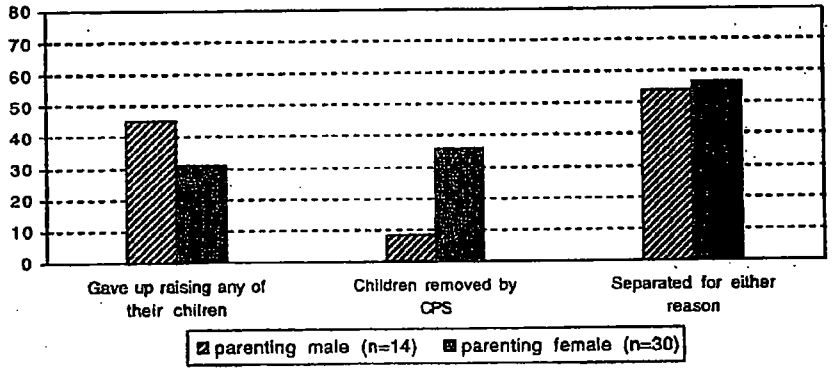
16.2 History of Secondary Disabilities: Comparing non-parenting and parenting clients



Fifty-nine percent of the parents were diagnosed FAS or FAE after they had become parents; 30% before they became parents and for the rest, the two ages were too close to evaluate in this regard. The 44 parents had produced 76 children, at least half of whom were no longer being cared for by the client (Figure 16.3). Thirty six percent of female clients had their children removed from their care by CPS, whereas 45% of male clients had given up raising their children.

Parenting clients were more likely to be married (23% versus 4%). They were more likely to have ever been on welfare (81% versus 48%), but somewhat less likely to have ever received SSI than non-parents (44% versus 50%); 10% of the parents had applied for and received eligibility for DDD versus 20% of the non-parents.

16.3 Separation of parent from child among parenting clients: Comparing male and female clients



An alarming 40% of the female parents were drinking during pregnancy; 17% had a child who had been diagnosed FAS or FAE, and another 13% had children that the respondents suspected of being fetal alcohol affected.

17 Recap and Recommendations

In this chapter, we summarize some of the main findings from this report, draw conclusions, and make recommendations for overcoming the Secondary Disabilities we have documented. While causation can't be inferred from these data, they nevertheless can suggest appropriate courses of action. First we focus on the "extrinsic" risk factors—those that suggest environmental influences, specific programs and so forth. Then we go to the "intrinsic" risk factors—those that reveal typical client characteristics that would demand special protection or consideration by caretakers and service providers.

17.1 Facilitate an early diagnosis of FAS/FAE

Although an early FAS/FAE diagnosis is a strong "universal" protective factor for all Secondary Disabilities (Figures 6.1), only 11% of the clients had a diagnosis prior to age 6 (Table 6.1). An early FAS/FAE diagnosis may help the family better understand the root of their child's developmental delays and behavioral problems and empower the family to advocate for appropriate services particularly in the crucial first years at school. A diagnosis is an effective communication tool that enables caregivers to educate others about the special needs of alcohol-affected individuals.

Many actions could be taken to foster an early diagnosis. These include:

- A statewide network of FAS Diagnostic Clinics such as is already underway in Washington State through the University of Washington Medical School (Clarren & Astley, 1997).
- A referral network system whereby children at risk of having FAS/FAE are screened during infancy and the preschool period for possible FAS/FAE. Those children identified at risk (i.e., from those being adopted from alcohol-abusing mothers, those who are under the supervision of Children's Protective Services for neglect or abuse, and those entering the foster care system) are referred to their local FAS Diagnostic Clinics. A foster care screening program is presently underway in King County, Washington.
- Routinely obtaining prenatal exposure information at each point at which the child and family are evaluated by the system (prenatal care, delivery, and pediatric care school entry, criminal justice, mental health, and so forth).
- Training school nurses to screen for FAS/FAE as part of the school entrance examination. The effectiveness of this program in two counties of Washington State has already been demonstrated.

17.2 Encourage stable long-lasting placements in nurturant homes of "good quality"

Desirable characteristics of the client's home life (stability, nurturance, "good quality") are universal protective factors for all Secondary Disabilities (Figures 6.1). This common sense protective factor is a fundamental right for the well being of all children. The fact that many alcohol-affected individuals have come from dysfunctional, transient, and abusive living situations demands that we reiterate the need for more effective community and family support services, perhaps modeled on the Birth to 3 program (Grant et al., 1996).

Environmental risk factors for secondary disabilities that must be modified if we are to prevent or decrease secondary disabilities are exemplified by the following problems revealed by this study:

- Half of our clients over 12 years of age have *not* lived at least 72% of life in a stable and nurturant environment.
- Half have *not* lived in a home with at least 10 of 12 positive qualities between the ages of 8 and 12 years;
- Half had *not* stayed in each living situation for an average of more than 2 years; and
- Half of our clients 12 years and older, had *not* had their basic (food and shelter) needs met for more than 12% of life.

By identifying environmental risk factors, service providers can work together with the family to prevent secondary disabilities in alcohol affected individuals.

- Early identification of children born to "high risk" women with alcohol and drug problems should draw community resources to these children and their families, ideally during the prenatal period, the postpartum period, and onward into infancy and childhood.
- Advocates/case managers in close personal contact with high risk families can work to improve the quality of the home. When the child's safety in the home cannot be safeguarded, the child should be moved to an alternative long-term home or placed for permanent adoption as early as possible.
- An early diagnosis can identify children with FAS/FAE living in high risk homes, so that biological families can obtain the same state resources that should be available for foster and adoptive families (family support services, medical coverage, home visits, respite care, and case management opportunities and so forth).
- On a policy level, requiring state personnel to disclose the full medical/mental health background history before placing a child in foster care or adoptive placement should insure that the parents will know about the special needs of their children with FAS/FAE from the onset of parenting.
- A better system for educating and training parents about children with FAS/FAE is needed, so that parents and all caregivers can use their energies most efficaciously on behalf of their children.
- Group homes providing stable, long-term residences with personal and job supervision appropriate to the special needs of youth and adults with FAS/FAE are urgently needed. As a start, a model program should be funded, implemented, and evaluated.

17.3 Improve the circumstances of children in alcoholic/drug abusing homes

Living with people who have alcohol/drug problems was a risk factor for five of the eight Secondary Disabilities studied: Alcohol and Drug Problems, Confinement, Disrupted School Experience, Trouble with the Law, and Inappropriate Sexual Behavior.

Half of the clients who were 12 years old and older, had spent at least 30% of their lives living with a person who had an alcohol/drug problem.

Based on our findings, we recommend that efforts be focused on:

- Detecting high risk alcohol and drug abusing mothers and providing appropriate services (during prenatal care, at delivery, at FAS Diagnostic Clinics, and at community treatment centers).
- Expanding alcohol and drug treatment services for women so that they can keep their children with them and their families intact during inpatient treatment.
- Developing advocacy services for alcohol/drug abusing mothers and using advocates as a liaison with CPS for monitoring the safety of children in alcohol/drug-abusing homes (as has been successfully accomplished in the Seattle Birth to 3 Program; Grant et al, 1996).

17.4 Intervene to prevent violence against people with FAS/FAE

Violence against the client is almost a universal risk factor for all Secondary Disabilities, impacting six of the eight studied (Figure 6.1). It is alarming to find that 72% of the clients with FAS/FAE who were 12 years and older have experienced violence (either physical or sexual abuse, and/or domestic violence), and that many have experienced several types of violence in their lifetimes.

Having experienced violence is by far the strongest risk factor for Inappropriate Sexual Behavior. At 45%, Inappropriate Sexual Behavior is the second most prevalent Secondary Disability in people with FAS/FAE across the lifespan (Figure 7.1). Regardless of age, between 40% and 52% of clients in the three main age groups studied had engaged in Inappropriate Sexual Behavior. In turn, Inappropriate Sexual Behavior has the strongest relationship to two other Secondary Disabilities, namely, Trouble with the Law and Confinement. These latter are the most costly, serious, and disruptive societal response to unacceptable, misunderstood, or problem behaviors (Figure 6.2). Clearly, there are strong economic and humanistic reasons for reducing all of these Secondary Disabilities and the risk factors related to them.

Both children and adults with FAS/FAE are vulnerable to being victims of violent physical and sexual behavior, perhaps as a result of both their prenatal brain damage and the high risk environments in which they often live.

Based on our findings, we recommend the following efforts be made to prevent violence against these vulnerable clients:

- Advocates, such as those described in the Birth to 3 program, can serve as an effective liaison with Children's Protective Services (CPS) for monitoring the safety of children and adults with FAS/FAE in high risk homes.
- Several groups of professionals must be informed about the presence of people with FAS/FAE among their client populations, so that the most efficacious treatment models can be developed and implemented. These groups include: sexual deviancy counselors, the sexual abuse treatment field, and those dealing with domestic violence.
- The possibility of prenatal alcohol exposure should be explored (along with a history of physical and sexual abuse) among all patients coming into trouble with the law, mental health, or judicial confinement settings.
- Further research on the most effective response of the criminal justice system to the expression of Inappropriate Sexual Behavior by people of all ages with FAS/FAE is urgently needed. Sentencing alternatives, methods of enhancing familial supervision, and programs to enhance social skills and job skills are needed.

17.5 Develop, evaluate, and implement methods to detect subgroups of people with FAS/FAE who are in special need of interventions

The recommendations in 17.1–17.4 pertain to environmental influences resulting from the discoveries in this study about the "extrinsic" risk factors for secondary disabilities. This study has also addressed the relationship of "intrinsic" characteristics of certain subgroups of people with FAS/FAE that might put them at special risk of developing secondary disabilities. These include:

- people with FAE, who do not have the full facial stigmata of FAS and thus may not readily qualify for services,
- people with FAS or FAE whose IQ level is above 70 and who consequently often fail to qualify for special services,
- people with FAS/FAE who have a marked discrepancy between their IQ levels and their level of Adaptive Behavior, and
- people with FAS/FAE who have a particular cluster of characteristic behaviors as identified by a high FABS score on the Fetal Alcohol Behavior Scale.

Recommendations follow:

- Further research is urgently needed to quantify the prenatal brain damage from alcohol in people who do not have the physical signs of FAS and/or who do not have an IQ score permitting classification as mentally retarded (i.e., IQ below 70).
- The identification and evaluation of new quantification methods should facilitate diagnosis as well as permitting the development of more appropriate guidelines for providing needed services.
- One promising new scale developed for this study that needs further evaluation is the Fetal Alcohol Behavior Scale (FABS). As a high FABS score is a risk factor for numerous secondary disabilities, providing specialized services for this subset of clients might be particularly cost effective.
- Another promising marker derives from the "IQ/Adaptive Behavior Discrepancy" score which is also associated with secondary disabilities. Further study of this and the use of other techniques for evaluating specific cognitive, linguistic, and neuropsychological problems in people with both FAS and FAE, particularly those with IQ scores too high to readily qualify for services, should be a high priority for future research.
- Finally, of course, effective strategies for remedial interventions appropriate to the needs of people with FAS/FAE need to be developed, evaluated, and implemented.

17.6 Promote communities, families, and clients with FAS/FAE working together

Just as all children need immunizations against diseases, people with FAS/FAE appear to need special "immunization" against many inadequacies and dangers in our society. Their functioning is often far below their apparent intelligence. Many people with FAS/FAE appear to learn only inadequately through normal channels about their environment and how to interact with it. Working around the disabilities that these clients are born with is not the family's responsibility alone, but neither is it solely the government's or community's responsibility. Communities, families, and clients working together toward this mutual goal of reduction of Secondary Disabilities may foster better optimization of quality of life for both people with FAS/FAE and their communities.

The Secondary Disabilities data indicate that clients with FAS/FAE come in contact with many different community professionals and agencies. For example: over 90% deal with Mental Health professionals, 60% with the criminal justice system, 23% with alcohol and drug treatment personnel (Figures 8.1, 10.1, 13.3), and 100% with school personnel. The finding that an early diagnosis is a universal protective factor against *all* Secondary Disabilities suggests that information about the diagnosis is useful to have in responding appropriately to a child's problems. People with FAS/FAE and their families could facilitate more beneficial and humane services by sharing the diagnostic information with professionals they work with.

Half of the clients 12 years and older display Inappropriate Sexual Behavior (Figure 12.1). Although the rates of these behaviors are about the same for females and males, the outcome is very different. Thirty nine percent of the males 12 years and older with Inappropriate Sexual Behaviors are "in Trouble with the Law" over these behaviors, while about 40% of the females go to treatment. The inappropriate sexual behavior that gets males into trouble with the law is "inappropriate touching". Females most frequently go to treatment for promiscuity, compulsions, and sexual advances. People with FAS/FAE and their families should know that Inappropriate Sexual Behavior does not just begin in adolescence. Among 6-11 year olds, approximately 37% of girls and 41% of boys are already exhibiting these behaviors. Families, criminal justice, and mental health professionals need to work together to develop new intervention strategies that will truly reduce rather than increase the level of Secondary Disabilities.

Alcohol problems occur in 33% of clients with FAS/FAE over the age 12 (Figure 13.1)—of these, 65% have gone on to abuse street drugs, at an average of two years after the onset of alcohol abuse. Early parental recognition and intervention of alcohol problems among youth with FAS/FAE could result in the prevention of other drug abuse problems. Families should also be apprised that female clients have as high a rate of alcohol problems as male clients, and that females are much less likely to receive treatment for their alcohol problems. Clients with FAS/FAE and their families should be aware of the special vulnerability to alcohol problems that is carried by all children of alcoholics, and develop appropriate early family practices to guard against the onset of alcohol problems among alcohol-affected individuals.

Approximately 80% of the adults with FAS/FAE in this study do not appear to be achieving either an independent living status or problem-free employment (Figures 14.1, 15.1). People with FAS/FAE, their families, school personnel, and mental health professionals need to start planning long before adulthood for a lifetime involving some degree of support. Training of appropriate living and working skills needs to start long before adulthood.

Many recommendations have been made regarding what the government agencies can do to help lower the rates of Secondary Disabilities observed in this client sample. Families can do a lot too. Certainly, communities also need better education about people with FAS/FAE. But it is the cooperation among families, communities, government agencies, and clients that permits a focusing of combined energies toward solutions that are beneficial to all.

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19 Glossary of Terms and Abbreviations

• **ABC score**	Adaptive Behavior Composite—the summary score from the VABS, derived from combining the four domains of adaptivity measured: Communication Skills, Daily Living Skills, and Socialization. The ABC is a standard score, like IQ, which is set to the same parameters as the Wechsler IQ tests: 100=normal and 15=one standard deviation
• **ADP**	Alcohol and Drug Problems
• **Arith**	Arithmetic—subtest of Wechsler IQ tests
• **ARND**	Alcohol Related Neurodevelopmental Disorder
• **Birth to 3**	The Seattle Advocacy Model Program for high risk mothers abusing alcohol and drugs. (See Grant et al., 1996)
• **BlkDes**	Block Designs—subtest of Wechsler IQ tests
• **CDC**	Centers for Disease Control and Prevention
• **CFN**	Confinement
• **CNS**	Central Nervous System
• **Coding**	Coding—subtest of Wechsler IQ tests
• **Comp**	Comprehension—subtest of Wechsler IQ tests
• **CPS**	Children's Protective Services
• **DDD**	Division of Developmental Disabilities
• **DigSp**	Digit Span—subtest of Wechsler IQ tests
• **DK**	Don't Know
• **DPL**	Dependent Living
• **DSE**	Disrupted School Experience
• **DSM-IV**	Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition
• **DVR**	Department of Vocational Rehabilitation
• **Dx**	Diagnosis
• **Extrinsic RPF**	A Risk and Protective Factor that describes something in the client's environment or something that has happened to the client
• **FABS**	Fetal Alcohol Behavior Scale—The FABS is a list of 36 behaviors (selected out of 72 items on a Personal Behaviors Checklist) that parents and caretakers have frequently used to describe people with FAS. The 36 items are characteristic of people with FAS irrespective of age (except infants and the elderly) and IQ (except the profoundly retarded). (See Streissguth, Barr and Press, 1996)
• **FADU**	Fetal Alcohol and Drug Unit
• **FAE**	Fetal Alcohol Effects
• **FAS**	Fetal Alcohol Syndrome
• **Info**	Information—subtests of Wechsler IQ tests

- **Intrinsic RPF** A Risk and Protective Factor that involves some characteristic of the clients themselves (like IQ)
- **IOM** Institute of Medicine
- **IQ** Intelligence Quotient. This is a standard score, with the population mean=100, and 15=one standard deviation.
- **ISB** Inappropriate Sexual Behaviors
- **LHI** Life History Interview
- **Max** Maximum
- **Med** Median
- **MHP** Mental Health Problems
- **NPCP** National Perinatal Collaborative Project
- **ObjA** Object Assembly—subtest of Wechsler IQ tests
- **Odds Ratio** The ratio of two ratios describing the relation of a two-valued outcome (say, A and B) to a two-valued predictor (say, y and n). It is the ratio (yB/yA)/(nB/nA), or (yB*nA)/(yA*nB). (See Chapter 6 for examples)
- **PFAE** Possible or Probable Fetal Alcohol Effects
- **PFAS** Possible or Probable Fetal Alcohol Syndrome
- **PicA** Picture Arrangement—subtest of Wechsler IQ tests
- **PicC** Picture Completion—subtest of Wechsler IQ tests
- **PIQ** Performance Scale IQ—from the Wechsler IQ tests
- **PWE** Problems With Employment
- **RPF** Risk and Protective Factors
- **SD** Secondary Disabilities
- **Sim** Similarities—subtest of Wechsler IQ tests
- **Specific RPF** A Risk and Protective Factor that only applies to some of the eight Secondary Disabilities studied, or may be Risky for some and Protective for others.
- **SS** Standard Score: the rescaling of any numerical variable so it has an easily remembered standard deviation (in the case of Wechsler IQ tests, the WRAT-R and the VABS, the average=100 and the standard deviation around the average is 15 points).
- **SSI** Supplemental Security Income
- **TWL** Trouble With the Law
- **Tx** Treatment
- **Universal RPF** A Risk and Protective Factor which is uniformly protective or risky for all six of the main Secondary Disabilities described in Figure 6.1 and Table 6.1.
- **VABS** Vineland Adaptive Behavior Scale
- **VIQ** Verbal Scale IQ—from the Wechsler IQ tests
- **Vocab** Vocabulary—subtest of Wechsler IQ tests
- **WISC-R** Wechsler Intelligence Scale for Children-Revised
- **WPPSI-R** Wechsler Preschool and Primary Scale of Intelligence-Revised
- **WRAT-R** Wide Range Achievement Test-Revised

Adaptive behavior and Fetal Alcohol Spectrum Disorders

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Adaptive Behavior is a construct originally invented as a prong for diagnosing Mental Retardation, but it has come to also play a role in diagnosing Fetal Alcohol Spectrum Disorders (FASD). In this paper, we: (a) describe the meaning and measurement of adaptive behavior, including the need for improved measures (b) illustrate clinical and research uses of the construct, in establishing the diagnosis and behavioral profile of people who have FASD and (c) discuss the forensic uses of adaptive behavior for people with FASD in establishing a mitigation case in criminal proceedings, including possible extension of Atkins *(death penalty exemption) protection for people with organic impairments who function just as if they have mental retardation in spite of possessing IQ scores in the borderline range.*

KEY WORDS: *Adaptive behavior, FASD, mitigation,* Atkins.

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The term "adaptive behavior" (sometimes referred to as "adaptive functioning") grew out of the animal research literature, and refers to an organism's competence in the real world rather than in a formal and artificial assessment situation (such as mazes for rats, IQ tests for humans). In clinical psychology, special education, and criminal law, the construct is used mainly in diagnosing Mental Retardation (now beginning to be termed Intellectual Disabilities), as the second of three definitional prongs (the other two being low IQ and developmental onset before adulthood) found in both clinical manuals and state criminal statutes. However, adaptive behavior is being used increasingly in diagnosing FASD; for example, the diagnostic protocol used by a prominent FASD forensic consulting group calls for the use of the Vineland Adaptive Behavior Scales (VABS) as one of their diagnostic methods.

This article is divided into three sections. In the first section, we describe the meaning and measurement of adaptive behavior, including the need for improved measures, especially ones which better capture foolish (risk-unaware) social and practical conduct. In the second section, we illustrate clinical and research uses of the adaptive behavior construct in establishing the diagnosis and behavioral profile of people who have FASD, and discuss the forensic uses of adaptive behavior in establishing a mitigation case for people with FASD in criminal proceedings. In the third section, we discuss the role of adaptive behavior in establishing exemption from rigid IQ ceilings in diagnosing Mental Retardation and providing supports and protections for persons with FASD whose IQ scores may be in the borderline or even low normal range.

The meaning and measurement of adaptive behavior

The widespread use of the term adaptive behavior stems from its inclusion in the 1961 classification manual (Heber, 1961) of the American Association on Mental Deficiency (AAMD; later changed to the American Association on Mental Retar-

dation, AAMR; and, recently, to the American Association on Intellectual and Developmental Disability, AAIDD). The idea behind invention of the construct was to reduce the sole reliance on IQ scores when diagnosing mental retardation (a term which was also coined in 1961 to substitute for the no longer acceptable term "mental deficiency"). The driving force behind this development was concern about the over-assignment of the mental retardation label, particularly to minority or low socioeconomic status children and adults. The idea, therefore, was that adaptive behavior would be used to reduce the size of the mental retardation class to those who functioned ineptly both in the classroom (IQ) and outside the classroom (adaptive behavior).

The meaning of adaptive behavior

As is true of many psychological constructs (including intelligence), the term adaptive behavior began to be used in advance of a clear or consensually accepted definition. In such a case, what happens is that measures of the construct are developed, and the construct becomes defined, in circular fashion, by the measure. Thus, there is considerable truth in the humorous definition of intelligence as "that which is measured by IQ tests." The same can be said about adaptive behavior. The first such instrument was the AAMD (later AAMR) Adaptive Behavior Scale (ABS, not to be confused with the more recent ABAS). The ABS tapped two basic areas: activities of daily living, and social functioning.

A major problem with the ABS, and by extension the definition of adaptive behavior in use at that time, was that the social domain was mainly defined by items tapping "maladaptive behavior," i.e., externalizing psychopathology (striking out against self or others) or its absence. A related problem was that the ABS was developed in an institutional setting (Parsons State School, in Kansas) serving mainly lower-functioning individuals, and thus the daily living subscales had items aimed at a fairly primitive level (toileting, dressing, feeding, etc.). Thus, early measures and definitions of adaptive behavior were of limited utility in diagnosing or

specifying the service needs of higher-functioning individuals who, for the most part, were living in less- (or non-) restrictive community settings and who could do many of the things tapped by the ABS even if they had other areas of incompetence that were not tapped by the instrument. Not surprisingly, for a long time adaptive behavior was ignored by clinicians and agencies, a problem that was also attributable to the fact that the authors of the ABS intended the measure to be used descriptively, and thus refused to develop population-based norms.

Around three decades ago, the second author published a number of papers (Greenspan, 1979, 1981) calling for a new approach to defining adaptive behavior and, by extension, to defining mental retardation. Building on a tripartite model of intelligence first formulated by Edward Thorndike (1920), he suggested that IQ tapped the first of the three forms of intelligence (termed abstract intelligence by Thorndike, conceptual intelligence by Greenspan). The other two forms of intelligence, termed mechanical and behavioral intelligence by Thorndike, and practical and social intelligence by Greenspan, could serve as the adaptive behavior piece, and would provide the construct with the theoretical foundation it was then lacking. In this formulation, Mental Retardation would, thus, be defined as a relative absence of three forms of intelligence: conceptual intelligence (IQ), practical intelligence (the kinds of everyday tasks involved in daily living activities), and social intelligence.

This proposal has been influential, in that the current model of adaptive behavior formulated by AAIDD has adopted the tripartite model, but has not gone as far in integrating adaptive behavior with intelligence as was originally intended. A full discussion of this issue is beyond the scope of this article, but can be found in Greenspan (2006). A major concern raised in that and earlier papers has been the lack of sufficient emphasis in various conceptualizations and measures of adaptive behavior on social competence, particularly "social

intelligence" (interpersonal "with-it-ness" or "out-of-it-ness"). Certainly, absence of social intelligence (i.e., presence of bad judgment in dealing with people and social institutions) has been repeatedly noted as a core defining characteristic of people with FASD (Fast & Conroy, 2009; McGee & Riley, 2007; Schonfeld, Paley, Frankel, & O'Connor, 2006). Furthermore, such poor social judgment is especially likely to get someone with FASD into legal trouble, either as a victim or a defendant, in criminal proceedings.

Measurement of adaptive behavior

For at least two decades after its invention (Heber, 1961), adaptive behavior was (to use a legal expression) "honored more in the breach than in the observance." This was due to several factors, including habit (many psychologists continued to believe that IQ alone was sufficient for diagnosing mental retardation), convenience (adaptive behavior assessment, if done properly, requires considerable out-of-office time and effort) and the absence of adequate and norm-based measures. The original idea was to make the IQ criterion (prong one) fairly easy to meet (by setting the ceiling score quite high) and then use prong two (adaptive behavior) to funnel the numbers down. The failure of the field to use adaptive behavior, however, contributed to the decision in the 1973 AAMR manual revision to reduce the IQ cutting score from minus one standard deviation (IQ score of 85, or the 16th percentile) to minus two standard deviations (IQ score of 70, or the 2nd percentile). This had the effect of reducing the class too much, creating a problem of "false negatives" (earlier the concern was mainly about false positives), which is probably the main reason, more than the stated emphasis on instrument error, why the IQ ceiling was raised in the 1992, 2002 and 2010 AAMR/ AAIDD manuals to 75.

Today, formal adaptive behavior measures are much more likely to be used, a development attributable to the publication of more theoretically-grounded norm-based measures and also to the preference by courts in so-called *Atkins* (death

penalty exemption cases involving possible Mental Retardation (MR)) proceedings for prong two data. Technically, a formal measure of adaptive behavior is not a requirement for diagnosing MR in a criminal case. For example, section 1376 (a) of the California Penal Code provides a definition which is typical of those found in other statutes: "'mentally retarded' means the condition of significantly subaverage general intellectual functioning existing concurrently with deficits in adaptive behavior and manifested before the age of 18." There is no specification of how adaptive behavior is to be measured or of what criteria (such as cut-off scores) are to be used to decide whether the prong is met. In theory, therefore, one can produce qualitative/descriptive information to argue that the criterion is met or not met, and that in fact is often what is done. The problem, of course, with using such an approach is that there is a tendency for experts and attorneys on both sides to cherry pick isolated skills that an individual is reputed to be able to do or not do and to then argue for or against a diagnosis of Mental Retardation based on such isolated and nonnormative evidence. Among the many reasons why such an approach is inappropriate, the main one is that people whose intellectual impairments are at the upper, or mild end of the mental retardation spectrum can do many things, including activities (such as working, having sexual partners, driving cars, etc.) that are often wrongly pointed to as evidence that someone cannot have Mental Retardation.

Descriptive information can be useful, however, in gathering information about areas of possible incompetence that are not tapped adequately or at all by items on the adaptive behavior rating instruments described in the next paragraphs. This is particularly important, as noted in the last two AAMR/AAIDD manuals, with regard to gullibility, an area of social adaptive functioning not yet included in existing rating instruments. The importance of gullibility in diagnosing mental retardation (and we would add, FASD) was brought to the attention of the field by the second author (Greenspan, Loughlin, & Black, 2001), although in the 19th Century it was often mentioned

(under the rubric of "credulity") as a core behavioral feature of persons then labeled "mental defectives" (Ireland, 1877).

The most common approach to assessing adaptive behavior is the use of a third-party rating instrument. Currently the three most widely used measures are the Vineland Adaptive Behavior Scale, second edition (VABS-2), the Adaptive Behavior Assessment System, second edition (ABAS-2), and the Scales of Independent Behavior, revised edition (SIB-R). In addition, the AAMR/AAIDD, which in its earlier incarnation as AAMD developed an instrument, the ABS (not to be confused with the ABAS), but later turned it over to an outside publisher, is developing a Diagnostic Adaptive Behavior Scale. This instrument is so-named in order to differentiate it from other measures, which were originally developed mainly to devise individualized service plans, and thus are imperfectly suited to their frequent use in the diagnostic process.

These rating instruments have many items (200 or more), divided up according to various subscales. Raters are asked, either through filling it out themselves or, more typically, through an interview conducted by a trained and qualified professional, to indicate whether the person being rated can always, sometimes or never perform each of the described activities. Raw scores from these ratings are then converted to standard scores from tables which reflect norms gathered on different age cohorts of the general population. These standard scores resemble IQ scores, in that a score of 100 indicates that an individual is at the 50th percentile of the population, while a score of 70 indicates that he or she is at the 2nd percentile. Different forms and norms are available for different categories of raters. For example, the teacher version of the VABS-2 has different daily living activity items than the parent or caregiver version, for the simple reason that teachers generally do not make home visits and, thus, cannot be expected to know if one of their students can cook, clean, operate a washing machine, etc. As a rule, two or more

raters should be used, to correct for the possibility that a given rater's score may be biased or otherwise invalid. Ratings which are in close agreement can, thus, be kept—and used separately or in combination—while highly discrepant ratings (either way too high or way too low, when compared to others') can be confidently discarded.

The various adaptive behavior instruments differ somewhat in terms of items, methods and subscale names. For example, on the ABAS-2 all items are to be administered to an informant, while the VABS-2 (except for the teacher version) uses a difficulty gradation method, in which one establishes a "basal" (giving credit for all items below first occurrence of failure) and a "ceiling" (giving no credit for all items above where failure becomes frequent), thus eliminating the need to administer all items. Although subscales on various measures are differently named, they basically all produce four summary standard scores: Conceptual Adaptive Behavior, Practical Adaptive Behavior, Social Adaptive Behavior, and Composite Adaptive Behavior (summed overall, comparable to Full-Scale). In addition, each of these are divided up into narrower subscales; thus the ABAS-2 has 10 subscales corresponding to the 1992 AAMR adaptive behavior model, and these are then combined into the three broader standard scores (and the overall composite score). Scale scores (standardized scores but with a mean of 10–15 on the VABS-2—rather than 100) can be obtained for these subscales, which is useful in those states whose criminal statutes define mental retardation according to the operational criteria in DSM-IV (2 out of 10 or 11 subscale deficits) rather than AAIDD (1 out of 3 domain deficits) standards.

This latter issue refers to an important point, which is that clinical standards for meeting prong two (and legal standards in those few states with more detailed statutes) do not require global impairment in all areas of adaptive behavior. The explanation given in the 2010 "green book" AAIDD manual is as follows: "a generalized deficit [in adaptive behavior] is

assumed even if the score on only one [of the three adaptive behavior domains] meets the operational criterion of being two standard deviations below the mean" (Schalock et al., 2010, p. 46). Thus, one can qualify as having mental retardation with significant impairment in one or two domains of everyday functioning, while overall impairment (on the composite summary standard score) is not a requirement. Although the green book justification cited above is statistical in nature, there is another reason which may be more important, and that is that for individuals in the higher functioning range of intellectual impairment (where most defendants in criminal proceedings, including those with FASD, are likely to be found), there is typically a mixed pattern of everyday functioning, with significant deficits mixed in with areas of near-normal functioning.

It should be noted that while presence or absence of maladaptive behavior (psychopathology) is no longer used to define or diagnose Mental Retardation, a maladaptive subscale still can be found in some adaptive behavior rating instruments, particularly the VABS-2. This maladaptive subscale is in fact commonly used, along with the other subscales, when diagnosing FASD, as individuals with FASD are very likely to exhibit many maladaptive behaviors, often reflecting inability to say "no" to friends. In fact, it is the continued inclusion of a maladaptive subscale that likely explains the preference among FASD clinicians for the VABS-2, in contrast to mental retardation clinicians who, in our experience, are more likely to use the ABAS-2, which lacks a maladaptive subscale.

Need for improved measures

As noted earlier, a problem with prevailing models and measures of adaptive behavior is that they focus too much on the minutiae of self-care skills (operating a washing machine) and are not sufficiently "cognitive" (understanding the importance of clean clothes), social (understanding their importance for acceptability), and strategic (being able to master the many steps involved in the process besides merely operating the machine). Here is a small sampling of adaptive behavior

rating items, all of them from the ABAS-2, which seem to have little or no connection to cognitive functioning: "controls temper when disagreeing with friends," "has pleasant breath," "offers to lend belongings," "offers assistance to others," "shows positive attitude on job," etc.

Many of these behaviors are performed adequately by cognitively impaired individuals, while we all know intellectually superior individuals who for various reasons are unable or unwilling to do some of these things. Granted, one should not put excess emphasis on isolated items (that is why these measures have many items). Furthermore, people with Mental Retardation do, as a class, demonstrate marked impairment in their levels of adaptive functioning, as reflected in their summary standard scores on instruments such as the ABAS-2. However, the focusing in these instruments on narrow overt behaviors as opposed to underlying cognitive processes—while understandable (the former entities are easier to rate objectively)—is still problematic. That is because they fail to capture the qualities of bad judgment (particularly bad social judgment) which go to the heart of these disorders and which are what puts cognitively- and organically-impaired individuals at social (e.g., legal) and physical risk and in need of services, protections and accommodations.

There is evidence that more cognitively oriented "molar" items can be used reliably and validly in rating instruments. One instrument, used thus far mainly in Australia with elderly individuals, is the Social Vulnerability Scale (SVS) (Pinsker, Stone, Pachana, & Greenspan, 2006). The SVS taps gullibility (which is a big problem in people in early stages of Alzheimer's, as it is, we believe, for people with FASD) and contains items such as "been tricked into giving money or other objects," "been talked into taking the blame for something he did not do," and "easily fooled." It is reported (Novick-Brown, personal communication) that the SVS may hold promise as a diagnostic instrument in assessing the social vulnerability of individuals with FASD.

An example from the FASD literature can be found in the Fetal Alcohol Behavior Scale (FABS) (Streissguth, Bookstein, Barr, Press, & Sampson, 1998). Technically, the FABS is not an adaptive behavior instrument (mainly because it is not labeled as such) but it has many of the qualities of an adaptive behavior rating measure. The FABS is an inventory, which includes a wide range of symptoms that have been found empirically to characterize people with FASD. The importance of the FABS, as a supplement to more general adaptive behavior instruments, is that it captures qualities and symptoms that are specific to FASD. Many of these qualities, such as those reflecting deficiencies in everyday social and practical judgment, are missing from the VABS, ABAS, and other adaptive behavior instruments. More information about the FABS is provided in a later section.

An alternate, but at this point underutilized, assessment approach involves so-called direct adaptive behavior tests. These resemble IQ tests, in that examiners individually present verbal or pictorial stimuli to subjects who are then asked to solve problems posed in the stimuli. Two such instruments are occasionally used: the Street Survival Skills Questionnaire (SSSQ) and the Independent Living Scale (ILS). Both tap mainly the practical adaptive domain, through tasks such as indicating tools to be used for different jobs, selecting correct washing machine settings (both on the SSSQ), and filling out checks to pay bills and using a phone book (both on the ILS).

These two instruments are both in need of norm-updating, and are excessively easy (especially the SSSQ), but the method itself holds promise in our opinion, particularly in getting at the kinds of judgment deficiencies, especially in the social domain, that are central to understanding the everyday vulnerabilities of people with neurodevelomental disorders. One such instrument, currently under development (Greenspan & Woods, 2010), is the Common Sense Questionnaire (CSQ). The CSQ is essentially a problem-solving mea-

sure, in that practical and social dilemmas are presented and subjects are asked to pick or generate a preferred solution. A unique aspect of the CSQ is that characters in the scenarios are depicted leaning towards solutions which have significant risks likely to result from these actions. The subject is asked to indicate whether the proposed course of action is all right or not all right and if the latter, why that might be the case. The instrument is thus intended to determine whether a subject is able to recognize obvious practical or social risk.

The importance of such an approach to adaptive behavior is that a potential for engaging in "foolish" (i.e. risk-unaware) behavior is usually found in people with Mental Retardation and in our view is always found in people with FASD (Greenspan, 2009). Put another way, the instrument determines if an individual lacks "common sense," defined as awareness of obvious risk. The potential utility of the CSQ (not yet normed or ready for distribution) can be found in the following responses from "Roger," a man in his early 20's diagnosed with FASD and facing a possible death sentence.

One story involved a man named Stan who was in a bar when someone mentioned he had a car for sale in good running condition. Stan drove the car home with the understanding he would pay $500 in a few days when the seller signed over the papers. On the way home, Stan noticed the engine was smoking and figured he had been swindled. He decided to abandon the car (which was still running) on the street without telling the seller, as a way of showing the seller that Stan was not someone to be taken advantage of. When asked if this course of action was all right or not all right, Roger responded "all right, if someone does you wrong you need to do him wrong." When asked if he could think of any reason why someone would say Stan's action was not all right, Roger responded "I don't know why anyone would think it is not all right." In contrast, virtually every normally-developing adult interviewed responded along the lines of "not all right, Stan could get arrested if the seller reported it as stolen." Paren-

thetically, this, and most of the CSQ items, were derived from a true story, and the real-life Stan was charged with (and pled to) a felony after the seller reported the value of his missing car as over $1,000. So it is not surprising that Roger was someone who was always getting into trouble with the law, given his dramatic inability to take others' perspectives or to anticipate the potential consequences of his actions.

Clinical, empirical and forensic uses of adaptive behavior with FASD

In the preceding pages we have mentioned Mental Retardation more than FASD, for the simple reason that adaptive behavior was originally invented for use in diagnosing Mental Retardation, and this continues to be the main focus of adaptive behavior scholarship and application. However, the previous discussion does have considerable relevance to FASD, for two reasons: (a) FASD is a major cause of Mental Retardation, and a sizeable subset of people with FASD also qualify for a diagnosis of Mental Retardation, and (b) people with FASD and related neurodevelopmenal disorders, even with full-scale IQ's in the 80's or 90's, function in the world (especially in the social realm) as if they have Mental Retardation, and are often seen by others as having Mental Retardation. It is this "Mental Retardation equivalency" that is at the heart of why people with FASD are entitled to legal protections including *Atkins* (death penalty) immunity. In fact, in terms of their behavioral phenomenology, the main difference between adults with FASD and Mental Retardation and adults with FASD who do not qualify as having Mental Retardation, is their full-scale IQ scores.

In this section, we discuss the role of adaptive behavior measures in both research and clinical applications with FASD-affected individuals, including in forensic settings. FASD is a medical disorder, diagnosed by physicians, using biomedical diagnostic indicators when available. However, because those

biological indictors (such as facial anomalies) are variable and sometimes missing, behavioral indicators such as adaptive behavior data are of great importance. Among the standardized third-party adaptive behavior rating instruments, the Vineland Adaptive Behavior Scale, second edition (VABS-2) is undoubtedly the most widely used, in part because it contains a "maladaptive behavior" scale, in addition to three adaptive functioning domains (Communication, Daily Living, Social) that are comparable to the three domains (Conceptual, Practical, Social) on the ABAS-2 and the AAIDD "green book" (Schalock et al., 2010) diagnostic manual.

The Vineland has long been used as part of the FASD research program led by Ann Streissguth at the University of Washington. In her very earliest studies with young children who have FAS, published in 1973, the original Vineland Social Maturity Scale (the precursor to the VABS) was used. When receiving the Edgar Doll Award from Division 33 (Mental Retardation and Developmental Disabilities) of the American Psychological Association, Professor Streissguth reported that with the early sample of FAS children, maternal ratings from the original Vineland measure (appropriately authored by Edgar Doll) were more powerful indicators of FAS than were the children's IQ scores. Follow-up studies of 61 adolescents and adults who were diagnosed with FAS and FAE also provided strong evidence of the diagnostic importance of adaptive behavior data.

Streissguth and her colleagues looked at all of the psychological measures administered to this sample and asked which ones produced the greatest number of findings that were at least two standard deviations (standard score of 70 or less) below the general population mean on these measures. The VABS won hands down, with the two most important discriminators being the overall "adaptive behavior composite" (71% of the sample below minus two SDs) and the "socialization" subscale (64%). The Vineland's Maladaptive Behavior Index was also a strong indicator, with 62% of the sample

having a "significant" degree of impairment, 38% having intermediate impairment, and zero being nonimpaired. Maladaptive behaviors reflecting impulsivity were particularly common (as is true of individuals with brain damage generally) in this sample, while socialization items indicating insensitivity to social cues were common as well. Streissguth noted, interestingly, that none of the members of her sample qualified technically as having Mental Retardation in terms of their full-scale IQ scores, even though virtually all of them had very significant deficits in adaptive behavior. Streissguth (2006, p. 6) concluded by noting that "it is clear that for patients with FASD, the VABS revealed differences in adaptive functioning that were more profound than the deficits observed in either IQ or Achievement Tests."

Because of the subtle and variable nature of FASD physical symptoms, and the fact that many children and adults (especially with too-high IQs) were consequently not receiving needed educational and social services, Streissguth, Bookstein, Barr, Press, and Sampson (1998) developed a rating instrument intended to "capture the behavioral essence of FAS and FAE, regardless of age, race, sex, or IQ, and thus have utility across various populations and across the life span" (p. 325). The driving force behind the instrument was the observation that individuals with FASD were universally described as exhibiting socially incompetent behaviors not always found in other disorders. Examples of such descriptive comments cited by Streissguth et al. (1998, p. 326) are: "talks a lot but says little," "makes 'off the wall' comments," "overreacts to situations," and "often demands attention or monopolizes a conversation."

The instrument (Streissguth et al., 1998) which emerged from this effort to capture the FASD behavioral phenotype is termed the "Fetal Alcohol Behavior Scale" (FABS). It grew out of research with a large data base of almost 500 subjects, dating from the 1970's. On a subset of 134 subjects with FASD, Sreissguth and colleagues gathered information using

a lengthy "Personal Behaviors Checklist" (PBC) rating instrument filled out by parents or other caregivers. In two derivation studies using factor-analytic methods, the FABS—a more manageable instrument with 36 items—emerged, by taking only those items with high loadings on the first principal component of the PBC.

In a subsequent detection study, the FABS was filled out by prison staff on 81 incarcerated men in a special unit of the Washington state prison system for developmentally disabled and emotionally disturbed (but not psychotic) adult inmates. Information was independently gathered regarding whether the mothers of these inmates had consumed alcohol while pregnant. A normative study was also conducted, involving 186 mothers who were selected at random from a pediatric clinic waiting room.

The detection study found impressive separation on the FABS between the reference sample of almost 500 individuals known to have FASD and the incarcerated adults. While 85% of the FASD sample had FABS scores above 6 or 7 (on an earlier 26-item version of the FABS), 85% of the inmates had FABS scores below 6 or 7. Of the four highest FABS scores in the incarcerated group, three were reportedly born to women who were known to have consumed alcohol during pregnancy. The normative study produced similar results. While about 80% of the reference FASD sample has FABS scores above 11 or 12 (on the later, 36-item version), the mean score among a random sample of children rated by their mothers in a pediatric waiting room was under 7. However, for children born to women in this sample known to have a drinking problem, the FABS scores were in the range expected for FASD children in the reference sample.

In addition to discriminating between individuals with and without FASD, the FABS has also been found to predict degree of disability severity (amount of supports needed) within the FASD population. Adults with FASD living depen-

dently (n = 60) were compared with adults living independently (n = 10) on 36-item FABS ratings obtained several years earlier. The FASD adults living independently had earlier FABS scores at the low end of the FASD range (mean of 8.5), while the adults livingly dependently had FABS scores (mean of 21.5) that were extremely high. Thus, the FABS has been found to be quite valid in identifying individuals with FASD and in predicting later outcomes, in a manner which compares favorably to (and probably exceeds) results from formal measures of adaptive behavior.

An examination of individual item-to-scale rankings for the 36 PBC items selected for inclusion in the FABS is particularly interesting for the purpose of clarifying the precise nature of the FASD behavioral phenotype or "taxon" (a biological term meaning group or family in the classification of organisms). Here are the 13 FABS items most descriptive of people with FASD: "overreacts," "chats, no content," "unusual topics," "demands attention," "unaware of consequences," "incomplete tasks," "inappropriate outside," "likes to talk," "interrupts," "center of attention," "touches frequently," "can't play team," and "can't take hints." In contrast, here are the 13 FABS items that made the 36-item cut, but that were least descriptive of the FASD taxon, going from least to most powerful : "tries hard, but . . . ," "problems in sexual functioning," "poor attention," "mood swings," "noise sensitivity," "loses things," "overly friendly," "loud, unusual voice," "difficulty performing," "poor judgment," "fidgety," "superficial friendships," and "inappropriate home."

While over half of these 26 items involved social incompetence (in contrast to formal adaptive behavior measures), social incompetence items constituted about 80% of FABS items most indicative of the FASD taxon and constituted only about 30% of the items least indicative of the FASD taxon. Furthermore, within the social incompetence items, problems of poor judgment and inadequate perspective taking were involved in almost all of the items. Results from the FABS

development study provide strong evidence, therefore, for the view that FASD is a disorder characterized by social incompetence in general and poor social intelligence and perspective taking in particular. This view of the centrality of social incompetence was supported in a study by Whatley, O'Connor, and Gunderson (2001) who found that while children with FASD are impaired in all areas of adaptive functioning, their relative deficiencies in the "socialization" subscale of the VABS becomes more pronounced over time.

The utility of both the VABS-2 and the FABS in diagnosing FASD, in making a strong case for criminal mitigation, and in illustrating the nature of the FASD taxon, can be seen in a very disturbing case report by the first author (Edwards, 2010). The case involves a woman now 28 years old whom we call "Lisa Jones." Ms. Jones was born three-and-a-half months prematurely to a mother who was known to abuse alcohol and drugs. Lisa went through 27 years of her life, many of these spent in psychiatric settings, before she finally received a diagnosis of FASD a year ago. Lisa had seven foster care placements and 15 psychiatric hospitalizations since the age of four. Over the years, Lisa has been given the following diagnoses: Conduct Disorder, Antisocial Personality Disorder, Bipolar Disorder, Borderline Personality Disorder, Major Depression, Mood Disorder, Oppositional Defiant Disorder, Posttraumatic Stress Disorder, Schizoaffective Disorder, and Separation Anxiety Disorder. We do not dispute the appropriateness of some of these diagnoses (Lisa has serious emotional problems and, as a child, she suffered sexual and other forms of extreme abuse, including being placed in animal cages), but the case illustrates how unfortunately common it is for otherwise qualified mental health clinicians and facilities to fail to diagnose FASD, even when obvious risk factors and overt signs are present.

Among the risk factors and behavioral indicators in this case are the following: (a) Lisa's younger brother was diagnosed with FAS after being removed from the home by a child pro-

tective agency. Studies (Abel, 1988; Spohr, 1996) show that the overall risk for FASD is 170 per 1,000 live births (compared with 2 per 1,000 for the general population) for the older siblings of a person with FASD; (b) Lisa had several early behavioral indicators of FASD, including a sleep disorder (Jan et al., 2010; Landesman-Dwyer, Keller & Streissguth, 1978; Rosett, Snyder, & Sander, 1979; McGee & Riley, 2007), jitteriness or fidgeting (O'Connor & Paley, 2009), and feelings of detachment from others (O'Connor, Kogan, & Findlay, 2002); (c) Lisa was placed in special education classes from first grade on, with a label of severe emotional disturbance.

Like many children with FASD, Lisa had special difficulty in arithmetic calculation (dyscalculia)—that often is associated with very poor social skills (Coles, Kable, & Taddeo, 2009; Koopera-Frye, Dehaene, & Streissguth, 1996; Streissguth, Barr, & Sampson, 1990), (d) while possessing relatively normal full-scale intelligence, Lisa has many areas of specific neuropsychological deficit, including sporadic memory problems, cause-effect reasoning impairment, poor planning, impulse control, and deficient organizational skills (Carmichael Olson, Feldman, Streissguth, Sampson, & Bookstein, 1998; Mattson, Goodman, Caine, Delis, & Riley 1999; Fast & Conry, 2009; Schonfeld et al., 2006), in addition to problems of poor motor coordination and signs of poor executive functioning, including inability to track visual information (Mattson, Riley, Gramling, Delis, & Jones, 1998), develop and maintain appropriate problem-solving strategies, and solve problems in a timely manner. Her levels of executive function deficit are much more severe than would be expected based on her level of overall intellectual functioning but very much in line with the picture of someone with significant brain damage; (e) Lisa talks incessantly, but does not often make sense. This is congruent with the finding (Carmichael Olson, 1994; Burgess, Lasswell, & Streissguth, 1992) that individuals with FASD have a "discrepancy between their relatively good verbal skills and their poor ability to effectively communicate and function in social situa-

tions"; (f) Lisa has great difficulty in handling complexities of peer interaction (Streissguth et al., 2004), interpreting social clues (Coggins, Friet, & Morgan, 1997; Carmichael Olson, 1994) and conforming to social norms. Inept social behavior is in fact a hallmark of FASD and of other neuro-developmental disorders (Greenspan, 2009; Greenspan & Love, 1997); (g) Lisa has a long history of depression, self-injury, and self-loathing, including at least five suicide attempts, starting in childhood. Because of their history of failure and rejection, individuals with FASD have an elevated risk of depression and suicide (Huggins, Grant, O'Malley, & Streissguth, 2008), something that is also characteristic of individuals with "non-verbal learning disabilities," a neurodevelopmental impairment sharing many qualities with FASD (Rourke, Young; & Leenaars, 1989).

A major episode of depression occurred at age 18 after Lisa learned that her foster parents, the only people she has ever loved, decided against adopting her. When stressed, Lisa will regress, drinking out of a baby bottle, curling up and licking herself like an animal, barking at people and eating off the floor. Starting at age 14, she has experienced hallucinations. Lisa has been in many residential placements, typically running away from them or being expelled. Lisa has been receiving SSI payments for mental illness, but no longer has a conservator, causing her to be sometimes homeless, and involved in prostitution and drug use. While these behaviors and symptoms are by no means specific to FASD, they do have a higher likelihood of occurring in individuals with prenatal alcohol exposure (O'Connor & Kasari, 2000; Fryer, McGee, & Matt, 2007).

Lisa has been involved in at least two criminal incidents involving group home placements, one in which she got into an altercation and then attacked a responding police officer with a knife, and more recently, at age 22, when she set fire to an occupied group home from which she had earlier been expelled. After the last incident, Lisa was arrested and charged with arson. Without ever investigating the possibility

of FASD, a court-appointed attorney advised Lisa to take a three-year sentence in state prison. At age 23, while in prison, Lisa made her most severe suicide attempt. After several weeks in the hospital recovering from her self-inflicted injuries, Lisa was returned to prison where she was classified as a "mentally disordered offender" and transferred to a correctional hospital facility. Neither in the correctional system, nor in her many earlier psychiatric placements, was Lisa ever evaluated for the possibility of having FASD or related brain damage. This changed recently, when Lisa's current attorney had her evaluated by a competent psychiatrist who is extremely knowledgeable about the disorder.

An effort to obtain services for Lisa through her state's Department of Developmental Services is being blocked for the usual reason (to be discussed in the final section): too high full-scale IQ score. Over the years, Lisa has had many IQ tests administered to her and these fall generally in the dull-normal range (scores have fluctuated from low 80's to high 90's, with most recent testing approaching 100), but with some scatter (impairment or low average performance on subtests tapping number-symbol translation, arithmetic, short term attention, pattern recognition, and arranging images to create a story). There is also evidence of limitations in working memory (Burden, Jacobson, Sokol, & Jacobson, 2005).

In line with the earlier discussion, useful diagnostic information about Lisa was obtained from both the FABS and the VABS. The FABS was filled out by two raters: Lisa's foster mother and the social worker at her current residential placement. Lisa received scores at the extreme ceiling of the FABS, with virtually all items endorsed as applying to her. Here is the descriptive picture, highly indicative of FASD, which emerged from these combined (and highly congruent) ratings: highly impulsive, often acting before considering consequences; poor judgment in whom she trusts, overly friendly with strangers, and too easily lead by others (all three indicating gullibility); cannot take a hint and needs

strong, clear commands because the fine points escape her; unawareness of the consequences of her behavior, particularly the social consequences; establishes superficial friendships easily but has no close friends in her own age group; very hyperactive and always on the go, runs around a lot; has unusual conversational topics; dwells on one or two particular subjects or speaks about unrealistic topics; often overreacts to things; is fearless and lacks foresight into potential danger. A fuller and more accurate statement of the typical behavioral profile of someone with FASD, with much of it describing foolish (risk-unaware) and impulsive social behavior, would be difficult to provide.

On the VABS-2, with Lisa's foster sister as an informant, Ms. Jones obtained the following standard scores: Communication (conceptual adaptive skills) score of 52, Daily Living (practical adaptive skill) score of 57, Socialization (social adaptive skill) score of 40, and overall Adaptive Behavior Composite score of 49. These are all in the range of mild or moderate Mental Retardation, and provide a much more valid picture of Lisa's disabled level of everyday functioning (as reflected for example in her inability to ever hold a job) than can be obtained from her IQ scores. It is for this reason that several American states and Canadian provinces have moved towards loosening IQ ceilings in FASD and other organic brain disorders where there is very clear evidence of adaptive behavior deficits providing a picture of someone with "Mental Retardation equivalence." This suggestion is fleshed out in the following section.

Role of adaptive behavior in giving FASD cases intellectual disability status automatically

The just-discussed case of Lisa Jones illustrates the serious problems flowing from the current practice of giving prong one (IQ score) a primary role, and prong two (adaptive behavior) a secondary role, when determining eligibility for services and legal protections flowing from application of the Mental Retardation or intellectual disability label. In the case of Ms. Jones, granting of intellectual disability (as opposed to mental illness) status would likely have resulted in an alternative or mitigated sentence more suitable for someone with

her brain-based developmental limitations. It likely would also make her today eligible for a more appropriate range of residential and other supportive service options. If Lisa's arson attempt had been more successful and someone had died in the resulting fire, then the consequence of granting or denying her such status would have been even more serious, in that it would have determined whether or not the state could seek the death penalty.

Our concern about the rigid reliance on prong one to deny services and legal protections to individuals like Lisa Jones is not, however, driven by a blind feeling that anyone seeking developmental services eligibility should be granted it. Rather, it is driven by a concern that eligibility decisions should be fair and just. In the case of Ms. Jones, denial of intellectual disability status seems grossly unfair, in that she exhibits all (actually, more) of the adaptive behavior limitations and related support needs that are found in people who typically are ruled eligible. Furthermore, Lisa's brain-based impairments are clearly developmental in origin. Cases such as Lisa's are increasingly resulting in class action lawsuits, such as recently in British Columbia. These have caused some developmental service agencies to take measures to relax their eligibility criteria, especially when FASD or related neurodevelopmental disorders are medically established. This can be, and has been, done in one of two ways, and in both of them adaptive behavior plays a central role: (a) raise the IQ ceiling (in British Columbia to minus one standard deviation) for individuals with FASD and significant adaptive impairments or (b) rely primarily on adaptive behavior deficit when FASD or related organic disorders are present (this has been done in Colorado, Minnesota, and a few other places).

The idea here is to provide a vehicle for "intellectual disability equivalency" for individuals with neurodevelopmental disorders who exhibit all of the adaptive behavior deficits associated with intellectual disability but whose IQ scores are above the minus two standard deviations cut-off. This can be

seen in language in Washington state's developmental services eligibility law, which after listing various categories, such as "intellectual disability" (which it substituted for the former term "Mental Retardation") has this escape clause for persons such as Lisa Jones: "or another neurological or other condition closely related to intellectual disability or that requires treatment similar to that required for individuals with intellectual disabilities." If the state in which Lisa resides had passed a law as enlightened as Washington's, it is difficult to imagine that she (or others with FASD and a similar adaptive behavior profile) would be turned down for services.

The trend towards increased emphasis on prong two in civil eligibility procedures for individuals with FASD can also be found, to a lesser extent, in criminal matters. For example, the California Supreme Court, *In re Hawthorne* 35 Cal. 4th 40 (2005) refused to establish specific IQ cutoffs for *Atkins* death penalty exemption hearings. This policy was upheld again by the California Supreme Court in *People v. Superior Court* (Vidal) 40 Cal. 4th 999 (2007). In that case a Tulare County judge's *Atkins* finding that Jorge Junior Vidal had Mental Retardation, was made in spite of a full-scale IQ score well above the usual clinical cutoff. The judge's reasoning was that full-scale IQ is not an appropriate prong one standard to apply to Vidal, a man with a medical diagnosis of FASD and with severe adaptive deficits, including extreme gullibility. Instead, the judge found that Vidal's very low verbal IQ score (used to explain his gullibility) was a better measure of prong one deficit in his case. This ruling, in fact, was in line with a statement in the Mental Retardation section in DSM IV-TR to the effect that when there are severe verbal-performance splits, as is often true in brain-damaged individuals, full-scale IQ may be an unreliable index of actual intellectual functioning.

Similar reasoning was used by a Federal judge in 2008, in ordering a reconsideration of the *Atkins* claim of Yokamon Hearn, a condemned Texas man with FASD and very substantial prong two deficits. The original denial of *Atkins* relief

was based on Hearn's too-high full-scale IQ scores (those scores, including a sizable verbal-performance split, were almost identical to Vidal's). In making his ruling, the judge cited an affidavit by the second author, in which the argument was made that for adaptive behavior-impaired individuals with FASD or related neurodevelopmental disorders, the optimal clinical practice is to rely on executive functioning or other non-IQ indices of intellectual impairment.

On a final note, we should point out that the shift from use of the term "Mental Retardation" to the term "intellectual disabilities" offers the possibility of increasing the importance of adaptive behavior in bringing people with FASD under the diagnostic umbrella. That is because there is such a strong semantic association of Mental Retardation with IQ, while the new term intellectual disabilities does not yet have so strong an association. Unfortunately, the AAIDD in its 2010 "green book" did not capitalize on the name change to broaden the diagnostic umbrella. It is our understanding, however, that the committees writing the Intellectual Disability sections of the forthcoming DSM-V and ICD-11 manuals are considering a dual pathway diagnostic approach, with the second path giving more emphasis to adaptive behavior deficits and less to IQ score ceilings for individuals with FASD and related brain-based disorders. Such an approach would, in our view, be more in line with the well-established reality that for people with FASD, adaptive behavior provides the best indication of why these individuals need and deserve legal and other protections.

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A proposed model standard for forensic assessment of Fetal Alcohol Spectrum Disorders

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A model protocol is proposed for multidisciplinary assessment of Fetal Alcohol Spectrum Disorders (FASD) in the forensic context. Used effectively on both sides of the courtroom in the United States in criminal and post-conviction matters in state and federal courts, the model relies on the FASD literature and best-practice standards of care in terms of clinical as well as forensic evaluation. It is suggested that FASD diagnostic criteria for older adolescents and adults in the criminal system may need different emphasis if facial features have diminished with age and confirmation of prenatal exposure is impossible.

KEY WORDS: *FASD, multidisiplinary assessment.*

It has been known for many years that persons with Fetal Alcohol Spectrum Disorders (FASD) are at high risk of criminal behavior. According to criminal offender statistics maintained by the Department of Justice (Criminal Records

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Nationwide Criminal Search, 2008), the lifetime risk of going to state or federal prison in the United States is 3.4% (18.6% for blacks and 10% for Hispanics). While comparable statistics have never been compiled for individuals with FASD, a large government-sponsored study at the University of Washington in the mid-1990s found that approximately 60% of children, adolescents, and adults in this population had histories involving arrest, conviction and/or incarceration (Streissguth, Barr, Kogan, & Bookstein, 1996). Approximately 45% of these crimes involved crimes against persons, including assaults (17%) and domestic violence (15%). Since that historic study, only one comparative analysis has ever examined the prevalence of FASD within a discrete criminal justice population. In that analysis, all 287 youth remanded during a one-year period to a psychiatric facility in British Columbia were evaluated, and researchers (Fast, Conry, & Loock, 1999) found that 23.3% had FASD. Only 1% of those with FASD received a diagnosis of Fetal Alcohol Syndrome (FAS), and the rest were diagnosed with Alcohol Related Neurodevelopmental Disorder (ARND). Both conditions are included under the FASD umbrella and involve brain damage and associated neurocognitive dysfunction due to prenatal alcohol exposure.

Combined, the implications of the British Columbia study and the earlier University of Washington study are alarming. If, according to the Department of Justice, over 2 million adults are now incarcerated in U.S. prisons and jails (Leary, 2009), plus approximately 100,000 youth held in juvenile facilities (Sickmund, 2010), what is the significance of these rates with respect to juvenile and adult offenders who may be brain damaged due to FASD? Most importantly, what do these figures mean with respect to the 3261 men and women now on death row in the United States (Fins, 2010)? Acknowledging the extensive Central Nervous System (CNS) damage in this population that produces a constellation of neurocognitive deficits in memory, attention, language, motor skills, social skills, academic achievement, cognition, and

"executive control" difficulties in planning, organizing, learning from past mistakes, linking cause and effect, anticipating consequences, and taking the perspective of others, Moore and Green (2004) note:

> This constellation of deficits poses significant obstacles to the fair treatment of FASD persons in the criminal justice system. Persons with FASD, as a group, challenge the underlying premise that defendants understand the relationship between actions, outcomes, intentions, and punishment. The treatment of FASD defendants raises fundamental questions about how we assess individual responsibility, both at the guilt-determining and sentencing stages of the adjudicative process. (p. 3)

The Substance Abuse and Mental Health Services Administration Fetal Alcohol Spectrum Disorders Center for Excellence (SAMHSA; 2007, para. 6) agrees that FASD merits special legal consideration: "Individuals with FASD typically are impulsive and have trouble foreseeing the consequences of their actions. . . . [This] presents challenges throughout the judicial process—from questioning through arrest, hearings, sentencing, and detention." U.S. Courts are beginning to recognize that FASD deserves special consideration. For example, in *Dillbeck v. State*, 643 So.2d 1027 [Fla. 1994], the Court made a point that FASD merited consideration in both the guilt-innocence phase and mitigation or sentencing phase:

> Evidence concerning certain alcohol-related conditions has long been admissible during the guilt phase of criminal proceedings to show lack of specific intent . . . (I)f evidence of a self-induced condition such as voluntary intoxication is admissible, then so too should be evidence of other commonly understood conditions that are beyond one's control, such as epilepsy . . . Just as the harmful effect of alcohol on the mature brain of an adult imbiber is a matter within the common understanding, so too is the detrimental effect of this intoxicant on the delicate, evolving brain of a fetus held in utero. As with 'epilepsy, infancy or senility,' . . . we can envision few things more certainly beyond one's control than the drinking habits of a parent prior to one's birth. We perceive no significant legal distinction between the condition of epilepsy . . . and that of alcohol-related brain damage in issue here—both are specific, commonly recognized conditions that are beyond one's control.

It is important to note that FASD is relevant on both sides of the courtroom. Not only should defense teams explore the possibility of FASD in their cases if they suspect a defendant may have been exposed to alcohol in utero, but prosecutors as well may want to present evidence of victim vulnerability to the Court. In our own experience working as a multidisciplinary FASD assessment team with state and federal prosecutors, enhanced sentencing has been obtained for victims determined in forensic evaluation to have FASD. In one such case, an adult female who was raped by an adult male relative was shown to have FASD, which resulted in an upward departure in the defendant's sentence.

While there appears to be growing recognition that FASD is relevant in the criminal justice system, review of over 100 cases on the Legal Issues Resource Center website (http://depts.washington.edu/fadu/legalissues) maintained by the Fetal Alcohol and Drug Unit at the University of Washington indicates that FASD has been raised as a relevant defense issue in only slightly over 100 cases in the United States over the past 20 years. Given the disproportionately large number of defendants likely to have FASD, this small number suggests that many defendants are convicted each year without awareness by anyone that they have brain damage. Moreover, analysis of the case law that has been summarized on this website reveals an imperfect understanding of the diagnostic process, symptoms, and behavioral consequences of FASD (Wartnik, in press), which understandably affects outcome. There are many reasons for this misunderstanding, not the least of which is the complex nature of FASD and associated difficulty in adequately explaining diagnosis to the court in a way that makes the science understandable and case relevant. Other problems include lack of identification by legal teams that a case may involve FASD or difficulty in tracking down information on the prenatal exposure history of defendants (or alleged victims, for that matter). Other misconceptions that complicate identification and eventual forensic assessment include:

- persons with IQs in or near the average range couldn't have FASD and/or couldn't have neurocognitive deficits that cause them to function at levels similar to those with intellectual disabilities;
- superficially "good" verbal skills preclude FASD;
- the absence of facial abnormalities either rules out FASD or indicates that functional deficits cannot be severe;
- if a birth mother denies drinking and drug use during pregnancy, her denial should be taken at face value;
- a complete absence of maternal drinking evidence after extensive investigation precludes FASD (i.e., in the presence of all the facial features, growth deficit, and CNS abnormalities, FAS can be diagnosed even without confirmation of maternal drinking);
- acute substance intoxication and/or intentional antisocial conduct fully "explain" offense behavior in someone known to have been exposed prenatally to alcohol; or
- basic ability to "plan" pursuit of an offense objective is equivalent to the more complex cognitive process of determining an objective, reflecting on anything that might be relevant to pursuit of the objective (e.g., generalizing past mistakes that are similar to the present objective, foreseeing consequences, considering impacts on self and others, appreciating the implications of the act), and being able to stop oneself from acting on impulse.

In an effort to address some of these misconceptions and problems associated with bringing FASD into the courtroom, a coalition of mental health professionals associated with the Fetal Alcohol and Drug Unit at the University of Washington formed in 2007 to conduct forensic assessments. Calling ourselves "FASD Experts," our goal was to develop a protocol for forensic FASD assessment that could be replicated by other forensic groups so as to expand awareness of this significant medical condition within the forensic context and increase diagnostic accessibility. What follows in the balance of this article is a description of that protocol.

Multidisciplinary assessment

Since FASD is a medical condition, diagnosis must be made by a medical doctor. However, current diagnostic guidelines require functional as well as physical assessment. Consequently, other specialists also are required as noted in diagnostic guidelines published by the Centers for Disease Control (Bertrand et al., 2004, p. 3): FAS diagnosis (is) confirmed using dysmorphic and anthropometric assessment procedures along with appropriate neurodevelopmental data.

For almost 40 years, diagnosing a condition due to prenatal alcohol exposure has involved three primary criteria plus "confirmation" that the exposure actually occurred. Solid evidence of prenatal alcohol exposure may be difficult if not impossible to achieve in forensic situations involving older defendants with few living relatives (including birth mother) and incomplete records. In cases such as this, the presence of typical neurocognitive abnormalities without the standard clinical phenotype may be sufficient for an experienced diagnostician to make a case for FASD, especially if some of the facial abnormalities are still present and the birth mother was known to have abused alcohol at some point in life. In such cases, family history as well as pregnancy and birth records is critical, as well as good documentation about early infant and child development.

The primary FASD diagnostic criteria are: (a) three specific facial abnormalities (i.e., short palpebral fissures or eye openings, flat philtrum or groove below the nose, and small upper lip circumference); (b) prenatal or postnatal growth deficit (i.e., significant deficit in height and/or weight); and (c) Central Nervous System (CNS) damage. Medical doctors are responsible for assessing the physical manifestations of FASD (face and growth deficits). CNS damage is investigated in three ways: structural, neurological, or functional assessment. Of these, the generally accepted approach in clinical FASD diagnosis is functional assessment, which requires

cognitive-behavioral assessment by specialists (usually psychologists). Structural and neurological assessment usually falls within the domain of medical doctors. Functional assessment, on the other hand, may involve several specialists. For children, multidisciplinary teams may consist of social workers or RNs who handle case management, educators, speech and language professionals, and/or occupational and physical therapy technicians. For older adolescents and adults who are beyond the school years, mental health specialists conduct neuropsychological assessment and investigate the lifelong impact of prenatal exposure on functional capacity, including adaptive functioning.

There is no reason why FASD assessment in the legal context shouldn't model the standard of care in the clinical context, which means multidisciplinary team assessment including a trained medical doctor and psychologist at a minimum. There should be at least one forensically trained person on the multidisciplinary team who can explain to the Court how the neurocognitive deficits in FASD produce Cognitive Disorder Not Otherwise Specified, which in mental health diagnostic terminology is equivalent to the legal concept of "mental defect." Beyond the diagnosis itself, there also is the challenge of explaining how FASD affected a defendant's lifelong functioning and, in particular, his or her instant offense conduct. Explaining the relevance of FASD to the forensic issue at hand requires an expert with forensic expertise as well as expertise in FASD.

The central role of executive functioning

Much of the misunderstanding about the relevance of FASD in the criminal context, including how it affects criminal conduct, stems from a common misconception that individuals with FASD who have average or low average IQs also have corresponding functional capacity. However, while most persons with FASD have IQs in the average to borderline ranges

(Streissguth et al., 2004), they tend to function adaptively like individuals who are intellectually disabled. This is due in large part to the impact of prenatal alcohol exposure on frontal lobe development where executive functioning is controlled. Most individuals diagnosed with FASD have deficits in executive functioning (Connor et al., 2000; Burden et al., 2009; Vaurio, Riley, & Mattson, 2008; Fryer et al., 2007; Mattson, Calarco, & Lang, 2006). Executive functioning involves a set of higher order cognitive skills necessary for adaptive self-regulation. These skills include planning (while also considering consequences and lessons learned from previous mistakes), sensory processing (accurately perceiving and efficiently processing environmental information), decision making (weighing alternatives and choosing the most appropriate option while taking into consideration potential outcomes), and response inhibition (controlling behavioral impulses that are likely to result in negative outcomes). These executive skills are not only involved in the reflection process required for "deliberate" and "intentional" criminal conduct. Response inhibition is necessary for the final step in this cognitive process once reflection is completed: stopping oneself from engaging in a criminal act. There is a robust finding in the literature that among all the executive function deficits associated with prenatal alcohol exposure, most individuals with FASD have significant impulse control problems (Burden et al., 2009; Mattson et al., 2006).

In the criminal context, FASD-associated problems in reflection, forming intent, and carrying out effective goal-directed behavior are directly relevant to mental state. While this population tends to function adequately in familiar, highly structured situations (such as IQ test settings), their behavior often breaks down or decompensates in novel high-stress situations due to their multiple executive function limitations (Streissguth, 1997; Streissguth et al., 2004), which establish a biological ceiling on their capacity to self-regulate. This decompensation often leads to instinctive fight or flight reac-

tions: either fleeing from stressful confrontation or using excessive force to gain a primary objective. Although individuals with FASD are capable of simple planning (including offense behavior), they typically think only of the goal with a narrow, single-minded focus, neglecting previous learning experiences, consequences, and impact on themselves and others. This executive deficit in cause-and-effect thinking is observed in most individuals with FASD (Streissguth et al., 1991) because they have difficulty holding disparate bits of information in working memory and taking this information into account during the reflection process. Because this population also tends to lack the executive ability to monitor and appreciate where their behavior is leading them, perseveration is common. Perseveration used in this context refers to the inability to change course midstream, despite obvious negative consequences.

FASD-associated CNS deficits also affect behavior once individuals with FASD are in custody, which affects all aspects of the legal process. Because of their lack of cognitive sophistication, persons with FASD tend to be suggestible, prone to waiving their rights, and inclined to acquiesce with authority figures—all of which can affect the accuracy of any statements they might make. Rational understanding of the legal process and ability to assist counsel in defense also may be impaired. However, once individuals with FASD are incarcerated awaiting trial or in prison following sentencing, they typically do not present a significant risk of future dangerousness regardless of their instant offense because they tend to function adequately in highly controlled, supervised environments that reduce the need for personal decision making (SAMHSA, 2006).

Screening by the legal team

The first step in forensic FASD assessment is identification by a legal team that a client (or victim) may have been prenatally exposed to alcohol. For instance, suspicions might be

raised if it is known that a birth mother abused drugs or that a client was in Special Education during childhood. To date, only four empirically developed screening tools have been reported in the peer-reviewed literature (Astley & Clarren, 1996; Mutsvangwa et al., 2009; Burd et al., 1999; Streissguth, Bookstein, Barr, Press, & Sampson, 1998), but none of these tools apply to the forensic context. The FASD Youth Justice Project in Manitoba uses a brief tool to screen for possible FASD in youth 12 to 18 years of age who have confirmed prenatal alcohol exposure. Items on the tool include repeated failure to comply, lack of empathy, poor school experiences, difficulties within institutions (i.e., compliance, peers, academics), inability to connect actions with consequences, being unaffected by punishment, taking a follower rather than leader role in crime, and engaging in risky crimes with little potential gain. While screening with this tool was reportedly effective (Goh et al., 2008), with 50/178 individuals receiving diagnostic assessment and 29 of those 50 ultimately receiving an ARND diagnosis and 1 receiving an FAS diagnosis, the measure is not in general use outside the Manitoba province.

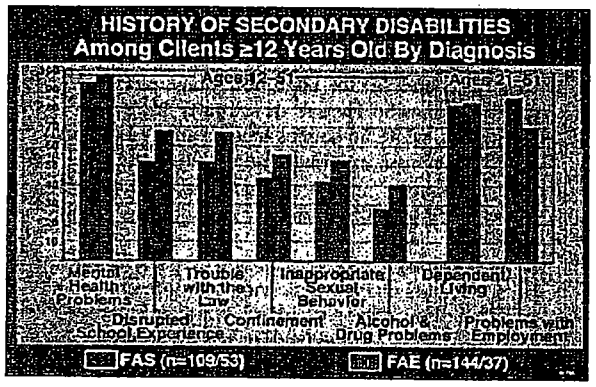
In British Columbia, the Asante Center is working with local probation officers to develop a screening tool and referral process for juveniles involved in the criminal justice system. Although the tool appears to be quite promising because items are anchored in the FASD literature and may have general forensic applicability, it is still in the developmental stage and has not yet been published. In lieu of a published forensic screening tool, FASD Experts suggests an informal checklist (Appendix A) that contains empirically validated factors known to be associated with FASD. There is no magic number of endorsed items on this checklist that guarantees a diagnosis of FASD. However, if several items are endorsed in each category, referral for a forensic FASD assessment is recommended—especially if the legal team has information suggesting the possibility of prenatal alcohol and/or drug exposure.

Adapting the clinical protocol to the forensic context

Beyond the importance of using a diagnostic protocol that is consistent with the clinical standard of care, several other factors are necessary in a forensic context, such as grounding assessment procedures in the FASD science. Toward that end, given the large amount of information now known about the neurocognitive deficits and behavior in persons with FASD, forensic assessment can incorporate best practice in scientific investigation: examining whether the individual's history matches a priori hypotheses about life course expectations in those with FASD. If a particular defendant (or victim) has FASD, then certain life course outcomes are expected if he or she has been exposed to adverse childhood experiences but has never received appropriate interventions. If life course history is not consistent with what is observed in this population, such a finding would tend to undermine the forensic hypothesis that the person has FASD. The chart in Exhibit 1 below depicts several negative life course outcomes that have been identified in individuals with FASD:

EXHIBIT 1

Adapted from Streissguth et al. (1996)



Because of the detailed findings in this large study (Streissguth, Barr, Kogan, & Bookstein, 1996), there can even be a priori hypotheses regarding the nature of criminal conduct in this population. For example, as the above chart depicts, while persons with FASD are at high risk of having trouble with the law, those at highest risk are not those with FAS but rather those with Fetal Alcohol Effects or FAE, which in today's terminology would include Partial FAS and ARND. With regard to the exact nature of the criminal conduct seen in this population, shoplifting is often the first documented crime (Streissguth et al., 2004). The most frequently described law violations were crimes against persons (45%), which included shoplifting/theft (36%), burglary (15%), assault (17%), and domestic violence (15%). Among adolescents and adults with histories of inappropriate sexual behavior, 18% had displayed sexual aggression. Other crimes included running away, property damage, and vehicular crimes. Trouble with the law began at a mean age of 12.8 years.

The high percentage of theft in the FASD population is likely due to executive function deficits (e.g., boundary confusion, poor judgment, impulse control problems, and difficulty learning from experience and foreseeing consequences) that affect self-regulation with respect to objects and personal space. With respect to the latter, executive deficits also affect the way individuals with FASD handle sexual urges, which accounts for the large percentage of individuals in this population who engage in sexually inappropriate behaviors. In fact, while no studies have examined this issue in adults who have been detained and/or committed as "sexually violent predators," it is likely that many of these individuals aren't so much sexually deviant or antisocial as brain damaged.

Although a large number of persons with FASD commit assaults and domestic violence, it is unknown how many commit homicide. As with other crimes committed by persons with FASD, executive function deficits typically worsen in unstructured, high stress situations and are likely central to such

crimes. For example, relevant deficits may involve the misperception of environmental stimuli (typically, visual and auditory stimuli), problematic integration and interpretation of that information, failure to modulate emotional responses, and ultimately, lack of impulse control. Of course, this entire flawed information processing/control process typically takes place in a split second, which adds two additional elements to the equation for individuals with FASD who also have delayed processing speed paired with an overly rapid response tendency (Burden et al., 2009; Burden, Jacobson, & Jacobson, 2005). The more of these information processing and control processes that are impaired, the more likely there will be a catastrophic outcome. Of over 40 cases assessed by FASD Experts from 2007 to the present, almost half have involved individuals either in the trial stage or post-conviction appeal stage for capital murder. Nearly all of these individuals displayed significant neuropsychological deficits in executive functioning—especially in unstructured versus structured contexts.

Another important factor in the forensic application of FASD is the importance of using procedures that meet evidentiary requirements as case law requires a showing that expert testimony is admissible as evidence. The two most significant tests in this regard are the *Daubert* and *Frye* rulings that deal with the scientific reliability of an expert's testimony. Therefore, testing within the forensic context should involve standardized measures that have been documented as reliable. Another important aspect of sound forensic investigation within the mental health sector is convergent evidence from multiple sources for legally relevant conclusions. The FASD assessment protocol itself should be transparent as well, with general consistency and standardization from case to case.

Diagnostic guidelines

There are three diagnostic protocols commonly used in the United States: Institute of Medicine (IOM) (Stratton, Howe,

& Battaglia, 1996), 4-Digit Code (Astley, 2004; Astley & Clarren, 1997, 1999), and the Centers for Disease Control (CDC) (Bertrand et al., 2004). Clinical providers in Canada often use the 4-Digit Code along with their own diagnostic protocol (Chudley et al., 2005), which is similar in many respects to the CDC protocol in the United States. All of these diagnostic protocols are based on multidisciplinary assessment, and all require reliable evidence of facial, growth, and CNS abnormalities. However, confirmation of prenatal alcohol exposure is handled somewhat differently. For example, IOM criteria (Stratton et al., 1996) describe significant alcohol exposure as a pattern of excessive intake characterized by substantial, regular intake or conversely, heavy episodic drinking. Evidence of such a pattern might include frequent episodes of intoxication, development of tolerance or withdrawal, drinking-related social or legal problems, engaging in physically hazardous behavior while drinking, or alcohol-related medical problems such as hepatic disease.

The 4-Digit Code (Astley, 2004; Astley & Clarren, 1997, 1999) defines four levels of prenatal alcohol exposure. Rank 4 is equivalent to confirmed exposure to high levels of alcohol; Rank 3 involves confirmed exposure at a level less than Rank 4, or the level is unknown; Rank 2 is assigned if exposure is unknown (neither confirmed absent nor confirmed present); and Rank 1 indicates confirmed absence of exposure from conception to birth. High exposure is defined generally as alcohol equivalent to six to eight beers consumed weekly, early in pregnancy, by a woman weighing 55 kg. or approximately 120 pounds. Astley (2004) provides examples of these rankings. For instance, Rank 4 exposure would be assigned to a birth mother who reports drinking to the point of intoxication weekly throughout pregnancy. Rank 3 would include a birth mother observed to be drinking an unknown amount during pregnancy or a birth mother who reported drinking a glass of wine weekly but stopped drinking at three months gestation when she learned she was pregnant. Rank 2 exposure, which involves unknown exposure history, might

include adopted individuals for whom no relevant records exist or individuals whose mothers were known to have a drinking problem, but there are no records or direct observation of her drinking during the index pregnancy. Rank 1 would include a birth mother who never drinks or planned pregnancies where the birth mother was intentionally abstinent during the entire time she was trying to conceive.

The CDC guidelines state:

- FAS with confirmed prenatal alcohol exposure requires documentation of the alcohol consumption patterns of the birth mother during the index pregnancy on the basis of clinical observation; self-reports; reports of heavy alcohol use during pregnancy by a reliable informant; medical records documenting positive blood alcohol levels or alcohol treatment; or other social, legal, or medical problems related to drinking during the index pregnancy.
- FAS with unknown prenatal alcohol exposure indicates neither a confirmed presence nor a confirmed absence of exposure. Examples include situations in which the child is adopted, and any prenatal exposure is unknown; the birth mother is an alcoholic, but confirmed evidence of exposure during pregnancy does not exist; or conflicting reports regarding exposure exist that cannot be reliably resolved. (CDC, 2005, p. 3)

The Canadian diagnostic guidelines define prenatal exposure as follows:

Prenatal alcohol exposure requires confirmation of alcohol consumption by the mother during the index pregnancy based on reliable clinical observation, self-report, reports by a reliable source or medical records documenting positive blood alcohol, alcohol treatment, or other social, legal or medical problems related to drinking during the pregnancy. The number and type(s) of alcoholic beverages consumed (dose), the pattern of drinking and the frequency of drinking should all be documented if available" (Chudley et al., 2005, p. S11).

The Canadian guidelines (Chudley et al., 2005) also recommend recording of other factors that may interact with neurocognitive deficits and affect functioning, such as co-occurring disorders and psychosocial stressors and prenatal exposure to other substances (e.g., smoking, licit or illicit drugs).

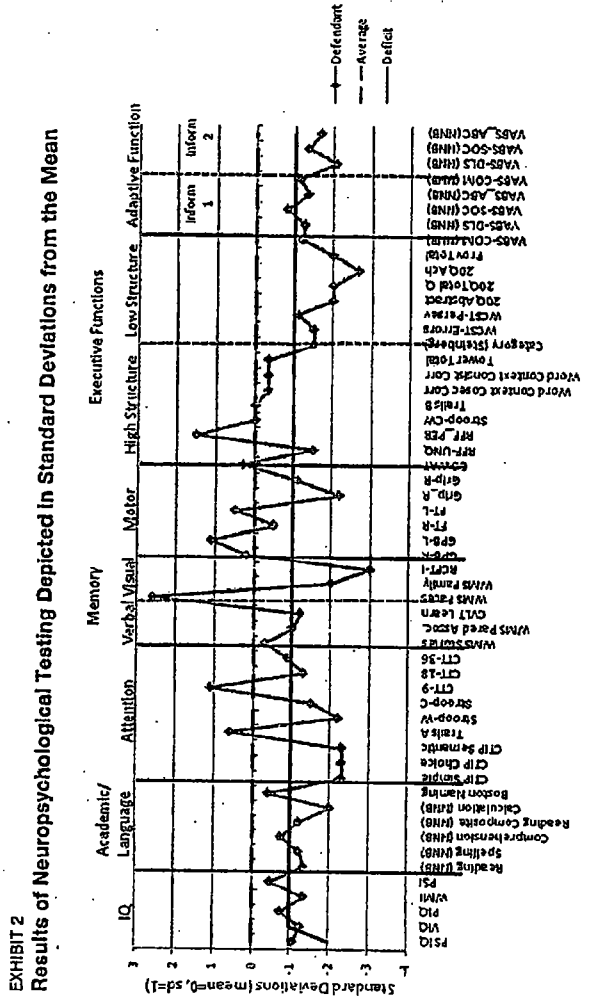
Multidisciplinary assessment process

Once a referral has been made to a forensic diagnostic team, we recommend that the assessment process begin with neuropsychological testing. As FASD involves structural brain damage with presumably lifelong neurocognitive deficits, if there are no current deficits, then there likely is no FASD. According to the CDC guidelines (CDC, 2005), there must be significant impairments which are defined as either a global or full scale IQ below 70 and/or impairments in at least three other functional domains (e.g., cognition, memory, attention/hyperactivity, academic achievement/learning, information processing, sensory integration, speech and language, adaptive/social functioning, executive functioning) that fall 1 or more standard deviations below the mean on standardized testing. Given the forensic context, measures of malingering should be employed in the neuropsychological test battery. The chart in Exhibit 2 below, excerpted from a recent FASD Experts case involving a defendant diagnosed with Partial FAS, summarizes neuropsychological test data in a format where neurocognitive deficits can be easily identified and counted. The tests in this exhibit are listed on the abscissa axis. The horizontal green line represents the mean for each test (a standard score of "0") and the horizontal red line represents the diagnostic threshold that distinguishes deficient test performance from average performance. For full scale IQ where the red line dips, the diagnostic threshold is 2 standard deviations below the mean; for all other functional domains, the threshold is 1 standard deviation below the mean.

As shown in Exhibit 2, this defendant had a mixed pattern of neurocognitive functioning with relative strengths mixed with relative weaknesses. This mixed pattern often is observed in FASD (Connor et al., 2000) as the teratogenic effects of alcohol affect whatever developmental process is occurring in the fetus (or embryo) at the time of exposure. Also evident and forensically relevant in the chart is the discrepancy between IQ and other functional abilities. While this defendant had a full scale

EXHIBIT 2

Results of Neuropsychological Testing Depicted in Standard Deviations from the Mean



IQ that fell approximately 1 standard deviation below the mean or in the low average range (approximately 85), he had multiple neurocognitive deficits in other functional domains that fell 1 to 3 standard deviations below the mean. One of the forensically relevant aspects of this profile is his significantly deficient academic achievement, which indicates that he not only had difficulty learning academic subjects like reading and arithmetic, he also was unable to learn from other lessons such as life experience. While deficient working memory appears to have had some impact on his ability to learn, his neuropsychological profile indicates that other neurocognitive skills also affected his learning capacity such as inability to generalize or apply retained information to new problems or contexts. The ability to generalize is an abstract executive skill.

Given the relevance of many neurocognitive skills to forensic issues, the test battery used in neuropsychological evaluation should incorporate adaptive assessment as well as intellectual, academic achievement, attention and impulse control, memory, motor skills, and executive functioning. The latter should include tests found in FASD research to be particularly susceptible and relevant to FASD-associated impairments. The following list contains some of the tests used in our assessment process:

1. Intellectual functioning: The *Wechsler Adult Intelligence Scale—4th Edition* (WAIS-IV) (Wechsler, 2008) assesses four sub-domains of intellectual functioning as well as full scale IQ: verbal comprehension, perceptual organization, working memory, and processing speed.
2. Academic achievement: The *Wide Range Achievement Test—4th Edition* (WRAT-4) (Wilkinson & Robertson, 2006) measures the ability to learn academic skills in four areas: word reading, reading comprehension, spelling, and arithmetic.
3. Learning and memory: Auditory learning and memory are assessed with the *California Verbal Learning Test* (CVLT) (Delis, Kramer, Kaplan, & Ober, 1999), which involves a list learning task. Nonverbal memory and visuospatial construction are assessed with the *Rey Complex Figure Test* (RCFT) (Meyers & Meyers, 1995).

4. Attention: Attention is assessed in a visual modality by using the *Conners' Continuous Performance Test* (CPT) (Conners, 2004), which is a computerized test that measures sustained attention and impulsivity. Of special interest on this test is the assessment of variability of response time as this has been demonstrated to be an issue in research on FASD (Connor et al., 1999; Coles, Platzman, Lynch, & Freides, 2002).

5. Motor coordination: This domain is assessed through multiple measures. *Grooved Pegboard* is used to assess speeded eye-hand coordination, *Finger Tapping* is used to assess speeded finger movement, and *Grip Strength* is used to assess general strength. Research has demonstrated that coordinated motor functioning is often impacted in FASD.

6. Executive functioning: This is a domain that involves assessment of multiple domains, including problem solving, learning from mistakes, generation of ideas, flexibility, and multitasking. Because executive functions are multidimensional, several tests are administered to assess it. Scanning and switching of attentional rules are assessed with the *Trail Making Test* (TMT). Fluency in generation of ideas is assessed with the *Controlled Oral Word Association Test* (COWAT) in the verbal modality and *Ruff's Figural Fluency Test* (RFF) (Ruff, 1996) in the nonverbal modality. Response inhibition is measured with the *Stroop Test* (Golden & Freshwater, 2002). Working memory and multitasking are assessed with the *Consonant Trigrams Test* (CTT) (Stuss, Stethem, & Pelchat, 1988; Stuss, Stethem, & Poirier, 1987). Planning, problem solving, rule switching, and learning from past mistakes are measured with three tests: *Wisconsin Card Sorting Test* (WCST) (Heaton & PAR Staff, 2005), *Delis-Kaplan Executive Function System* (D-KEFS) (Delis, Kramer, & Kaplan, 2001) *Tower Test* and *Proverbs Test*.

7. Adaptive functioning: Adaptive functioning is assessed with the *Vineland Adaptive Behavior Scale* (VABS) (Sparrow, Cicchetti, & Balla, 2005), which involves a detailed interview of an individual with considerable knowledge of the defendant's daily functioning. Research with individuals with FASD has demonstrated that adaptive functioning tends to be significantly more deficient than IQ in this population and is indicative of real-world deficits in appropriate functioning (Streissguth et al., 1996).

If malingering and other factors that might affect an examinee's test performance are ruled out, results obtained on neu-

ropsychological tests may fall near the top of an individual's skill range. This "maximum potential" outcome is due to the structure that must be employed during testing (e.g., minimizing environmental and social distractions, and test protocols that dictate the specific manner of responding) to ensure reliability. Test conditions, which are typically described in detail in test manuals, must resemble to the extent possible those used during test development, even when testing is done in jails or prisons. Even under structured testing situations, some tests allow for a little more cognitive control by the subject. As can be seen in the profile shown above, the defendant in this case example had significant difficulty performing executive function tests that gave him relatively more control versus those that were tightly structured. Such disparity depicts in a graphic way what happens in the real world when stressful, fast-paced events occur in unfamiliar situations where there is no predetermined structure or routine. The relevance of this dynamic in terms of offense conduct cannot be overstated.

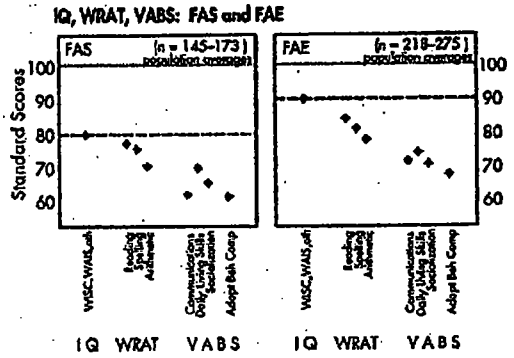
The above chart shows that this defendant had numerous executive skill deficits. As previously noted, executive functioning affects all aspects of information processing, from perception to integration and interpretation to action or inaction. Thus, if perceptual skills are impaired by attention deficits, it follows that inaccurate information will feed into the next step in the process, integration. Integration relies heavily upon the ability to recall important related information or experiences, which enables contextual assessment of the meaning and significance of input, including its relation to previous information and relevance to anticipated events. Both perception and integration affect the next stage in the information processing cycle: interpretation. If brain structures responsible for different elements of self-regulation do not communicate efficiently, then interpretation of input that has been perceived and integrated also may involve another step (emotion modulation), particularly if the input is interpreted as threatening or dangerous. If all of these cognitive processes are impaired and/or slowed, the individual may resort to instinctive fight or

flight. In this particular defendant's case, he killed his wife in a heated argument. His neuropsychological chart displays his difficulty in processing information in unstructured versus structured situations, demonstrating graphically the importance of social context in his case.

Another way of looking at this performance difficulty in those with FASD is through the disparity or "downward slope" found in IQ and adaptive functioning scores. Using IQ as a benchmark that establishes an upward limit on how someone with FASD might be expected to function, the charts in Exhibit 3 below (Streissguth et al., 1996) depict how individuals in this population typically function. The chart on the left shows test results for individuals diagnosed with FAS and a mean IQ of 79, and the chart on the right shows test results for individuals diagnosed with FAE (i.e., Partial FAS or ARND) and a mean IQ of 90. For individuals without FASD, standard scores for academic achievement in reading, spelling, and math should roughly resemble IQ. Thus, whether the IQ is 79, 90, or above, standard scores on the Wide Range Achievement Test (WRAT) typically will fall around the same level as the IQ, if the individual does not have FASD. The same correlation applies to other functional abilities as well, including adaptive functioning (e.g., communication, daily living, social skills), which is typically measured via standardized informant measures such as the Vineland Adaptive Behavior Scale (VABS). Consequently, in individuals without FASD, standard scores for adaptive skills also should resemble IQ.

As can be seen in Exhibit 3, a downward slope regardless of specific FASD diagnosis from IQ to academic achievement and finally to adaptive functioning appears to be characteristic for this population. This downward slope has relevance in the forensic context because IQ scores are so ubiquitous and powerful in terms of how a legal defendant is viewed in court. If a defendant's IQ is average or low average, there is a general expectation on the part of all individuals in the legal context that functional capacity also will be average or low

EXHIBIT 3
Adapted from Streissguth et al. (1996)



average. As Greenspan has eloquently argued in the *Hearn* case, IQ is merely a benchmark and no more:

> The use of IQ scores (is) an attempt to create an illusion of scientific certainty in identifying a disorder whose causes and manifestation are often hidden and subtle. . . . The problem is that when the artificial number fails to fit with the disability as it is experienced and documented by others, which criterion should be used? Typically, clinicians and government entities find it easier to "go by the book," but there are times when that results in a wrong and, possibly, unjust decision.

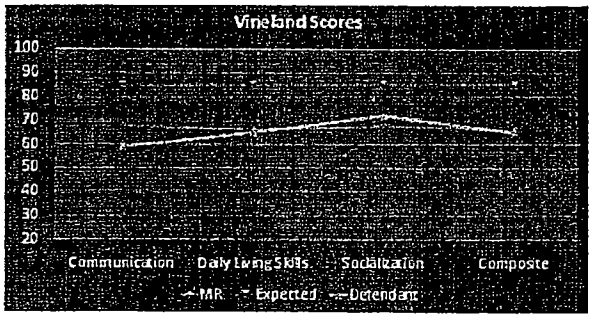
Greenspan further noted that emphasizing IQ score over adaptive functioning in determining "intellectual disability" or "mental retardation" eliminates the need for clinical judgment and essentially reduces the mental health professional's forensic role to that of a psychometrist.

In other words, it is adaptive ability and not IQ that should determine whether someone is able to meet culturally age-appropriate standards of personal independence and social responsibility. According to the literature (Streissguth et al., 2004), while most individuals with FASD have IQs over 70,

their adaptive abilities as measured by adaptive assessments such as the Vineland tend to fall near or in the intellectually deficient range. In fact, as shown in the chart above, the Adaptive Behavior Composite, which averages the three adaptive subskill scores, is close to 60 for individuals with FAS and slightly below 70 for those with Partial FAS or ARND. In both groups, adaptive functioning tends to fall approximately 20 points below full scale IQ. Bottom line, regardless of IQ, individuals with FASD tend to function as if they were intellectually deficient. The chart in Exhibit 4 below displays this phenomenon using scores from the Vineland to contrast how the defendant in our case example would be expected to perform in terms of adaptive functioning if he didn't have FASD (i.e., adaptive skill standard scores around his IQ score of 85) and how he actually performs compared to individuals diagnosed with mental retardation (MR). As can be seen in this chart, his Adaptive Composite score is almost identical to the MR sample despite his IQ—a performance deficit due in large part to his multiple executive function skills, which control how more basic neurocognitive abilities are used.

EXHIBIT 4

Adaptive functioning of a defendant with FASD compared to mean scores for individuals with mental retardation and those with average IQ



After it has been established through neuropsychological testing that the functional criterion for CNS damage in FASD has been met, the next step in the forensic assessment process is to determine if life course history is consistent with deficits identified in current testing. While school records are a primary source of reliable information for this task, other sources of information (e.g., medical, juvenile justice, adoption, child welfare, treatment, birth mother's records) also may be relevant. If the individual received Special Education services during the school years, school records may be a rich source of standardized test data, with scores that can be interpreted in terms of standard deviations from the mean (the measurement metric used in FASD diagnostic protocols). If the individual is an adult, other records may contain reliable data about behavioral history (e.g., employment, criminal history, Department of Corrections, probation and parole).

Objective life course analysis involves a search for positive as well as negative outcomes. For example, discovering that an individual completed college and maintained a long-term job requiring considerable cognitive skills would likely rule out FASD. On the other hand, a history of employment problems and inability to live independently is consistent with FASD. As in other phases of FASD assessment, life course analysis involves a comparison of the life history with a priori expectations based on consistent secondary disabilities findings by multiple researchers (Löser, Bierstedt, & Blum, 1999; Spohr, Willms, & Steinhaussen, 2007; Streissguth et al., 1996). The question at hand is whether a defendant eventually developed the maladaptive coping patterns (i.e., mental illness, substance abuse, disrupted schooling, inappropriate sexual behaviors, trouble with the law, confinement, employment problems, and inability to live independently) predicted in the research. As also seen in the research, risk of these secondary disabilities diminishes if the defendant was exposed to protective factors (e.g., FASD diagnosis in early child-

hood, developmental disabilities services, and nurturing and protective caregiving through most of childhood).

Functional FASD assessment also involves differential diagnosis. For the neuropsychologist, competing etiologies for any functional deficits found in testing must be addressed and explained. This could include head injuries or chronic substance abuse. For the specialist who conducts the lifelong functional assessment, differential diagnosis should address conduct and personality disorders or any other mental health disorder with symptoms that overlap FASD. For instance, antisocial personality disorder is an adult disorder defined in the DSM-IV-TR by a "pervasive pattern" since age 15 of disregarding and violating the rights of others by means of three or more of the following behaviors: failure to conform to social norms with respect to lawful behaviors as indicated by repeatedly performing illegal acts; deceitfulness; impulsivity; irritability and aggressiveness; reckless disregard for safety of self or others; consistent irresponsibility, as indicated by repeated failure to sustain consistent work behavior or honor financial obligations; and lack of remorse. Of course, since many of these behaviors also are seen in neurocognitively impaired individuals with FASD, differential diagnosis can be challenging. In particular, it is important in FASD assessment to assess exposure to traumatic environmental events such as neglect and abuse as well as neurocognitive impairments since interactions between these impairments and social experiences can create an antisocial orientation. Neurocognitive impairments diminish a child's ability to cope adequately with traumatic experiences, thereby leading to maladaptive behavior patterns. This situation has been described in the FASD literature as "double jeopardy" (Carmichael-Olson, Oti, Gelo, & Beck, 1999).

Along with life course analysis, case referral also may involve testing of abilities directly relevant to the legal context. For example, since many individuals with FASD tend to be suggestible (Pollard et al., 2004), assessment of this trait

with the Gudjonsson Suggestibility Scale (Gudjonsson, 1997) is important. Likewise, many persons with FASD claim to understand and then waive their rights to silence when arrested even though they may have difficulty understanding some of the abstract terms and implications in the Miranda waiver. The Grisso Miranda tests (Grisso, 1998) assess rights waiver competency. If a suggestible person with FASD is interrogated, there is risk of inaccurate statements, including false confession (Fast, Conry, & Loock, 1999; Moore & Green, 2004). Thus, competency to proceed also may be an issue, which should include standardized assessment with an appropriate measure. Measures involving forced-choice test formats may be insufficient because they limit ability to develop a rich understanding of a defendant's functional competence.

If personality testing is done, interpretations should be offered with the caveat that there are no current measures which include persons with FASD in their development samples, which makes interpretation of test results potentially misleading. The same caveat applies to actuarial assessment to determine risk of future violence, including sexual violence. Malingering tests should be administered as part of a competency test battery. Depending upon the nature of the alleged offense, risk of future dangerousness also may be important. In addition to any standardized measures that might be used to measure risk of aggression, it will be important to examine how violence-prone a defendant has been over the course of his or her life, especially in high stress contexts. Of course, given that persons with FASD tend to function best in structured settings, this factor should be taken into consideration during risk analysis.

Once members of the multidisciplinary team have assessed the defendant and provided their findings to the team, the medical diagnostician's responsibility is to review medical records and conduct a thorough medical assessment. Physical examination of the face is necessary in order to determine if there are abnormalities. Facial assessment also may include

computerized facial analysis using software developed by Astley and Clarren (1996). Growth deficits may be observable at the time of the assessment. If not, review of medical records may establish deficits in childhood. Physical examination also is important in differential diagnosis in order to rule out competing etiologies. The team's diagnostician may want to refer the defendant for structural brain imaging as well. While brain imaging is not necessary for diagnosis, graphic depiction of damage to a specific brain structure can sometimes be helpful in illustrating the impact of FASD within the courtroom setting.

Finally, after an FASD diagnosis has been made, a team member with forensic expertise should conduct an analysis of the alleged offense behavior and offense history to determine if there are any inconsistencies with the diagnosis. For example, sophisticated planning (particularly with regard to elaborate escape plans) would be inconsistent with FASD. On the other hand, certain offense behaviors are often seen in this population and therefore consistent with FASD (Streissguth et al., 2004). These offenses include:

- running away
- shoplifting/theft (often involving items of little value)
- burglary
- drug offenses
- property damage
- vehicular crimes
- domestic violence
- assault
- illegal sexual behavior
- probation/parole violations

Post-arrest behaviors consistent with FASD include:

- rights waiver after initial denial of culpability and subsequent "over" confession (i.e., providing more information during interrogation than is typical)

- naivete and gullibility
- immaturity
- stubbornness (e.g., clinging to foolish decisions that are not in the defendant's best interest and consistently refusing to agree with defense team suggestions and advice)
- grandiose posturing (e.g., bragging about offense behavior and/or abilities even when such statements are self-sabotaging)
- magical thinking (e.g., believing that something or someone will suddenly make the legal problem go away)
- unquestioning faith in defense attorney's ability to obtain the best outcome (e.g., failure to ask any questions)
- inability to appreciate the impact of the alleged offense either on the victim or on himself/herself (e.g., unemotional or "flat" response to situation)
- inability to provide a coherent, sequential narrative
- memory deficits (e.g., forgetting important defense-related information from interview to interview, inability to recall many offense-related details)

Testimony

Explaining a complicated diagnosis like FASD to the Court requires PowerPoint-rich testimony, with many visual slides to illustrate points being made. At a minimum, two experts should testify regardless of the size of the diagnostic team: one to explain the diagnosis, and another to explain how the diagnosis impacted life history, including the instant offense. Order of testimonial presentation is important. For example, the medical doctor should explain the diagnosis prior to another expert explaining why the diagnosis is relevant to offense conduct. Not only can the diagnostician explain FASD science to the Court, which lays the groundwork for testimony regarding how the defendant meets diagnostic criteria, he or she also can integrate functional findings from other team members. The latter prepares the Court for the nexus testimony: how the FASD impacted the offense conduct. It is very

important for the expert who addresses the nexus to have forensic expertise. Beyond communicating the diagnosis and why it matters to offense conduct, testimonial goals also might include: (a) explaining why standardized assessment trumps subjective analysis in terms of reliability, (b) pointing out areas of convergent validity (i.e., consistency over time among tests and test examiners and consistency among informants), (c) describing indices that portray neurocognitive profiles in FASD (e.g., downward slope, secondary disabilities), (d) integrating standardized test results with behavioral information across the defendant's lifespan (drawing parallels between test deficits and actual behavior), and if appropriate, (e) explaining how FASD affects future dangerousness.

If the results of the FASD assessment will be used in plea negotiation or contested competency hearings, then it will be important in the functional reports to address how specific deficits affected behavior directly relevant to these issues as well as to mental state. For instance, did attention deficits and impulsivity compound the defendant's self-regulation difficulty to such an extent that he or she was unable to resist an urge to act? Did such deficits worsen a tendency to make hasty decisions, such as consenting to rights waiver without adequate understanding? Do memory deficits as well as poor sequencing ability preclude ability to provide relevant exculpatory details to the defense attorney? Do communication deficits affect understanding and ability to communicate adequately in terms of Miranda rights, police interrogation, and the legal process? Do executive function deficits affect abilities to learn from experience, to engage in the abstract thinking required to generalize from previous experiences to the present while simultaneously foreseeing consequences of actions (i.e., aspects of "reflection"), and to make independent decisions, delay gratification, consider alternatives, appreciate the impact of one's behavior on self and others, resist others' efforts to lead and manipulate, control emotions and impulses, and show remorse?

In summary, FASD assessment is complex and challenging, even in the clinical context. In the forensic context, these complexities only increase. However, with each member of a multidisciplinary team working as part of the ensemble to handle discrete aspects of the assessment process, the end result can be a rich analysis of a defendant's functional capacities and how those capacities influenced pre-assessment events (i.e., offense conduct and rights waiver) and are likely to affect future conduct (e.g., competency to proceed to trial and future risk of dangerousness). The end result in Court is a thorough understanding of the defendant's capacity to behave, which can only enhance the legal process.

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APPENDIX A

FASD EXPERTS SCREENING QUESTIONNAIRE ✓

Offense Conduct

- Impulsive and illogical actions with high risk of detection ☐
- "Simple" offense plan (focus is only on the objective) ☐
- Poor exit strategy
- Aggressive over-reaction to unforeseen events ("fight or flight") ☐
- More sophisticated/experienced co-defendants ☐

Arrest Conduct

- Immediately or easily waives rights ☐
- Over-confesses (suggestible) ☐
- Brags about prowess or takes full responsibility if co-defendants ☐
- Emotionally detached from crime (shows little remorse or guilt) ☐
- Behavioral regression (breaks down in tears, infantile behavior) ☐

Interview with Client

- Short stature (not always) ☐
- Unstable lifestyle ☐
- Socially inept, immature, and naïve ☐
- Eager to please or stubbornly resists the obvious ☐
- Can't provide coherent, detailed narrative ☐
- Can't concentrate ☐
- Doesn't add much to discussion ☐
- Doesn't seem to remember what you tell him/her from appointment to appointment ☐

Prior Legal History

- Easily led by more sophisticated peers ☐
- Multiple low-grade offenses in teen years, often with co-defendants ☐
- Lots of stealing ☐
- Illogical offenses (e.g., stealing something with little value) ☐
- Oblivious to risk ☐
- Impulsive, opportunistic crimes ☐
- Probation violations ☐

Life History

- Mom abuses alcohol/drugs ☐
- Involvement with child welfare
- Adoption/foster or relative placements/juvenile commitment ☐
- Special Education / learning disabilities in school ☐
- Mental health diagnoses in childhood (especially ADD/ADHD) ☐
- Anger control problem ☐
- Rule-breaking behaviors in childhood (lying, cheating, stealing, fighting) ☐
- Disrupted education ☐
- Substance abuse ☐
- Poor understanding of personal boundaries ☐
- Difficulty living independently ☐
- Poor employment history ☐

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Risk Factors for Adverse Life Outcomes in Fetal Alcohol Syndrome and Fetal Alcohol Effects

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ABSTRACT. Clinical descriptions of patients with Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE) suggest major problems with adaptive behavior. Five operationally defined adverse outcomes and 18 associated risk/protective factors were examined using a Life History Interview with knowledgeable informants of 415 patients with FAS or FAE (median age 14 years, range 6–51; median IQ 86, range 29–126). Eighty percent of these patients were not raised by their biological mothers. For adolescents and adults, the life span prevalence was 61% for Disrupted School Experiences, 60% for Trouble with the Law, 50% for Confinement (in detention, jail, prison, or a psychiatric or alcohol/drug inpatient setting), 49% for Inappropriate Sexual Behaviors on repeated occasions, and 35% for Alcohol/Drug Problems. The odds of escaping these adverse life outcomes are increased 2- to 4-fold by receiving the diagnosis of FAS or FAE at an earlier age and by being reared in good stable environments. *J Dev Behav Pediatr 25:228–238, 2004.* Index terms: *fetal alcohol syndrome, fetal alcohol effects, risk factors for adverse life outcomes, life span studies, Fetal Alcohol Spectrum Disorders (FASD).*

Since Fetal Alcohol Syndrome (FAS) was described[1,2] and named,[3] enormous progress has been made in verifying that FAS is a birth defect caused by prenatal alcohol exposure and in identifying patients with FAS in many countries where women drink alcohol.[4] Reported incidence rates of 3 per 1000 births[5,6] would result in 12,000 children with FAS born annually in the United States.[7] The first quantitative assessment of physical, cognitive, and behavioral outcomes of a large group of adolescents and adults with FAS and a few without the full syndrome, documented deficits in intellectual, academic, and adaptive living skills.[8] Although a high rate of maladaptive behaviors was noted, that paper made no attempt to understand the etiology of such problems or to evaluate their consequences in a community context. The present study extends this previous work in several directions and examines not only patients with FAS but also a large number with Fetal Alcohol Effects (FAE) who also have heavy prenatal alcohol exposure but do not manifest the full physical features of FAS. The focus of this paper is on the consequences of maladaptive behaviors and on the adverse life outcomes experienced by patients with FAS/FAE. Additionally, we examine some key risk/protective factors, both personal and environmental, that might exacerbate or ameliorate these adverse life outcomes.

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Although the teratogenic effects of alcohol and their mechanisms are well-documented,[9] there are almost no studies of the psychosocial milieu into which children with FAS or FAE are born, or of how this might affect their lives at different stages of development. Each person with FAS or FAE has a mother who abused alcohol to some extent. Among the environmental circumstances frequently found in association with prenatal alcohol damage are early maternal death,[8,10] living with an alcoholic parent, child abuse and neglect,[11-12] being removed from the home by authorities, experiencing repetitive periods of foster care and other transient home placements, and being raised by adoptive or foster families. Rearing families are highly variable. They may have no knowledge of the prenatal exposure history, making it difficult to obtain an accurate diagnosis of an alcohol-related problem. Or, they may be the birth families themselves, with a mother who may or may not be in recovery. To date these environmental characteristics have not been quantified and examined in light of their contribution to adverse psychosocial outcomes for the child with FAS or FAE.

The objective of the present study is to examine adverse life outcomes not previously documented in patients with FAS or FAE, and to evaluate the impact of a variety of personal and environmental characteristics that have the potential to alter the nature and level of adverse life outcomes in these patients. We consider five such adverse outcomes: Inappropriate Sexual Behavior (ISB), Disrupted School Experience (DSE), Trouble With the Law (TWL), Confinement (CNF), and Alcohol and Drug Problems (ADP). We examine them in light of ten influential personal and environmental characteristics that we call "risk and protective factors." Three age groups (children, adolescents, and adults) were evaluated to give the study a lifetime perspective.

METHODS

Subjects

This study involves 415 patients enrolled in the Fetal Alcohol Follow-up Study of the University of Washington's Fetal Alcohol and Drug Unit (FADU) who lived in the Pacific Northwest, who were at least 6 years old between August and December 1995, and for whom a consenting informant could be located. Ninety-one otherwise eligible patients were not represented due to unavailability of an informant or to refusals. These 91 did not differ from the 415 in terms of diagnosis, age at diagnosis, ethnicity, IQ, or sex.

Diagnoses were by dysmorphologists associated with the University of Washington (UW)[2-4,13-17] at the time of original enrollment in the Follow-up Study between 1972 and 1995. Fetal Alcohol Syndrome (FAS) was diagnosed when patients had a positive history of maternal alcohol abuse during pregnancy and (1) growth deficiency of prenatal origin (for height and/or weight); (2) a pattern of specific minor anomalies that included a characteristic face (generally defined by short palpebral fissures; midface hypoplasia, smooth and/or long philtrum, and thin upper lip); and (3) Central Nervous System (CNS) manifestations, including microcephaly or history of delayed development, hyperactivity, attention deficits, learning disabilities, intellectual deficits, or seizures.[8] The term Fetal Alcohol Effects (FAE) was used for individuals who were exposed prenatally to significant amounts of alcohol, were examined by the same dysmorphologists, and had some but not all of the characteristics of FAS.[4,8] Appropriately informed consent was obtained from each subject or guardian at the time of examination and from each informant at the time of interview. The study was approved by the University of Washington Human Subjects Review Board.

Procedures

Data on diagnosis (FAS vs FAE), age at diagnosis, date of birth, and sex were obtained from patient records. Performance data were from standardized tests administered at FADU. Test scores (see Table 1) were derived from age-appropriate IQ, achievement, and adaptive behavior tests, administered with standard procedures at the FADU: Wechsler Intelligence Scale for Children-Revised,[18] Wechsler Adult Intelligence Scale-Revised,[19] the Wide Range Achievement Test-Revised,[20] and the Vineland Adaptive Behavior Scales.[21]

Life History Interview (LHI). The rest of the data were from the LHI administered by telephone between August and December 1995 to informants such as parents or guardians (see below) who had known the patients for many years. Interviewers were "blind" to contents of the patient records including diagnosis and performance on standardized tests. Designed for this study, much of the LHI is analogous to the "current status and history" questions used in clinical settings; it is suitable for subjects at least 6 years old and produces data for both the risk/protective factors and the adverse life outcomes. The LHI is a 36-page structured interview comprising 450 questions about past and current events that is organized to provide a clear visual guide for the interviewer in standardizing queries and facilitating accurate coding. Each administered interview was reviewed collectively by the team and each coded interview reviewed by the project director. Mean administration time was 70 minutes.

Potential Risk and Protective Factors. Our clinical experience and a pilot study review of our clinical research records[22] produced the list of 10 risk/protective factors shown in Table 1, which include both patient characteristics and life experiences. Coding systems were built into the LHI to quantify them.

Specifically, variables described as "percent of life" are intended to be an explicit quantification. For these, the informant was asked to think back over the history of living situations that the patient had experienced and list for the interviewer the inclusive ages during which the patient's living situation was judged to be "stable and nurturing," the patient had "lived with a person who abused alcohol or drugs," or the patient had "not had their basic needs met." For each of these, a computer algorithm calculated the total years that each patient lived with the relevant risk or protective factor and divided by the patient's age (times 100) to determine the percentage of life that the factor was experienced. "Ever a victim of physical or sexual abuse or

Table 1. Potential Risk/Protective Factors, Adverse Life Outcomes, and Test Performance for Patients with Fetal Alcohol Syndrome (FAS) or Fetal Alcohol Effects (FAE): by Three Age Groups

	Age 6–11.9 yr					Age 12–20.9 yr					Age ≥ 21 yr				
	(n)	%	Mean	Med	SD	(n)	%	Mean	Med	SD	(n)	%	Mean	Med	SD
Risk Protective Factors															
Males (vs females)	(162)	56.2				(163)	62.6				(90)	47.8			
FAS diagnosis (vs FAE)	(162)	28.4				(163)	34.4				(90)	58.9			
Age at FAS/FAE diagnosis (years)	(162)		6.5	6.8	2.5	(163)		12.5	13.4	3.9	(90)		19.6	21.1	10.8
Age at Life History Interview (LHI)	(162)		8.8	8.7	1.8	(163)		16.0	15.8	2.4	(90)		28.4	25.7	7.2
Percent of life in stable/nurturing home	(158)		72.6	82.0	26.3	(161)		68.0	75.0	30.9	(86)		62.9	65.5	31.8
Percent of life with person abusing alcohol/drugs	(153)		35.5	33.0	26.2	(156)		32.9	28.0	27.9	(87)		33.0	30.0	26.7
Percent of life basic needs not met	(151)		26.5	23.5	22.0	(159)		21.1	24.0	14.0	(90)		12.5	15.9	6.0
Ever physical, sexual abuse or domestic violence	(157)	61.1				(161)	70.8				(88)	73.9			
Average years per household by age 18	(160)		2.7	2.1	2.1	(162)		3.8	3.0	3.0	(88)		6.3	6.0	4.6
Good Quality Home Scale for ages 8–12 yr	(162)		6.3	9.0	5.3	(161)		9.4	10.0	3.1	(90)		9.2	10.0	3.3
IQ < 70	(138)	13.8				(149)	10.7				(81)	17.3			
Adverse Life Outcomes															
Inappropriate Sexual Behaviors	(161)	39.1				(160)	47.5				(87)	51.7			
Disrupted School Experiences	(161)	14.3				(162)	62.3				(88)	59.1			
Trouble With the Law	(161)	14.3				(162)	61.1				(89)	58.4			
Confinements	(161)	8.1				(161)	46.0				(90)	57.8			
Alcohol/Drug Problems	(162)	0.0				(162)	29.0				(90)	45.6			
Test Performance															
Wechsler IQ	(134)		86.9	88.5	15.8	(145)		88.1	89.0	15.1	(80)		80.7	80.0	15.9
WRAT Reading standard score	(127)		77.7	81.0	18.2	(133)		83.1	86.0	17.7	(77)		82.1	80.0	19.0
WRAT Spelling standard score	(125)		74.7	77.0	18.5	(132)		80.0	80.0	18.1	(77)		81.2	81.0	16.6
WRAT Arithmetic standard score	(126)		74.1	75.0	16.4	(132)		74.6	76.0	18.7	(77)		74.3	74.0	15.0
VABS Communication standard score	(140)		73.3	71.5	15.1	(146)		66.2	63.5	18.1	(72)		57.8	53.0	24.0
VABS Daily Living Skills standard score	(140)		70.7	70.0	16.7	(146)		67.8	67.5	18.1	(72)		74.2	77.5	22.8
VABS Socialization standard score	(140)		71.7	69.0	13.2	(146)		63.3	60.5	16.9	(72)		63.4	61.5	20.5
VABS Adaptive Behavior Composite standard score	(140)		67.2	66.0	13.6	(146)		61.1	59.5	14.9	(72)		61.1	58.5	20.1

Med, median; WRAT, Wide Range Achievement Test; VABS, Vineland Adaptive Behavior Scales.

domestic violence," a dichotomy, was obtained by asking about each of these individually, and counting "yes" if any of the three were affirmed. "Average years per household by age 18" was obtained by asking about each of the different households in which the patient had lived before the age of 18 and dividing the total count by 18 or by the patient's age if under 18 years. The "Good Quality Home scale for age 8 to 12 years" is a list of 12 home qualities selected a priori to be "good" in terms of potential to prevent adverse outcomes in the pre-adolescent years. Possible scores range from "0" to "12." The items are "people tried to help each other; the environment was reasonably normal; there was quite a bit of structure; the days were generally predictable and not frenetic; there was enough to keep [patient] busy; people worked at fairly regular jobs; someone was in trouble with the law; a parent had a serious mental illness; a parent had or was suspected of having FAS or FAE; the family had enough resources to manage ok; the family was actively involved in a church or a religious group like that; and there were specific household rules and consequences for their enforcement." (Three items are in reverse order for scoring; data on this scale were coded only for patients who were 9 years and older.)

Adverse Life Outcomes. The five adverse life outcomes (Table 1) also derive from the LHI. Criteria include the following: Inappropriate Sexual Behaviors (ISB): "repeated problems" with one or more of the following 10 inappropriate sexual behaviors: making inappropriate sexual advances, inappropriate sexual touching, promiscuous sexual behavior, exposing behavior, compulsive sexual behavior, voyeuristic behavior (i.e., peeping), masturbating in public, in trouble for incest behavior, making obscene phone calls, having sex with animals, or any other unusual or worrisome sexual behavior; or ever having been in trouble with the law for a sexual offense even once; Disrupted School Experience (DSE): ever suspended, expelled, or dropped out; Trouble With the Law (TWL): ever charged, arrested, convicted, or otherwise in trouble with the law; Confinement (CNF): ever in juvenile detention, jail, prison,

psychiatric hospital, or in inpatient alcohol/drug treatment; Alcohol/Drug Problems (ADP): ever had alcohol or drug problems or received treatment for alcohol, drugs, or both.

Analysis

Odds ratios were calculated for the 9 risk factors as binary variables individually across the five life outcomes (Table 2) for the subsample of subjects aged 12 years and older (n = 253). The quantity tabulated is the ratio of the odds of adverse life outcomes in the subgroup with the factor (or falling above some level of the factor) to the odds of adverse life outcomes in the subgroup lacking the factor. For the purpose of computing odds ratios, factors that are continuous, other than age, were made dichotomous by dividing the distribution at the median. Approximate 95% confidence intervals for these odds ratios were computed by transforming conventional (symmetric) intervals for log odds ratios.

In order to examine the multiple risk factors jointly (and without simplifying to binary categorizations as in the median scores in Table 2) we carried out a number of logistic regression analyses (Table 3). These analyses examine the odds of adverse life outcomes as functions of the environmental and diagnostic risk factors listed in Table 2, including sex. As the nature and prevalence of adverse outcomes is fundamentally different for children, the models of Table 3 address only the patients who are 12 years and older, as does Table 2. These models were built using stepwise procedures for "generalized additive models" as implemented in the S-PLUS statistical analysis system.[23,24] The effects of "Average years per household," age of the patient at diagnosis, and the age of the patient at the time of the LHI were all represented by nonlinear effects in the regression models, the latter two predictors jointly in a single two-dimensional nonlinear surface. To facilitate interpretation, Table 3 reports model-based odds ratios for selected values of the risk/protective factors. The Appendix describes the logistic regression analysis procedures in more detail.

RESULTS

Demographics and Clinical Findings

The 415 subjects include 155 patients with FAS and 260 with FAE, 236 males and 179 females. The majority (60%) of the subjects were white, 25% were Native American, 7% black, and 6% Hispanic. The sample included 162 children 6.0 to 11.9 years (median 8.8 years), 163 adolescents 12.0 to 20.9 years (median 16.0 years), and 90 adults 21 to 51 years (median 28.4 years). Respondents included adoptive mothers (33%); foster mothers, biological fathers, or stepmothers (25%); biological mothers (17%); other relatives, or current or former caretakers (20%); spouse or partner (1%); and others (4%). Most respondents (80%) had known the patients for at least half of their lives and 42% had known the patients their entire lives. Almost all respondents felt knowledgeable about the patients' life histories. Questions that respondents could not answer were scored to indicate absence of knowledge.

As a group, the entire sample had a median age of 14 years and a median age of 10 years at diagnosis. Sixty-seven percent had been the victim of physical or sexual abuse or of domestic violence. Their median percent of life in a stable/nurturing home was 75% of their lives; median years per living situation (up through age 18) was 2.7 years (Table 1). Overall, 42% of these patients were reported

Table 2. Unadjusted Odds Ratios of Risk Factors Across Five Adverse Life Outcomes as Binary Variables Among Patients with Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE), 12 Years and Older

Risk Factor	Inappropriate Sexual Behavior	Disrupted School Exp.	Trouble with the Law	Confinements	Alcohol/Drug Problems
Environmental Factors					
Over 12 yrs of age at FAS/FAE diagnosis	2.25 (1.27, 4.00)	3.27 (1.85, 5.78)	2.92 (1.66, 5.15)	3.03 (1.69, 5.41)	4.16 (2.07, 8.36)
"Low" percent of life in stable/nurturing home	3.13 (1.83, 5.36)	3.10 (1.79, 5.37)	2.16 (1.27, 3.69)	2.97 (1.75, 5.04)	2.44 (1.40, 4.26)
"Fewer" years per household by age 18 yrs	2.22 (1.30, 3.81)	2.20 (1.25, 3.87)	2.10 (1.20, 3.66)	2.27 (1.32, 3.89)	2.19 (1.27, 3.78)
IQ ≥ 70	0.75 (0.34, 1.64)	2.43 (1.07, 5.52)	1.99 (0.90, 4.38)	1.69 (0.76, 3.76)	3.72 (1.24, 11.14)
Victim of physical, sexual abuse/domestic violence	4.36 (2.31, 8.24)	1.37 (0.77, 2.44)	2.13 (1.20, 3.78)	2.03 (1.14, 3.62)	2.71 (1.38, 5.30)
"Poor" Quality Home for ages 8–12 years	1.51 (0.88, 2.59)	3.03 (1.65, 5.56)	2.01 (1.14, 3.54)	1.92 (1.12, 3.30)	1.89 (1.09, 3.28)
"High" percent of life with person abusing alc/drugs	1.42 (0.84, 2.39)	1.84 (1.07, 3.14)	1.45 (0.86, 2.45)	1.82 (1.08, 3.06)	2.22 (1.28, 3.86)
"High" percent of life basic needs not met	1.93 (1.14, 3.25)	1.24 (0.73, 2.10)	1.85 (1.09, 3.14)	1.97 (1.17, 3.30)	1.40 (0.81, 2.40)
Personal Characteristics					
Being male	0.93 (0.56, 1.56)	1.92 (1.13, 3.25)	1.84 (1.09, 3.10)	1.83 (1.09, 3.06)	0.85 (0.50, 1.45)
Being FAE	1.48 (0.88, 2.49)	1.92 (1.13, 3.25)	1.97 (1.17, 3.33)	1.55 (0.93, 2.59)	1.80 (1.04, 3.11)

Odds ratios less than 1 = protection, greater than 1 = risk. A 2-tailed log-symmetric 95% confidence interval is to the right of each odds ratio. Lower confidence interval limits above 1.0 correspond to conventional significance levels of $p \leq .05$. "Low," "High," "Fewer," and "Poor" refer to above or below the median for this sample.

Table 3. Adjusted Odds Ratios (with approximate *p* values) for Selected Values of Risk Factors in Nonlinear Logistic Regression Models for Five Adverse Life Outcomes Among Patients with Fetal Alcohol Syndrome (FAS) or Fetal Alcohol Effects (FAE), 12 Years and Older

	ISB (n = 240)	DSE (n = 244)	TWL (n = 245)	CNF (n = 248)	ADP (n = 242)
Environmental Factors					
Low percent life in stable/ nurturing home (18% vs 100%)	4.06 (.0006)	4.67 (.0003)	2.69 (.01)	—	4.10 (.001)
Fewer years per household[a] by age 18 yrs (1.2 vs 9 yrs)	—	—	—	7.35 (.001)	—
Victim of physical, sexual abuse/domestic violence	3.37 (.0004)	—	—	—	2.56 (.02)
Age at diagnosis[a]					
(17 vs 9 yr at Age at LHI = 18)	1.28	1.70	1.92	1.81	0.95
(17 vs 9 yr at Age at LHI = 26)	2.07 (.002)	2.58 (.05)	1.48 (.01)	1.46 (.04)	6.69 (.02)
Personal Characteristics					
Being male	1.04 (.89)	2.31 (.005)	2.23 (.005)	2.35 (.004)	1.30 (.43)
Being FAE	1.15 (.66)	1.90 (.04)	2.26 (.009)	1.55 (.17)	3.07 (.002)

ISB, Inappropriate Sexual Behavior; DSE, Disrupted School Experiences; TWL, Trouble With the Law; CNF, Confinement; ADP, Alcohol/Drug Problems; LHI, Life History Interview.
[a]Factor was included as a nonlinear term.
Odds ratios were computed from fitted nonlinear logistic regression models (see Appendix). All models include a component that is a two-dimensional nonlinear function of Age at FAS/FAE Diagnosis and Age at LHI. Points at which to evaluate odds for Age at FAS/FAE Diagnosis were selected as ages of interest. Points at which to evaluate odds for the first two Environmental Factors were taken as 10th and 90th percentiles of observed distributions on these variables. Other Environmental Factors listed in Table 2 did not contribute significantly given the effects of those listed above. We note that there are competing models using alternate environmental factors that fit the data nearly equally as well; the stepwise algorithm selection is sensitive to particulars of the degree of nonlinear smoothing. However, the effects of the diagnostic factors are stable under alternative parameterizations. Approximate *p* values, given in parentheses, use conventional chi-square approximations to changes in the log-likelihood (or "deviance") divided by degrees of freedom. The *p* value for Age at FAS/FAE Diagnosis compares the two-dimensional nonlinear function of Age at Diagnosis and Age at LHI (representing approximately 6 d.f.) and the smaller (but not "nested") model nonlinear function only of Age at LHI (representing approximately 4 d.f.).

to have been in special education; 66% had been in a "resource room"; about 65% had received remedial help in each of reading and arithmetic; 29% had received life skills training; 21% had received occupational or physical therapy; 11% had therapeutic day care; and 7% had "infant stimulation."

IQ scores for patients with Fetal Alcohol Syndrome (FAS) and for those with Fetal Alcohol Effects (FAE) represent a wide range of intellectual abilities from profoundly retarded to above average IQ (Figure 1). There is an 8-point mean IQ discrepancy, with FAS lower than FAE. According to the usual cutoff score for developmental disability services (IQ < 70), only 13% of these patients with FAS/FAE would be served in the absence of other qualifying criteria (Figure 1). Group means are considerably below population norms for IQ, achievement, and adaptive behavior tests (Figure 2). Arithmetic and the Vineland Adaptive Behavior Scales (VABS) composite score are the areas of greatest functional deficit for these patients. For Arithmetic, the group means for FAS and FAE are each 2/3 of a normative standard deviation below their own mean IQ; for the Adaptive Behavior Composite, the group means for FAS and for FAE are about 1 to 1.5 normative standard deviations below their own mean IQ score (Figure 2).

Descriptive Data on Adverse Life Outcomes

Inappropriate Sexual Behaviors (ISB) on repeated occasions is the most frequent adverse life outcome across the life span (Figure 3a), increasing slightly with each age category from 39% in children to 48% in adolescents and 52% in adults. Among children, the most frequently mentioned repeated inappropriate sexual behaviors are exposing (20%) and inappropriate sexual touching (19%). Among adolescents and adults, those most frequently mentioned are promiscuity (26%) and inappropriate sexual advances (18%). Across all ages, there is little difference in the overall prevalence of ISB for males compared to females or for FAS versus FAE. However, promiscuity is

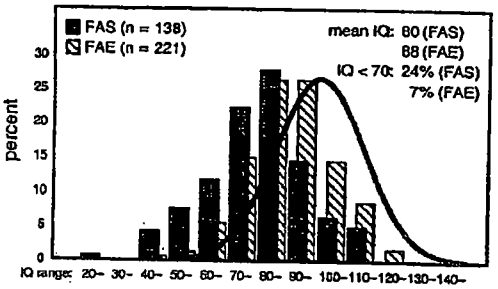


FIGURE 1. IQ Scores of Patients with Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE) superimposed on the normative curve for IQ. An IQ score of 100 is the mean of the population for IQ. The sample size for IQ is below 415 because IQ testing was not funded for this study, but was retrieved from research records as available.

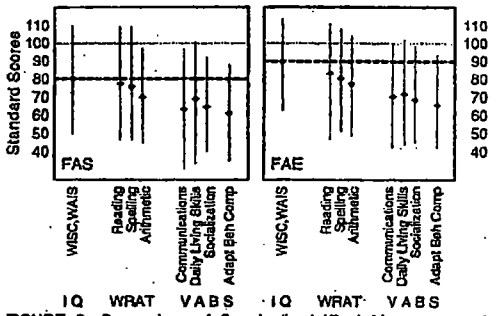


FIGURE 2. Comparison of Standardized IQ, Achievement, and Adaptive Behavior Scores for Patients with Fetal Alcohol Syndrome (FAS) (on the left) and those with Fetal Alcohol Effects (FAE) (on the right). Diamonds represent sample means for each group on each test. Vertical lines connect the 5th and 95th percentile for each group on each test. Bold dashed lines reflect sample IQ means for each group to facilitate comparison with sample means on achievement and adaptive behavior tests for each group. The dotted lines represent the population mean for IQ, achievement, and adaptive behavior tests. Data are from all subjects who have a Wechsler IQ score and additionally have at least one valid Wide Range Achievement Test (WRAT) or Vineland Adaptive Behavior Scale (VABS) score. N = 123 to 138 FAS, and 193 to 221 FAE.

mentioned for twice the fraction of females (22%) as males (11%). Among adolescents and adults, Trouble With the Law (TWL) for ISB was twice as frequent among males (19%) as females (8%).

Disrupted School Experience (DSE) was reported for 14% of school children and 61% of adolescents and adults (Figure 3a). About 53% of the adolescents with FAS/FAE had been suspended from school, 29% had been expelled, and 25% had dropped out. The most frequently mentioned learning problems were attention problems (70%) and repeatedly incomplete schoolwork (58%). The most frequently mentioned behavior problems in school were repeatedly having difficulty getting along with peers (58%) and repeatedly being disruptive in class (55%). The fraction of patients with DSE for whom these problems were mentioned was twice the fraction for the patients without DSE.

Trouble With the Law (TWL) is reported overall for 14% of children and 60% of adolescents and adults (Figure 3a). Among adolescent/adults the most frequently mentioned category of law violations are crimes against persons (45%), which includes shoplifting/theft (36%), assault (17%), burglary (15%), and domestic violence (15%). Among those with TWL, the fraction actually charged, arrested, and/or convicted increases with age from 13% for children to 67% for adolescents and 87% for adults.

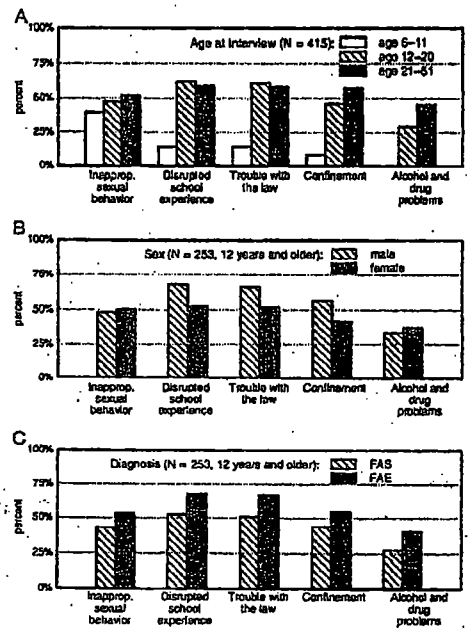


FIGURE 3. Lifespan Prevalence of Five Adverse Life Outcomes: (a) by Three Age Groups, (b) by Male vs Female, and (c) by Fetal Alcohol Syndrome (FAS) vs Fetal Alcohol Effects (FAE). Age group is defined by age at time of Life History Interview (LHI). n = 161 to 162 in the 6- to 11-year-old range, 160 to 163 in the 12- to 20-year-old range, and 87 to 90 in the 21 and older range. For adolescents/adults: n = 141 to 145 males and 106 to 108 females; n = 106 to 109 with FAS and 141 to 144 with FAE.

Confinement (CNF) is reported for 8% of children (all for psychiatric hospitalizations) and 50% of adolescents and adults (Figure 3a). Among adolescents and adults, the fraction reported ever incarcerated for a crime is 35%; ever hospitalized for psychiatric problems, 23%; ever hospitalized for alcohol and drug treatment, 15%.

Alcohol and Drug Problems (ADP) are mentioned for 35% of the patients 12 years and older (29% of the adolescents and 46% of the adults [Figure 3a]). Alcohol problems are mentioned more frequently than are drug problems (33% vs 23%). Almost all of the 57 patients reported to have used street drugs also are reported to have had alcohol problems. Among patients with both problems, alcohol abuse began on average two years before street drug use; 65% of those who abused alcohol went on to use street drugs.

The onset of these adverse outcomes began early for these patients. Problems at school generally began early in their schooling. The mean age of onset of ISBs was 9.6 years; of TWL, 12.8 years; and of ADP, 13.4 years.

Odds Ratios of Risk Factors for Adverse Life Outcomes

In these analyses, the risk factors are treated as binary variables in examining their impact on the five adverse life outcomes in adolescents and adults.

The odds of all five adverse life outcomes are increased 2- to 4-fold for Diagnosis After Age 12 (Table 2). Likewise, odds of all or almost all adverse outcomes are increased with a low percent of life in a Stable/Nurturing Home, fewer Years per Household by age 18, or ever being a Victim of Physical, Sexual Abuse, or Domestic Violence.

Logistic Regression Analysis

In these analyses, the environmental risk factors are treated as continuous (rather then binary) data and, through multiple regression analyses, we are able to adjust one for the other. Personal risk factors (male/female; FAS/FAE) are obviously binary variables.

Results of the logistic regression analyses (Table 3) examine more closely, and generally reinforce, the bivariate relationships depicted in Table 2. As subjects' age at diagnosis and age at the time of the Life History Interview (LHI) are confounded in this study design (the age at LHI being necessarily greater than the age at diagnosis), these analyses model the effects of that pair of age factors jointly. Environmental factors were selected by stepwise regression procedures. As in any multiple regression, the models estimate effects of diagnosis factors adjusting for significant environmental factors, and vice versa. See Appendix for details.

Table 3 shows that environmental factors are clearly important (highly significant) predictors of all five outcomes. For purposes of illustration, Table 3 reports estimated odds ratios pertaining to relatively extreme values (the 10th and 90th percentiles) of the first two quantitative environmental risk factors. However, the *p* values pertain to the *overall* contributions of the risk factors in the logistic regressions; they are not a function of the particular levels of the risk factors chosen for the computation of the odds ratios in Table 3.

A stable/nurturing home is the most influential protective factor in these analyses, reducing by three- or four-fold the risk of four of the five adverse outcomes examined. In other words, the smaller the percent of life that patients with FAS or FAE spent in stable/nurturing homes, the greater the risk that these patients as adolescents and adults would have more ISB, DSE, ADP, and TWL. Being a victim of sexual, physical abuse or violence also increases the odds for two adverse outcomes (Inappropriate Sexual Behavior and Alcohol/Drug Problems). Years per household was the single significant environmental risk factor for one adverse outcome, namely Confinement.

The nonlinear modeling[23,24] of the effect of age at diagnosis (see Appendix) shows the greatest change in risk roughly over the period from 9 to 17 years, hence our choice of these particular years for computing odds ratios. As the effect of age at diagnosis depends on age at LHI, (which is necessarily greater than the age at diagnosis) we report the estimated odds ratio for an adolescent 18 years old at the time of the LHI and for an adult 26 years old at the LHI.

For an adolescent at 18 years of age, older age at diagnosis (17 vs 9 years) nearly doubles the estimated odds of DSE, TWL, and CNF. For an adult of 26 years of age, older age at diagnosis more than doubles the estimated odds of ISB, DSE, and ADP. (The relatively high odds ratio for ADP for adults is significant but not precisely estimated.)

The coefficients for personal characteristics in Table 3 agree well enough with the simple odds ratios in Table 2. Specifically, "being FAE" and "being male" puts one at higher risk of DSE and TWL. Those with FAE are also at higher risk of ADP while males are also at higher risk of CNF. There is no significant difference between males and females with respect to their risk for ISB and ADP, and there is no difference between FAS and FAE in their risk of ISB and CNF. No interactions were detected between the FAS vs FAE diagnosis factor and any of the other factors in the logistic regression models. Note that IQ < 70 is not in these regressions. That is because it is confounded with FAS in some cases. Note from Table 2 that the effect of IQ is actually protective: the lower the IQ, the better the outcome for 4 of the 5 adverse outcomes.

DISCUSSION

Evaluation of these 415 patients who have either Fetal Alcohol Syndrome (FAS) or Fetal Alcohol Effects (FAE) reveals a lifetime prevalence of adverse life outcomes that documents more specifically the poor prognosis that has been suggested by previous studies.[8,25–29] The understanding that both FAS and FAE are birth defects caused by maternal alcohol abuse, and that 80% of these patients are not raised by their biological mothers, makes it impossible to consider the outcomes of these patients without simultaneously considering their rearing environments. One of the strongest correlates of adverse outcomes is lack of an early diagnosis: the longer the delay in receiving the diagnostic information, the greater the odds of adverse

outcomes. An early diagnosis allows capable caring families to effectively advocate for their children's needs.

Psychological testing of this large group of patients revealed two striking deficits that have been described previously in small samples,[8,30–32] namely specific arithmetic disability and specific problems with adaptive behavior. Although group means for Reading and Spelling are in line with expectations based on mean IQ, the mean Arithmetic standard scores are 2/3 of a standard deviation below mean IQ scores (Figure 2). This finding, which was found for both patients with FAS and those with FAE, suggests a functional deficit in problem solving that should trigger specific interventions. Even more striking is the finding that the mean Adaptive Behavior Composites for FAS and FAE respectively, are far below the normative population mean and 1 and 1.5 normative standard deviations below their respective mean IQ scores (Figure 2). This is a debilitating functional deficit that would probably not be detected in routine IQ testing. This profile suggests a group of patients born with a birth defect who are in apparent need of special services in school and of specialized life skills and job skills training as young adults. As only 24% of those with FAS and 7% of those with FAE have an IQ below 70 (Figure 1) (the traditional qualification for special services), it is clear that these patients will have difficulty getting appropriate services in their schools and in their communities. The 60% rate of Disrupted School Experiences (DSE) among adolescents and adults in this study substantiates what has happened to them historically.

The finding that patients with a diagnosis of FAE, who lack the primary physical stigmata of FAS, nevertheless have clear intellectual, academic, and adaptive behavior deficits, confirms earlier reports [8,33,34] and highlights the particular need for broader recognition and understanding of the full spectrum of fetal alcohol effects. As Figure 3b demonstrates, patients with FAE have higher rates of all five adverse outcomes compared to those with FAS. The finding that having FAE (compared to FAS) doubles the odds of Trouble With the Law (TWL) and Alcohol and Drug Problems (ADP) (Tables 2 and 3) is puzzling until one considers that for many years FAE was thought to be essentially a "nondiagnosis" in contrast to FAS. While beyond the scope of this paper, diagnostic nomenclature is an area in great need of further clarification. This may occur with improved brain/behavior studies such as those now underway[35,36] showing good sensitivity and specificity for detecting patients with both FAS and FAE from age and sex matched controls, and showing comparable deficits in both FAS and FAE.

The only adverse life outcomes we measured that continue to increase into adulthood are Alcohol/Drug Problems (ADP) and Confinement (CNF) (Figure 3a). We identified three strong protective factors against ADP: high percent of life in a Stable/Nurturing Home; younger age at diagnosis, and a diagnosis of FAS (vs FAE) (Table 3). These findings are congruent with clinical experience, where we find that stable, nurturing families with a child diagnosed early, are better able to plan effectively for the transition from adolescence to adulthood, and to maintain as close a relationship with their child in young adulthood as seems necessary. Furthermore, patients with FAS often have an easier time qualifying for developmental disabilities services and SSI benefits than do those with FAE.

One specific entry in Table 3 is of great societal importance: the contribution of being a victim of physical/sexual abuse and violence to Inappropriate Sexual Behaviors (ISB). Physicians who encounter Inappropriate Sexual Behaviors (ISB) among patients with either FAS or FAE should consider the possibility of victimization. The relation between being a victim of violence and being a subsequent perpetrator of violence is a particularly problematic clinical area in the management of these organically brain-damaged adolescents and adults.

Females and males in this study have comparable rates of Inappropriate Sexual Behaviors (Figure 3b), but the associated consequences may differ, as nearly as we can evaluate them from these data and from our clinical experience. Almost all (50 of 53, or 94%) of the females who had Inappropriate Sexual Behaviors also had experienced sexual, physical abuse or violence against themselves, and 57% of these also had Alcohol/Drug Problems. The types of inappropriate sexual behaviors most often reported for these adolescent and adult females (promiscuity and inappropriate sexual advances) put them at risk of unplanned pregnancies. Thus, Inappropriate Sexual Behaviors, along with the increased odds of Alcohol/Drug Problems, increase the chances of females with either FAS or FAE producing additional alcohol-affected children.[37] In clinical practice, either the child or the mother or both might be fetal alcohol affected and in need of help with family planning as well as other special services.

Males have a higher rate of Disrupted School Experience, Trouble With The Law, and Confinement than do females (Tables 2 and 3), and these three adverse outcomes appear linked. We have complete data on 140 male adolescents and adults: of the 67% who have disrupted school experiences, 83% have been in trouble with the law and, among these, 69% have been incarcerated in jail or prison. In contrast, of the 33% males who have not had Disrupted School Experiences, only 30% have been in Trouble With the Law, and 36% of these have been incarcerated. Support for families advocating to keep adolescents in school and advocating for schools to meet the special needs of these patients, may well be protective against other adverse life outcomes. A recent follow-up study of patients with FAS in Germany, where support services for all disabled children are more comprehensive than those in the United States, found that the patients were not unlike ours in many developmental outcomes but were seldom in trouble with the law.[29]

While these findings have some degree of face validity, caution is always necessary in interpretation. Cause and effect are not clear in cross sectional studies of correlated events. For example, there may be no causality between confinement and household changes (Table 3): being removed from a household and being confined may be competing societal responses to the same undesirable or maladaptive behaviors representing Central Nervous System (CNS) deficits from prenatal alcohol exposure. Fine-grained temporal causality cannot be established by retrospective interviews or multiple regression analyses. The inevitable interactions among the adverse outcomes

themselves also limit interpretation. A person who has dropped out of school is probably more likely to develop alcohol and drug problems and then to get in trouble with the law even in the absence of either FAS or FAE.

Finally, we remind the reader that this is a referred clinical sample, and as such, cannot be considered representative of all people born with FAS or FAE, or even all of those who have a diagnosis of FAS or FAE; this limits the generalizability of the findings. (For example, an adverse life outcome could have been the reason for referral to a diagnostic evaluation in the first place.) Except for those diagnosed at birth whose alcohol exposure history was the trigger for evaluation, there is always the possibility that the manifest problems of the patients brought them to the attention of a diagnostician. Additionally, both the inherent developmental disabilities and the personal characteristics associated with this CNS condition may have contributed to those affected offspring who are brought to a FAS clinic for evaluation. Looking at our data historically, we see that the severely retarded patients were disproportionately diagnosed in the 1970s when the diagnosis was new and before the Surgeon General's (1981) warning on abstaining from alcohol during a pregnancy or when planning a pregnancy.[38] Retarded patients who are already being adequately served in the community may be less likely to be brought in for a diagnostic evaluation now.

Despite the apparent simplicity of Table 2, the family rearing environment for these patients is diverse and multidimensional. Consistent with other studies,[8,39] only 20% of these children were raised by their biological mothers. We found that some mothers achieved sobriety, while others did not. Some achieved sobriety because they had an affected child, others died before knowing their child was affected. Some adoptions occurred at birth, others years later. Some foster families raised children to adulthood without officially adopting them. Other children were in a succession of different households for a variety of reasons, some of which (like behavior problems) stemmed from the child, while others (like aging foster parents) were not child-related. The families, whether biologic, adoptive, or foster, varied widely in both education and finances. To capture and quantify this complexity and richness of life experience, this study demanded an instrument that would produce, on the one hand, a current "snapshot" of each patient's life, and, on the other, a "moving picture" quantifying each patient's unique environmental and personal characteristics from past to present. As we could not find a suitable instrument, we developed our own Life History Interview (LHI). The fact that we did not use a standardized instrument is a limitation in terms of the absence of reliability and validity data, and in terms of replication, but a study of this scope and magnitude could not have been done without it.

While this study has by design focused on adverse outcomes and risk/protective factors in the environment and in the patients themselves, the larger goal of the study is to help families and communities raise the next generation of people born with this birth defect with more understanding, to protect them from the hazardous straits into which they are born, and to provide access to appropriate services so that each can develop to his/her own best potential. This paper suggests that a fetal alcohol diagnosis is important to this goal. We believe that more physicians can play a central role in detecting FAS and FAE and in either making the diagnosis or taking steps to obtain a diagnosis.

We have previously argued that the physician can play an important role in the recognition of FAS among disabled youth and adults.[8] With the work of the past decade, we now propose that the physician be sensitive to the possibility of fetal alcohol problems among children of all ages, as well as youth and adults. We also propose that the physician not just focus on patients with the full FAS, but also be sensitive to the clinical population of patients who are here classified as FAE. They may not display the full clinical characteristics of FAS, yet may have equally heavy prenatal alcohol exposure and be equally impaired. A word on nosology is important here. FAS is characterized by a unique pattern of facial features, growth deficiency of prenatal origin, and some manifestation of central nervous system (CNS) dysfunction. The children here classified as FAE by the dysmorphologists who originally examined them were most usually missing the full facial features and/or the growth deficiency. This paper and others, have demonstrated the importance of examining those suspected of either FAS or FAE, as both may have CNS deficits, and as this paper shows, both may have adverse life outcomes that could presumably be prevented or ameliorated by appropriate interventions. Since these patients were diagnosed, the term FAE is less frequently used. The IOM[7] suggested the term "partial FAS" for patients meeting certain criteria, but also coined the new term "Alcohol-Related Neurodevelopmental Disorders" (ARND) to describe children who had heavy prenatal alcohol exposure along with CNS, neurodevelopmental abnormalities, and/or a complex pattern of behavior or cognitive abnormalities shown through clinical or animal research to be linked to prenatal alcohol. We have found ARND to be a helpful descriptive term in a study of FAS incidence and ARND prevalence.[5] Another classification system developed by Astley and Clarren,[40] the 4-Digit Diagnostic Code, attempts to quantify four primary characteristics (face, growth, CNS, and exposure) according to specific criteria with the idea of developing a consistent "case definition" across clinicians. The system yields a diagnosis of FAS and of static encephalopathy, alcohol exposed. While the two latter systems are attempts at defining specific diagnostic categories, another new term has been coined to allow one to speak of individuals classified by any of these systems as alcohol-affected. The term "Fetal Alcohol Spectrum Disorders" (FASD)[41] is an umbrella term for FAS, FAE, ARND, Partial FAS, and Static Encephalopathy, Alcohol Exposed. The children described in this paper could be called FASD, however, it seemed more accurate to describe them by their original diagnostic nomenclature. While this paper is not about how to diagnose, it clearly points to the value of a diagnosis in ameliorating the postnatal risk factors for adverse life outcomes.

In summary, this study documents the adverse postnatal environments and the corresponding risk of adverse life outcomes among many patients diagnosed FAS or FAE. These include major disruptions in schooling, trouble with the law, inappropriate sexual behaviors, extensive confinements,

and alcohol and drug problems. Adverse life outcomes are not restricted to those with or without the classic facial features of FAS or to those with or without mental retardation. We find that good stable families, with enduring relationships with their children with FAS/FAE, appear to be a critical protective factor for helping children avoid adverse life outcomes.

We also observed a significant reduction in the risk of adverse life outcomes with an earlier diagnosis. We therefore believe that physicians can be important facilitators of improved outcome for patients born with FASD: by systematic querying about maternal alcohol history in patients of any age suspected of alcohol-related disabilities, by making the diagnosis themselves and/or by making appropriate referrals as indicated, by encouraging parents to take an active role in advocating for their children at school and in the community, and by using their experience to improve community supports.

These results suggest that the risk of adverse outcomes might be reduced by families, communities and physicians working together to assure that children with FASD are raised in long-lasting, stable, nurturing homes, and by providing the rearing families with a meaningful diagnosis for their child as early as possible in life.

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APPENDIX

We fitted nonlinear logistic regression models using stepwise model building procedures for "generalized additive models" available in the S-PLUS statistical analysis system.[23,24] These models consider the log odds of adverse life outcomes as possibly nonlinear functions of quantitative risk/protective factors using nonparametric regression procedures, rather than assuming that the log odds are linearly related to a variable such as age of diagnosis (the assumption in conventional logistic regressions). They permit us to identify ranges in the values of a risk/protective factor over which the log odds of an adverse outcome increase or decrease most rapidly, or do not change at all. The degree of smoothness or nonlinearity was specified subjectively. The stepwise model-building algorithm chooses a "best" set of predictors from among those listed in Table 2 on the basis of the common Akaike Information Criterion (AIC). It chooses not only whether to include a predictor, but in the case of quantitative factors, whether to include it as a linear or a nonlinear effect. As this model-building selects from among competing, correlated risk/protective factors on empirical (not theoretical) grounds, the end result should not be considered a "true model" but an analysis useful for assessing the effects of one risk/protective factor adjusting for other competing or confounding variables.

Two of the variables listed in Table 2, age of diagnosis and age at the Life History Interview (LHI), are necessarily strongly correlated in this study design as age at the LHI is greater than or equal to the age at diagnosis. Furthermore, the effects of these two variables should not be considered separately (as one does in the usual "additive" regression model without interaction terms) as the possible effect of age at diagnosis on the log odds of an adverse life outcome will almost certainly depend, to some extent, on the age of the subject at the LHI interview, especially in view of the older adult subjects. We therefore used the "loess" function in S-PLUS to characterize log odds as a general (nonadditive) function of these two age-related variables. For computational reasons, we chose to estimate this joint effect in terms of age at diagnosis and years postdiagnosis (age at LHI minus age at diagnosis).

The joint effect of age at diagnosis and age at the time of the LHI, sex, the indicator of the diagnosis Fetal Alcohol Syndrome (FAS) versus Fetal Alcohol Effects (FAE), and the interaction of these two binary factors (representing 4 sex/diagnosis cohorts) were included in all models. The stepwise regression procedure was used only to determine a "best" set of other environmental measures to include for explanation of the rate of adverse life events. Once a model with environmental effects was selected, we assessed whether the other risk/protective factors played equivalent roles for both FAS and FAE subjects by examining interactions between the FAS/FAE diagnosis indicator and other terms in the model.

The significance of terms in the regression models, reported in Table 3 with p values, was assessed using the conventional chi-squared approximation to the contribution of the term to the fit of the model—the change in log-likelihood or "deviance"—divided by the degrees of freedom for the term. The nonlinear terms in these models (the number of years per household and the joint effects of age at diagnosis and age at the LHI) are represented by non-integer degrees of freedom as explained in Hastie and Tibshirani.[24] To assess the significance of age at diagnosis we compared the model with the joint effect of age at diagnosis and age at LHI with a model including only a (nonlinear) effect of age at LHI.

Finally, in order to facilitate interpretation of the resulting multiple regression models and to compare these with the unadjusted odds ratios in Table 2, we computed model-based odds ratios with respect to selected pairs of values of the risk/protective factors (e.g., to compute an odds ratio comparing patients diagnosed at age 9 with patients diagnosed at age 17).

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Direct and Indirect Effects of Prenatal Alcohol Damage on Executive Function

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Patients with Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE) often have difficulty functioning appropriately in everyday life and seem to employ poor problem-solving strategies. Tests of executive function are relevant for quantifying the functional deficits and underlying real-life problems associated with prenatal alcohol exposure. This study considers two pathways for the effects of prenatal alcohol on executive function: a direct effect and an indirect effect through prenatal alcohol's effect on IQ. We compared 30 men who had been diagnosed with FAS or FAE with young adults participating in a longitudinal prospective study ($n = 419$) and 15 control participants that comprised a comparison group. This study is unique in its analysis of the same battery of assessments of executive function in both a large low dose longitudinal study sample and a clinically diagnosed group. Participants were evaluated on 9

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tests (including 58 scores) of executive function. For some but not all of the tests in this executive function battery, the decrement in the alcohol exposure group is greater than would be predicted from their IQ scores. We found that 3 of 6 Stroop scores, 2 of 4 Trails scores, 9 of 16 Wisconsin Card Sorting scores, 1 of 2 Ruff's Figural Fluency scores, and 2 of 4 Consonant Trigrams scores appear to be particularly sensitive to the direct effects of prenatal alcohol damage for patients with FAS and FAE. The findings suggest that these executive function tests would be particularly useful in clinical evaluations of persons suspected of fetal alcohol damage because they would not simply reflect deficits in IQ or facial stigmata.

Patients with Fetal Alcohol Syndrome (FAS) and Fetal Alcohol Effects (FAE) are often clinically characterized by behaviors resembling those for patients diagnosed with frontal lobe lesions. This is exemplified by descriptions of two individuals, one of a young man diagnosed with FAS and another of a young woman with known frontal lobe damage. RW is a 26-year-old man in our study who was born to a mother who abused alcohol during pregnancy. He was diagnosed with FAS at the age of 3. DT was 7 years old when she underwent surgery for a left frontal subarachnoid hemorrhage (Eslinger, Grattan, Damasio, & Damasio, 1992). When tested as adults, both RW and DT had IQ scores broadly within the average range. However, both RW and DT had difficulties with cognitive flexibility, maintaining set, planning and organizing information, utilizing feedback to alter behavior, and they displayed a perseverative response style. Behaviorally and socially, both had great difficulty maintaining employment because of poor judgement and inability to stay focused on tasks, and required assistance with many daily living skills including hygiene, structuring leisure time, and managing finances. Both RW and DT had great difficulty with relationships due to poor impulse control and decision-making skills. The pervasive impact of RWs disorder seems quite similar to that of DTs, no matter the difference in etiology, and a central theme appears to be an extensive deficit of executive function (EF), regardless of level of measured intelligence.

A number of researchers have defined EF as a group of cognitive abilities that include (a) self-regulation of behaviors, (b) sequencing of behaviors, (c) cognitive flexibility, (d) response inhibition, (e) planning, and (f) organization of behavior (Eslinger, 1996). Denckla (1996) suggested that executive functions are "future-oriented" processes. Most researchers agree that EFs are not basic cognitive processes such as motor activity, sensation, perception, attention, or memory, but instead involve the integration of the more basic cognitive processes and thus depend on their development. In other words, good EF depends on intact basic cognitive functioning. EF is of particular interest in evaluating people with prenatal alcohol effects because poor judgement and failure to consider consequences are often described clinically in this population.

According to Denckla (1996), EF is an integrative process, so it can sometimes be affected by performance on other tasks that reflect general intelligence and

other cognitive processes. For example, in the presence of high IQ, tasks of EF may be too easy and thus may not be sensitive to EF deficits that are subtle. Denckla described a complex overlap between general intelligence and EF, noting that Performance IQ, which emphasizes fluid intelligence and timed tasks, may be more closely related to EF than Verbal IQ, with its emphasis on more crystallized intelligence. In addition to the effects of IQ, individual tasks considered to tap EF, such as the Stroop task, can be impacted by other abilities, such as literacy.

EF deficits have been identified in many clinical populations (Arnett et al., 1994; Channon, 1996; Dise & Lohr, 1998; Eslinger et al., 1992; Foong et al., 1997; Hutton et al., 1998; Levin et al., 1997; Pantelis et al., 1997; Raskin & Rearick, 1996; Romans, Roeltgen, Kushner, & Ross, 1997; Shoqeirat, Mayes, MacDonald, Meudell, & Pickering, 1990). In addition, several studies have assessed EF deficits in populations of individuals prenatally exposed to alcohol. Kodituwakku and colleagues utilized the Wisconsin Card Sorting Test (WCST), Controlled Oral Word Association Test (COWAT), and a tower planning test with a small group of children with FAS and FAE (Kodituwakku, Handmaker, Cutler, Weathersby, & Handmaker, 1995). They found that children with FAS and FAE correctly identified significantly fewer categories and made significantly more perseverative errors on the WCST, generated significantly fewer words on the COWAT, and had significantly poorer performance on the tower test than did controls. A study using the D–KEFS (a battery of tests including versions of Trail Making, Stroop, Tower, and Word Context tests) was administered to children diagnosed with FAS or Prenatal Exposure to Alcohol and nonexposed controls (Mattson, Goodman, Caine, Delis, & Riley, 1999). This study found that alcohol-exposed participants, regardless of diagnosis, performed more poorly than control participants, with specific impairments in planning, response inhibition, abstract thinking, and flexibility. Another study compared performance on the WCST of children exposed to alcohol prenatally, children diagnosed with attention deficit hyperactivity disorder (ADHD), and control children (Coles et al., 1997). Children diagnosed with FAS or FAE completed significantly fewer categories than did either children with ADHD or controls. Adolescents and adults with FAS and FAE made many more bizarre responses than did control participants on a cognitive estimation task (Kopera-Frye, Dehaene, & Streissguth, 1996).

Very few of the studies we reviewed considered IQ effects and their complex overlap with EF (Denckla, 1996). However, we know from the literature that prenatal alcohol directly depresses IQ (Mattson, Riley, Grambling, Delis, & Jones, 1997; Streissguth et al., 1991; Streissguth, Barr, Sampson, Darby, & Martin, 1989) and thereby may indirectly depress EF. In addition, alcohol may directly affect at least some aspects of EF even more than would be predicted by the alcohol-related IQ deficit alone. Therefore, it is important to assess not only the indirect effects of EF through the mediation of IQ but also to determine the direct effects of alcohol damage on EF.

This study examines the direct effect of prenatal alcohol damage on EF, and also the indirect effect via IQ, in two groups of adult participants. One group, from a large population-based study, has available measures of prenatal alcohol exposure; the other group, from a clinic-based study, includes adults previously diagnosed with FAS or FAE, along with controls. All participants were examined on nine tests thought to be sensitive to EF and on a standardized IQ test. These nine tests yielded 58 EF outcome measures (Table 1). Each of these tests was examined with respect to IQ using data from both studies to compute coefficients reflecting the direct effects of prenatal alcohol damage as expressed in the FAS and FAE diagnosis according to a simple path model. These coefficients were used as weights to compute a composite EF outcome score embodying direct as well as indirect effects (Table 2).

METHOD

Participants

Participants from two studies who were administered an identical battery of tests were included in this analysis. The first study, a longitudinal prospective study, examined a cohort of 419 young adults between 20.8 and 23.4 years of age who had been followed since birth. Participants were originally selected based on maternal report of alcohol use during pregnancy. Pregnant women who were receiving prenatal care were interviewed in the 5th month of pregnancy about alcohol use patterns before pregnancy and during pregnancy. The participants from the prospective study have widely varying prenatal alcohol exposure levels, across the whole spectrum of use (Streissguth, Barr, Bookstein, Sampson, & Carmichael Olson, 1999), with relatively few at levels believed necessary for outcomes that can be diagnosed as FAS or FAE (Sampson et al., 1997). Only two members of this cohort had been identified at birth with FAS.

The second study, a clinic-based study, examined 45 men, including 15 control participants, 15 participants who were diagnosed with FAS, and 15 who were diagnosed with FAE, by experienced dysmorphologists according to established criteria (Clarren & Smith, 1978). The diagnosis of FAS was made when the participant presented with a triad of symptoms including specific facial stigmata, growth deficiency, and evidence of central nervous system (CNS) dysfunction or damage in the presence of significant alcohol exposure during pregnancy. The facial stigmata of FAS include short palpebral fissures (smaller than normal eye openings), a thin upper lip, and an indistinct philtrum (ridges just above the middle of the upper lip; Stratton, Howe, & Battaglia, 1996). By contrast, FAE was diagnosed when there was evidence of significant prenatal alcohol exposure and the presence of CNS dysfunction or damage but there was not sufficient evidence of facial anomalies or

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TABLE 1
Means and Standard Deviations of Executive Functioning Scores for the FAS and FAE Study ($N = 45$) and the Longitudinal Prospective Study

	FAS and FAE Study							
	FAS[a]		*FAE*[a]		*Controls*[a]		*Longitudinal Prospective Study*[b]	
Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Wechsler Adult Intelligence Scale–Revised (WAIS–R)								
VIQ	80.3	10.4	84.7	8.9	111.3	15.5	99.4	12.0
PIQ	91.7	17.1	93.7	14.9	111.1	16.3	104.8	13.6
FSIQ	83.9	12.9	87.1	9.8	112.7	16.6	101.9	12.5
Wisconsin Card Sorting Test (WCST)								
WCST1stCAT	15.7	8.5	15.1	7.8	14.4	6.9	13.5	8.7
WCSTSETFAIL	0.9	1.6	1.2	1.5	1.1	1.3	0.8	1.1
WCSTLTOL	−6.1	11.9	−3.7	7.1	0.9	2.6	−0.7	4.2
WCST#CAT	4.1	2.0	5.0	1.6	6.0	0.0	5.8	0.9
WCST#COR	70.3	19.1	73.8	12.4	75.3	10.8	71.7	9.6
WCST#CONCEP	58.6	27.1	64.5	15.7	72.3	10.3	68.0	10.0
WCST#ERROR	43.1	28.8	33.5	21.1	16.6	7.2	17.0	13.3
WCST#NPERROR	24.7	18.4	17.9	12.2	9.5	5.3	8.7	8.3
WCST#PERROR	18.4	13.5	15.6	11.0	7.1	3.6	8.3	5.8
WCST#PRESP	20.0	15.6	17.3	12.7	7.3	3.9	8.8	6.7
WCST%CONCEP	55.1	28.1	62.7	18.5	79.3	8.3	78.8	12.9
WCST%ERROR	35.6	20.6	29.6	14.7	17.5	5.9	17.7	9.2
WCST%NPERROR	20.4	13.5	15.7	8.7	9.9	4.6	8.9	6.1
WCST%PERROR	15.3	9.8	13.9	7.6	7.7	3.5	8.8	4.1
WCST%PRESP	16.6	11.3	15.5	9.0	7.9	3.8	9.3	4.8
WCST#TRIALS	113.4	20.0	107.3	18.8	91.9	15.1	88.6	18.0

(*continued*)

TABLE 1 (*Continued*)

	FAS and FAE Study							
	FAS[a]		*FAE*[a]		*Controls*[a]		*Longitudinal Prospective Study*[b]	
Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Cognitive Estimation (CE; folded rank scores)								
CE-01	119.5	81.2	76.2	64.7	94.6	68.2	104.6	61.0
CE-02	155.1	95.7	153.4	85.6	96.2	99.1	89.9	87.7
CE-03	141.9	55.8	119.3	46.5	64.2	42.6	102.5	64.2
CE-04	106.2	92.0	139.4	71.6	106.8	79.9	106.2	68.6
CE-05	127.5	71.3	114.6	59.5	118.3	62.4	106.0	59.7
CE-06	123.7	65.3	107.4	54.0	96.3	61.0	104.4	60.1
CE-07	56.4	96.8	0.0	0.0	0.0	0.0	1.5	18.0
CE-08	143.0	74.7	129.5	47.4	121.6	59.8	103.9	67.5
CE-09	132.7	71.1	127.2	89.8	103.7	70.6	101.2	73.1
CE-10	141.0	66.9	90.8	91.9	86.8	76.4	89.9	76.4
CE-11	122.8	74.2	111.1	64.9	97.6	54.4	105.9	60.2
CE-12	147.7	80.6	141.6	55.8	86.4	76.2	103.6	65.7
CE-13	127.7	66.8	116.8	78.6	122.8	77.2	106.2	67.9
CE-14	110.5	72.2	108.8	77.1	93.4	66.1	104.5	65.4
CE-15	135.4	70.7	124.5	72.5	79.3	64.0	101.7	68.8
Controlled Oral Word Association Test (COWAT)								
COWATF	9.2	3.8	11.7	5.0	15.3	3.9	13.9	4.3
COWATA	7.8	2.5	8.7	3.8	13.3	4.2	11.7	4.2
COWATS	11.3	4.4	11.6	3.6	16.9	5.5	15.4	4.5
COWATTOT	28.3	9.1	32.1	10.6	45.5	11.8	40.9	11.1
COWATANIM	17.9	5.0	19.5	5.5	24.4	4.6	23.1	5.2

Ruff's Figural Fluency Test (RFF)								
RFFPERSEV	8.1	10.4	6.8	7.7	5.1	4.9	5.3	5.3
RFFUNIQUE	65.3	20.7	65.3	24.7	111.3	20.7	96.0	22.7
Trail Making Test								
TRLAERROR	0.1	0.3	0.5	0.6	0.2	0.6	0.2	0.5
TRLASECONDS	38.1	15.9	36.1	11.6	23.3	6.6	25.0	8.1
TRLBERROR	0.9	1.8	1.1	1.2	0.1	0.3	0.4	0.8
TRLBSECONDS	100.2	51.2	103.1	28.4	55.2	20.5	57.8	24.6
Stroop Color–Word Test								
STROOPW	85.5	16.2	80.1	13.8	105.7	14.6	103.2	15.5
STROOPWERR	0.7	1.2	1.1	1.0	0.5	0.8	0.5	0.9
STROOPC	60.8	16.4	57.7	12.0	74.5	9.7	74.8	12.4
STROOPCERR	1.8	1.7	1.4	1.8	0.9	0.9	1.0	1.1
STROOPCW	33.9	10.7	31.1	8.3	45.6	7.9	45.8	10.0
STROOPCWERR	2.2	1.7	2.5	2.7	1.2	1.1	1.6	1.5
Consonant Trigrams Test (CTT)								
CTT03	10.9	3.4	10.5	2.3	13.2	2.5	12.8	2.2
CTT09	7.1	2.9	8.1	3.0	11.0	3.1	10.4	3.0
CTT18	5.1	2.8	5.5	2.5	8.8	3.1	8.8	3.3
CTTPOS	32.9	8.1	33.2	6.3	44.7	8.6	43.3	8.0
Digit Span (DS; WAIS–R)								
DSSCALE	7.0	2.9	7.5	2.2	12.0	2.9	10.5	2.6

(continued)

TABLE 1 (*Continued*)

	FAS and FAE Study							
	FAS[a]		*FAE*[a]		*Controls*[a]		*Longitudinal Prospective Study*[b]	
Measures	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
California Verbal Learning Test (CVLT)								
CVLTPERSEV	5.4	5.0	5.7	4.4	5.6	4.6	5.5	4.8
CVLTASEM	14.9	7.4	11.1	8.4	21.7	9.3	25.5	13.2
CVLTLDSEM	3.1	3.3	4.3	2.7	7.5	2.7	7.5	3.3
CVLTSDSEM	2.8	2.8	3.9	3.3	6.7	2.7	6.9	3.2
CVLTINTRUS	9.4	10.8	4.7	4.2	1.7	1.5	2.5	3.9

Note. FAS = Fetal Alcohol Syndrome; FAE = Fetal Alcohol Effects; VIQ = verbal IQ; PIQ = performance IQ; FSIQ = full scale IQ; WCST1stCAT = number of first category correctly identified; WCSTSETFAIL = number of set breaks; WCSTLTOL = the learning to learn factor; WCST#CAT = number of categories correctly identified; WCST#COR = number of correct responses; WCST#CONCEP = number of conceptual level responses; WCST#ERROR = number of errors; WCST#NPERROR = number of nonperseverative errors; WCST#PERROR = number of perseverative errors; WCST#PRESP = number of perseverative responses; WCST%CONCEP = percentage of conceptual level responses; WCST%ERROR = percentage of errors; WCST%NPERROR = percentage of nonperseverative errors; WCST%PERROR = percentage of perseverative errors; WCST%PRESP = percentage of perseverative responses; WCST#TRIALS = number of trials completed; COWATF = number of words: "F" trial; COWATA = number of words: "A" trial; COWATS = number of words: "S" trial; COWATTOT = total words generated from letters; COWATANIM = number of animal words generated; RFFPERSEV = total number of perseverative errors; RFFUNIQUE = number of unique designs; TRLAERROR = number of errors for the Trails A; TRLASECONDS = total amount of time to reach the goal; TRLBERROR = number of errors for the Trails B; TRLBSECONDS = total amount of time to reach the goal; STROOPW = number of correct responses in a 45-sec time period when reading colors; STROOPWERR = number of errors reading color words; STROOPC = number of correct responses naming color of ink; STROOPCERR = number of errors made naming colors; STROOPCW = number of correct responses when naming colors ignoring printed words; STROOPCWERR = number of errors naming colors ignoring printed words; CTT03 = total number of letters remembered, 3-sec delay; CTT09 = total number of letters remembered, 9-sec delay; CTT18 = total number of letters remembered, 18-sec delay; CTTPOS = total number of letters placed in the correct sequence; DSSCALE = scaled score of both conditions combined; CVLTPERSEV = number of perseverations; CVLTASEM = semantic clustering; CVLTLDSEM = long-delay semantic clustering; CVLTSDSEM = short-delayed semantic clustering; CVLTINTRUS = number of intrusions.

[a] $n = 15$. [b] $n = 419$.

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TABLE 2
Performance Versus Expectation Given IQ for 58 Executive Function Scores and Their Weights

Test Score	*Better Than Expected*	*Worse Than Expected*	*Weight*[a]
STROOPW	5	24	0.71
STROOPCW	5	24	0.68
TRLBSECONDS	6	23	0.65
TRLASECONDS	9	20	0.65
WCST#ERROR	6	23	0.60
WCST#NPERROR	7	22	0.58
WCST%ERROR	6	23	0.57
WCST%NPERROR	7	22	0.55
WCST#TRIALS	9	20	0.55
WCST%CONCEP	6	23	0.55
RFFUNIQUE	8	21	0.54
WCST#PERROR	9	20	0.53
STROOPC	6	23	0.53
CTTPOS	7	22	0.52
WCST#PRESP	9	20	0.52
WCST#CAT	15	14	0.50
CTT18	6	23	0.49
WCST%PERROR	9	20	0.46
WCST%PRESP	8	21	0.45
CVLTASEM	11	18	0.44
CVLTSDSEM	10	19	0.44
CVLTLDSEM	11	18	0.43
DSSCALE	8	21	0.43
CVLTINTRUS	12	17	0.41
CE–08	10	19	0.38
CE–15	8	21	0.36
COWATTOT	12	17	0.35
CTT09	10	19	0.35
WCSTLTOL	7	22	0.32
CE–05	12	17	0.31
COWATS	13	16	0.30
COWATANIM	10	19	0.26
WCST1STCAT	14	15	0.26
COWATF	11	18	0.25
CE–07	25	4	0.23
RFFPERSEV	13	16	0.22
CE–02	13	16	0.18
COWATA	13	16	0.17
CE–12	13	16	0.16
CTT03	9	20	0.14
CE–11	14	15	0.11
CE–03	12	17	0.11

(*continued*)

TABLE 2 (*Continued*)

Test Score	*Better Than Expected*	*Worse Than Expected*	*Weight*[a]
CE–10	12	17	0.09
STROOPWERR	14	15	0.08
STROOPCWERR	14	15	0.07
WCSTSETFAIL	14	15	0.07
CE–13	16	13	0.06
WCST#CONCEP	16	13	0.03
TRLBERROR	17	12	0.02
CE–04	12	17	0.02
CE–09	18	11	0.02
WCST#COR	20	9	0.01
TRLAERROR	22	7	0.01
CE–14	14	15	–0.03
STROOPCERR	17	12	–0.03
CE–06	19	10	–0.25
CVLTPERSEV	20	9	–0.42
CE–01	21	8	–0.62

Note. STROOPW = number of correct responses in a 45-sec time period when reading colors; STROOPCW = number of correct responses when naming colors ignoring printed words in a 45-sec time period; TRLBSECONDS = total amount of time to reach the goal; TRLASECONDS = total amount of time to reach the goal; WCST#ERROR = number of errors; WCST#NPERROR = number of nonperseverative errors; WCST%ERROR = percentage of errors; WCST%NPERROR = percentage of nonperseverative errors; WCST#TRIALS = number of trials completed; WCST%CONCEP = percentage of conceptual level responses; RFFUNIQUE = number of unique designs; WCST#PERROR = number of perseverative errors; STROOPC = number of correct responses in a 45-sec time period when reading color of ink; CTTPOS = total number of letters placed in the correct sequence; WCST#PRESP = number of perseverative responses; WCST#CAT = number of categories correctly identified; CTT18 = total number of letters remembered during 18-sec delay period; WCST%PERROR = percentage of perseverative errors; WCST%PRESP = percentage of perseverative responses; CVLTASEM = semantic clustering; CVLTSDSEM = short-delayed semantic clustering; CVLTLDSEM = long-delay semantic clustering; DSSCALE = scaled score of both conditions combined; CVLTINTRUS = number of intrusions; CE–08, CE–15, CE–05, CE–07, CE–02, CE–12, CE–11, CE–03, CE–10, CE–13, CE–04, CE–09, CE–14, CE–06, CE–01 = scoring in the Cognitive Estimation questionnaire; COWATTOT = total words generated from letters; WCSTLTOL = the learning to learn factor; COWATS = number of words: "S" trial; COWATANIM = number of animal words generated; WCST1stCAT = number of trials to first category correctly identified; COWATF = number of words: "F" trial; RFFPERSEV = total number of perseverative errors; COWATA = number of words: "A" trial; CTT03 = total number of letters remembered during 3-sec delay period; STROOPWERR = number of errors reading colors; STROOPCWERR = number of errors when naming colors ignoring printed words; WCSTSETFAIL = number of set breaks; WCST#CONCEP = number of conceptual level responses; TRLBERROR = number of errors for the trail; WCST#COR = number of correct responses; TRLAERROR = number of errors for the trail; STROOPCERR = number of errors made; CVLTPERSEV = number of perseverations.

[a]Weights are computed as the mean of the deviations of Fetal Alcohol Syndrome and Fetal Alcohol Effects cases from the loess regression curves for each of the items, as illustrated for WCST%CONCEP and COWATA in Figure 2.

growth deficiency. All of the participants with FAS or FAE were selected from a study of secondary disabilities conducted in our lab for the Centers for Disease Control and Prevention (Streissguth, Barr, Kogan, & Bookstein, 1996). All of these diagnosed participants have been involved in follow-up studies in this lab for many years. Many of them were identified at birth, whereas others were identified later in life by the diagnostic clinic at the University of Washington, Seattle.

Control participants for the clinic-based study were recruited by advertisements placed at local universities, community colleges, and the University Medical Center. They were matched for age and ethnicity. Potential control participants were screened for prenatal alcohol exposure, head trauma involving loss of consciousness, history of neurological disturbances (i.e., seizures, Parkinson's disease, Alzheimer's disease, and birth defects involving damage to the brain), and current psychiatric problems (i.e. hearing voices, seeing visions, and alcohol or drug abuse problems). Control participants reporting any of these symptoms were not included in the study.

The participants from the longitudinal prospective study ($n = 419$) and control participants from the clinic-based study ($n = 15$) were compared with the participants diagnosed with either FAS or FAE from the clinic-based study. Demographic characteristics of the participants diagnosed with FAS or FAE, the 15 control participants from the clinic-based study, and 419 comparison participants from the longitudinal prospective study are presented in Table 3.

Participants from both studies were administered an identical group of neuropsychological tests. One of the 15 participants with FAS was unable to complete the EF battery; therefore the results reported are based on 29 patients with FAS or FAE, 15 control participants from the clinic-based sample, and 419 comparison participants from the longitudinal prospective study.

Measures

Nine tests of EF with a total of 58 scores were recorded and administered under identical conditions to participants in both studies.

Wisconsin Card Sorting Test (WCST). The WCST (Heaton & Staff, 1993) is a computerized task which requires participants to match individual response cards to one of four key cards (one red triangle, two green stars, three yellow crosses, and four blue circles). The participant is required to correctly identify the sorting principle (i.e., color, shape, or number) on 10 consecutive trials at which point the sorting principle changes. The three different sorting principles are each repeated twice over the 128 possible trials. Some controversy is attached to the use of the computerized version of the WCST versus the standard administration. In one

TABLE 3
Comparison of Demographic Characteristics by Group: FAS and FAE Study ($N = 45$) and the Longitudinal Prospective Study

	FAS and FAE Study			
Characteristics	*FAS*[a]	*FAE*[a]	*Control*[a]	*Longitudinal Prospective Study*[b]
Sex				
Male	15 (100%)	15 (100%)	15 (100%)	220 (53%)
Female	—	—	—	199 (47%)
Ethnicity				
White	9 (60%)	9 (60%)	10 (67%)	347 (83%)
Black	—	2 (13%)	2 (13%)	31 (7%)
Native American	6 (40%)	4 (27%)	3 (20%)	12 (3%)
Other	—	—	—	29 (7%)
Age at exam				
18–24.9 years	10 (67%)	10 (67%)	10 (67%)	419 (100%)
25–31.9 years	4 (26%)	4 (26%)	4 (26%)	—
32–38.9 years	1 (7%)	1 (7%)	1 (7%)	—
Highest grade completed				
High school incomplete	6 (40%)	5 (33%)	1 (6%)	48 (11%)
High school graduate	7 (47%)	9 (60%)	1 (6%)	92 (22%)
Beyond high school	2 (13%)	1 (6%)	13 (87%)	279 (67%)

Note. FAS = Fetal Alcohol Syndrome; FAE = Fetal Alcohol Effects.
[a] $n = 15$. [b] $n = 419$.

study of autistic and nonautistic children, the two forms of the test were found to have low to moderate alternate forms validity, and a somewhat attenuated effect was found with the computerized version (Ozonoff, 1995). In contrast, another study of neurologically intact participants found the two versions of the test to be quite comparable (Artiola-i-Fortuny & Heaton, 1996). Computerized administration offers the benefit that all of the tests are identically administered and reliably scored, thus removing much examiner error that could occur in hand administration. Scores for this task include the number of trials completed, number correct responses, number and percentage of errors, number and percentage of perseverative responses, number and percentage of perseverative errors, number and percentage of nonperseverative errors, number of categories correctly identified, number of trials to completion of the first category, number and percentage of conceptual level responses, number of set breaks (making an incorrect response after a run of six correct responses), and the "learning to learn" factor.

Cognitive Estimation (CE). This 15-item questionnaire was originally based on the questionnaire developed by Shallice and Evans (Shallice & Evans,

1978). The current questions were derived from a previous study in our unit (Kopera-Frye et al., 1996). The participant is asked to make a reasonable guess about such questions as the length of a dollar bill, weight of the heaviest dog, and the height of the tallest tree in the world. The original scoring system devised by Shallice and Evans was a 4-point scale based on the extremeness of the response (Shallice & Evans, 1978). This method of scoring is quite dependent on the size of the normative group as extremeness was based on the response being outside the range of the full normative sample. Instead, in this study each participant's responses were rank ordered and the folded ranks (distance from the median rank) used for scoring.

Controlled Oral Word Association Test (COWAT). This is a task of verbal fluency in which the participant is given 1 min to name as many words as possible beginning with the letters "F," "A," and "S," and as many animals as possible (Spreen & Strauss, 1991). Scores were the number of words generated for each condition in the 1-min period.

Ruff's Figural Fluency Test (RFF). This nonverbal analog of COWAT requires the participant to create unique designs by connecting dots with and without distracting interference (Evans, Ruff, & Gualtieri, 1985). Scores recorded are the numbers of unique designs over the five trials and the total number of perseverative errors.

Trail Making Test. This test, especially Trails B, is a measure of shifting attentional set (Spreen & Strauss, 1991). Trails B requires the participant to switch between numbers and letters when connecting dots to reach the goal. Scores for this test are the total amount of time to reach the goal and the number of errors for each of the trails.

Stroop Color–Word Test. The three-part Golden version of the task (Golden, 1978) requires the participant to read the names of colors, name the color of ink, and name the color of ink when words printed are in contrast to the colored ink (interference task). The interference task is a measure of response inhibition. Scores for this test are the numbers of correct responses named in a 45-sec time period and the number of errors made.

Consonant Trigrams Test (CTT). This is a test of letter memory in the presence of a distraction task and is a measure of working memory (Stuss, Stethem,

& Poirier, 1987). In this task the participant is given a string of three consonants and is asked to remember them while counting backward by threes for delay periods of 3, 9, and 18 sec. Scores recorded are the total number of letters remembered at each of the delay periods and the total number of letters placed in the correct sequence.

Digit Span (DS). This subtest from the Wechsler Adult Intelligence Scale–Revised (WAIS–R) is a rudimentary measure of working memory. On this task, the participant is required to repeat numbers presented first forwards and then backwards. The score recorded is the scaled score of both conditions combined.

California Verbal Learning Test (CVLT). This test of list learning over repeated trials (Delis, Kramer, Kaplan, & Ober, 1987) has a component that measures the number of items the participant clusters through semantic methods (items within the same category). The clustering of information can be seen as a form of working memory. The test also measures the number of intrusions and perseverations. Scores recorded for this test include List A semantic clustering, short-delayed semantic clustering, long-delay semantic clustering, number of intrusions, and the number of perseverations.

Wechsler Adult Intelligence Scale–Revised(WAIS–R). In addition to the EF tests, a measure of intellectual functioning was administered. For these studies a prorated form of the WAIS–R was utilized (Wechsler, 1981). This prorated form of the WAIS–R eliminated the vocabulary and comprehension verbal subtests to shorten administration times. Correlation between prorated Full Scale IQ (FSIQ) and the traditional FSIQ was .96 and correlation between the prorated Verbal IQ (VIQ) and traditional VIQ was .95. Scores recorded from this test included the prorated VIQ, Performance IQ, and the FSIQ.

Statistical Analysis

The analysis in this report utilizes a unique strategy for combining data from a population-based longitudinal prospective study with corresponding data from a comparative clinic-based study of FAS and FAE. We first studied the relation between IQ and the individual EF measures using a composite pool of "control" participants, taken to be the 15 control participants from the comparative study, together with those participants from the longitudinal prospective study not having the highest levels of prenatal alcohol exposure. Those participants in the top 10% on a composite prenatal alcohol exposure score (computed as an "alcohol latent variable" according to the method explained in Streissguth, Bookstein, Sampson, & Barr,

1993) were excluded. This exclusion of participants was to ensure that the IQ–EF relations observed were not influenced by participants who could be judged clinically to have FAE. The second stage of analysis combined the EF measures into a composite score that best expresses the alcohol-related deficits in EF that are not explained by corresponding deficits in IQ.

One convenient way of disentangling systems involving multiple causal systems like this is the method of path analysis originally developed by Sewall Wright for applications in genetics (Bookstein et al., 1985). A path analysis is an alternative to multiple regression analysis in which each of a variety of postulated causal mechanisms is examined with the others controlled as well as possible by explicit subtabulations. In view of the complex overlap between the constructs of IQ and EF (Denckla, 1996), two paths of effect can be identified (Figure 1). In one, prenatal alcohol damage would indirectly affect EF through the mediation of IQ, and in another, the effects of prenatal alcohol damage would directly affect EF irrespective of IQ. When the fact of alcohol damage is encoded as a binary indicator distinguishing the FAS and FAE patients from the comparison control group, the coefficient representing the direct effect of prenatal alcohol according to the path diagram of Figure 1 is just the difference between the mean of the IQ-adjusted EF scores for the FAS and FAE patients and the mean of the IQ-adjusted EF scores for the comparison group. This logic is identical with Wright's original strategy of controlling for the effect of "general factors" (Bookstein et al., 1985). For any given EF score, the IQ-adjusted scores are the differences between the EF score and a regression-based prediction of EF from IQ computed from the pooled comparison or control group. We compute this regression relation using only the composite comparison group (longitudinal study participants plus 15 control participants from the comparison study), for whom the mean IQ-adjusted score (the mean residual) will then be zero. The direct effect coefficient is then just the mean of the IQ-adjusted EF scores for the FAS and FAE patients.

To account for varying types of distributions of the 58 individual EF scores, including features such as skewness, outliers, and ceiling effects, we chose to replace the original EF scores by a transformation of their ranks known as "normal scores," except that we constrained the (nonlinear) predictions of each EF outcome

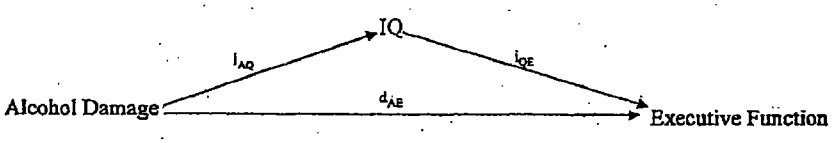


FIGURE 1 Path model for alcohol effect on executive function (EF). There is a direct effect of alcohol, and also an indirect effect through IQ. In the usual notation of path analysis, the direct effect is measured by d_{AE}, the indirect effect by the product of direct effects i_{AQ} of alcohol exposure on IQ and i_{QE} of IQ deficit on EF deficit.

not to exceed the 98th percentile of the actual outcome distribution because of occasional overextrapolation of unconstrained regressions due to "ceiling effects." This replaces, for example, the 40th lowest score by the equivalent Gaussian quantile: the 40th lowest score that would be expected under a Gaussian (normal) distribution. For each of the 58 EF measures, we plotted its normal scores against FSIQ, and to account for nonlinearity in the IQ–EF relations, estimated the regression relations for IQ adjustment using the fit of a nonlinear scatterplot smoother ("loess" in S-plus; StatSci, 1993).

For each of the 58 normal-equivalent EF measures, the computed nonlinear regression on IQ was used to adjust the scores of the FAS and FAE participants (by simple subtraction of the regression prediction, given IQ, from the normal scores of the EF measures). The direct effect of FAS and FAE, measure by measure, is then the average over the 30 cases of these IQ-adjusted normal scores. Any collection of direct effect coefficients like these can be used to form the estimate of a "latent variable" (Bookstein et al., 1985). We used the 58 direct effects here to generate a weighted combination of the 58 normal-equivalent EF items; a diagnosis-informed composite EF score that is explicitly controlled for IQ by the nature of its construction. A scatterplot of this EF composite against IQ lets us assess the variation of this net EF performance over the cases.

RESULTS

Means and standard deviations of the 58 EF scores are presented in Table 1 for each of the three groups in the FAS and FAE study and the longitudinal prospective study. Note that, in nearly all cases, patients with FAS or FAE performed more poorly than either controls or participants in the longitudinal prospective study. Average IQ scores for each group were consistent with previous findings in which patients with FAS had a mean FSIQ score of 79 and patients with FAE had a mean score of 90 (Streissguth et al., 1996).

Scatterplots of FSIQ versus each of the 58 EF measures, each transformed to "normal scores" as previously explained, were examined in our analysis of the complex overlap between IQ and EF. Figure 2 shows two of the resulting 58 scatterplot analyses: for COWAT words named in the A condition (COWATA), and for the percentage of conceptual responses on the WCST (WCST%CONCEP). We show scatters for both the original measurement units and the normal score equivalents.

The regression predictions on the scatterplots generated residuals for each FAS and FAE patient, deviations from the regression curve indicating whether the patient's EF is better or worse than would be expected given his or her IQ. Table 2 tabulates this classification of FAS and FAE residuals for all 58 EF measures. The plot for

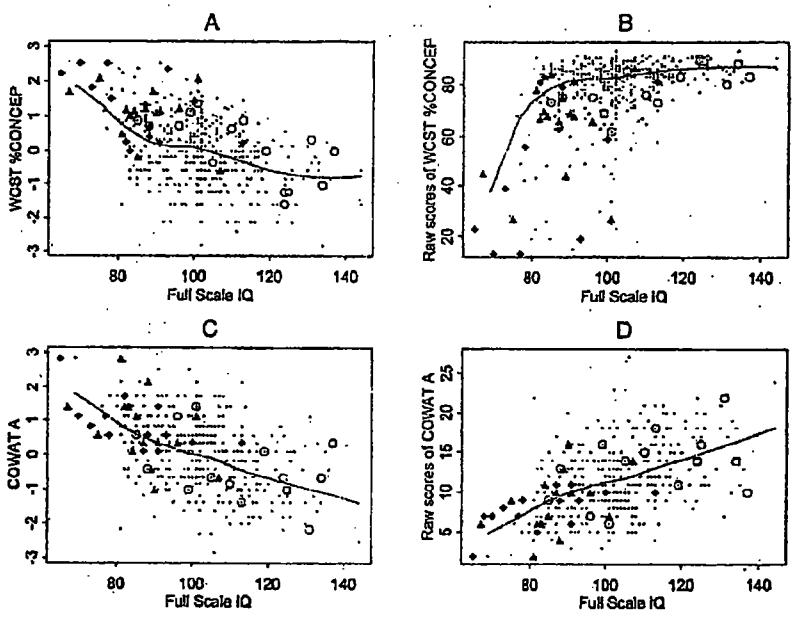


FIGURE 2 Examples of nonlinear regressions of executive function (EF) normal scores and raw scores on IQ. Controls, Fetal Alcohol Effects (FAE), and Fetal Alcohol Syndrome (FAS) participants are indicated by circles, triangles, and diamonds, respectively, as in the legend on Figure 3. The regression curve is based on 377 participants from the longitudinal study (dots) plus the 15 control participants for the FAS and FAE study (circles). Wisconsin Card Sorting Test percentage of conceptual responses was one of the best measures to identify worse than expected performance based on IQ and thus the direct effect of prenatal alcohol damage on EF. Controlled Oral Word Association Test letter "A" condition exemplifies one of the tasks in which almost half of the patients performed better than expected and half performed worse than

the variable COWATA (Figure 2c) shows that 13 patients lie below the regression curve of the performance expected given their IQ, and 16 lie above. In this sense, the COWATA EF score may be considered "uninformed by fetal alcohol diagnosis." By contrast, a typical Wisconsin outcome performance score, WCST%CONCEP, is better than expected for IQ in only six patients, versus 23 in which it is worse than expected (Figure 2a). As previously explained, the computation of a coefficient representing the direct effect of prenatal alcohol damage according to the simple diagram of Figure 1 (d_{AE}) is just the mean of the residuals of the FAS and FAE patients (because the mean of the residuals of the comparison participants is zero by fitting of the regression to just these cases). These coefficients appear as "weights" in the third column of Table 2. Table 2 orders the 58 EF measures by the mean residual score of the patients, the weights in the third column. As previously explained, we used these

weights to compute a diagnosis-informed EF composite score that emphasizes those EF measures, indicating the greatest direct effects of prenatal alcohol exposure. This composite is scattered against IQ in Figure 3. Shown here is the usual "complex overlap" (Denckla, 1996) between the constructs. The Fetal Alcohol diagnosed patients, however, are consistently in more extreme deficit as in the individual scatters for many of the Wisconsin, Stroop, Trails, RFF, and CTT measures. Note, in particular, that for IQ below 80 there seems to be no real possibility of acceptable performance on the EF composite. In addition, the results show that there is a complete intermingling of the patients with FAS and FAE, with no difference between the two groups on measures of EF. This can be seen in the similarity of group means for the patients with FAS and FAE displayed in Table 1 and in the intermingling of the two groups in Figure 3.

DISCUSSION

In view of the complex overlap between the constructs of IQ and EF (Denckla, 1996), two paths of effect can be identified (Figure 1). In one, prenatal alcohol dam-

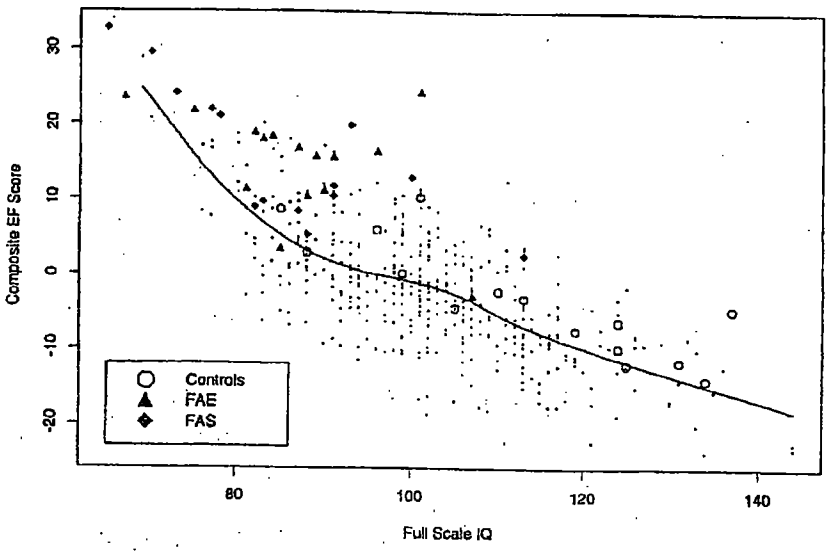


FIGURE 3 Summary of alcohol sensitive executive function (EF) composite score by IQ. Most of the participants with Fetal Alcohol Syndrome and Fetal Alcohol Effects have EF performance worse than expected based on the IQ regression curve.

age would indirectly affect EF through the mediation of IQ, and in another, the effects of prenatal alcohol damage would directly affect EF irrespective of IQ. We have in effect sorted the EF block based on a path analysis model of direct versus indirect effects. For more on path analysis, see Bookstein et al., 1985, section 4.2.

In this way we have sorted the EF outcome block along a continuum of apparent direct effects of alcohol. The EF scores most specific to alcohol damage, those appearing first in Table 2's ordination of the EF outcome battery, all seem to pertain to the WCST, the Stroop performance scores, Trails, RFF production, or CTT. This is analogous to the better known phenomenon that adaptive behavior scores such as the Vineland are in deficit in patients with FAS and FAE to an extent even greater than is predicted by their deficits in IQ (Streissguth et al., 1996). By contrast, the EF deficits in patients with FAS and FAE shown by the COWAT scores, the Stroop error scores, CVLT and RFF perseveration scores, Trails error scores, and cognitive estimation scores, can be wholly explained by IQ deficit.

EFs have long been associated with frontal lobe integrity. Patient DT (discussed earlier) presented with left frontal subarachnoid hemorrhaging and had concomitant EF deficits (Eslinger et al., 1992). Functional neuroimaging studies of working memory have consistently shown dorsolateral prefrontal cortex (DLFPC) activation in control participants (Cohen et al., 1994; Mellers et al., 1995; Smith & Jonides, 1999). Using this same working memory paradigm, Weinberger and colleagues found that participants with schizophrenia had significantly less activation of the DLFPC compared to controls (Weinberger et al., 1996). Although, to our knowledge, no studies of participants with FAS or FAE, in either humans or animal models, have demonstrated structural damage to the frontal lobe, it seems reasonable to predict that in accordance with the EF deficits evidenced, participants with FAS and FAE will have structural or functional changes to the frontal lobes or to pathways that link frontal lobe functions with the rest of the brain.

Several studies have addressed plasticity of the frontal lobes following early lesions in a rat model (Kolb & Gibb, 1993; Kolb & Whishaw, 1985). In one study, rats were given medial frontal lesions on postnatal day 1 or postnatal day 10 (Kolb & Gibb, 1993). Both groups of rats performed equally poorly on the Morris water task shortly after surgery. However, the rats receiving surgery on day 10 improved their performance on this task after longer recovery, whereas the rats receiving surgery on day 1 did not improve their performance postsurgery. In addition, rats with later lesions showed generation of more dendritic spines per unit dendritic length in the parietal layer II to III pyramidal cells than did rats undergoing surgery on postnatal day 1. This suggests that early damage to the brain, as is caused by prenatal alcohol exposure, may not result in significant brain plasticity and recovery of function as might be seen after lesions acquired later in childhood.

The results of this study suggest a lack of relation between EF performance and the facial features of FAS. The performance of participants with FAS and FAE on EF measures in this study was completely intermingled, suggesting that the facial features of FAS do not and should not indicate that the damage caused by prenatal alcohol exposure to patients with FAS is any more severe than the damage caused to patients with FAE. This has been noted in a number of studies (Mattson et al., 1997; Mattson, Riley, Gramling, Delis, & Jones, 1998).

Findings from previous studies in our laboratory have not consistently demonstrated deficits on WCST performance in participants exposed to alcohol in utero (Carmichael et al., 1998; Sampson, Streissguth, Barr, & Bookstein, 1989; Streissguth et al., 1999). However, those reports with one exception (Carmichael et al., 1998), were based on a longitudinal prospective study of participants exposed to "social" levels of drinking during pregnancy. In that study, very few participants were diagnosed with FAS and only a small subset can be classified as FAE or Alcohol Related Neurodevelopmental Disorder (Sampson et al., 1997). The relative inability of WCST to correctly identify participants exposed to lower doses of alcohol suggests that this test is not as sensitive at measuring EF functions in relatively "normal" populations with subtle alcohol effects. In addition, the version of the WCST used previously in our labs was unable to calculate some of the scores that may be more sensitive to these subtle effects.

Despite Denckla's (1996) admonition that intelligence must be taken into account when assessing EFs because of the complex overlap with IQ, the majority of studies we reviewed did not address this issue. Previous studies of EF in FAS and FAE populations did not specifically assess the complex overlap between general intelligence and EF (Coles et al., 1997; Kodituwakku et al., 1995; Kopera-Frye et al., 1996), and so were not able to determine which EF tasks are directly affected by alcohol exposure, and which are only indirectly affected through the mediation of IQ. Several studies assessing EF in a variety of other disorders have made some attempts to address the overlap between EF and IQ (Foong et al., 1997; Rumsey, 1985; Welsh, Pennington, Ozonoff, Rouse, & McCabe, 1990). However, in each case statistical errors precluded identification of both the direct effects of the disorder on EF and the indirect effects through the mediation of IQ. For instance, it is inappropriate to adjust an alcohol–EF regression coefficient for IQ (as one of the other studies in another population did), as this is actually the partialling of a correlation for a mediating variable.

This study emphasizes that one cannot assess alcohol-affected patients (diamonds vs. triangles in Figure 3) by only focusing on the degree of mental retardation. In fact, very few of the participants in this study had IQs within the borderline to mentally retarded range. Instead, it is important when assessing alcohol-affected patients to measure the full range of EF skills, emphasizing those representing the direct effects of exposure, as this study demonstrates those are the tests for which FAS and FAE performance is typically worse than would be predicted from the concomitant IQ deficit alone.

The tests that are directly affected by prenatal alcohol damage appear to be assessing the ability to shift tasks, maintain complex attention, perform visuospatially mediated tasks, and maintain and manipulate information in working memory despite distraction. EF tasks that are mediated by the effects of IQ appear to reflect errors.

Patients with FAS and FAE who present the specific EF deficits of direct effects of alcohol have difficulties shifting or changing strategies, especially when there is uncertainty about which rules to employ. They may have difficulty maintaining important information in distracting situations and may need to be reminded frequently. Patients who present with EF deficits that are mediated by IQ will likely be seen as performing commensurate with their IQ and are likely to be more error prone. Employment settings in which there is a pressure for rapid production and in which tasks shift frequently with some uncertainty about the requirements of the task would not be a good fit for a person with these deficits. In interpersonal situations, patients with these problems may have difficulties with the subtle and complex interactions that underlie personal relationships. Thus, these participants may feel isolated or may be prone to inappropriate behaviors that could get them into trouble both socially and legally. It is therefore important to take EF deficits into account when working with patients with FAS and FAE. Discussions and instructions should be concrete with little ambiguity. Information will probably need to be repeated several times, and multiple modalities of presentation should be considered. Also, because of the problems of abstraction, planning, and problem solving with which these patients present, it is important to plan out contingencies for many possible problems that they may face during the course of their daily lives. This suggests that many patients with FAS and FAE who display EF deficits will have great difficulties with independent living and will often need a caretaker to assist them with many life activities.

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Bringing morphometrics into the fetal alcohol report: Statistical language for the forensic neurologist or psychiatrist

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The components of a diagnosis in the fetal alcohol spectrum consider, among other domains, evidence of prenatal alcohol exposure, facial features, neurological or neuropsychological deficits, and neuroanatomical form. Most of these assessments are based on traditional sorts of quantifications (e.g., length measures, IQ scores, quantity of alcohol drunk by the mother), but for one of the domains, the morphometric assessment of neuroanatomical form as abnormal in a way suggesting fetal alcohol damage, the associated quantifications are relatively recent. For purposes of a forensic report, this morphometric component of the diagnostic judgment would seem to go best in the form of one classic statistical quantity, the likelihood ratio (odds ratio) of two

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hypotheses as they both attempt to explain the form of a particular structure within the defendant's brain (in our approach, the midcurve of the corpus callosum). On one hypothesis, the subject is presumed to be from a normal population; on the other, he is from a group all characterized by diagnosable fetal alcohol damage. Our article explains in simple terms how the forensic physician might wish to couch that component of the FASD diagnostic system for the purpose of aiding those in the courtroom who have the task of determining guilt or culpability.

KEY WORDS: *FASD, morphometrics.*

A diagnosis in the Fetal Alcohol Spectrum can be viewed as a complicated task of information processing that combines data from a variety of channels. The list of these channels includes, but is not limited to, birth size measurements, facial form assessments, assorted somatic dysmorphologies, psychological test profiles, personality or behavioral characteristics, prenatal medical history, neonatal or pediatric medical history, neurological or neuropsychiatric symptomatology, and neuroanatomy.

For each of these domains, there are rules or conventions for extracting the necessary information from raw clinical or medical records, rules that are variously more or less explicit and more or less quantitative. Some of these rules are familiar both to physicians and to the prosecutors, judges, or members of juries who have to consider the testimony of the expert(s) conveying these observations. For instance, neonatal head circumference, if available, can be interpreted as soon as the accompanying gestational age is known, and a report of maternal alcoholism during pregnancy from any of a wide range of informants can be calibrated against the ver-

bal standards of either the Institute of Medicine report that introduced the category of "alcohol-related neurodevelopmental disorder" (Stratton, Howe, & Battaglia, 1996) or the National Center for Birth Defects and Developmental Disabilities (NCBDDD) standards (NCBDDD, 2004). Similarly, the psychological test scores or their profiles that the standards mention can be referred to published norms; neurological hard or soft signs and dysmorphology, to the clinician's own experience; measurements of specific fetal-alcohol-related facial features, again, to published normative standards.

The one notable exception to this generalization is the extraction of information from the neuroanatomical image. The information borne in a structural magnetic resonance image (MRI) for a fetal alcohol diagnosis is not conveyed by any single quantification, but instead as a summary rating of significant clinical abnormality as indicated by these phrases from original versions of the two principal diagnostic guidelines that underlie our work: "Evidence of CNS [central nervous system] developmental abnormalities, as in . . . structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia), neurological hard or soft signs" (Stratton et al., 1996), and then eight years later, after publication of the Bookstein et al. papers to be cited below, "Clinically significant brain abnormalities observable through imaging techniques (e.g., reduction in size or change in shape of the corpus callosum, cerebellum, or basal ganglia) as assessed by an appropriately trained professional" (NCBDDD, 2004). Neither of these declarations is accompanied by any explicit methodology, and neither list of examples (what follows the "e.g.") incorporates any description of how to continue the list. It is the task of this article to sketch the missing instructions for measurements of the corpus callosum, the easiest of these structures to measure for purposes of a fetal alcohol diagnosis. The kind of measurements to which these instructions are suited are measurements of its midcurve. To understand the midcurve, imagine an ordinary hourglass that has

been knocked over onto its side. The midcurve is a curve on the callosal surface that relates to the overall form of the callosum somewhat as the constriction of that tipped hourglass relates to the sand compartments that now lie to the left and the right: it is the curve around which this structure diverges nearly symmetrically outward on both sides. The analogy becomes even more apt if you imagine that the middle of the hourglass takes the form of the downward-pointing C in Figure 3 instead of a circle. We will be measuring the shape of this curve using standard statistical tools for the purpose.

Background: The corpus callosum and its import for FASD diagnosis

The corpus callosum, one of the structures specifically noted by both the Institute of Medicine and the NCBDDD as appropriate for inspection in the course of an FASD differential diagnosis, is a structure of neural fibers that serves mainly to connect cell bodies within and across the two sides of the cortex (the evolutionarily higher brain, the component that fills the upper part of the skull). Its morphogenesis (the emergence of its form) begins early in gestation, around the 6th week post-conception (i.e., prior to the middle of the first trimester of pregnancy, before the pregnant woman knows she has missed two consecutive menstrual periods), and continues until roughly the middle of the second trimester. The principles according to which it develops normally are known, from before birth to late adolescence, and also, in general terms, the mechanisms by which alcohol disrupts this normal morphogenesis (cf. Guerri, Rubert, & Pasqual, 2006).

Attention turned to the corpus callosum early in the course of fetal alcohol science. Riley et al. (1995) usefully reviewed the early literature here, as did Swayze et al. (1997). Our awareness of the sensitivity of this structure to prenatal alcohol damage came originally from studies on stillbirths—several early investigators reported an unusual incidence of

obvious callosal problems—and later from the Riley 1995 study, which was the first to apply conventional biostatistical methods to images of this structure in the living patient. Riley's successful study was the foundation of a project by the Seattle group under Ann Streissguth that applied more contemporary methods for analysis of curving form in three dimensions. The findings of this study in respect of the corpus callosum were published in three peer-reviewed articles early in the new century (Bookstein, Sampson, Streissguth, & Connor, 2001; Bookstein, Sampson, Connor, & Streissguth, 2002; Booksein, Streissguth, Sampson, Connor, & Barr, 2002). As Riley himself noted recently (Fryer, McGee, Spadoni, & Riley, 2006), his study was in the form of a conventional comparison of averages, unexposed children or adolescents vs. patients diagnosed with what we now call FASD, whereas the Seattle study was of a different design, the attempt to construct an actual discriminatory protocol for differentiating FASD patients from unaffected adolescents and adults by a more extensive numerical characterization of this outline form. The Seattle approach is thus fairly well-suited for the purpose at which this paper is directed, namely, the representation of information from the corpus callosum in a way that helps the diagnostician to determine whether the evidence about this particular defendant, taken as a whole, supports a diagnosis of fetal alcohol damage, and, if so, which diagnosis and with what degree of confidence.

Statistics for discrimination: An illustrated lecture

The following material is part of the standard curriculum in biometrical statistics. We are explaining it here using diagrams, not formulas, just to ease the communication of the underlying principles.

When the task at hand is one of discrimination between two or more groups (in our case, FASD versus "unaffected"), and when the information at hand is quantitative, the preferred

biometric approach works through a *likelihood ratio.* In the likelihood ratio approach, each possible group assignment is treated as a *hypothesis,* and from the distribution of the quantitative information within the groups at hand we can compute a likelihood of arriving at the given data for the client on either of these hypotheses (FASD or unaffected). The likelihood ratio is simply the ratio of these two probabilities ("likelihoods"). If forced to assign a group, one would assign the group that has the higher probability; as a figure of one's confidence in that assignment one usually uses the likelihood ratio itself. A ratio of 7 or so corresponds to the classic 5% significance level from the more familiar context of "null hypothesis testing," an approach that deals with only one hypothesis, instead of the two (or more) under consideration in the forensic context here.

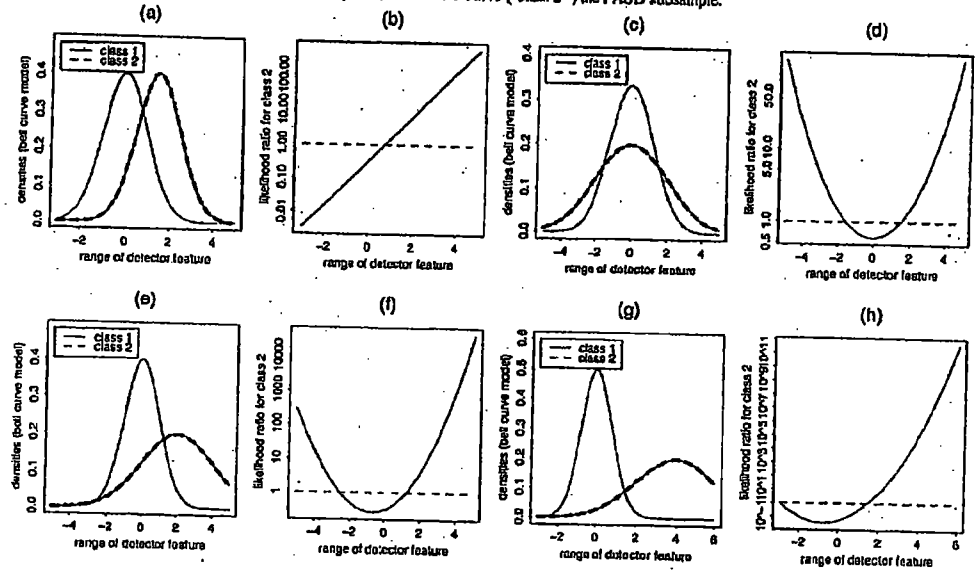
A likelihood ratio for a classification problem like this can be considered a function of three things: the distribution of some list of descriptors in the first group, the distribution of the same descriptors in a second group and the values of the descriptors for the case at hand whose group assignment is not yet known.

Figure 1 shows the situation when only one quantity has been measured. Here the two groups are indicated by the two bell curves, in two different line types. In panels (a) and (b) is the situation that the textbooks usually present first. This is the *linear discriminant function* made famous by Sir Ronald A. Fisher, one of the greatest of twentieth-century statistical geneticists. In this scenario, panel (a), the distributions of the feature of interest in two groups have the same *standard deviation* (half the distance between the "shoulders" of the bell curve, the place on either side of the peak where it changes from convex to concave), but they have different averages. If all the data are distributed with probabilities that exactly match the bell-curve formulas, meaning that the *logarithm* of the probability of any particular score is an upside-down parabola with a peak at the average, then panel (b) shows the

FIGURE 1

How likelihood ratios drive a discriminant classification: the case of one measurement only

(a-b) Classic case, same standard deviation but different averages. The likelihood ratio is loglinear in the measurement. (c-d) One situation occasionally encountered in FASD studies, same averages but different standard deviations. The log of the likelihood ratio is a quadratic (squared) function of the measurement. (e-f) Typical composite case: mean shift and variance shift. (g-h) More extreme composite case, greater mean shift. Notice that in all of these scenarios except the one in panels (a-b), which is Fisher's original "linear" discriminant analysis, the points classified into one group (the group of smaller standard deviation) come from a finite interval. In panels (b), (d), (f), (h), the dashed line is the level of equal likelihood (likelihood ratio 1). In panel (h) the vertical axis actually goes from 0.1 to 100,000,000,000 on a log scale. In each of panels (a), (c), (e), (g), the reader might think of the solid curve ("class 1") as representing the normal subsample and the dashed curve ("class 2") the FASD subsample.



corresponding likelihood ratio on a log scale. Its logarithm is a linear function of the measured value, hence the word "linear" in the common name of the technique. Cases to the left of the average of the group averages are classified into group 1, cases to the right into group 2, and cases that fall exactly at this average of averages have a likelihood ratio of 1, so they can't be classified either way. The horizontal axis where the two probability curves cross in panel (a) is the same as the location where the diagonal line crosses the horizontal line of height 1.0 in panel (b): the value of the measurement for which the likelihoods on the two hypotheses are equal.

This situation was quite unrealistic in presuming equal standard deviations between the groups. In panel (c) is the other special case, the same averages but different standard deviations. Now cases with measured values closer to the grand average are classified into the group of smaller standard deviation, and cases with measured values farther away are classified into the group with larger standard deviation. The logarithm of the likelihood ratio, panel (b), is now a parabola (a quadratic equation) pointing upward (in this case of group 2's standard deviation being the larger one) with its bottom (its vertex) at the grand mean. The crossovers where likelihood switches from favoring the first group to favoring the second, panel (d), are the places where the two distribution functions, panel (c), have the same height. Again this is where the curves cross; when standard deviations differ this is at two points, not one. Where the crossing point is improbable on either hypothesis, you will have trouble locating it graphically, but algebraically it is certain to be there.

In the bottom row of this same figure are two examples, panels (e) and (g) of the general case, the one you will most frequently encounter in real data: two groups that have different averages and also different standard deviations. In such cases, the likelihood ratio curve, in the log plot we are using here, takes the form, panels (f) and (h), of the sum of a parabola and a straight line; but this is just another parabola, so they all look

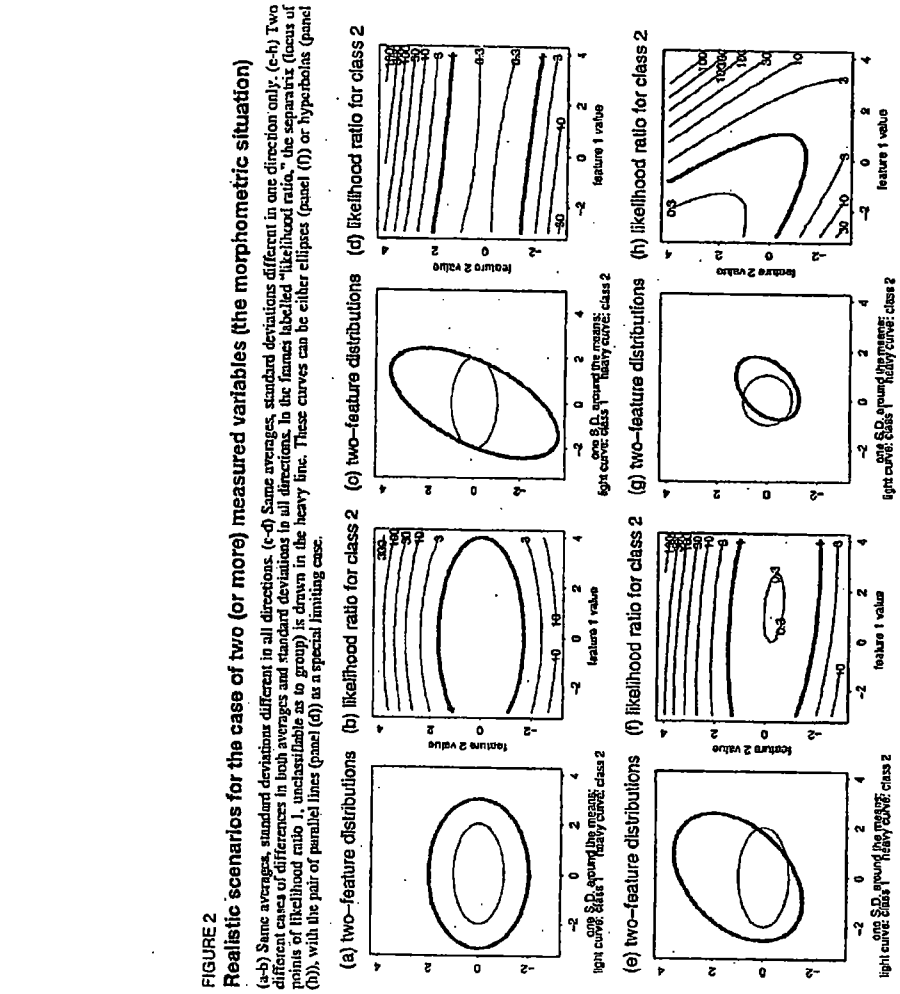
qualitatively similar. Each parabola dips below the line of likelihood ratio 1, i.e. classifies data to the group with the smaller standard deviation (here, group 1), in a finite region that is bounded by the two points where the two distribution functions cross; everything outside this region gets classified into group 2. Except for panels (a-b), the linear discriminant function, all of these are examples of the more general formula, the quadratic discriminant function.

Figure 2 shows what happens when you have measured more than one variable. The mathematics is quite similar here, but the geometry of the picture is different. Now we represent each group by a bell-shaped surface, not a curve. In the pictures, each of these surfaces is only suggested by the elliptical "waist" where it changes from convex to concave (just like the "shoulders" of the univariate version). The shape and size of this waist curve together specify what the statisticians call the *covariance matrix* for each group separately. You can think of each of panels (a), (c), (e), (g) as a drawing of the standard deviation in each different direction around an average that is just the center of the circle or the ellipse in question. To the right of each such scene, in panels (b), (d), (f), (h), is a likelihood ratio surface drawn by some contours that are roughly equally spaced on a log scale. The *separatrix*, the locus that separates the points going to be classified into group 1 from the points that are going to be classified into group 2, is now not a pair of dots, the way it was in Figure 1, but instead a curve all its own, which can be an ellipse, a hyperbola, a pair of parallel lines, or any of a variety of other possibilities. In this figure, all of the examples will involve different standard deviations, and so every computation is a quadratic discriminant function, not a linear one.

In panels (a-b) in this figure is one relatively common special case, in which the two groups have the same averages (on both of the measured variables) but different standard deviations in every direction. Then the likelihood ratio in every direction looks just like the corresponding version of panel

FIGURE 2
Realistic scenarios for the case of two (or more) measured variables (the morphometric situation)

(a–h) Same averages, standard deviations different in all directions. (c–d) Same averages, standard deviations different in one direction only. (e–h) Two different cases of differences in both averages and standard deviations in all directions. In the frames labeled "likelihood ratio," the separatrix (locus of points of likelihood ratio 1, unclassifiable as to group) is drawn in the heavy line. These curves can be either ellipses (panel (f)) or hyperbolas (panel (h)), with the pair of parallel lines (panel (d)) as a special limiting case.



(d) in Figure 1. Points close enough to the averages will be classified into the group of smaller standard deviation, and points farther away into the group of larger standard deviation. The separatrix curve is itself an ellipse lying somewhere near the curve of larger standard deviation.

In panels (c-d) of Figure 2 is a situation that is even more specialized. Here, the groups have the same average and the same standard deviation in one direction but not any other direction. The separatrix (panel [d]) is now a pair of parallel lines. Points between the lines are assigned to one group, and points outside the lines in either direction to the other group.

In the bottom row are a pair of complementary examples of the completely general situation, with different averages and also different standard deviations. In panels (e-f), the standard deviations of the one group are always less than those of the other, and the separatrix is an ellipse. In panels (g-h), one group has the lower standard deviation in one direction, and the other group has the lower standard deviation in another direction; the separatrix is now a hyperbola.

This ends the statistical part of this article. We have left out all the formulas, but nothing is missing that is required for an adequate intuitive understanding of what is going on here. The fetal alcohol spectrum disorders are diseases that show excess of variation in just about every profile that has ever been measured, and so our discriminators will usually look like the cases at left in Figure 2, a pair of ellipses of which one (for the FASD group) is always wider than the other (for the unexposed group of some reference sample). They may have the same averages (panel [a]) or different averages (panel [e]), but, still, forms that are close to the grand average will be called "normal" and forms that are far enough away (how far is enough depends on the direction) will have likelihood ratios suggesting an FASD diagnosis.

Discriminating fetal alcohol spectrum disorders by callosal shape

The NCBDDD advisory quoted above suggests that in persons having an FASD diagnosis, or deserving one, abnormalities that offer significant amounts of information can often be observed in the form of the corpus callosum, among other neuroanatomical structures. The preceding statistical lecture indicated in general terms how we might proceed to examine specific instances (specific defendants) in a search for evidence of this sort. The research papers inform us that the distribution of measures of callosal form for persons with a preexisting FASD diagnosis is, in fact, strikingly different from that for persons who were unexposed to prenatal alcohol. Those papers, particularly the three Bookstein et al. papers of 2001–02, emphasize the difference between the *variation* of the two distributions, not necessarily their means, meaning that it is the second of the two versions of discrimination mentioned above, the case we've called "quadratic," that obtains here. (But the finding of hypervariability here is not original to the Bookstein study; it is already visible in the summary charts of the original study of the Riley group published in 1995.)

Two issues arise: how to produce the appropriate likelihood ratios corresponding to these detections, and how to report such findings to a decider of fact or of culpability. A callosal finding alone cannot exhaust the data base to which the expert refers. There must of course be additional information about the prenatal exposure. We agree with the advice of the FASDExperts group that the appropriate expert summary should refer to all relevant available observations pertaining to the defendant. That summary ought to construct a composite FASD diagnosis based on all the channels of information for which there are tabulations or graphics for FASD and for unexposed groups in the research literature(s) of the various fields. Thus a report of an abnormality of callosal form, should it obtain, will accompany a report of an abnormal pro-

file of cognitive test scores, or a measurement of the facial signs of FASD in a childhood photograph, or a note of a ventricular septal defect on a neonatal medical record. The callosal abnormality serves as evidence from a time early in the history of the causal path from dose (exposure) to response (behavioral deficits). It reifies the damage as physically tangible, not subjective, and reaffirms that it was produced prior to birth. Ultimately its role is as much to render plausible an alternate narrative of the instant offense as to serve in this developmental explanatory context.

The method introduced in the three cited papers is based on all the usual protocols of sound research techniques that apply everywhere in neuroimaging. The structural image has to be produced under conditions of a controlled machine protocol (the "pulse sequence"), the callosal outline curve has to be digitized in a rigorously systematized protocol that is the same for all clients and all research subjects, and the search for descriptors of an abnormality needs to come under the general supervision of the published research findings. The reduction of the callosal midcurve data to a list of discrete quantities goes forward using the rules of contemporary geometric morphometrics as published, for instance, in the textbook *Statistical Shape Analysis* by Dryden and Mardia (1998). In practice our work examines findings approximately in the order in which they were published: first, examination of the relation of the callosal outline as a whole to the intercommissural line, corresponding to the large-scale finding in Bookstein et al., 2001; second, examination of the large-scale features of the callosal curve per se, such as overall arch height or overall thinning, as in the findings of Bookstein, Sampson, et al., 2002; and finally, attention to points of particular thinning or thickening of the outline, as demonstrated by Riley (using a different method) and as highlighted in the analyses of Bookstein et al. (2002) already mentioned. The typical fetal-alcohol-affected brain has a callosum that is abnormal in at least one of these ways but not necessarily in all of them. For each such comparison a likeli-

hood ratio may be constructed along the lines explained in the previous section.

For the method to go forward we need those ellipses and the other apparatus of the background information. For this purpose we turn to the Seattle study of Streissguth's already mentioned, the one spurred by Riley's 1995 publication and resulting in the three cited Bookstein publications. The Streissguth study involved a total of 180 human subjects, of whom 177 had usable callosal outline information (the other three were missing parts), of which a total of two instances were called "clinically abnormal" by the neuroradiologist who was part of the study (a neuroradiologist, furthermore, who knew that the hypothesis was that we could tell the FASD callosa from the normal ones). Thus when we found that we could correctly classify 100 of those 117 FASD corpora callosa while misclassifying only 6 of the 60 normals, we were quite satisfied.

The subjects of the Seattle study divided into four groups in one way and into three groups in another way. The four groups were adult males, adult females, adolescent (aged 14-17) males, and adolescent females, 45 of each; the threeway classification was unexposed (to prenatal alcohol), Fetal Alcohol Effects, or Fetal Alcohol Syndrome, 15 of each within each of the four subgroups of 45. Fetal Alcohol Effects (FAE) is one of the names that used to be given to what we now call alcohol-related neurodevelopmental disorder, the presence of the central nervous system damage without the full spectrum of physical and facial features; Fetal Alcohol Syndrome (FAS) means (and has always meant) the combination of the brain damage with those physical signs. Both categories, of course, require separate evidence of exposure. The Seattle analyses all combined the FAS and FAE groups after checking that they did not differ meaningfully on any measurements of the corpus callosum, which is our subject here. This combination is usually called "Fetal Alcohol Spectrum Disorder" or FASD, as we have already been referring to it in this article.

For forensic purposes we care mostly about the 45 Seattle adult males. We have measurements of their corpora callosa; we also have extensive psychometric information, some 260 measured variables, on all of these men. The forensic workup here is limited to only the morphometric component, the callosal outlines, although our publications dealt with "endophrenology" as well (Bookstein, Streissguth, et al., 2002b): Callosal findings like these are indeed correlated with some patterns of cognitive deficit. We emphasize that the diagnoses of all 120 of the syndromal subjects in this study had been in place before they were recruited into the study—we produced no new diagnoses. The diagnoses of these syndromal subjects had been based on the qualitative standards of the Institute of Medicine report or its predecessors, or on quantitative consideration of test scores and measurements of size or facial form, all of which were standard protocols for FASD diagnoses across most clinics at the time.

Example

From about 40 forensic examples with which the first author has been involved to date (five of them in collaboration with the second author) we have selected one of the earliest. The following analysis concerns the thickness of the corpus callosum across the *isthmus*, the locus that is typically thinnest and for which the original paper by the Riley team found the greatest difference between the groups by his area measurements. The callosal outline is digitized as in the plus signs of Figure 3 below. These points are digitized in 3D, not in any 2D plane; the image behind the outline, a plane near the curve in question, is included simply to be helpful for purposes of orientation. From a suitable graphical comparison of this curve to the average for the Seattle unexposed adult males, Figure 4, we determine that the defendant in this case shows a thinning of the callosum just where it is most commonly encountered, there in the isthmus. We quantify in terms of a vector (drawn as an arrow) cutting across this sector from lower left to upper right, as in the inset in Figure 5. For the rules about how we

line up such a vector from image to image, see the cited Bookstein papers or the Dryden–Mardia textbook.

FIGURE 3
Callosal midcurve polygon, selected defendant, superimposed over a nearby plane section of his MRI.

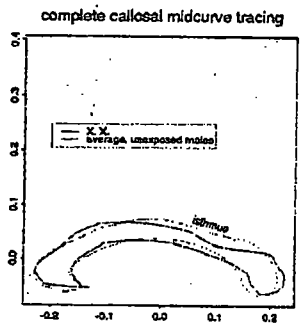


The isthmus of the corpus callosum (narrowing to right of center) looks too narrow. Our task is to communicate the strength of evidence of this observation, which is the most common of all observations in morphometric examination of fetal-alcohol-exposed subjects in a forensic context. The isthmus to which the text refers is labelled on Figure 4, and the conventional measure of its width at its narrowest is across the arrow in the inset of Figure 5.

The Figure 5 inset showed this transect vector as it cuts across the average outline of those 15 Seattle adult male normals. The main panel of the same figure displays that vector instead as if it sprung from the same location (the solid black square) in all 45 of the adult males of our Seattle study and likewise in the defendant here. We see that the defendant's descriptor vector (at the XX) is located in the midst of a considerable pool of Seattle FASD cases and far from any Seattle unexposed case. Thus it is certainly plausible to declare this callosum to be "indicative of fetal alcohol damage" in a qualitative way. The task at hand is to go further, to put some explicit numerical weight of evidence behind that assertion. To this problem the methods of Figure 2, applied to this pair

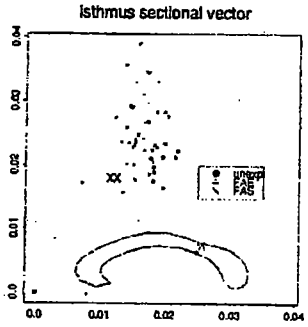
FIGURE 4

The same callosal midcurve, now superimposed over the average for the 15 unexposed adult males of our Seattle study.



In this plot and the next, both axes are in units of Procrustes distance, a formal scale for graphing shape statistics that does not reduce to any conventional unit such as centimeters or degrees. See the Appendix of Bookstein et al., 2001, or the extended discussion in Dryden and Mardia, 1998.

FIGURE 5



Underlay for a figure like those in Figure 2: raw distributions of the transect vector across isthmus, this defendant (XX) and all 45 adult males of the Seattle study. The likelihood ratio for the hypothesis of FASD group membership vs. membership in the group of unexposed is about 800 to one.

of variables, yield an immediate solution aligned with the example in panels (c-d) there. In that schematic, the likelihood ratios vary up to about 100, but as applied to these fetal alcohol callosa, where the normals are so tightly stereotyped, the same formulas give us a likelihood ratio of fully 800 to 1 in favor of the hypothesis of belonging to the group of fetal-alcohol-affected adult males.

For this particular defendant, it was the first author who explained all of this to the jury, including a hint or two of the bell surface machinery that lay behind that numerical value of 800. The instant offense was a typical FASD-like crime, a case of impulsively following idiotic instructions without pausing to reflect. The mitigation case of which the morphometric detection of the damage here was part ended in a jury's decision to impose a sentence of life without parole rather than a death sentence. Absent jury exit interviews, the specific role played by the morphometric argument is unknown, but we doubt that it was construed as favoring the prosecution.

The research finding is not restricted to this transect alone. While in this application we selected it for examination right from the beginning—it corresponds to the relative-area finding of Riley et al., 1995, for instance, and so is the first place one would typically look—in other cases we may search for the maximum such effect all around the callosum. In such computations the nominal odds ratio needs to be divided by the mathematical constant 2.7183 (a diminution by precisely one unit on the log scale) before it is reported—this is, again, the standard rule (the so-called Akaike Information Criterion) we teach our students for when they are working with likelihood ratios in a multivariate setting. Here that would have led to an "equivalent" likelihood ratio (for the case of no optimization of search) of about 294 to 1. Likelihood ratios of 20 to 1 or better for the morphometric component correspond to a strength of evidence at least as strong as the conventional 5% significance level even after such a correction.

An overly skeptical reader might cavil at how much diagnostic weight we are resting on a comparison with a group of only 45 Seattle adult men. To such a complaint there are three separate stout rebuttals. First, our published finding, of excess variance in most measurements comparing the FASD brain to the normal, is not any sort of scientific outlier. Our study is itself a replication of the earlier study by Riley et al. (1995) from a different age group in a different city. We used more modern statistical methods, and so the finding takes a more usable form: a classification with accompanying likelihood-ratio, not a mere test of significance. Second, the finding of hypervariability—classification by variation from a mean—is found in all four of our age-sex subgroups, not just these adult males: a total sample size of 177, not just 45. Nor do the findings appear to differ by race. Riley had already found this hypervariance, and we have found it subsequently in the same structure at birth (Bookstein et al., 2007) and in another quantifiable brain region, the cerebellum (Bookstein, Streissguth, Connor, & Sampson, 2006). Third, we are certainly not claiming that particular callosal shapes predict the specific actions with which a criminal defendant may have been charged, nor that any diagnostic procedure can rely solely on corpus callosum shape. Rather, the logic is that two quite distinct classes of observations, the callosal form and the behavioral deficits, have both arisen as consequences of the same cause, which is the prenatal alcohol exposure. The morphometric observations are meant to be combined, then, with information on measureable behavioral deficits, on facial form (preferably in childhood), on neurological symptoms, on pediatric medical history, and so on; all these would serve as additional effects of the same documented common cause. The morphometric component serves here as the appropriate summary of information about similarity to one or the other subgroup of callosa in the Bookstein publications cited. The report of such a differential similarity, in the company of analogous similarities as detected in other measurement domains, is highly relevant to assessing the plausibility of a diagnosis of FASD and thereby to the decision (e.g., full

or diminished culpability) at which the decider of fact has been directed to arrive. We return to this point in our concluding comment.

Another application: The Fetal Alcohol Behavior Scale

To this point, our argument has dealt with likelihood ratios that arise in the course of examining one particular channel of data, the callosal midline curve as treated morphometrically. While the quantification of that domain is somewhat specialized, the logic of the subsequent statistical inferences is entirely general, so it applies as well to data arising from other measurement domains. For instance, the Fetal Alcohol Behavior Scale [FABS] score, Streissguth et al., 1998, is a summary score, applicable to children, adolescents, or adults, that counts the total number of characteristics reported to be present out of a list of 35 each of which has been rated by a knowledgeable caregiver as either "present" or "absent." Among the ratings on this list that show the highest frequency in the FASD sample are being unaware of the consequences of actions, showing poor attention, being too easily led, overreacting, and showing poor judgment. The scale distills the clinical insights of one early investigator (the same Ann Streissguth who ran the brain study), a collation of reports she had heard from parents and other caregivers of her hundreds of patients, all filtered through the fairly demanding machinery of a psychometric statistical analysis.

In the one published study permitting analysis at the necessary level of detail, we found total scores ranging from 0 to 35 for 472 subjects with a pre-existing FASD diagnosis and ranging from 0 to 27 for 186 age- and sex-comparable subjects from an unselected comparison sample. The total scores for the FASD subsample averaged 20; for the 186 unselected comparison group, the average was 6.6. When we say "unselected," we mean, for instance, that thirteen mothers of persons in this comparison sample reported themselves as having

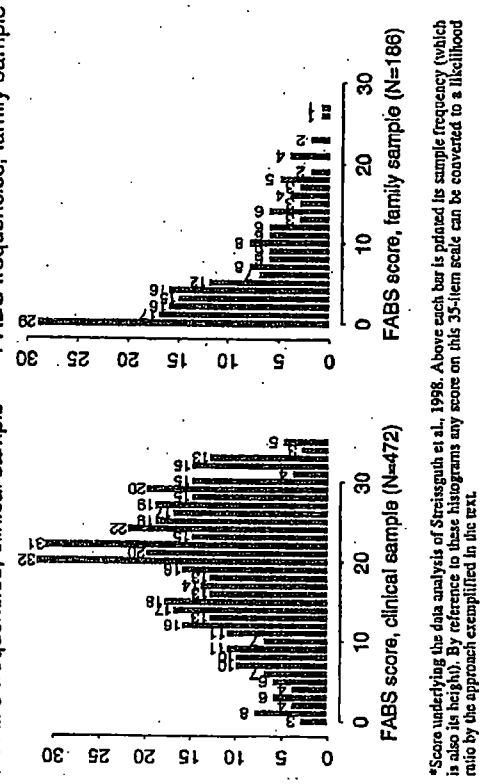
"problems with alcohol"—this comparison group may well have included a few subjects with alcohol damage that had not been recognized by a diagnosis.

Figure 6, an elaboration of one published in the original Streissguth article, expedites the computation of likelihood ratios corresponding to any observed FABS score for a client of interest. These data do not appear to be distributed as bell curves, so we proceed by using actual counts instead of relying on the formulas that would otherwise obtain. For instance, an observed FABS score of 20 or greater (20 is the mean score for the whole group of diagnosed patients) is found in 265 out of 472 of these patients, but only 8 out of 186 subjects from the unselected sample, for a likelihood ratio of 20 to 1, with no correction factor required. An approach along these lines will function to produce ratios like these regardless of whether the test score under consideration is distributed in a bell-curve (Gaussian) distribution, as long as findings for a group of patients and a reasonably comparable group of ostensibly normal subjects are available to be compared in this way. All these items, without exception, are more frequently encountered in the FASD sample of the data underlying Figure 6 than in the normative sample. The item-specific likelihood ratios range from about 1.5 to over 10. For more information on the use of the FABS in this forensic context, please consult FASDExperts, www.FASDExperts.com.

Concluding comment

We have shown how information about corpus callosum form, information not originally in a format familiar to the typical medical or psychiatric expert, can nevertheless be converted into a quantification (the likelihood ratio) compatible with reports of the relevant information conveyed by other channels, such as cognitive test scores, single conventional quantitative measurements of birth status or facial form, or behavioral summaries such as the Fetal Alcohol

FIGURE 6
Distributions of the FABS (Fetal Alcohol Behavior Scale)*



*Score underlying the data analysis of Streissguth et al., 1998. Above each bar is printed its sample frequency (which is also its height). By reference to these histograms any score on this 35-item scale can be converted to a likelihood ratio by the approach exemplified in the text.

Behavior Scale. The combination of information from all of these domains of observation in a context informed by reports of the prenatal exposure is one aspect of the expert's expertise in this forensic area. Another, equally important, is the narrative of the crime as it typifies specific aspects of "the fetal alcohol behavior profile." Because each separate FABS item itself characterizes the FASD pool more than the normative pool, as noted above, such a narrative often includes mention of individual items from the FABS.

In this way the morphometric report becomes part of a standard forensic clinical report for defendants potentially classifiable under one of the FASD categories. Brain structural abnormalities detected by the comparisons here are among the relatively more reliable modes of data available to the medical expert, as the structure itself is laid down so early in infancy (Bookstein et al., 2007) and because so few postnatal causes can affect its form without leaving traces elsewhere, so deep and well-protected in the brain does it lie. In terms of its probative value for a diagnosis of FASD, its information content is rather more like that of childhood facial indicators, which are very nearly pathognomonic, than psychometric deficits, which are so often multifactorial instead. The task of the medical expert is to weave together all of the evidence into an authoritative diagnosis, one specific "color" on the FASD spectrum, and then to draw out the consequences of this diagnosis for the behaviors of the defendant as manifested under whatever conditions of confusion or stress characterized the offense with which he has been charged. Persons diagnosed with an FASD have a great many cognitive and behavioral deficits and a startlingly high incidence of histories of incarceration (Streissguth et al., 1996, 2004). While the morphometric component of an FASD diagnosis arrived at in this way is not designed to predict whatever specific behaviors may lie at the root of the criminal charge, still it helps account for the offending behaviors—just by helping to establish that diagnosis—as consequences of a cause operating before the defendant was even born.

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AMERICAN BAR ASSOCIATION

Co-Sponsors: ABA Commission on Youth at Risk, Criminal Justice Section, Commission on Disability Rights, Commission on Homelessness and Poverty, Death Penalty Representation Project, Health Law Section, Judicial Division, Alaska Bar Association, and the American Judicature Society

RESOLUTION APPROVED BY THE ABA HOUSE OF DELEGATES – AUGUST 7, 2012

RESOLUTION

RESOLVED, That the American Bar Association urges attorneys and judges, state, local, and specialty bar associations, and law school clinical programs to help identify and respond effectively to Fetal Alcohol Spectrum Disorders (FASD) in children and adults, through training to enhance awareness of FASD and its impact on individuals in the child welfare, juvenile justice, and adult criminal justice systems and the value of collaboration with medical, mental health, and disability experts.

FURTHER RESOLVED, That the American Bar Association urges the passage of laws, and adoption of policies at all levels of government, that acknowledge and treat the effects of prenatal alcohol exposure and better assist individuals with FASD.

REPORT

Introduction

Fetal Alcohol Spectrum Disorders (FASD) is a serious problem in the U.S., adversely affecting a very large number of children and families, and in recognition of that the U.S. Senate has annually, since 2004, passed a resolution designating September 9th as National Fetal Alcohol Spectrum Disorders Awareness Day. In 2011 Senate Resolution 259[1] called upon the people of the United States to promote awareness of the effects of prenatal exposure to alcohol, to increase compassion for individuals affected by prenatal exposure to alcohol, to minimize the effects of prenatal exposure to alcohol to ensure healthier communities across the United States, and to observe a moment of reflection during the ninth hour of September 9, 2011 to remember that during her nine months of pregnancy a woman should not consume alcohol.

The Report provides background on FASD and discusses its impact on the justice system, the child welfare system, and the disability benefits system. It also describes approaches to improving the various problems identified in individuals with FASD who are in these systems. In addition, it highlights current initiatives that are in place to benefit individuals with FASD.

The essential focus of this Resolution is to encourage: improvement in the civil, juvenile, and criminal legal representation for persons with FASD; increased access to FASD expert screening and assessment; attention to the over-abundance of FASD-affected persons in foster care, juvenile delinquency cases, adult criminal proceedings, and correctional facilities; and the use of FASD knowledge in court for the mitigation of sentencing and alternatives to incarceration and execution, including therapy and comprehensive services to rehabilitate and reduce recidivism. *Neither the Resolution nor this Report should be construed as suggesting that use of alcohol during pregnancy is, or should be, a criminal act.*

[1] The language of the Senate Resolution includes these observations: "...fetal alcohol spectrum disorders are the leading cause of cognitive disability in Western civilization, including the United States, and are 100 percent preventable...fetal alcohol spectrum disorders are a major cause of numerous social disorders, including learning disabilities, school failure, juvenile delinquency, homelessness, unemployment, mental illness, and crime".

The ABA House of Delegates has not previously addressed the issue of FASD. FASD is a disability that cuts across all age ranges, and it is a lifelong disability. There are unique concerns and problems facing children and youth with FASD that need to be addressed. This includes the very large numbers of children with FASD in both the foster care and juvenile justice system, as well as in the adult criminal justice system and correctional institutions. The focus of the ABA Commission on Youth at Risk is on youth, and that is the reason for its principal sponsorship of this Resolution. Given the unique nature of FASD, this Resolution, while addressing and focusing on issues that impact children with FASD, also contains suggestions for actions that would benefit adults with FASD.

This Resolution and its accompanying Report provides a road map, for legal professionals, lawmakers, and those in government who deal with youth at risk, to increase awareness of FASD. It also encourages federal, state, territorial, tribal, and local law and policy makers to implement laws and policies that reflect the serious effects of prenatal alcohol exposure.

Types of Fetal Alcohol Spectrum Disorders

FASD is a group of conditions that can occur in individuals whose mother drank alcohol during pregnancy.[2] FASD can result in birth defects, growth and development deficits, cognitive and learning issues, executive functioning problems, difficulty remaining attentive, and problems socializing, as well as other behavioral issues.[3]

The correlation between maternal alcohol consumption during pregnancy and deficits in physical and mental development were first identified in the early 1970s.[4] The first common symptoms identified among children who had been prenatally exposed to alcohol were growth deficiencies and developmental delays.[5] Shortly thereafter Kenneth L. Jones, et al., found similar physical characteristics in these individuals.[6] They included similar patterns of craniofacial, cardiovascular, and limb defects.[7]

A follow-up study followed 11 children whose mothers drank heavily during pregnancy and found many common features among this cohort.[8] Among this small sample population, all children displayed growth deficiencies (for height, weight, and head circumference), their abnormal craniofacial features had not changed over the past decade, and they all had below-normal intellectual development.[9] Most of these children had low IQ scores and 8 were either mildly or severely handicapped.[10]

Since that time, several disorders related to fetal alcohol exposure have been identified. There are several types of FASD, including Fetal Alcohol Syndrome (FAS), Partial FAS, Alcohol Related Neurodevelopmental Disorders (ARND), and Alcohol Related Birth Defects (ARBD).[11] These four diagnoses share certain

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[2] National Institute on Alcohol Abuse and Alcoholism, Alcohol Alert, Fetal Alcohol Spectrum Disorders: Understanding the Effects of Prenatal Alcohol Exposure, No. 82.

[3] *Id.* On March 29, 2012 U.S. Senators Johnson, Murkowski, Inoye, and Begich introduced S.2262, the *Advancing FASD Research, Prevention, and Services Act*, that addresses many of the issues included in this Resolution and Report.

[4] Kenneth L. Jones, David W. Smith, Christy N. Ulleland, & Ann Pytkowicz Streissguth, *Pattern of Malformation in Offspring of Chronic Alcoholic Mothers*, THE LANCET, June 9, 1973, at 7815.

[5] C. N. Ulleland, *The Offspring of Alcoholic Mothers*, 197 *ANN. NY ACAD. SCI.* 197 (1972).

[6] Medically referred to as aberrant morphogenesis.

[7] Jones et al., *supra* note 4.

[8] Ann Pytkowicz Streissguth, Sterling Keith Clarren, & Kenneth Lyons Jones, *Natural History of the Fetal Alcohol Syndrome: A 10-Year Follow-up of Eleven Patients*, THE LANCET, July 13, 1985, at 85.

[9] *Id.*

[10] *Id.*

[11] National Institute on Alcohol Abuse and Alcoholism, *supra* note 2.

characteristics and fall within the broader category of FASD.[12] All but ARBD involve significant brain damage.

FAS is the most severe of the conditions that constitute FASD. An FAS diagnosis requires three specific deficits:[13] a characteristic pattern of facial abnormalities known as facial dysmorphology, such as a smooth ridge between the nose and upper lip;[14] growth deficits, such as lower than average weight and/or height;[15] and central nervous system abnormalities or brain damage.[16] The latter may include a diagnosis of Attention Deficit Disorder (ADD) or Attention Deficit Hyperactive Disorder (ADHD).[17] While generally, individuals with FAS tend to be the most impaired, with more severe physical and cognitive issues than individuals with other forms of FASD,[18] those with Partial FAS and ARND tend to show the most severe secondary disabilities.[19] FAS alone costs the United States approximately $5.4 billion each year in direct and indirect costs.[20] It is also the leading cause of non-genetic intellectual disability in the United States.[21]

Partial FAS includes some of the signs and symptoms of full FAS but not all.[22] Individuals with Partial FAS typically still present with physical and emotional deficits, but do not have all of the physical characteristics listed in the FAS diagnostic guidelines. ARND includes central nervous system abnormalities and other cognitive/behavioral problems, but none of the outward physical abnormalities.[23] Individuals with partial FAS and ARND present with cognitive issues that can be as severe as those seen in FAS. ARBD is a rarely-used diagnosis that only identifies alcohol related physical abnormalities outside the central nervous systems (e.g., skeletal or organ abnormalities).[24]

The prevalence of full FAS and FASD has been examined in several studies. In one, the prevalence of full FAS in the U.S. was estimated at 0.5-2.0 cases per 1000 births.[25] The estimated rate of alcohol-affected births was estimated to be 5 to 10 times higher, close to 1% of newborns.[26] A more recent study reported the FAS prevalence in the U.S. to be at least 2 to 7 cases per 1000 births, with all levels of FASD estimated as high as 2-5% among younger school children.[27] There is currently an NIAA initiative to establish more accurate estimates of FASD prevalence.

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[12] It is important to note that FASD is not a diagnosis; it is a broader category of related diagnoses.
[13] National Institute on Alcohol Abuse and Alcoholism, *supra* note 2.
[14] *Id.* at 2.
[15] *Id.* at 1.
[16] *Id.*
[17] *Id.*
[18] *Id.*
[19] Streissguth, A., Barr, H., Kogan, J., & Bookstein, F. (1966). Understanding the occurrence of secondary disabilities in clients with fetal alcohol syndrome (FAS) and fetal alcohol effects (FAE). Final Report: Centers for Disease Control and Prevention Grant No. R04/CCR008515.
[20] National Organization on Fetal Alcohol Syndrome, FASD: What Everyone Should Know, http://www.nofas.org/MediaFiles/PDFs/factsheets/everyone.pdf.
[21] National Institute on Alcohol and Abuse and Alcoholism, *The 10th Special Report to the U.S. Congress on Alcohol and Health: Prenatal Exposure to Alcohol*, No. Publication No. 00-151583 (2000); Amy M. Schonfeld, Blari Paley, Fred Frankel, and Mary J. O'Connor, *Executive Functioning Predicts Social Skills Following Prenatal Alcohol Exposure*, 12 CHILD NEUROPSYCHOLOGY 439 (2006).
[22] National Institute on Alcohol Abuse and Alcoholism, *supra* note 2, at 1.
[23] *Id.*
[24] *Id.*
[25] NIH Fact sheet; P.A. May & J. P. Gossage, *Estimating the prevalence of Fetal Alcohol Syndrome: A Summary*, 25 ALCOHOL RESEARCH & HEALTH 159 (2001).
[26] Diane V. Malbin, *Fetal Alcohol Spectrum Disorder (FASD) and the Role of Family Court Judges in Improving Outcomes for Children and Families*, JUVENILE & FAM. CT. J. 52 (2004).
[27] Philip A. May, J. Phillip Gossage, Wendy O. Kalbert, Luther K. Robinson, David Buckley, Melanie Manning, and H. Eugene Hoyme, *Prevalence and epidemiologic characteristics of FASD from various research methods with an emphasis on recent in-school studies*. Dev Disabil Res Revs, 15: 176-192 doi: 10; 1002/ddrr.68 (2009).

The intent of this Resolution is to spur development of programs for those living with FASD and their families. This should especially include youth transitioning from foster care and juvenile justice systems, since those with FASD are especially vulnerable to physical and sexual abuse. One study of over 400 individuals with FASD found that 72% had been abused, either physically or sexually.[28]

Cause of FASD

FASD is caused by prenatal alcohol exposure. There is no safe amount of alcohol to drink during pregnancy, and even small amounts of alcohol may have an impact on fetal brain development.[29] A U.S. Surgeon General's 2005 advisory states that: pregnant women should *not* consume alcohol during pregnancy; pregnant women who have already consumed alcohol while pregnant should *stop* to minimize risk; and women who are *considering* becoming pregnant should not drink alcohol.[30] In addition, former Surgeon General Carmona recommended that health professionals routinely ask women of child bearing age about their alcohol consumption and advise them not to drink during pregnancy, noting that this is of particular importance since about half the births in the United States are unplanned.[31] Many professional medical association guidelines also indicate that women should not drink alcohol during pregnancy.[32]

Although any prenatal alcohol exposure presents a risk, there does seem to be a correlation between the amount of alcohol a woman drinks during pregnancy and the likelihood her child will have FASD.[33] Some women who consume alcohol heavily during pregnancy do not have a child with FASD.[34] However, typically, the more a woman drinks during pregnancy, the higher the risk her child will have FASD and the more severe that child's symptoms will be.[35]

Diagnosis and Treatment

There are several diagnostic guidelines for FASD, each of which list characteristics required for a FAS, partial FAS, ARND, or ARBD diagnosis. Despite these guidelines, there are still challenges to diagnosing individuals with these disorders. It may be particularly difficult to diagnosis less severe cases of FASD. Individuals with FASD do not always present with observable physical characteristics, making it more difficult to diagnose them.[36] In addition, one of the most helpful ways to diagnose FASD is when there is confirmed prenatal alcohol exposure; however, mothers are often reluctant to admit they drank alcohol during pregnancy due to a sense of guilt or shame.[37]

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[28] Ann Streissguth, *Attaining Human Rights, Civil Rights, and Criminal Justice for People with Fetal Alcohol Syndrome*, TASH NEWSLETTER, September 1998, at 18.
[29] U.S. Surgeon General, Surgeon General's Advisory on Alcohol and Pregnancy, 2005, *available at* http://www.surgeongeneral.gov/pressreleases/sg02222005.html; Claire Coles, *Discriminating the Effects of Prenatal Alcohol Exposure From Other Behavioral and Learning Disorders*, 34 ALCOHOL RESEARCH AND HEALTH 42 (2011).
[30] U.S. Surgeon General, Surgeon General's Advisory on Alcohol and Pregnancy, 2005, *available at* http://www.surgeongeneral.gov/pressreleases/sg02222005.html.
[31] *Id.*
[32] *See e.g.*, American College of Obstetricians and Gynecologists.
[33] Susan E. Maier & James R. West, *Patterns and Alcohol-Related Birth Defects*, National Institute on Alcohol Abuse and Alcoholism, *available at* http://pubs.niaaa.nih.gov/publications/arh25-3/168-174.htm.
[34] This may be due to a number of factors including, but not limited to, genetic susceptibility, maternal metabolism, drinking patterns,
[35] Maier, *supra* note 33.
[36] Malbin, *supra* note 26.
[37] Kenneth Lyons Jones & Ann P. Streissguth, *Fetal Alcohol Syndrome and Fetal Alcohol Spectrum Disorders*, 38 J. PSYCH & L. 373 (2010).

Individuals with FASD may have IQs that are in the average range and appear to have good verbal skills, making it more difficult to recognize FASD.[38] Although these individuals may not have obvious symptoms or characteristics, they typically exhibit adaptive behavior and other deficits that make it difficult for them to function at an age appropriate level.[39]

There is no cure for FASD, and the deficits associated with these disorders follow children into adulthood.[40] That being said, early intervention and treatment services may improve a child's development. Strategies include medication for some symptoms, behavioral and educational therapies, special education, social services, and the support of a nurturing and loving environment.[41] These interventions are most effective when a child is diagnosed before age 6.[42] Children with FASD who were involved in intervention programs to improve social skills showed improvements in both knowledge and behavior.[43]

It is critical that comprehensive resources be allocated for early identification, diagnosis, intervention, and treatment for those with FASD. Given the array of problems individuals with FASD face, and the importance of early intervention, it is vital that professionals become aware of the issue so that appropriate services can be provided.

Impact on the Justice System

Children with FASD are at high risk of getting into trouble with law. One study looked at FASD in alleged juvenile offenders in British Columbia, Canada during 1995. All youth from the juvenile court who were sent to the Inpatient Assessment Unit for purposes of a psychological and psychiatric study were also assessed for what was then called FAS/FAE and is now called FASD. Of the 287 youth seen over a year's time, 23.3 % had an alcohol-related diagnosis (FASD). The authors also found, among youth in juvenile facilities, an astounding 40 times the expected rate of individuals with FASD.[44]

Individuals with FASD have executive functioning issues that result in difficulties socializing with peers, sharing, and managing conflicts.[45] This can result in rejection, which leads to a higher probability of individuals with FASD associating with other rejected children and a higher probability that they will be involved in delinquent behaviors and have problems with the law.[46]

A large study at the University of Washington found that about 60% of individuals with FASD had a history of trouble with the law and 50% had a history of confinement in a jail, prison, residential drug treatment facility, or

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[38] Natalie Novick Brown, Anthony P. Wartnik, Paul D. Connor, and Richard S. Adler, *A Proposed Model Standard for Forensic Assessment of Fetal Alcohol Spectrum Disorders*, 38 J. OF PSYCH. & L. 383, 387 (2010).
[39] Kathryn Page, *The Invisible Havoc of Prenatal Alcohol Damage*, J. CENTER FOR FAM. CHILD. & CTS. 1, 10-11 (2002).
[40] Blair Paley and Mary J. O'Connor, *Neurocognitive and Neurobehavioral Impairments in Individuals with Fetal Alcohol Spectrum Disorders: Recognition and Assessment*, 6 INT'L J. DISABIL. HUM. DEV. 127, 130 (2007).
[41] *Id.*
[42] *Id.*
[43] Mary O'Connor et al., *A Controlled Social Skills Training for Children with Fetal Alcohol Spectrum Disorders*, 74 J. CONSULTING & CLINICAL PSYCH. 639, 646 (2006).
[44] Malbin, *supra* note 26, citing Julianne Conry and Diane K. Fast, *Fetal Alcohol Syndrome and Criminal Justice*, BC: Fetal Alcohol Syndrome Resource Society (2000); *see also*, Diane K. Fast, Julianne Conry, and Christine A. Loock, *Identifying Fetal Alcohol Syndrome Among Youth in the Criminal Justice System*, Developmental and Behavioral Pediatrics, v. 20(5), October 1999.
[45] Schonfeld, et al., *supra* note 21, at 450.
[46] *Id.*

psychiatric hospital.[47] The average age children with FASD begin having trouble with the law is 12.8 years.[48] This may be in part due to the fact that children with FASD are easily led by others and tend to be impulsive.[49] Individuals with FASD have various characteristics that put them at a greater risk of ending up in the criminal justice system. For example, as was mentioned, they are typically impulsive and have difficulty predicting the consequences of their actions.[50]

In addition, given their executive functioning problems, these individuals may not always have the legal capacity to commit deliberate/intentional crimes.[51] Given these characteristics:

...[i]n the criminal context, FASD-associated problems in reflection, forming intent, and carrying out effective goal-directed behavior are directly relevant to mental state...their behavior often breaks down or decomposes in novel high-stress situations...This decomposition often leads to instinctive fight or flight reaction...neglecting previous learning experiences, consequences, and impact on themselves and others.[52]

Several issues surrounding individuals with FASD in the justice system include competency to stand trial, validity of expert testimony,[53] and mitigation during sentencing.[54] Individuals with FASD may not understand charges.[55] Since individuals with FASD cannot always form the requisite intent required for certain crimes and do not fully understand the consequences of their actions, defendants with FASD may face diminished capacity issues.

In *Dillbeck v. State*, the court held that FASD should be considered in the guilt/innocence phase of the trial as well as in sentencing, noting that:

... Evidence concerning certain alcohol-related conditions has long been admissible during the guilt phase of criminal proceedings to show lack of intent ... then so too should evidence of other commonly understood conditions that are beyond one's control ... [w]e perceive no significant legal distinction between the condition of epilepsy... and that of alcohol-related brain damage in issue here – both are specific, commonly recognized conditions that are beyond one's control.[56]

This is significant because the court recognized the benefits of considering FASD during both the trial phase and during sentencing.

Confirming a diagnosis of FASD may be critical in designing a sentence or sentencing alternative that will be effective in reducing the risk of recidivism and will avoid causing far greater harm to a defendant with FASD than to a defendant without this disability. Because of their impairments, individuals with FASD, when confined in a jail or prison, can be more vulnerable than those who are not disabled to physical and sexual abuse, and consequently more adversely affected than others.[57]

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[47] Natalie Novick Brown, Anthony P. Wartnik, Paul D. Connor, and Richard S. Adler, *A Proposed Model Standard for Forensic Assessment of Fetal Alcohol Spectrum Disorders*, 38 J. OF PSYCH. & L. 383, 384 (2010).

[48] *Id.*

[49] Natalie Novick Brown, Gisli Gudjonsson, & Paul D. Connor, *Suggestibility and Fetal Alcohol Spectrum Disorders: I'll Tell You Anything You Want to Hear*, 39 J. OF PSYCH. & L. 39 (2011).

[50] Substance Abuse and Mental Health Services Administration: A Fetal Alcohol Spectrum Disorders Center for Excellence. *What You Need To Know: Fetal Alcohol Spectrum Disorders and Juvenile Justice: How Professionals Can Make a Difference*. DHHS Pub. No. (SMA)-06-4240 (Rockville, MD: 2007).

[51] Brown, et al., *supra* note 49.

[52] Brown et al., *supra* note 47.

[53] *See* State v. Brett, 126 Wash. 2d 136, 892 P.2d 29 (1995); Castro v. State of Oklahoma, 71 F.3d 1502 (10th Cir. 1995) (discussing who can determine a whether an individual has FASD when it in unknown if the mother drank alcohol during pregnancy).

[54] U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services, Fetal Alcohol Spectrum Disorders and the Criminal Justice System, *available at* www.samhsa.gov.

[55] *See* Dunn v. Johnson, 162 F.3d 302 (5th Cir. 1998); People v. Flemming, 2003 WL 21675890 (Mich. App.); State v. Lee, 220 Wis. 2d 716, 583 N.W. 2d 674 (Ct. App. Wisc. 1998).

[56] Dillbeck v. State, 643 So. 2d 1027 (Fla.).

[57] Kathryn A. Kelly, "Fetal Alcohol Spectrum Disorders and the Law," in Prenatal Alcohol Use and FASD: Diagnosis, Assessment and New Directions in Research and Multimodal Treatment (Susan A. Adubato and Deborah E. Cohen, eds), 2011.

In recent years, FASD has been offered as a mitigating factor during sentencing; there are many death penalty cases in which the defendant's history suggests FASD and a diagnosis of FASD has been persuasive to juries as an explanation for otherwise inexplicable behavior. Also, in *Atkins v. Virginia,* the U.S. Supreme Court held that executing an individual with an intellectual disability violates the Eighth Amendment.
In some cases this will exclude the defendant with FASD from eligibility for the death penalty. However, if an I.Q. score of 70 or below, as many states define intellectual disability, is what disqualifies a defendant from the death penalty then most of those with FASD, because of their typically higher scores, would not be covered by *Atkins.*[58]

Children and adults with FASD are becoming involved in the justice system at an alarming rate. The unique characteristics of these individuals warrant additional attention.[59] This Resolution encourages increased awareness of FASD among attorneys, judges, other court professionals, and court appointed advocates. These individuals are urged to utilize existing screening tools to identify clients with FASD.

Attorneys, judges, bar associations and law school clinical programs, as well as all other persons and entities involved with youth at risk, juvenile justice, or adult criminal court, should also support training and develop enhanced awareness and understanding of FASD. This can be accomplished by collaborating with medical, mental health, and disability experts on training to enhance representation for individuals with FASD. Current initiatives that strive to achieve such successes are discussed later in this Report and can be used as potential models.

Courts should also be considering FASD disability as a factor in mitigation with juvenile and adult offenders during sentencing, particularly where the death penalty is an option. This mitigating factor should also be applied when considering alternatives to incarceration, including therapy, community-based programs, and other non-custodial measures, in order to rehabilitate the individual and reduce recidivism.

Impact on the Child Welfare System

A large study of over 400 individuals with FASD, ranging from age 6-51, resulted in various concerns about human rights, civil rights, and criminal justice system involvement for individuals with FASD.[60] An astounding 80% of children in that study had not been primarily raised by a biological parent.[61] In addition, 12% of children and 60% of adolescents experienced significant school disruptions.[62] This statistic is especially disturbing since children with FASD already face heightened academic challenges based on several of the mental and behavioral characteristics associated with FASD. It is clear that children with FASD are part of the "at-risk" population and should have access to special programs and services.

There are a disproportionate number of children with FASD in the foster care system; the rate of FAS in the foster care system is 10-15 times higher than in the general population.[63] In the United States, an estimated 70% of children in foster care are affected by some type of prenatal alcohol exposure.[64] This is particularly

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[58] Atkins v. Virginia, 536 U.S. 304 (2002).
[59] Timothy E. Moore & Melvyn Green, *Fetal Alcohol Spectrum Disorder (FASD): A Need for Closer Examination by the Criminal Justice System*, 19 CRIM. REPORTS 99 (2004).
[60] Ann Streissguth, *Attaining Human Rights, Civil Rights, and Criminal Justice for People with Fetal Alcohol Syndrome*, TASH NEWSLETTER, September 1998, at 18.
[61] *Id.*
[62] *Id.*
[63] SJ Astley, J Stachowiak, SK Clarren, & C Clausen, *Application of the fetal alcohol syndrome facial photographic Screening Tool in a Foster Care Population*, 141 J. Pediatrics 712 (2002).
[64] The National Organization on Fetal Alcohol Syndrome, FASD in the Foster Care System, http://adp.ca.gov/women/pdf/FASD_in_the_Foster_Care_System.pdf

worrisome because children with FASD benefit from having a stable environment, a comfort that is, unfortunately, not available for many children in foster care.

Children in foster care are already at high risk for educational disruptions as well as behavioral and developmental issues. This is exacerbated when that child has FASD. It would be beneficial to provide training and education to foster parents on how to identify the behaviors associated with FASD, how to seek a diagnosis of the disability, and how to appropriately respond to characteristics and behaviors associated with FASD. This training could provide foster parents with information about services and programs available for children with FASD.

Full implementation is also necessary for a provision of the federal *Child Abuse Prevention and Treatment Act*[65] that was intended to provide for more effective screening and referral processes for individuals with FASD, in recognition of the importance of addressing the over-abundance of children with FASD in the child welfare system. Fully implementing that Act's existing mandate of screening and referral processes for children with FASD can help assure their early identification and hopefully prompt access to successful treatment.

Impact on Disability Benefits

Individuals with disabilities may be eligible for medical and cash benefits, such as Social Security Disability, based on their disability. Children and adults with FASD may be eligible for such benefits, but a diagnosis of FASD does not create an automatic entitlement[66] and they will be required to meet statutory criteria, and establish that such impairment exists.[67] In some cases, individuals will be required to prove that FASD severely limits their work activities, which may not be the case for many individuals with FASD.[68]

Individuals with FAS or more severe FASD symptoms will have an easier time proving they are eligible for benefits. That being said, even individuals with severe cases of FAS may face difficulties establishing their eligibility because the system is complex and often difficult to navigate.[69] Also, state law and policy may not list FASD related impairments within their definitions of "developmental disabilities."[70] Individuals who seek attorney representation may have an easier time establishing eligibility and gaining access to disability benefits that will improve their quality of life.

It is important that FASD, alcohol-related neurological disorders, alcohol-related birth defects, and the effects of fetal alcohol exposure generally, be included within statutory definitions of developmental disabilities and listing of conditions that provide medical and other benefit coverage for screening, diagnosis, and treatment for those with these conditions. Law and policy makers should ensure that individuals with FASD are eligible for disability benefits and appropriate medical services. Eligibility and utilization of these benefits and services will help improve the lives of individuals with FASD, especially youth and those transitioning out of the foster care or juvenile justice system.

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[65] 42 U.S.C. 5106a (b)(2)(B).

[66] Amy Gilbrough, *Eligibility for Social Security Benefits: Fetal Alcohol Spectrum Disorders, in* Alcohol Related Birth Disorders and the Law: How Should Attorneys & Judges Respond to FASD?, Continuing Legal Education Materials (Feb. 3, 2012).

[67] *Id.*

[68] *Id.*

[69] *Id.*

[70] One state law that does include FASD as a "related condition" within the definition of developmental disabilities is Minnesota Statutes §252.27.

Current Initiatives

There are many beneficial programs and initiatives in the United States focusing on increasing awareness of FASD among legal professionals, identifying individuals with FASD in the justice system, and ensuring that these individuals receive appropriate and necessary services. This section highlights two of these initiatives.

Seventeenth Judicial District Juvenile Court FASD Project

The Juvenile Court of Colorado's 17th Judicial District's FASD Project is working to increase awareness of FASD among judicial officers, attorneys, and court appointed advocates and use referrals as an effective tool for children in the child welfare system.[71] The FASD Project screens children in the Juvenile Delinquency and Child Welfare Courts of Adams and Broomfield Counties for prenatal substance exposure.[72] By integrating FASD screening, diagnosis, and intervention within the court system, the FASD Project is working to improve the lives of children and youth with FASD.

The FASD Project's key strategies are:

- To integrate FASD screening into Juvenile Court;
- To refer children to a diagnostic center for evaluation and a possible diagnosis;
- To meet with key players in the child's life, including parents, case workers, and probation officers, to develop individualized case plans for children with FASD; and
- To track data and monitor the success of this project.

The FASD project is so important because "it is identifying, supporting and tracking outcomes for children and youths who would otherwise have a high likelihood of failing in school, experiencing multiple placements, and re-offending or violating probation."[73] This project provides the necessary care for children with FASD while increasing awareness among professionals who work with these individuals to ensure that the appropriate care and services are provided.

Alaska FASD Partnership

The Alaska FASD Partnership is a statewide coalition of over 75 organizations and individuals working to prevent FASD and improve access to services for individuals with FASD. The mission of the partnership is "[t]o promote awareness, prevention, and effective life-long interventions for those affected by prenatal exposure to alcohol and their families."[74] Through seven workgroups, the partnership has been able to identify gaps and barriers to services for individuals with FASD.[75] The workgroups develop policy and funding recommendations and strive to increase awareness about this issue.

In 2010, the partnership's first year, they were integral in establishing state funding for substance abuse treatment programs for pregnant women, parent navigation services, and greater access to services for individuals with FASD.[76] The workgroups are currently addressing several important issues, including prevention of FASD, diagnosis and access to services, the impact of FASD in the legal and education systems, and professional development.[77] Identifying these issues and establishing best practices will ensure better services and care for individuals with FASD.

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[71] Seventeenth Judicial District Juvenile Court FASD Project, Project Summary, http://www.fasdcenter.com/files/17thJudicialDistrictColorado-Article.pdf.
[72] *Id.*
[73] *Id.*
[74] Advisory Board on Alcoholism and Drug Abuse, Alaska FASD Partnership, http://www.hss.state.ak.us/abada/fasd.htm.
[75] *Id.*
[76] *Id.*
[77] *Id.*

The Seventeenth Judicial District FASD Project and the Alaska FASD Partnership are two initiatives that exemplify meaningful work being done around the country to serve individuals with FASD. As highlighted by this Report, legal professionals and individuals involved with youth at risk should be supporting training and awareness of FASD. In addition, lawmakers should be addressing various issues surrounding FASD to ensure that individuals with FASD have access to appropriate services and are treated fairly.

Conclusion

Given the alarming data, and the unique characteristics of individuals with FASD, there is a clear need for increased awareness of FASD. Attorneys, judges, bar associations, law schools, and other entities involved with at-risk youth, juvenile justice, and the adult criminal court should support training and awareness of FASD. These individuals should collaborate with medical, mental health and FASD disability experts to promote appropriate legal representation and advocacy for individuals with FASD and to address the over-abundance of individuals with FASD in the foster care system, the juvenile justice system and the adult criminal court.[78] In addition, courts should use FASD as a mitigating factor for juveniles and adults during criminal sentencing.

Federal, state and local law and policy makers should also enact laws and policies that reflect the serious effects of prenatal alcohol use. It is vital to increase public awareness, especially for women who are substance abusers, pregnant, or of childbearing age, about FASD to both prevent it's occurrence and to ensure that individuals with FASD, and specifically children and youth with FASD, have access to appropriate services and, if they are involved in the courts, skilled legal representation.

[78] In August 2010 the Council of the Canadian Bar Association approved resolution 10-02-A, which called for the "initiative of Federal, Provincial and Territorial Ministers responsible for Justice with respect to access to justice for people with FASD," urged "all levels of government to allocate additional resources for alternatives to the current practice of criminalizing individuals with FASD," urged "federal, territorial and provincial governments to develop policies designed to assist and enhance the lives of those with FASD and to prevent persistent over-representation of FASD affected individuals in the criminal justice system," and urged "the federal government to amend criminal sentencing laws to accommodate the disability of those with FASD."

STATE OF SOUTH CAROLINA
County of Greenville

In the Court of Common Pleas
2010-CP-23-9792

CHARLES CHRISTOPHER WILLIAMS, Applicant,

V.

THE STATE OF SOUTH CAROLINA, Respondent.

AFFIDAVIT OF DR. DAVID A. GRIESEMER

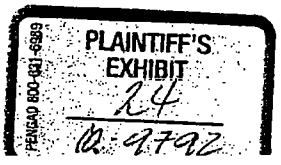
Dr. David A. Griesemer, who appeared personally before me, affirms and states the following:

I am a neurologist presently working at Tufts Medical Center in Boston. From 1993 through 2010 I was employed full time at the Medical University of South Carolina in Charleston. I interviewed and evaluated Charles Christopher Williams for two hours on January 27, 2005 at the request of Mr. Williams' attorneys, and I wrote the attached report. It is now my practice to date when the report is drafted as well as when the examination is performed, but I did not date this final report and cannot say when it was delivered to the attorneys. I do not recall whether I talked to either of Mr. Williams' attorneys or anyone else on the defense team about the findings of my examination. I have no independent recollection of the evaluation of Mr. Williams, but I have reviewed my report and subsequent neuropsychological testing done by Dr. Jim Evans.

In reviewing my report, I immediately noted that the evaluation was based exclusively upon interview and examination of Mr. Williams, and that no medical, educational, or judicial records were made available for review. I reported this fact on page 4 of my report, "No school-based psychoeducational test results or court-ordered psychological assessments were available for review." I also noted it on page 5 of my report, "These above opinions are based upon limited information presently available; should there be additional data (particularly that which addresses educational, psychological, or adaptive capabilities) which alter my opinions, I will supplement this report."

I also noted on page 5 of my report that Mr. Williams should be evaluated for high blood pressure and a scalp condition, and, "More importantly, given subtle (but likely stable) neurologic abnormalities, including slight asymmetry of eye movements and balance, formal neuropsychiatric evaluation may be helpful."

I have since reviewed additional information from the defense team, including notes from Jan Vogelsang regarding performance on neuropsychological testing, a brief history of school



performance, and new history that Mr. Williams' mother drank alcohol throughout her pregnancy with him.

Had this additional information been made available to me, I would have structured my report and my recommendations differently. The information that was available to me at the time of evaluation was certainly less information than I normally have when completing a patient evaluation. To begin with I was unaware that there was neuropsychological testing results available for review. I was also unaware that Mr. Williams suffered a head injury. I was also unaware that his mother consumed alcohol during her pregnancy, or that Mr. Willaims had poor performance in school.

Despite this lack of history, I did note that he had delayed recall and difficulty with frontal lobe flexibility. These are indicative of cognitive problems. Were I to have seen neuropsychological test results that I did not see, characterized by a 15 point spread between Verbal IQ and Performance IQ, and and by evidence of frontal lobe difficulty and rigidity, I would have expressed greater concerne about Mr. Williams' neurological functioning.

Particularly important would have been the results of the Wisconsin Card Sorting test. During this test the rules change as the patient progresses through the test and part of the evaluation is whether the subject has the ability to detect changes in the rules. Mr. Williams took 18 trials to understand the very first rule; this poor performance placed him in bottom three percentof test takers.

The mother's drinking is a significant piece of history. If this were made known to me, I would have certainly informed the team to investigate fetal alcohol syndrome or to at least rule out fetal alcohol effects.

I have never testified as an expert on fetal alcohol syndrome (FAS) and I was not aware of the CDC Guidelines regarding FAS until speaking to Mr. Enderlin. While I have patients with FAS that I follow for neurologic issues, I would not consider myself an expert on FAS.. Furthermore, regardless of the FAS diagnosis, had I been given the neuropsychological testing information as well as other relevant data, I would have noted compelling evidence of problems with judgment and cognitive functioning that might be significant in understanding his actions.

David A. Griesemer

Boston, Massachusetts

Sworn to me this 27 day of September, 2012

Notary Public for MA. My commission expires: 11/9/18

Neurological Evaluation

Name:	**Charles Christopher Williams**
Date of Birth:	[redacted]
MUSC ID:	[redacted]
Evaluation Date:	**January 27, 2005**

Referred By:	Billing To:
Mr. William Nettles 914 Richland Street Suite A-102 Columbia, SC 29201 (803) 779-9966	Greenville Court Public Defenders Office Attn: Mr. John Mauldin P.O. Box 10264 Greenville, SC 29603

Sources of Information

The following evaluation is based exclusively upon interview and examination of Mr. Charles Christopher Williams. No medical, educational, psychological, or judicial records are available for review.

Venue of Examination

Mr. Williams was transported for evaluation from the Greenville County Detention Center, where he has been incarcerated for the past 18 months, to the Medical University of South Carolina, Rutledge Tower, 135 Rutledge Avenue. He arrived at 10:30 AM with evaluation taking place between 11:00 AM and 1:00 PM. Un-handcuffed and unshackled, Mr. Williams met with me privately in a patient examination room, with security guards outside the room. The available time was divided between review of neurological history, testing of cognitive abilities, and physical examination of general and neuromuscular function.

At the outset of the evaluation we established that Mr. Williams understood the purpose of my evaluation and the absence of typical patient confidentiality; that he could hear my questions and would ask for clarification if he did not fully understand them; that he could adequately see me, my gestures, and materials that I showed him; that he would seek to clarify any confusion that he encountered during the evaluation; and that there would be no questions about the criminal act for which legal proceedings are ongoing.

Current Medical Concerns

Mr. Williams indicated that he had no medical problems or concerns at the present time. However, he acknowledged that he had been hospitalized in June, 2003, at Greenville Regional Medical Center with an overdose of **Klonopin**. He indicated that is last medical evaluation was in December, 2003, when he was given **Risperdal** for depression. He said that he is no longer taking the medication because he did not sleep as well while taking it, even though during the day it made him sleepy and it "mentally slowed (him) down."

Review of Systems

Review of all organ systems was negative, indicating that Mr. Williams reported no abnormal symptoms. Hew specifically denied prior head injuries, seizures, loss of consciousness, or fainting spells. When asked about sleep, he indicated that in high school – because of his need to work – he only slept 4 or 5 hours a night, struggling to wake up when

called and often falling asleep in class. (He purchased a car and was carrying both the car payment and the $200/month insurance payment; he "didn't want to shift responsibility" to his parents; he considered this his personal "obligation, otherwise I would have.") He is sleeping satisfactorily now.

When asked about sadness, he indicated that he has experienced no problems recently. However, before he was arrested (June through September, 2003) he was very "irritable," feeling himself to be "a loner. . . unfriendly." This surprised him as he considers himself to usually be a "high energy" person. He said he was "definitely a big spender . . . money would flow out of my pocket." He acknowledged that he "oversteps boundaries at times." When asked how he handles anger, he indicated that he "separates himself from the situation and from people" and that he is "never violent."

Family Profile

Mr. Williams stated that his mother, Daisy Huckaby, is of German-Polish descent. Now 56 years old, she separated from Mr. Williams' father in 1991 and remarried in 1998. She is a cosmetologist who graduated from high school and college. He is aware the she takes medication for high blood pressure and "nerve pills" for what may be bipolar illness.

Mr. Williams stated that his father, Dwight Miller, is African-American and also 56 years old. A graduate of high school and the University of Maryland, he met Mr. Williams' mother in Germany, when he worked for the Army as an aircraft mechanic. Now retired from the military, he has worked in the mortgage loan business for 7 or 8 years. Mr. Williams describes his relationship with his father as "irregular," with communication occurring "off and on." They may talk as often as once a month, or they may allow 6 to 18 months to pass between conversations. Mr. Williams said that his father was "fun to be around" and taught him "ethics and morals." Mr. Williams recalls that "it was a task to get him to spend time with me" and that he was "irregular with (playing) sports or videogames (with me)."

Mr. Williams has one sister, Maureen Bangoyo, who is 33 years old and a registered nurse. Mr. Williams recalls that his "sister did as much as his mom" in raising him. His step-father is Rodney Huckaby, who was married previously but had no children.

Mr. Williams grew up in Taylors, South Carolina (in Greenville County) because his father was working as an aircraft mechanic at Lockheed. He recalls that his parents were always fighting that there was physical abuse involved. When his mother called the police, his father left and it was necessary for them to sell their house. Mr. Williams recalls having to move every 2 to 3 months, going "from place to place, to the Women's Shelter in Spartanburg" until his mother married Rodney Huckaby.

Psychosocial Profile

Mr. Williams described his strengths as being "loyal" and "dependable," noting that he would both *finish* his work and *do it well.* He felt his weakness was being "stubborn (but agreeable)," especially "about what I wanted to do." He described his sense of humor as "pretty decent . . . no comedian, but I try to make people laugh."

Educational History

Mr. Williams stated that he dropped out of school during his 9th grade year for financial reasons, even though he felt he was "good in class." He recognized the importance of getting his GED, however, and he took preparatory classes at Greenville Tech. Ironically, he never took the test because he was "too nervous" about how he would do.

In elementary and middle school, Mr. Williams got "in trouble daily for talking. . . the things I did before were just not funny anymore." He noted that his grades were "average or

slightly below at the beginning of the year, and got much better at the end of the year when tested." All things considered, Mr. Williams concluded: "I know I had the ability to be a good student" and "I could have been a lot better than I was at the time."

Cognitive Evaluation

Mr. Williams was focused, respectful, restrained, and cooperative throughout the visit. He answered questions promptly, clearly and with detail, revealing no evidence of confusion or delay in processing what he heard. There were no lapses of eye contact or vigilance that might suggest seizure activity. There was no suggestion of fatigue in his attention, effort or cooperation, and there was no suggestion of distractibility at any time during the 2-hour session. There were no restless movements or tics. Mr. Williams' affect was appropriate; he demonstrated no jocularity, excessive familiarity, suspicion or apathy. Although subdued and perhaps mildly dysthymic, he demonstrated a normal range of emotions, without lability, anger, or euphoria. Intensity and congruence of emotions were appropriate. Mr. Williams' speech was fluent, his vocabulary was good, and his sentences were moderately complex and appropriate for casual conversation.

Mr. Williams' cognitive skills were evaluated using the mental status assessment recommended by the American Academy of Neurology[1], supplemented by elements from the Neurobehavioral Cognitive Status Examination[2]. Mr. Williams' *global attention*, as measured by orientation to time and by A-vigilance, was normal. *Speech comprehension* assessed by verbal commands (token test) was also normal. *Language screening*, as assessed by verbal repetition and category fluency were normal. His writing to dictation was normal, with clear, neat printed letters and correct use of capital and lower case forms. *Visual naming to confrontation* was marginal; he called the stem of a watch the "winder" or "dial" and he called a belt buckle a "belt loop." *Memory screening* using the reverse digit span was normal; Mr. Atkins could correctly recall 5 digits backwards. A more detailed test of memory, the serial word list learning test, was also administered; this is a sensitive screening device for frontal lobe dysfunction and left mesial temporal lobe dysfunction. In this test a normal response is to recall five of 10 unrelated nouns on the first trial and nine of the same 10 nouns by the fourth trial. Mr. Williams recalled seven of 10 on the first trial, nine of 10 on the second trial, and ten of 10 on the third trial. On a 5-minute delayed recall, Mr. Williams had borderline normal performance, recalling seven of the 10 words, but with correct recognition of only one of the two nouns that he had forgotten; he correctly rejected four of 5 distracter nouns offered.

Higher cognitive visual perception was assessed finger constructions and clock-drawing, both of which were normal. Abstract construction ability with design tiles was very good, with designs optimally completed within 30 seconds, taking 13, 20 and 22 seconds. Calculation skills were average. Without use of paper, he was able to correctly answer screening questions, including 39 divided by 3, 31 minus 8, and 5 times 13.

Executive function screening was done with the Luria 3-step test ("fist-edge-palm" test) which evaluates premotor, prefrontal and parietal cortex, as well as visual working memory; his performance was normal bilaterally. He demonstrated no abnormal grasp response, imitation behavior, or problem with anti-saccades. Additional tests of executive function included 3-finger go-no go test and parapet drawing, both of which were normal. Design fluency, in which an individual is asked to draw as many nameless designs as possible, was between normal and abnormal. Mr. Williams produced nine designs in the allotted two minutes; whereas most people produce 6 or more different designs, Mr. Williams produced four designs which involved intersecting lines, three designs that were ameba-shaped, and one design that was a variation on a demonstration example. This performance suggests some difficulty with frontal lobe flexibility. *Reasoning* was assessed using word similarities. He correctly identified painting and music as

"art;" all other word pairs were quickly and correctly categorized. *Judgment* was assessed with situational responses. When asked what he would do if he was walking along the sidewalk of a busy street and saw a 2-year-old by himself playing in the street, he provided the following sequence: he would first rescue the child and then "call the police; it makes no sense to ask a 2-year-old where his parents are." When asked what he would do if he came to his mother's house and found the kitchen flooded, he indicated that he would turn off the water, soak up the water with towels, and then call the insurance company. Only with prodding did he consider calling a plumber to fix the leak.

Physical Examination

Mr. Williams is a 22-year-old right-handed African-American male, whose height was 161.5 cm and whose weight was 67.8 kg. His head circumference was 58 cm and roundly shaped. Vital signs included an elevated blood pressure of 167/90 in the left arm and 155/100 in the right arm' his pulse was 88. He had a prominent raised, erythematous rash over much of his scalp, which appears to be chronic because of associated hair thinning in the region of rash. Scalp lesions were occasionally pustular. Mr. Williams had no dysmorphic facial features. There were no café-au-lait spots or other neurocutaneous abnormalities, although there was a small area of depigmentation above his right eyebrow. *HEENT* examination was normal except for erythema of the right external auditory canal and erythema in the center of the right tympanic membrane. Ophthalmoscopy revealed unremarkable fundi with no A-V nicking. Examination of the nose, mouth, dentition, palate, and pinnae was normal. No thyroid enlargement or adenopathy was seen. *Chest* examination revealed normal cardiac rhythm and rate without murmers. Lungs were clear. *Abdominal* and *genitourinary* examinations were not performed. *Skeletal* examination revealed no joint abnormalities or bony malformations. Posture, station and gait were normal.

Neurological Examination

Cranial nerve examination revealed normal visual fields by confrontation and finger counting. Eye movements were full with smooth pursuits and normal saccades. There was, however, fine end-gaze nystagmus with movement of the eyes to the left. Pupils were briskly and symmetrically reactive to light. There was no asymmetry of facial sensation or musculature. Hearing was adequate to hear the rubbing of fingers. Bulbar function was normal.

Motor examination showed normal muscle bulk and resistance to passive movement. There was no suggestion of rigidity or spasticity. Strength was symmetric and normal throughout, in fact quite impressive for his medium physique. There was no drift or pronation of outstretched arms while the eyes were closed; however, there was a fine tremor of both hands. Rapid alternating movements of fingers and feet were well performed. There were no mirror movements, tics, or tremors. Finger-to-nose testing was normal. Muscle stretch reflexes were bilaterally symmetric, measuring trace at the chin, 3++ at the biceps, 3+ at the brachioradialis, and 2+ at the triceps. In the lower extremities, knee jerks were 3++ bilaterally, with 3+ at the ankles; a trace crossed adductor reflex could be elicited. Plantar responses were downgoing.

Screening sensory examination, focusing on touch and position sense, was normal. Romberg sign was present, with falling to the left. Mr. Williams, however, was able to stand on either foot alone, briefly with eyes closed.

Review of Neuropsychological Testing

No school-based psychoeducational test results or court-ordered psychological assessments were available for review.

Review of Neuroimaging Studies

Following examination, Mr. Williams underwent magnetic resonance imaging imaging of the brain. This demonstrated no structural abnormalities upon my review. Independent interpretation by Dr. Ranjiv Saini was normal.

Diagnostic Impressions

Physical Assessment

(1) Hypertension, untreated
(2) Mild hyperreflexia with crossed leg adductor reflex, suggesting minimal impairment of corticospinal tract function
(3) Mild "cerebellar" dysfunction with asymmetric end-gaze nystagmus and Romberg sign and symmetric tremor of outstretched hands
(4) Scalp rash with secondary alopecia
(5) Mild right otitis externa

Cognitive Assessment

(1) Average intelligence with effective communication skills
(2) Good visual-motor speed despite fine motor impairment

Psychosocial Assessment

(1) Impaired reciprocal relationships with parents
(2) School drop-out *without* academic failure
(3) History of behavior reflecting extreme moods, suggesting bipolar illness or anxiety disorder

Recommendations

Mr. Williams should be evaluated and treated medically for significantly elevated high blood pressure, which is not likely related to a stressful environment. In addition, dermatologic evaluation for chronic scalp rash and hair loss should be considered. More importantly, given subtle (but likely stable) neurologic abnormalities, including slight asymmetry of eye movements and balance, formal neuropsychiatric evaluation may be helpful. Finally, formal psychiatric evaluation may provide diagnostic evidence of manic-depressive illness or anxiety disorder.

This above opinions are based upon limited information presently available; should there be additional data (particularly that which addresses educational, psychological, or adaptive capabilities) which alter my opinions, I will supplement this report.

David A. Griesemer, M.D.
Professor of Neurosciences and Pediatrics
Medical University of South Carolina
Charleston, SC

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[1] Grabowski TJ, Anderson SW, Cooper GE. Disorders of Cognitive Function. **Continuum** 8:130-158, 2002

[2] Manual for the Neurobehavioral Cognitive Status Examination. (Fairfax, CA: Northern California Neurobehavioral Group, 1988)

Formule C

1	Staat/État/Country	**Bundesrepublik Deutschland**
2	Standesamtsbehörde Service de l'état civil de Civil Registry Office of	Röthenbach a.d.Pegnitz
3	**Auszug aus dem Sterbeeintrag Nr.** **Extrait de l'acte de décès n°** **Extract from death registration no.**	S 3/2008

4	Tag und Ort des Todes Date et lieu du décès/Date and place of death	Jo Mo An 16 01 2008 Röthenbach a.d.Pegnitz, Deutschland
5	Name Nom/Name	Ateşçi, geb. Scheller
6	Vornamen Prénoms/Forenames	Silvia Michaela
7	Geschlecht Sexe/Sesi	?
8	Tag und Ort der Geburt Date et lieu de naissance/Date and place of birth	Jo Mo An [redacted] Nürnberg, Deutschland
9	Name des letzten Ehegatten Nom du dernier conjoint/Name of the last spouse	Ateşçi
10	Vornamen des letzten Ehegatten Prénoms du dernier conjoint/Forenames of the last spouse	Yakup

		12 Vater Père/Father	13 Mutter Mère/Mother
5	Name Nom/Name		
6	Vornamen Prénoms/Forenames		
11	Tag der Ausstellung Date de délivrance/Date of issue	Jo Mo An 24 01 2013	

(Roch)
Unterschrift/Signature/Signature

Siegel/Sceau/Seal

SYMBOLES/ZEICHEN/SYMBOLS/SIMBOLOS/ΣΥΜΒΟΛΑ/SIMBOLI/SYMBOLEN/SÍMBOLOS/ŞARELER/ΣΗΜΕΙΑ/SYMBOLE/SIMBOLIAI/SIMBOLURI/SÜMBOLID

Jo: Jour/Tag/Day/Día/Ημέρα/Giorno/Dag/Dia/Gün/Dan/Dzień/Diena/Zi/Päev

Mo: Mois/Monat/Month/Mes/Μήνας/Mese/Maand/Mês/Ay/Месец/Miesiąc/Mēnuo/Luna/Kuu

An: Année/Jahr/Year/Año/Έτος/Anno/Jaar/Ano/Yıl/Godina/Rok/Metai/Aasta

M: Masculin/Männlich/Masculine/Masculino/Αρρεν/Maschile/Mannelijk/Masculino/Erkek/Muški/Męska/Vyras/Masculin/Mees

F: Féminin/Weiblich/Feminine/Femenino/Θήλυ/Femminile/Vrouwelijk/Feminino/Kadın/Ženski/Żeńska/Moteris/Feminin/Naine

15/15 Internationale Sterbeurkunde Seite 1 von 2 Vordruck für Standesämtern GmbH, Frankfurt am Main - Berlin 2012

PLAINTIFF'S EXHIBIT 25 10-9702

PENGAD 800-631-6989

EXTRAIT DÉLIVRÉ EN APPLICATION DE LA CONVENTION SIGNÉE À VIENNE LE 8 SEPTEMBRE 1976 / AUSZUG AUSGESTELLT GEMÄSS DEM ÜBEREINKOMMEN VON WIEN VOM 8. SEPTEMBER 1976* / EXTRACT ISSUED IN PURSUANCE OF THE CONVENTION SIGNED AT VIENNA ON SEPTEMBER 8, 1976 / CERTIFICACIÓN EXPEDIDA EN APLICACIÓN DEL CONVENIO FIRMADO EN VIENA EL 8 DE SEPTIEMBRE DE 1976 / ΑΠΟΣΠΑΣΜΑ ΧΟΡΗΓΟΥΜΕΝΟΝ ΚΑΤ' ΕΦΑΡΜΟΓΗΝ ΤΗΣ ΣΥΜΒΑΣΕΩΣ ΤΗΣ ΒΙΕΝΝΗΣ ΤΗΣ 8 ΣΕΠΤΕΜΒΡΙΟΥ 1976 / ESTRATTO RILASCIATO IN APPLICAZIONE DELLA CONVENZIONE FIRMATA A VIENNA IL 8 SETTEMBRE 1976 / UITTREKSEL AFGEGEVEN INGEVOLGE DE OVEREENKOMST ONDERTEKEND TE WENEN OP 8 SEPTEMBER 1976 / CERTIDÃO EMITIDA AO ABRIGO DA CONVENÇÃO ASSINADA EM VIENA A 8 DE SETEMBRO DE 1976 / 8 EYLÜL 1976 TARİHİNDE İMZALANAN SÖZLEŞME UYARINCA VERİLEN ÖRNEK / IZVOD IZDAT NA OSNOVU PRIMENE KONVENCIJE POTPISANE U BEČU 8. SEPTEMBRA 1976 / ODPIS SKRÓCONY WYDANY ZGODNIE Z KONWENCJĄ PODPISANĄ W WIEDNIU DNIA 8 WRZEŚNIA 1976 R. / IŠRAŠAS, IŠDUOTAS TAIKANT 1976 M. RUGSĖJO 8 D. VIENOJE PASIRAŠYTĄ KONVENCIJĄ / EXTRAS ELIBERAT ÎN CONFORMITATE CU CONVENȚIA SEMNATĂ LA VIENA LA 8 SEPTEMBRIE 1976 / TÕEND ANTAKSE VÄLJA VIINI 8. SEPTEMBRIL 1976. AASTA KONVENTSIOONI ALUSEL:

1	Staat/Country/Estado/Κράτος/Stato/Staat/Estado/Devlet/Država/Państwo/Valstybė/Stát/Riik
2	Standesamtsbehörde/Civil Registry Office of/Registro Civil de/Αρχή του (ή της ή των)/Servizio dello stato civile/Dienst van de burgerlijke stand van/Serviços do registo civil de/Nátlus Idarea/Matična služba/Urząd Stanu Cywilnego w/Civilinės būklės aktų įrašų tarnyba/Organ de stare civilă/Perekonnaseisusutus
3	Auszug aus dem Sterbeeintrag Nr./Extract from death registration no./Certificación del acta de defunción núm./Απόσπασμα ληξιαρχικής πράξεως θανάτου αριθ./Estratto dell'atto di morte n./Uittreksel uit de overlijdensakte nr./Certidão de assento de óbito nº/Ölüm sicil örneği No./Izvod iz matične knjige umrlih br./Odpis skrócony aktu zgonu nr/Extras de pe actul de decese nr./Surmatõend number
4	Tag und Ort des Todes/Date and place of death/Fecha y lugar de la defunción/Χρονολογία και τόπος θανάτου/Data e luogo della morte/Datum en plaats van overlijden/Data e lugar do óbito/Ölüm yeri ve tarihi/Datum i mesto smrti/Dato i miejsce zgonu/Mirties data ir vieta/Data şi locul decesului/Surmaaeg ja -koht
5	Name/Name/Apellidos/Επώνυμον/Cognome/Naam/Apelidos/Soyadı/Priezime/Nazwisko/Pavardė/Numele/Perekonnanimi
6	Vornamen/Forenames/Nombre propio/Ονόματα/Prenomi/Voornamen/Nome próprio/Adı/Imena/Imiona/Vardai/Prenumele/Eesnimed
7	Geschlecht/Sex/Sexo/Φύλον/Sesso/Geslacht/Sexo/Cinsiyeti/Pol/Lytis/Sex/Sugu
8	Tag und Ort der Geburt/Date and place of birth/Fecha y lugar de nacimiento/Χρονολογία και τόπος γεννήσεως/Data e luogo di nascita/Geboortedatum en -plaats/Data e lugar do nascimento/Doğum yeri ve tarihi/Datum i mesto rođenja/Data i miejsce urodzenia/Gimimo data ir vieta/Data şi locul naşterii/Sünniaeg ja -koht
9	Name des letzten Ehegatten/Name of the last spouse/Apellidos del último cónyuge/Επώνυμον του τελευταίου συζύγου/Cognome dell'ultimo coniuge/Naam van de laatste echtgenoot/Apelidos do último cônjuge/Son eşin soyadı/Prezime posljednjeg supružnika/Nazwisko ostatniego małżonka/Paskutinio sutuoktinio vardas/Numele ultimului soț/Viimase abikaasa perekonnanimi
10	Vornamen des letzten Ehegatten/Forenames of the last spouse/Nombre propio del último cónyuge/Ονόματα του τελευταίου συζύγου/Prenomi dell'ultimo coniuge/Voornamen van de laatste echtgenoot/Nome próprio do último cônjuge/Son eşin adı/Ime posljednjeg supružnika/Imiona ostatniego małżonka/Paskutinio sutuoktinio vardai/Prenumele ultimului soț/Viimase abikaasa eesnimed
11	Tag der Ausstellung, Unterschrift, Siegel/Date of issue, signature, seal/Fecha de expedición, firma, sello/Χρονολογία έκδόσεως, υπογραφή, σφραγίς/Data di rilascio, firma, bollo/Datum van afgifte, handtekening, zegel/Data de emissão, assinatura, selo/Verilş tarihi, imza, mühür/Datum izdavanja, potpis, pečat/Data wydania, podpis, pieczęć/Išdavimo data, parašas, antspaudas/Data eliberării, semnătura, ştampila/Väljaandmise kuupäev, allkiri, pitser
12	Vater/Father/Padre/Πατήρ/Padre/Vader/Pai/Baba/Otac/Ojciec/Tėvas/Tata/Isa
13	Mutter/Mother/Madre/Μήτηρ/Madre/Moeder/Mãe/Ana/Majka/Matka/Mama/Mama/Ema

*Nach den Artikeln 3, 4, 5 und 7 des Übereinkommens

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15/16 Internationale Standesurkunde Satz 2 - Jan 2012 Verlag für Standesamtswesen GmbH, Frankfurt am Main - Berlin 2012

BUNDESREPUBLIK DEUTSCHLAND

Staatsangehörigkeitsausweis

Vorname(n), Familienname, Geburtsname
Charles Christopher Gerard Williams

geboren am [redacted] in Greenville, South Carolina

Wohnort
Ridgeville, South Carolina

Ist deutsche(r) Staatsangehörige(r).

Dieser Ausweis gilt bis zum
entfällt

Ort, Datum
Köln, 16. Juli 2012

BUNDESVERWALTUNGSAMT
Az.: IIIB3-201110280057-F

Im Auftrag

Hachel

BUNDESVERWALTUNGSAMT 191

PLAINTIFF'S EXHIBIT 26 10-9792

Art.-Nr. 10 005

Bundesdruckerei

Consulate General of the
Federal Republic of Germany
Atlanta

MR. DEREK J. ENDERLIN
PARTNER
ROSS AND ENDERLIN, PA
330 EAST COFFEE STREET
GREENVILLE, SC 29601

ADDRESS:
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Federal Republic of Germany
Marquis Two Tower, Suite 901
285 Peachtree Center Avenue, NE
Atlanta, GA 30303-1221

INTERNET: www.germany.info/atlanta
TEL + 404-659-4760
FAX + 404-649-1280
From the desk of: Kerstin Haken Flautt
TEL-direct 404-979-6419
rk-s1@atla.diplo.de

Certificate of Citizenship for Charles Christopher Gerard Williams
File no. (please quote in reply): Rk 531 SE Williams
Atlanta, August 23, 2012

Dear Mr. Enderlin,

Attached please find certificate of citizenship for your client Charles Christopher Gerard Williams.

Sincerely

Kerstin Flautt

Consulate General of the
Federal Republic of Germany
Atlanta

Durchschrift zur Kenntnis

Ref. Mr 13/9

Ref.

Abgang SEP 13 2012

ADDRESS:
Consulate General of the
Federal Republic of Germany
Marquis Two Tower, Suite 901
285 Peachtree Center Avenue, NE
Atlanta, GA 30303-1221

INTERNET: www.germany.info/atlanta
TEL + 404-659-4760
FAX + 404-659-1280
From the desk of Gerrit Moerking
TEL direct 404-979-6413
RK-1@atla.diplo.de

File No.: RK 531 SE Williams
Atlanta, September 13, 2012

To whom it may concern

This is to certify that the certificate of citizenship for the German citizen Charles Christopher Gerard Williams, issued on July 16, 2012 by the Bundesverwaltungsamt in Köln/Cologne, is an authentic and accurate document. It was issued by the competent German authority. The signature and the official position of the attesting person is genuine.

Sincerely,

Mr 13/9

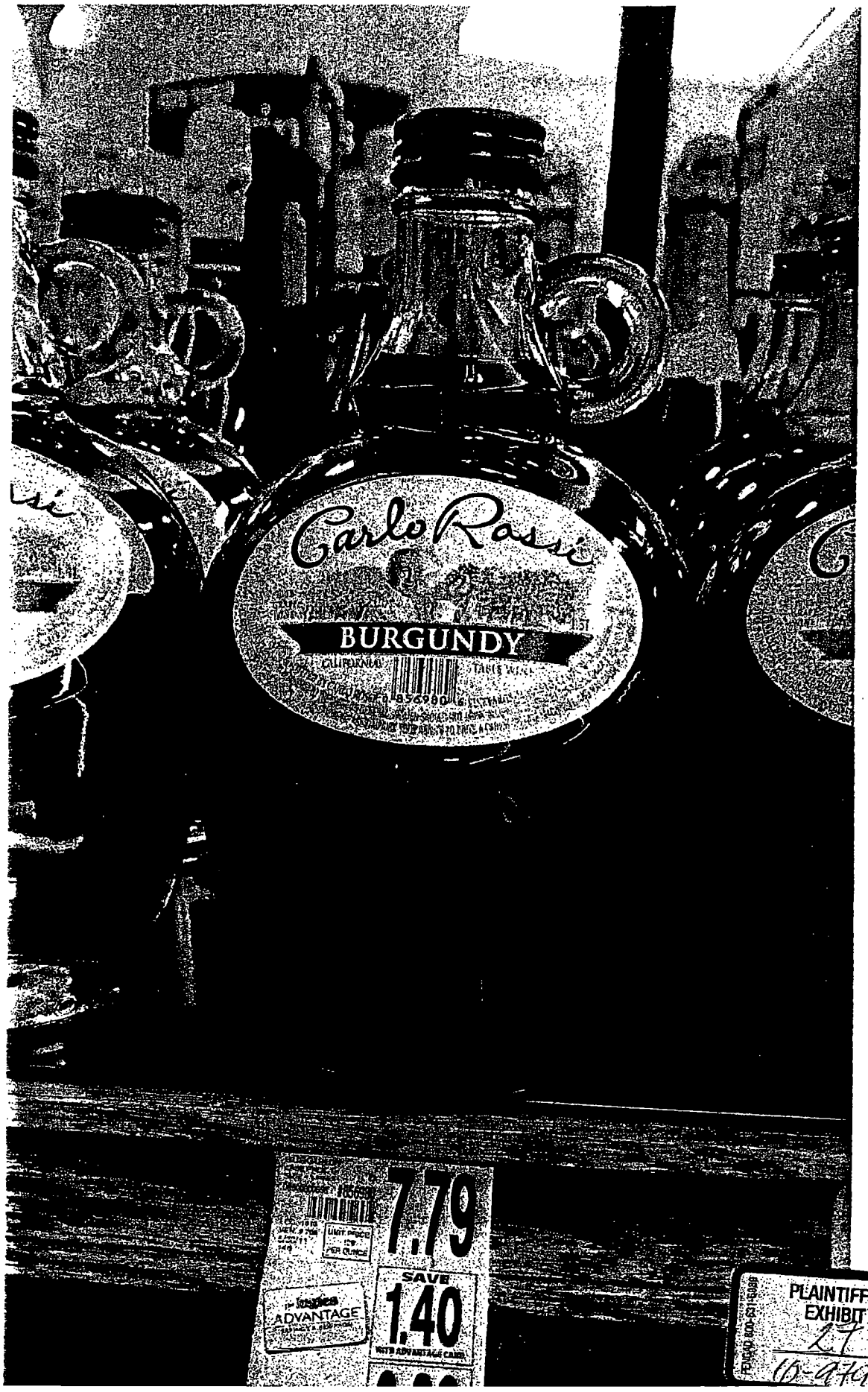
Gerrit Moerking, Consul

1) AL

2) WV 10.10.12

10.12.12 Mr 10/10

Wiedervorgelegt
am 10. [illegible] 2012


Carlo Rossi
BURGUNDY
CALIFORNIA
7.79
SAVE
1.40
ADVANTAGE
PLAINTIFF'S
EXHIBIT
27
05-9477

CURRICULUM VITAE

Paul David Connor

EDUCATION

1988	B.S., University of Washington, Seattle, WA, Psychology
1995	Ph.D., Brigham Young University, Provo, UT, Clinical Psychology/Neuropsychology

POSTGRADUATE TRAINING

1994-95	Internship, Henry Ford Health System, Detroit, MI, Neuropsychology
1995-1999	Senior Postdoctoral Fellow, University of Washington, Seattle, WA, Fetal Alcohol Research and Neuropsychology

FACULTY POSITIONS

1999-2002	Acting Instructor, University of Washington, Psychiatry and Behavioral Sciences, Fetal Alcohol and Drug Unit
2002-2005	Acting Assistant Professor, University of Washington, Psychiatry and Behavioral Sciences, Fetal Alcohol and Drug Unit
2005-2008	Assistant Professor, University of Washington, Psychiatry and Behavioral Sciences, Fetal Alcohol and Drug Unit
2002-Present	Research Affiliate of the Center on Human Development and Disability
2008-Present	Courtesy Clinical Assistant Professor, University of Washington, Psychiatry and Behavioral Sciences, Fetal Alcohol and Drug Unit

LICENSURE TO PRACTICE

1997	Licensed Psychologist #PY00002050, Washington State

PROFESSIONAL ORGANIZATIONS

American Psychological Association
International Neuropsychological Society
National Academy of Neuropsychology, Program Committee 2007-2008
American Academy of Clinical Neuropsychology
Pacific Northwest Neuropsychological Society, President 2004-2005
Research Society on Alcoholism

SPECIAL LOCAL and UNIVERSITY RESPONSIBILITIES

1998-Present	UW Substance Abuse Theme Committee
2003-Present	UW MSRTP Application Committee
2003-Present	Board Member of Fetal Alcohol Syndrome Information Service

October 17, 2012



2004-Present UW FASD Training Alliance
2004-Present Dept of Psychiatry and Behavioral Sciences Addictions and Trauma Work Group

SCIENTIFIC REVIEW COMMITTEES

June 2002 NIAAA Biomedical Research Review Committee ZAA1-DD-20
Feb 2004 NIAAA Neurotoxicology and Alcohol Study Section
Nov 2004 UW Research Royalty Fund
Jun 2007 ADAI Small Grant Review Committee
July 2007 NIAAA Review Committee ZAA1 CC 13
Jun 2009 ADAI Small Grants Review Committee

AD-HOC MANUSCRIPT REVIEWS FOR JOURNALS

Neuropsychology Review
Alcoholism: Clinical and Experimental Research
Child Neuropsychology
Alcohol Health and Research World
Developmental Neuropsychology
Brain Injury and Behavior

RESEARCH FUNDING

1995-1999 National Institute on Alcohol Abuse and Alcoholism, Grant No. R01 AA01455-20-23. "Alcohol Intake During Pregnancy: Offspring Development." Ann P. Streissguth, principal investigator. Total direct costs: $1,363,749.

1999-2000 UW ADAI, Grant. "Regional Metabolic Changes in Brain Function Associated with Acute Alcohol Intoxication: A Functional Neuroimaging Study". Kenneth Maravilla, principal investigator.

1996-2002 National Institute on Alcohol Abuse and Alcoholism, Grant No. R01 AA10836-01-06. "Neuroanatomic-Psychologic Analyses of FAS/FAE Deficits." Ann P. Streissguth, principal investigator. Total direct costs: $888,175.

1999-2004 National Institute on Alcohol Abuse and Alcoholism, Grant No. R37 AA01455-24-28. "Alcohol Intake During Pregnancy: Offspring Development." Ann P. Streissguth, principal investigator. Total direct costs: $1,363,749.

2002-2005 National Institute on Alcohol Abuse and Alcoholism, Grant No. R01 A/HD12419-01A2. "Functional MRI of Cognitive Activation in FAS/FAE." Paul D. Connor, principal investigator. Total direct costs: $600,000.

2003-2006 National Institute on Alcohol Abuse and Alcoholism, Grant No. R01 AA10836-07-10. "Neuroanatomic-Psychologic Analyses of FAS/FAE Deficits." Ann P. Streissguth, principal investigator. Total direct costs: $750,000.

2004-2006 National Institute on Alcohol Abuse and Alcoholism, Grant No. U01. "Detecting FASD from Newborn Ultrasound and Infant Behavior" Ann P. Streissguth, principal investigator. Part of The Riley Consortium "Crosscultural Longitudinal Assessment of FASD".

2004-2009 National Institute on Alcohol Abuse and Alcoholism, Grant No. R37 AA01455-29-34. "Alcohol Intake During Pregnancy: Offspring Development." Ann P. Streissguth, principal investigator.

MANUSCRIPTS IN REFEREED JOURNALS

1. Connor, P.D. and Streissguth, A.P. (1996). Effects of prenatal exposure to alcohol across the life span. Alcohol Health and Research World. 20(3), 170-174.

2. Connor, P.D., Streissguth, A.P., Sampson, P.D., Bookstein, F.L., Barr, H.M. (1999). Individual differences in auditory and visual attention among fetal alcohol-affected adults. Alcoholism: Clinical and Experimental Research. 23(8), 1395-1402.

3. Connor, P.D., Sampson, P.D., Bookstein, F.L., Barr, H.M., Streissguth, A.P. (2000). Direct and indirect effects of prenatal alcohol damage on executive function. Developmental Neuropsychology. 18(3), 331-354.

4. Bookstein, F.L., Sampson, P.D., Streissguth, A.P., Connor, P.D. (2001). Geometric morphometrics of corpus callosum and subcortical structures in the fetal- alcohol-affected brain. Teratology. 64, 4-32.

5. Bookstein, F.L., Streissguth, A.P., Sampson, P.D., Connor, P.D., Barr, H.M. (2002). Corpus callosum shape and neuropsychological deficits in adult males with heavy fetal alcohol exposure. NeuroImage. 15, 233-251.

6. Bookstein, F.L., Sampson, P.D., Connor, P.D., Streissguth, A.P. (2002). The midline corpus callosum is a neuroanatomical focus of fetal alcohol damage. The New Anatomist. 269, 162-174.

7. Baer, J.S., Sampson, P.D., Barr, H.M., Connor, P.D., Streissguth, A.P. (2003). A 21-year longitudinal analysis of the effects of prenatal alcohol exposure on young adult drinking. Archives of General Psychiatry. 60, 377-385.

8. Grant, T., Huggins, J., Connor, P., Pedersen, J., Whitney, N., Streissguth, A. (2004). A pilot community intervention for young women with fetal alcohol spectrum disorders. Community Mental Health Journal. 40 (6), 499-511.

9. Grant, T., Huggins, J., Connor, P., Streissguth, A. (2005). Quality of life and psychosocial profile among young women with fetal alcohol spectrum disorders. Mental Health Aspects of Developmental Disabilities. 8(2), 33-39.

10. Bookstein, FL, Connor, PD, Covell, KD, Barr, HM, Gleason, CA, Sze, RW, McBroom, JA, Streissguth, AP (2005). Preliminary evidence that prenatal alcohol damage may be visible in averaged ultrasound images of the neonatal human corpus callosum. Alcohol: An International Biomedical Journal. 36(3), 151-160.

11. Connor, PD, Sampson, PD, Streissguth, AP, Bookstein, FL, Barr, HM (2006). Effects of prenatal alcohol exposure on fine motor coordination and balance: A study of two adult samples. Neuropsychologia, 44(5), 744-751.

12. Barr, HM, Bookstein, FL, O'Malley, KD, Connor, PD, Huggins, JE, Streissguth, AP (2006). Binge drinking during pregnancy as a predictor of psychiatric disorders on the structured clinical interview for DSM-IV in young adult offspring. The American Journal of Psychiatry, 163(6), 1061-1065.

13. Bookstein, FL, Streissguth, AP, Connor, PD, Sampson, PD. (2006). Damage to the human cerebellum from prenatal alcohol exposure: The anatomy of a simple biometrical explanation. The New Anatomist. 289B, 195-209.

14. Bookstein, FL, Connor, PD, Huggins, JE, Barr, HM, Pimentel, KD, Streissguth, AP. (2007). Many infants prenatally exposed to high levels of alcohol show one particular anomaly of the corpus callosum. Alcoholism: Clinical and Experimental Research, 31(5), 1-12.

15. Brown, NN, Gudjonsson, G, & Connor, P. (2011). Suggestibility and fetal alcohol spectrum disorders (FASD): I'll tell you anything you want to hear. International Journal of Psychiatry and Law.

16. Brown, NN, Wartnik, AP, Connor, PD, & Adler, RS (2011). A proposed model standard for forensic assessment of FASD. International Journal of Psychiatry and Law.

17. Brown, N. N., Adler, R. S., & Connor, P. D. (in press). Conduct-disordered adolescents with fetal alcohol spectrum disorder: Intervention in secure treatment settings. *Criminal Justice and Behavior*.

BOOK CHAPTERS and OTHER PUBLICATIONS

1. Kopera-Frye, K., Connor, P.D., Streissguth, A.P. (2000). Recent advances in fetal alcohol syndrome: Implications for diagnosis, intervention, and prevention. In Seitz, H.K., Lieber, C.S., & Simanowski, U.A. (eds.), Handbuch Alkohol, Alkoholismus, Alkoholbedingte Organschäden (2nd Ed.). Heidelberg, Germany: Johann Ambrosius Barth Verlag.

2. Streissguth, A.P., Connor, P.D. (2001). Fetal alcohol syndrome and other effects of prenatal alcohol: Developmental cognitive neuroscience implications. In Nelson, C.A. & Luciana M. (Eds.) The Handbook of Developmental and Cognitive Neuroscience. Cambridge, MA: The MIT Press.

3. Connor, P.D., Huggins, J.E. (2006). Prenatal development: Fetal alcohol spectrum disorders. In Thies, K.M., Travers, J.F. (Eds.). The Handbook of Human Development for Health Professionals. Thorofare, NJ: SLACK, Inc.

4. Connor, P.D. (2001). Synthesizing animal and human studies of prenatal alcohol exposure. Journal of the International Neuropsychological Society. 7(5), 648-649.

5. Connor, P.D. (2004). Prison: A warehouse for individuals with FASD. Iceberg. 14 (2). http://www.fasiceberg.org/newsletters/Vol14Num2_Apr2004.htm#warehouse.

PUBLISHED ABSTRACTS

1. Norman, M.A., Lajiness-O'Neill, R.R., Nilsson, D.E., Walker, M.L., Boyer, R.S., Connor, P. D., and Bigler, E.D.(1994). Quantitative neuroimaging outcome in benign hydrocephalus. Archives of Clinical Neuropsychology. 9, 168-169.

2. Connor, P.D., Gale, S.D., Johnson, S.C., Anderson, C.V., Bigler, E.D., and Blatter, D.D. (1995). Cingulate degeneration following traumatic brain injury: Quantitative neuroimaging, memory and executive functions. Journal of the International Neuropsychological Society. 1(2), 174.

3. Connor, P.D. and Streissguth, A.P. (1997). Auditory and visual attention in adults with fetal alcohol syndrome. Journal of the International Neuropsychological Society. 3(1), 56.

4. Connor, P.D. and Streissguth, A.P. (1997) Verbal fluency, word reading and color naming in adults with fetal alcohol syndrome and fetal alcohol effects. Alcoholism: Clinical and Experimental Research, 21(3 Suppl), 116A.

5. Connor, P.D., Streissguth, A.P., Sampson, P.D., Bookstein, F.L., Barr, H., and Wegelin, J. (1998). Fluency and speed of information processing as related to quantitative neuroimaging in fetal alcohol syndrome and fetal alcohol effects: Preliminary findings. Journal of the International Neuropsychological Society, 4(1), 21.

6. Connor, P.D., Streissguth, A.P., Barr, H.M., Sampson, P.D., and Bookstein, F.L. (1998). Psychiatric symptomatology and characteristic behaviors in adults with fetal alcohol syndrome and fetal alcohol effects. Alcoholism: Clinical and Experimental Research, 22(3 Suppl), 61A.

7. Streissguth, A.P., Connor, P.D., Sampson, P.D., Barr, H.M., Bookstein, F.L., and Wegelin, J. (1998). Fetal alcohol syndrome: Relationships between CNS structure and function. Alcoholism: Clinical and Experimental Research, 22(3 Suppl), 58A.

8. Connor, P.D., Streissguth, A.P., Sampson, P.D., Bookstein, F.L., Barr, H.M. (1999). Executive functioning deficits in adults prenatally exposed to alcohol. Alcoholism: Clinical and Experimental Research. 23(5), 31A.

9. Bookstein, F.L., Streissguth, A.P., Sampson, P.D., Connor, P.D., Barr, H.M. (1999). Corpus callosum shape and neuropsychological deficits in adult males with heavy alcohol exposure. Alcoholism: Clinical and Experimental Research. 23(5), 28A.

10. Streissguth, A.P., Barr, H.M., Sampson, P.D., Bookstein, F.L., Connor, P.D. (1999). Adult IQ as related to prenatal alcohol exposure, FAS, FAE, ARND, and child IQ in a large sample. Alcoholism: Clinical and Experimental Research. 23(5), 107A.

11. Streissguth, A.P., Barr, H.M., Connor, P., Sampson, P.D., Bookstein, F.L. (1999). The fetal alcohol syndrome: Relationship between CNS structure and neuropsychological performance. In R.A. Saul & M.C. Phelan (Eds.), Proceedings of the Greenwood Genetics Center, (18:147-148). Greenville, S.C.: Keys Printing, Inc.

12. Connor, P.D., Streissguth, A.P., Sampson, P.D., Bookstein, F.L., Barr, H.M., Toth, S.K. (2000). Motor coordination in adult males with fetal alcohol syndrome and fetal alcohol effects. Journal of the International Neuropsychological Society, 6(2), 237-238.

13. Connor, P., & Mahurin, R. (2001). A preliminary study of working memory in fetal alcohol damage using fMRI. International Neuropsychological Society, 7(2), 206.

14. Bookstein, F.L., Sampson, P.D., Connor, P.D., Streissguth, A.P. (2001). Excess variance of subcortical brain shape in fetal alcohol spectrum disorders: An analysis by landmark location data. Teratology, 63(6), 251.

15. Huggins, J.E., Connor, P.D., Barr, H.M., Streissguth, A.P. (2001). Quality of life in adults with fetal alcohol spectrum disorders (FASD). Alcoholism: Clinical and Experimental Research, 25(5), 125A.

16. Huggins, J.E., Connor, P.D., O'Malley, K., Barr, H.M., Streissguth, A.P. (2001). Suicide/parasuicide behavior in adults with fetal alcohol spectrum disorders (FASD). Alcoholism: Clinical and Experimental Research, 25(5), 123A.

17. Baer, J.S., Sampson, P.D., Barr, H.M., Connor, P.D., Streissguth, A.P. (2001). Prenatal alcohol exposure and young adult drinking: A 21-year longitudinal analysis. Alcoholism: Clinical and Experimental Research, 25(5), 123A.

18. Streissguth, AP, Barr, HM, Sampson, PD, Connor, PD, Bookstein, FL. (2001) Young adult outcomes of prenatal alcohol exposure from a large population-based study. Alcohol and Alcoholism, 36(5), 437.

19. Connor, PD, Bookstein, FL, Barr, HM, Sampson, PD, Streissguth, AP. (2002). Assessing prenatal alcohol damage: Test of Nonverbal Intelligence-2 (TONI-2) vs. WAIS-R. Alcoholism: Clinical and Experimental Research, 26(5), 175A.

20. Streissguth, AP, Sampson, PD, Bookstein, FL, Connor, PD, Barr, HM. (2002). 21-year dose-response effects of prenatal alcohol exposure on cognition. Alcoholism: Clinical and Experimental Research, 26(5), 93A.

21. Connor, PD, Barr, HM, Streissguth, AP. (2003). Two scoring methods of the Rey-Osterreith in adults with fetal alcohol damage. Journal of the International Neuropsychological Association, 9(2), 201.

22. Connor, P., Mahurin, RK, Dehaene, S. (2004). Subtraction ability in patients with fetal alcohol spectrum disorders: an fMRI study. Archives of Clinical Neuropsychology, 19, 917.

23. Connor, PD., Mahurin, R.K. (2005). Working memory in patients with fetal alcohol spectrum disorders: An fMRI study. Journal of the International Neuropsychological Society.

24. Connor, PD, Mahurin, RK (2005). Decreased cerebellar activation in patients with fetal alcohol spectrum disorders. Alcoholism: Clinical and Experimental Research, 29(5), 45A.

INVITED ADDRESSES

Sep 1996	Northwest Oregon FAS Conference, Forest Grove, OR. "Implications of secondary disabilities of fetal alcohol syndrome and fetal alcohol effects on intervention strategies."
Apr 1997	Juvenile Court Workers Conference, Lake Chelan, WA. "Fetal Alcohol Syndrome and Fetal Alcohol Effects: Brain Damage, Long-Term Consequences, and Treatment Concerns."
Jun 1998	FAS Study Group meetings, Hilton Head SC. "Perseverative styles of performance: Troubles learning from mistakes in patients with FAS and FAE."
Oct 1998	Seattle Mental Health, Seattle, WA. "Fetal Alcohol Syndrome and Fetal Alcohol Effects: Brain Damage, Long-Term Consequences, and Treatment Concerns."
Apr 1999	Association of the Advanced Practice of Psychiatric Nurses, Tacoma, WA. "Fetal Alcohol Syndrome and Fetal Alcohol Effects: Brain Damage, Long-Term Consequences, and Treatment Concerns."
Jun 1999	Department of Health and Welfare Idaho, Twin Falls, ID. "Fetal Alcohol Syndrome and Fetal Alcohol Effects: Diagnosis, Neuroanatomy, and Neuropsychology"
Nov 2001	Valley Family Care Residents Training for March of Dimes Grant (Preventing Double Jeopardy). "Developing Successful Interventions for Clients with FAS or ARND."
Dec 2001	Carolyn Downs Clinic, Obstetrics didactics training for March of Dimes Grant (Preventing Double Jeopardy). "Developing Successful Interventions for Clients with FAS or ARND."
Dec 2002	Monroe State Prison Special Offenders Unit "Fetal Alcohol Spectrum Disorders: Diagnosis, Prevalence, Neuropsychological and Mental Health Implications."
Oct 2003	11th Annual MeritCare Pediatric Neuroscience Conference, Fargo, ND. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences." 6-hour CE course.

Dec 2003	Forensic Psychiatric Services Commission's Colloquium on Cognitive Impairment, Simon Fraser University, Vancouver, BC. "Efficacy of Neuroimaging and Neuropsychology for Patients with Fetal Alcohol Spectrum Disorders."
Feb 2004	Edmonds Community College, Human Sexuality. "Fetal Alcohol Spectrum Disorders."
Mar 2004	Western State Hospital, Grand Rounds. "Fetal Alcohol Spectrum Disorders: Diagnosis, Prevalence, Neuropsychological and Mental Health Implications."
Apr 2004	Joint Meeting of the Mental Health Advisory Board and the Chemical Dependency Administrative Board. "Developing Successful Interventions for Clients with FAS or ARND."
Aug 2004	Seattle Mental Health. "Fetal Alcohol Spectrum Disorders: An Interdisciplinary Perspective Identification, Treatment, and Community Involvement."
Oct 2004	School of Nursing , Graduate course 556. "Fetal Alcohol Spectrum Disorders."
Mar 2005	13th Annual Children's Justice Conference, Seattle, WA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
June 2005	Center for Sexual Assault and Traumatic Stress, Seattle, WA. "Fetal Alcohol Spectrum Disorders: Brain Cognition, and Long-Term Consequences."
July 2005	fMRI/Brain Imaging Interest Group, Department of Radiology, University of Washington. "Neuroimaging Studies of FASD."
Oct 2005	Idaho Health Care Conference, Idaho State University. Plenary session "FASD Across the Lifespan" Psychosocial Workshop "Assessment and Treatment of FASD: A Neuropsychological Approach".
Nov 2005	Northwest Chapter of the American Association of Neuroscience Nurses, Swedish Hospital. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Nov 2005	School of Nursing, Graduate course 556. "Fetal Alcohol Spectrum Disorders."
May 2006	DDD Region 1 Training, Lecture and Workshop Series. Two full day work shops in Wenatchee and Spokane, WA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
May 2006	Spruce Street Secure Crisis Residential Center didactics course, Seattle. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Jun 2006	Mercy Medical Center 1st Annual FASD Conference, Sioux City, IA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Jun 2006	National Association of Drug Court Professionals, Seattle, WA. "Law and Disorder: Understanding Fetal Alcohol Spectrum Disorders within the Legal System."

Jun 2006	After an FASD Diagnosis: Then What? Strategies for effective interventions with youth with FASD and their families. Mount Vernon, WA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Sep 2006	National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect. Atlanta, GA. "Neuroimaging Studies of Infants, Adolescents, and Adults with FASD: Perspectives from the Far Northwest."
Oct 2006	Washington WIC: Exceptional and Extraordinary, Bellevue, WA. "What Happens to a Fetus Exposed to Alcohol? Long-Term Effects of Prenatal Alcohol Exposure on the Brain and Behavior."
Nov 2006	School of Nursing, Graduate course 556. "Fetal Alcohol Spectrum Disorders."
Mar 2007	Substance Use and Brain Development Conference, Eugene, OR. "The Impact of Prenatal Alcohol Exposure on Brain and Behavior Development."
Apr 2007	Juvenile Justice at risk youth and truancy court, Seattle, WA. "Fetal Alcohol Spectrum Disorders and the Justice System."
Apr 2007	Annual MRFASTC Booster Session, St. Louis, MO. "The Brain and Behavior in Individuals with FASD."
Apr 2007	Alaskan Dietetics Association Annual Meeting, Anchorage, AK. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
May 2007	SATUCI Impact of Fetal Alcohol Spectrum Disorders On Schools, Families, and Communities, Marshalltown, IA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Aug 2007	Merrill Scott Symposium, Yakima, WA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Sep 2007	Elements of Success: Helping Individuals with FASD and Co-occurring Disorders Conference, Federal Way, WA. "Adults with FASD: From Research to Clinical Practice."
Sep 2007	Indian Health Services FASD Training, Seattle, WA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Oct 2007	Spokane Child Abuse and Neglect Prevention Center Training, Spokane, WA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Mar 2008	Nurses' Advisory Committee for Perinatal Education "Pot O' Gold Conference: Perinatal/Neonatal Challenges 2008", Spokane, WA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Apr 2008	NorCal SOT 2008 Spring Meeting, Menlo Park, CA. "As if Brain damage Wasn't Enough – The Lifelong Impact of FASD on Social Interaction."
Apr 2008	The Forensic FASD Diagnostic Team: What? When? Who? How? Why? – a 3 day meeting with New Zealand practitioners. Seattle, WA. "The Role of Neuropsychological Testing in FASD."

Apr 2008	Sound Mental Health, Seattle, WA Psychology Practicum and Intern Didactics "Neuropsychological Assessment and Service Implications for Individuals with FASD."
Sep 2008	Alcohol HealthWatch, Auckland New Zealand. Three day training of New Zealand neuropsychologists on neuropsychological assessment of adolescents and adults for FASD especially in forensic settings.
Sep 2008	FASDay Celebration Auckland, New Zealand. "International FASD Awareness Day: FASD and Mental Health"
Sep 2008	Pediatricians Breakfast Meeting, Auckland General Hospital Auckland, New Zealand. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Sep 2008	New Zealand Ministry of Health hosted meeting, Wellington, New Zealand. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Jan 2009	Oregon State Hospital Psychology Didactics, Salem, OR. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Mar 2009	3rd International Conference on FASD, Victoria, BC, Canada. "FASD in the Legal System: A Multidisciplinary Assessment Model for Adults."
Apr 2009	Fetal Alcohol Spectrum Disorders and the Justice System , Kiana Lodge, Suquamish, WA. All day in service on assessment and diagnosis of FASD in the forensic setting.
Sep 2009	Tribal Communities Transforming Mental Health, Great Wolf Lodge, WA "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Sep 2009	FASD Discovering, Connecting, and Creating Change. Saskatoon, Saskatchewan, Canada. Plenary Session "Poking the Brain: What 30 Years of Research Tells Us About FASD."
Sep 2009	FASD Discovering, Connecting, and Creating Change. Saskatoon, Saskatchewan, Canada. Concurrent Session. "Assessing FASD in Legal Settings."
Feb 2010	Washington Death Penalty Assistance Center, 3rd Interdisciplinary Program, Expertise You Need to Defend a Capital Case in 2010. Seattle, WA. "Fetal Alcohol Syndrome: Practical Tools."
Feb 2010	Texas Criminal Defense Lawyers Association Capital Trial and Habeas Conference. Austin, TX. "FASD: Application of the Scientific Method to the Forensic Setting."
Mar 2010	2010 Aboriginal Health Forum Fetal Alcohol Spectrum Disorder, Aboriginal People, & the Criminal Justice System, Toronto, ON, Canada. "FASD and the Criminal Justice System."
Mar 2010	2010 Aboriginal Health Forum Fetal Alcohol Spectrum Disorder, Aboriginal People, & the Criminal Justice System, Toronto, ON, Canada. "FASD in the Legal System: A Multidisciplinary Assessment Model."
Apr 2010	Fourth National Biennial Conference on Adolescents and Adults with Fetal Alcohol Spectrum Disorder. Vancouver, BC. "Suggestibility in FASD: Forensic Assessment and Implications."

Apr 2010	7th National Seminar on the Development and Integration of Mitigation Evidence. Seattle, WA. "Forensic Assessment of Fetal Alcohol Spectrum Disorders with State-of-the-Art Facial Analysis, Diffusion Tensor Imaging, and MRIs."
Oct 2010	Annual Western All-Star Conference and Confabulation Annual Death Penalty Day. Boise, ID. "Forensic Assessment of Fetal Alcohol Spectrum Disorders in the Habeas Context."
Sep 2011	Indian Health Services FASD Training, Seattle, WA. "Fetal Alcohol Spectrum Disorders: Brain, Cognition, and Long-Term Consequences."
Jul 2012	American Institute for the Advancement of Forensic Studies. St. Paul, MN. "Forensic Assessment of Developmental Disabilities."
Sep 2012	Indian Health Services FASD Training, Seattle, WA. "Fetal Alcohol Spectrum Disorders: Effects on the Brain (Structure and Function)."
Oct 2012	Co-Occurring Disorders Conference, Yakima, WA. "Fetal Alcohol Spectrum Disorders and the Justice System."

DEPARTMENTAL TEACHING

Feb 2000	Department of Psychiatry and Behavioral Sciences, University of Washington, Psychology Residency didactics. "A Neuropsychological Perspective on Fetal Alcohol Syndrome"
Feb 2001	Department of Psychiatry and Behavioral Sciences, University of Washington, Psychology Residency didactics. "A Neuropsychological Perspective on Fetal Alcohol Effects."
Mar 2002	Department of Psychiatry and Behavioral Sciences, University of Washington, Psychology Residency didactics. "A Neuropsychological Perspective on Fetal Alcohol Effects."
Sep 2002	Department of Psychiatry and Behavioral Sciences, University of Washington, R3 Medical School Didactics "Disorders of Brain Development: Fetal Alcohol Syndrome & Fetal Alcohol Effects."
Nov 2002	Children's Child Psychiatry Didactics for Psychiatry Residents and Developmental Pediatricians "Development of Executive Functions: Specific Examples in Fetal Alcohol Spectrum Disorders."
Mar 2003	Department of Psychiatry and Behavioral Sciences, University of Washington, Psychology Residency didactics. "A Neuropsychological Perspective on Fetal Alcohol Effects."
Aug 2003	Department of Psychiatry and Behavioral Sciences, University of Washington, R3 Medical School Didactics "Disorders of Brain Development: Fetal Alcohol Syndrome & Fetal Alcohol Effects."
Feb 2004	Department of Psychiatry and Behavioral Sciences, University of Washington, Child Psychiatry Residents Didactics. "Brain Development: Executive Functions in Childhood and Adolescents."
Apr 2004	Department of Psychiatry and Behavioral Sciences, University of Washington, Psychology Residency didactics. "A Neuropsychological Perspective on Fetal Alcohol Effects."

Feb 2005	Department of Psychiatry and Behavioral Sciences, University of Washington, Child Psychiatry Residents Didactics. "Brain Development: Executive Functions in Childhood and Adolescents."
Mar 2005	Department of Psychiatry and Behavioral Sciences, University of Washington, Neurosciences Seminar. "Neuroimaging Studies of FASD."
Mar 2005	Department of Psychiatry and Behavioral Sciences, University of Washington, Psychology Residency didactics. "A Neuropsychological Perspective on Fetal Alcohol Effects."
Dec 2005	Department of Psychiatry and Behavioral Sciences, University of Washington, Child Psychiatry Residents Didactics. "Development of the Central Nervous System from the Prenatal Period On: The Special Example of Fetal Alcohol Spectrum Disorders."
Dec 2005	Department of Psychiatry and Behavioral Sciences, University of Washington, Friday Noon Conference. "Fetal Alcohol Spectrum Disorders."
Mar 2006	Department of Psychiatry and Behavioral Sciences, University of Washington, Psychology Residency didactics. "A Neuropsychological Perspective on Fetal Alcohol Effects."
Oct 2006	Department of Psychiatry and Behavioral Sciences, University of Washington, Child Psychiatry Residents Didactics. "Development of the Central Nervous System from the Prenatal Period On: The Special Example of Fetal Alcohol Spectrum Disorders."

PAPERS PRESENTED AT SCIENTIFIC MEETINGS

Feb 1995	International Neuropsychological Society, Seattle, WA. "Cingulate degeneration following traumatic brain injury: Quantitative neuroimaging, memory and executive functions."
Feb 1997	International Neuropsychological Society, Honolulu, HI. "Fluency and speed of information processing as related to quantitative neuroimaging in fetal alcohol syndrome and fetal alcohol effects: Preliminary findings."
Jul 1997	FAS Study Group, San Francisco, CA. "Brain study: Preliminary findings of a study of patients with FAS and FAE."
Jun 1999	FAS Study Group, Santa Barbara, CA. "Executive Function Deficits in Adults Prenatally Exposed to Alcohol."
Nov 2001	National Academy of Neuropsychology, San Francisco, CA. CE course entitled "Fetal Alcohol Syndrome: Brain and Cognition."
Jul 2007	Research Society on Alcoholism, Chicago, IL. "Screening for Fetal Alcohol Exposure in Human Neonates Using the Corpus Callosum."
May 2009	Scientific Workshop on Cognitive Deficits, Neurobehavioral Problems, and Psychopathology due to Prenatal Alcohol Exposure, Rockville, MD. ICCFASD meeting to discuss diagnostic guidelines for ARND. "Adults with FASD: From Research to Clinical Practice."

MEDIA PRESENTATIONS

Nov 2000	Filmed Interview for Decouverte, a Science and Technology Canadian Broadcasting Company TV program from Montreal.
Feb 2004	Interview with Laurie McHale for CHDD Outlook article.
Mar 2004	Filmed interview with Michael Skinner for video "Recovering Hope Mothers Speak Out about Fetal Alcohol Spectrum Disorders" sponsored by SAMHSA's FASD Center for Excellence.
May 2007	Interviewed by Chicago Tribune for article on FASD and DDD in Illinois

Nov 2000	Filmed Interview for Decouverte, a Science and Technology Canadian Broadcasting Company TV program from Montreal.
Feb 2004	Interview with Laurie McHale for CHDD Outlook article.
Mar 2004	Filmed interview with Michael Skinner for video "Recovering Hope Mothers Speak Out about Fetal Alcohol Spectrum Disorders" sponsored by SAMHSA's FASD Center for Excellence.
May 2007	Interviewed by Chicago Tribune for article on FASD and DDD in Illinois

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natnovickbrown@gmail.com

Curriculum Vitae

Forensic Specialties

- **FETAL ALCOHOL SPECTRUM DISORDERS (FASD) and other DEVELOPMENTAL DISABILITIES**
- **SEX OFFENDER EVALUATION & RISK ASSESSMENT**
- **PARENTING EVALUATION and PSYCHOLOGICAL EVALUATION**

Licenses (Psychologist)

#PY1965 (Washington)

#PY6219 (Florida)

#LP183 (Oregon - temporary)

#20 (South Carolina - temporary)

Certifications

Certified Psychologist (CPQ #3258), Association of State & Provincial Psychology Boards

Certified Sex Offense Treatment Provider (Washington State SOTP #FC112)

Polygraph Examiner / Certified in Post-conviction Sex Offender Testing (PCSOT)

Certified Evaluator for Department of Corrections, Division of Developmental Disabilities, Department of Social & Health Services (Washington State)

Certified Parenting Evaluator, University of Washington Department of Psychiatry and Behavioral Sciences

Listed in the National Register of Health Service Providers in Psychology, #49892



Education and Training

Post-conviction Sex Offender Polygraph Training, 2004

International School of Polygraph, Fort Lauderdale, FL, 2003

Sex Offense Treatment Provider (SOTP), 1995

Post-Doctoral FASD Training, U. of Washington Fetal Alcohol and Drug Unit, 1995

Parenting Evaluation Training Program, University of Washington, 1994

Ph.D., Clinical Psychology, University of Washington, 1994

M.H.A., Health Care Administration, University of Washington, 1979

M.L.S., Library and Information Sciences, University of Washington, 1975

B.A., Sociology (Psychology minor), UCLA, 1968

Clinical Experience

2007 – present **Program Director / Chief Psychologist: FASD Experts**
Pre/post-conviction case review, assessment, consultation, and testimony re Fetal Alcohol Spectrum Disorders (FASD)

1994 – present **Clinical and Forensic Psychologist / Expert Witness**

Adult and juvenile sex offense and risk assessment evaluation (criminal state and federal prosecution; civil commitment cases under Sexually Violent Predator law]

Adult, adolescent, and child psychological evaluation (competency, dependency, FASD, developmental delay, Asperger Syndrome, Shaken Baby Syndrome, abuse and neglect allegations)

Parenting evaluation (court-appointed and stipulated cases involving such issues as physical and sexual abuse allegations, neglect, parental alienation, and relocation)

Private therapy practice, 1994-2000, 2004-present (adult and adolescent therapy involving multiple issues, including sexual offending and SVP, developmental delay, and FASD)

2005 – present **Clinical Assistant Professor,** Department of Psychiatry and Behavioral Sciences, University of Washington, Seattle

Assessment of recidivists referred by King County Mental Health Court and King County Drug Court to screen for FASD/organic brain impairment (Fetal Alcohol and Drug Unit); consultation regarding FASD secondary disabilities; supervise doctoral students

1994 - 1995 **Psychologist, Postdoctoral / Faculty Appointment,** Fetal Alcohol and Drug Unit (Dr. Ann Streissguth), University of Washington

Evaluation/treatment of FAS/FAE patients with sex offense issues; research on Secondary Disabilities related to FAS/FAE in Washington State Prison system

1992 - 1994 **Pre-doctoral Internships**, University of Washington

- Specialized training and certification in forensic evaluation and expert testimony
- Specialized training in pain management, Pain Clinic, University of Washington
- Specialized training in individual psychotherapy, Group Health Cooperative, Seattle
- Specialized training in rehabilitation therapy (including traumatic brain injury), University Hospital, University of Washington

Pre-Doctoral Work Experience

1979 – 1989 Hospital CEO / Clinic Administrator

1975 – 1979 Hospital Medical Librarian

Research

2005 – present Clinical Assistant Professor (courtesy appointment), Fetal Alcohol and Drug Abuse Unit, Department of Psychiatry and Behavioral Medicine, University of Washington.

Consultation involves FASD research (FASD prevention), training, assessment of individuals referred by King County Mental Health and Drug Courts

1994 - 1995 Postdoctoral Fellow: Fetal Alcohol Unit, University of Washington

	Developed and conducted FAS research project in Washington State prison system (men's and women's correctional facilities at Shelton and Purdy)
1991 - 1994	Dissertation: Relation between Psychological Correlates of Alcoholism Risk and Stress-Response Dampening Across the Blood Alcohol Curve
1991 - 1993	Research Coordinator: Prediction of High Risk Drinking
1990 - 1992	Research Coordinator: Alcohol and Social Influence
1989 - 1991	Research Coordinator: Self-Esteem in Young Adults

Publications

Grant, T.M., Brown, N.N., Graham, J.C., & Ernst, C.E. (in press). Substance abuse treatment outcomes in women with fetal alcohol spectrum disorder. *International Journal of Alcohol and Drug Research.*

Brown, N.N., Burd, L., Grant, T. M., Edwards, W., Adler, R., & Streissguth, A. (in press). Prenatal alcohol exposure: An assessment strategy for the legal context. *International Journal of Alcohol and Drug Research.*

Grant, T.M., Brown, N.N., Dubovsky, D., Sparrow, J., & Ries, R. (in press). Understanding substance abuse in fetal alcohol spectrum disorders. *Journal of Addiction Medicine.*

Grant, T.M., Brown, N.N., Graham, J.C., Whitney, N., & Dubovsky, D. (in press). Screening in treatment programs for fetal alcohol spectrum disorders and associated life history problems. *International Journal of Alcohol and Drug Research.*

Brown, N.N., Adler, R.S., & Connor, P.D. (2012). Conduct-disordered adolescents with fetal alcohol spectrum disorder: Intervention in secure treatment settings. *Criminal Justice and Behavior, 39,* 789-812.

Brown, N.N., O'Malley, K., & Streissguth, A.P. (2012). FASD: Diagnostic dilemmas and challenges for a modern transgenerational management approach. In S. Adubato & D. Cohen (Eds.), *Prenatal Alcohol Use and Fetal Alcohol Spectrum Disorders: Diagnosis, Assessment, and New Directions in Research and Multimodal Treatment.* Bentham Online Publishing.

Brown, N.N., Gudjonsson, G., & Connor, P. (2011). Suggestibility and Fetal Alcohol Spectrum Disorders (FASD): I'll Tell You Anything You Want to Hear. *Journal of Psychiatry and Law, 39,* 39-71.

Brown, N.N. (Spring 2011). Evidence-based interventions in children with Fetal Alcohol Spectrum Disorders. *Paradigm, 16,* 12-17.

Brown, N.N., Wartnik, A.P., Connor, P.D., & Adler, R.S. (2010). A proposed model standard for forensic assessment of FASD. *Journal of Psychiatry and Law, 38,* 383-418.

Brown, N.N. (June 2008). FASD Experts: Multidisciplinary Forensic Assessment for a Multidimensional Condition. *Iceberg,* 18.

Brown, N.N. (2007). ADHD and FASD: Comorbidity and Its Effect on Sexual Behavior Problems. In K O'Malley (Ed.), *ADHD and FASD: Diagnosis, natural history, and therapeutic issues across the lifespan.* Hauppauge, NY: Nova Pub.

Novick (Brown), N.J. (1998). FAS: Preventing and treating sexual deviancy. In A.P. Streissguth & J. Kanter (Eds.), *The challenge of fetal alcohol syndrome: Overcoming secondary disabilities.* Seattle: University of Washington Press.

Novick (Brown), N.J. (1996). *Sexual victimization and inappropriate sexual behavior in children: Recommendations for evaluation and treatment.* Proceedings of 1996 International Conference on Fetal Alcohol Syndrome, Seattle, Washington.

Novick (Brown), N.J., & Streissguth, A.P. (1995). Identifying clients with possible fetal alcohol syndrome: Fetal alcohol effects in the treatment setting. *Treatment Today, 7(3),* 14-15.

Novick (Brown), NJ, & Streissguth, AP (1995). Some thoughts on the treatment of adults and adolescents impaired by fetal alcohol exposure. *Treatment Today, 7(4),* 20-21.

Novick (Brown), N.J., Cauce, A.M., & Grove, K. Competence self-concept. In B.A. Bracken (Ed.), *Handbook of self-concept.* New York: Wiley.

Novick (Brown), N.J., & Brown, J.D. (1992). *The influence of self-esteem on response to mood.* Paper presented, 100th Annual Convention of the American Psychological Association, Washington, D.C., August, 1992.

Brown, J.D., Novick (Brown), N.J., Lord, K.A., & Richards, J.M. (1992). When Gulliver travels: Social context, psychological relatedness, and self-appraisals. *Journal of Personality and Social Psychology, 62*, 717-727.

Norris, J, Novick (Brown), N.J., & Kerr, K.L. (1992). *Alcohol and violent pornography: Impact of social influence on sexual arousal.* Poster presented at the Research Society on Alcoholism Meeting, San Diego, California, June, 1992.

Brown, J.D., & Novick (Brown), N. (1991). *Social context, psychological relatedness, and self-appraisals.* Paper presented at the 99th Annual Convention of the American Psychological Association, San Francisco.

Invited Presentations, Workshops, and Trainings

07/20/12 Forensic Assessment of Developmental Disabilities. American Institute for the Advancement of Forensic Studies; St. Paul, MN

07/13/12 FASD and Competency. WI Association of Criminal Defense Lawyers; Stevens Point, WI

04/19/12 Changing Public Policy in the Juvenile Courts: What Works? Fifth National Biennial Conference on Adolescents and Adults with FASD: It's a Matter of Justice, Vancouver, BC, Canada

03/29/12 Fetal Alcohol Spectrum Disorders. Death Penalty Institute, Lexington, KY

02/03/12 Alcohol Related Birth Disorders and the Law. Mid-year ABA Conference, Interagency Coordinating Committee on FASD in Collaboration with U.S. Dept. of Justice and Minnesota Organization on FAS, New Orleans, LA

02/02/12 FASD and Neurobehavioral Issues in the Criminal Justice System. Capital Defense Project of SE Louisiana, New Orleans, LA

11/18/11 Assessing and Understand Fetal Alcohol Spectrum Disorders in Capital Clients. Virginia Bar Assoc., 19th Annual Capital Defense Workshop, Richmond, VA

10/07/11 FASD and the Criminal Justice System. Seattle City Attorney's Office and University of Washington, Seattle, WA

09/21/11 FASD: Preventing and Treating Sexual Deviancy. Indian Health Service FASD Training, Seattle, WA

07/09/11 FASD and Competency. Capital Mitigation – Beyond Atkins, Center for American and International Law; Houston, TX

06/23/11 FASD in the Courtroom. Ninth Annual Statewide Conference, Arizona Public Defenders Association; Tempe, AZ

05/20/11 FASD and Intellectual Disability/Mental Retardation. Metropolitan Public Defender, Oregon Capital Resource Center, Oregon Criminal Defense Lawyers Association; Portland, OR

03/11/11 Forensic Aspects of Fetal Alcohol Spectrum Disorders. Sponsored by Pathways Counseling Center, MOFAS, Minnesota DOC, MN Community Corrections Association, & American Institute for the Advancement of Forensic Studies; St. Paul, MN

10/27/10 FASD: Its Relevance Throughout the Legal Process from Competency to Stand Trial to Clemency. 2010 Appellate Judicial Attorneys Institute, Burlingame, CA

10/02/10 Forensic Assessment of FASD in the Habeas Context. Federal Defenders Annual Death Penalty Conference, Boise, ID

07/16/10 Team Approach to Litigating FASD (plenary). Center for American and International Law, Plano, TX

07/10/10 Fetal Alcohol Spectrum Disorder in the Courtroom: The 20th Anniversary of Dr. Ann Streissguth (plenary + break-out). NAACP LDF, Airlie, VA

04/22/10 Forensic Assessment of FASD with State-of-the-Art Facial Analysis, Diffusion Tensor Imaging and MRIs. 7th National Seminar on the Development and Integration of Mitigation Evidence (plenary). American Bar Association, Seattle, WA

04/17/10 Suggestibility in FASD: Forensic Assessment and Implications. 4th International Conference on Fetal Alcohol Spectrum Disorder, Vancouver, BC, Canada

03/31/10	Fetal Alcohol Spectrum Disorder and Justice. Alcohol Healthwatch, Parnell, New Zealand. (Abbreviated presentations also provided on 4-1-10 to New Zealand Ministry of Health and Ministry of Justice.)
02/25/10	Fetal Alcohol Spectrum Disorder (FASD). Texas Criminal Defense Lawyers Association, Austin, TX
02/12/10	FASD and Justice: A Multidisciplinary Assessment Model for Adults and Adolescents. CACJ/CPDA Capital Defense Seminar, Monterey, CA.
02/06/10	Fetal Alcohol Syndrome: Practical Tools. 3rd Interdisciplinary Program: UW School of Law & Washington Death Penalty Assistance Center, Seattle, WA.
03/11/09	FASD in the Legal System: A Multidisciplinary Assessment Model for Adults and Adolescents. 3rd International Conference on Fetal Alcohol Spectrum Disorder, Victoria, BC.
11/18/08	Screening for FASD in Family Practice. Family Practitioners, University of Washington/Swedish Hospital, Family Practice Medical Residents In-service.
10/25/08	Sex Offender Commitment Defense Association (SOCDA), Atlanta, GA Cross-Examination of Adverse Expert Witnesses in SVP Commitment Trials
05/30/08	Fetal Alcohol Syndrome and Fetal Alcohol Effect: Identifying Clients and Understanding Consequences. Fifth National Seminar on the Development and Integration of Mitigation Evidence, Habeas Assistance & Training Counsel Project, Baltimore, MD
11/03/07	Direct and Cross Examination of Experts in SVP Cases. Sex Offender Commitment Defense Association (SOCDA), San Diego, CA
08/18/07	Fetal Alcohol Syndrome / Fetal Alcohol Effects. 12th Annual Federal Habeas Corpus Seminar, Nashville, TN
05/23/07	Fetal Alcohol Spectrum Disorders: History, Diagnosis, and Mitigation Issues. Capital Federal Public Defender Unit (capital habeas and trial attorneys, Federal District of Nevada)

04/14/07	What Attorneys and Policy Makers Need to Know About FAS and FASD. American Bar Association/Harvard Law School National Conference on Children and the Law, Cambridge, MA
02/18/07	Fetal Alcohol Spectrum Disorders (FASD). California Attorneys for Criminal Justice/California Public Defender Association (CACJ/CPDA) Annual Death Penalty Conference, Monterey, CA
06/30/06	Sexually Violent Predator Evaluation, Risk Assessment, and Testimony. Florida Public Defenders Sexually Violent Predator Conference, Orlando, FL
04/19/06	Screening Protocol for Fetal Alcohol Spectrum Disorders (FASD). King County Mental Health / Drug Courts, Seattle, WA
02/26/05	FASD: Problems of Witness Suggestibility and False Confessions. International FASD Conference, Victoria, British Columbia, Canada

Professional Memberships

1990 – present	American Psychological Association (APA)
2008 – present	American Society-Law Society (APA)
2005 – present	Association for the Treatment of Sex Abusers (ATSA)
2004 – present	Association of Family & Conciliatory Courts (AFCC – National) (WA-AFCC - Washington State; Board of Directors, Treasurer)
2000 – present	American College of Forensic Examiners
2011 – present	Midwest Alliance on Shaken Baby Syndrome (Board of Directors)
2001 – 2003	Jacksonville Youth Authority Advisory Board
1996 – 2000	Chairman, Social Issues Committee, Washington State Psychological Association
1994 – 2000	Washington State Psychological Association, Board of Directors

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EDUCATION

1998 - 2000	**Senior Fellow in Forensic Psychiatry** University of Washington School of Medicine, Seattle, WA 98195
1986 - 1988	**Fellow in Child Psychiatry** Massachusetts General Hospital /Harvard Medical School, Boston, MA 02114
1984 - 1986	**Resident in Adult Psychiatry** McLean Hospital/Harvard Medical School, Belmont, MA 02478 *Included rotations in Consult – liaison,* *Psychiatry at Brigham & Women's Hospital,* *Forensic Psychiatry at Bridgewater State Hospital,* *Adolescent and Family Treatment Unit at McLean Hospital*
1983 - 1984	**Internship year in Adult Psychiatry** McLean Hospital/Harvard Medical School *Six months medical internship at Mt. Auburn* *MGH, two months of Inpatient Geriatric Psychiatry,* *two months of Substance Abuse Psychiatry*
1978 - 1983	**Albany Medical College of Union University (AMC)** Albany, NY 12208 Doctor of Medicine *Alpha Omega Alpha Honor Society* *Shaeffer Prize in Pathology* *Honor Committee Member 1978 - 1980*

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1980 - 1981 **Research in Gastrointestinal Pathology**
Brigham and Women's Hospital, Department of Pathology,
Ramzi Cotran, M.D., Chief
Boston, MA 02115
American Gastroenterological Association Award for Medical Student Research - Poster Presentation

1976 - 1982 **Rensselaer Polytechnic Institute (RPI)**
Troy, NY 12180
Bachelor of Science in Biology
Summa cum laude, RPI - AMC 6-year Biomedical Program

POSITIONS

5/08 – present **Medical Director**
FASD Experts.com - Interdisciplinary collaborative forensic and clinical evaluation of Fetal Alcohol Spectrum Disorders

2/97 - present **Private Practice**
Forensic and clinical psychiatric practice in child and adult psychiatry

2007 - present **Clinical Instructor**
University of Washington School of Medicine
Seattle, WA

2007 - present **Clinical Instructor**
Antioch University - Seattle
2326 6th Avenue, Seattle, WA 98121

3/00 - present **Speakers Bureau and National Advisory Board**
McNeil Consumer Healthcare, makers of Concerta ®

2006 - 2008 **Psychiatric Consultant**
Renton Academy
Employed by the Renton School District to provide on-site psychiatric services for a new specialty school informed by the Re-Education Approach pioneered by Nicholas Hobbs, Ph.D.

2006	**National Advisory Panel** Cephalon, Inc., makers of Sparlon ® (modafanil)
2/97 - 6/08	**Staff Psychiatrist** Children's Hospital & Medical Center, Seattle, WA *Patient evaluation and psychopharmacologic management, team consultation two days a week*
7/00 – 12/00	**Staff Psychiatrist** Western State Hospital Program for Forensic Evaluations in Corrections and the Community, Tacoma, WA 98498. *Carl Redick, Psy.D., Director*
7/98 – 6/00	**Acting Clinical Instructor (*)** Children's Hospital & Medical Center, Lake City Way Satellite Clinic, 12360 Lake City Way NE, Seattle, WA 98125 - (206) 368-4949 *Individual and group supervision of Child Psychiatry Fellows, Adult Psychiatry Residents, and Psychology Interns at a University of Washington Medical School teaching site.* *(*) During the Senior Fellowship in Forensic Psychiatry (see Education), University of Washington regulations require that the instructorship be regarded as "acting".*
1/98 - 7/98	**Clinical Instructor** Children's Hospital & Medical Center, Lake City Way Satellite Clinic
8/96 - 6/98	**Staff Psychiatrist** Seattle Mental Health Institute, 1600 East Olive Street, Seattle, WA 98122. (206) 324-2400 *Provided staff consultation and treatment to adults, children and adolescents, including The Deaf Services unit, in a large community mental health center (CMHC) with a wide continuum of care.*

8/91 - 7/96	**Medical Director & Founding President** Comprehensive Psychiatric Group, 120 East Main Street, Salisbury, MD 21801. *Region's largest multi-disciplinary mental health practice with Addictions, Eating Disorders and extensive Group Psychotherapy Programs.*
3/95 - 7/95	**Consultant - Behavioral Health Services** Peninsula Regional Medical Center (PRMC), 100 East Carroll Street, Salisbury, MD 21801. *Retained to design integrated and managed care-friendly inpatient, outpatient, consult-liaison and emergency mental health services.*
1/94 -12/95	**Community Affiliate** Peninsula Regional Medical Center
11/91-12/93	**Attending Psychiatrist** Peninsula Regional Medical Center *Inpatient, emergency and consult - liaison services*
7/88 - 6/92	**Medical Director, Mental Health Clinics** Somerset County Health Department, Westover, MD 21871. *National Health Service Corps Scholarship Placement; Included consultation to Colbourne Substance Abuse Center, Crisfield, MD, Somerset County Sheriff's Department, Public Schools, and Developmental Center.*
7/88 - 6/91	**Medical Director, Mental Health Clinics** Worcester County Health Department, Snow Hill, MD 21863. *National Health Service Corps Scholarship Placement*
7/86 - 6/88	**Part-time private practice** Boston, MA
10/86 - 6/88	**Ciba-Geigy Clinical Trial of Clomipramine for Obsessive-Compulsive Disorder in Children and Adolescents.** Massachusetts General Hospital, Boston, MA 02114. *Co-investigator with Joseph Biederman, M.D.*

1986 - 1990	**Doctor-on-Call** Pembroke Hospital, Pembroke, MA. *150 bed Private Psychiatric Hospital*
1985 - 1990	**Doctor-on-Call** Norwood Hospital, Norwood, MA. *21 bed locked psychiatric unit in a suburban community General hospital with Emergency Room and Consult-liaison consultation services.*
1985 - 1986	**Psychopharmacology Consultant** Cutler Counseling Center, Norwood, MA. *Non-profit, community outpatient facility*

LICENSES

1996 - present	Washington 33536 (3/12/96)
1988 - 1996	Maryland D36903 (6/18/88)
1984 - 1991	Massachusetts 54358 (6/1/84)
2012 - present	Georgia 68346 (7/30/12)

CERTIFICATIONS

1992	**Child and Adolescent Psychiatry** American Board of Psychiatry and Neurology, #2903
1990	**Adult Psychiatry** American Board of Psychiatry and Neurology, #33030
1984	**National Board of Medical Examiners**, #262751

MAJOR COMMITTEE ASSIGNMENTS

2005	**Chairman, Ethics Committee** Washington State Psychiatric Association

1998	**Managed Care Committee** Division of Child & Adolescent Psychiatry, Children's Hospital and Regional Medical Center, Seattle, WA.
1995	**Representative of the Maryland Psychiatric Society to the Rural Psychiatry Task Force** American Psychiatric Association Annual Meeting, May, Miami Beach, FL
1992 - 1996	**Board of Directors** Life Crisis Center, 216 East Main Street, Salisbury, *Chairman, Executive Director Search Committee (1994)*
1993	**Select Committee, Quality Council** Peninsula Regional Medical Center. Empowered to re-engineer Emergency Room Psychiatric Services. Developed satisfaction assessment tool
1992 - 1993	**Utilization Review Committee** Peninsula Regional Medical Center
1991 - 1993	**Founding President** Eastern Shore Psychiatrists' Group *Established to provide networking opportunities*
1991	**Physicians' Steering Committee** Atlantic General Hospital, Berlin, MD. *Providing input on design of new community General hospital*
1988 - 1992	**Utilization Review Committee** Peyton Psychogeriatric Unit, McCready Hospital, Crisfield, MD. *Specialty in-patient locked unit for chronically mentally ill geriatric patients*

MEMBERSHIPS

1996 - present	American Academy of Psychiatry and the Law
1984 - present	American Psychiatric Association
1991 - 1996	Eastern Shore Psychiatrists' Group
1988 - 1996	Maryland Psychiatric Society

1986 - 1988 Massachusetts Psychiatric Society

PUBLICATIONS & PRESENTATIONS

2012 **"Fetal Alcohol Spectrum Disorder Training: (1) What is FASD and How Does It Affect the Behavior of Your Client? (2) Getting a Diagnosis: Who Can Do It and What Is Involved?** Washington Defender Association - 2012 Defender Conference: Creating Better Outcomes for Our Clients, Winthrop, WA. April 28, 2012.

2011 **"Conduct-disordered Adolescents with Fetal Alcohol Spectrum Disorder: Intervention in Secure Treatment Settings"** Brown, N.N., Adler, R.S., & Connor, P.D. (in press). *Criminal Justice and Behavior.*

2011 **"Neuroimaging"** Ellis, J, Adler RS, Oregon Criminal Defense Lawyers Association: Death Penalty Defense: The Cutting Edge: New Developments in Law and Science, Pendleton, OR. October 21, 2011.

2011 **"Picking the Right Expert"** Oregon Criminal Defense Lawyers Association: Death Penalty Defense: The Cutting Edge: New Developments in Law and Science, Pendleton, OR. October 21, 2011.

2011 **"FASD: Diagnosis for the Legal Setting"** Seattle Municipal Attorney's Office: Fetal Alcohol Spectrum Disorders and the Criminal Justice System, Seattle City Hall. October 7, 2011.

2011 **"Prenatal Substance Exposure and Childhood Mental Health Issues"** UW School of Medicine Department of Psychiatry: Indian Health Service - Fetal Alcohol Spectrum Disorders (FASD) Training. Seattle, WA. September 21-22, 2011.

2011 **"Risk Factors for Mental Health Problems in Children"** Lummi Nation Summer Training. Bellingham, WA. September 16, 2011.

2011 **"Ethics"** NBI Conference: Use of Parenting Evaluations in Divorce. Washington State Convention and Trade Center. September 15, 2011.

2011 **"Introduction to Forensic Psychiatry"** UW School of Medicine Department of Psychiatry: Forensic Psychiatry R4 Seminar Series. University of Washington. September 8, 2011

2011 **"FASD: Diagnosis for the Legal Setting"** Snow R, Adler, RS, UW School of Law: Fetal Alcohol Spectrum Disorders and the Criminal Justice System. King County Courthouse. March 1, 2011.

2010 **"Fetal Alcohol Spectrum Syndrome: Its Relevance Throughout the Legal Process From Competency to Stand Trial to Clemency,"** Adler RS, Novick Brown N, 2010 Appellate Judicial Attorneys Institute, Administrative Office of the Courts: Education Division. Burlingame, CA. October 27, 2010. www2.courtinfo.gov

2010 **"Violence Risk Assessment in 2010: State of the Art as Seen Through a Wide Lens,"** Adler RS, Heavin S, McMinimee S. 2010 Oregon-Washington Bi-State School Psychology Conference. Vancouver, WA. October 14, 2010. www.WSASP.org.

2010 **"Creating a Tomorrow: Post-Mortem on a Death Verdict."** Panel Member/Faculty, Full-day Continuing Legal Education Program, Washington Death Penalty Assistance Center. Seattle, WA. September 22, 2010.

2010 **"Fetal Alcohol Spectrum Disorder in the Courtroom: The 20th Anniversary of Dr. Ann Streissguth at Airlie,"** (Plenary Session). Adler RS, Kase KM, Kelly K, Novick Brown N. NAACP Legal Defense Fund 31st Annual Meeting. Airlie Conference Center, Warrenton, VA. July 10, 2010.

2010 **"FASD: Practicalities,"** (Breakout Session). Novick Brown N, Adler RS, Kase KM, Kelly K. NAACP Legal Defense Fund 31st Annual Meeting. Airlie Conference Center, Warrenton, WA. July 10, 2010.

2010 **"Forensic Assessment of Fetal Alcohol Spectrum Disorders with State-Of-The-Art Facial Analysis, Diffusion Tensor Imaging, and MRIs,"** (Plenary Session). Adler RS, Novick Brown N, Connor PD, Wartnik AP. Habeas Assistance & Training Counsel Project, Seventh National Seminar on the Development and Integration of Mitigation Evidence: "New Science, New Strategies." Seattle, Washington, April 22, 2010.

2010 **"Suggestibility in FASD: Forensic Assessment and Implications,"** Adler RS, Novick Brown N, Connor PD, Wartnik AP. 4th National Biennial Conference on Adolescents and Adults with Fetal Alcohol Spectrum Disorder: Facing the Future Together: Where Do We Go From Here?. Vancouver, B.C., Canada. April 17, 2010

2010 **"FASD: Application of the Scientific Method to the Forensic Setting."** Adler RS, Novick Brown N, Connor PD. Texas Criminal Defense Lawyers Association Annual Capital Trial/Habeas Seminar. Austin, Texas. February 25, 2020.

2010 **"Fetal Alcohol Syndrome: Practical Tools"** Adler RS, Novick Brown N, Connor PD, Wartnik AP. 3rd Interdisciplinary Program, University of Washington School of Law & The Washington Death Penalty Assistance Center: Expertise You Need To Defend A Capital Case In 2010. Seattle, WA. February 6, 2010.

2009 **"Testamentary Capacity: Why, When and How to Use a Mental Health Expert."** King County Bar Association. 12th Annual Probate, Trust and TEDRA Litigation Conference. Rainier Square Conference Center, Seattle, WA. December, 4, 2009.

2008	**"Fetal Alcohol Spectrum Disorder in the Educational Context."** Lake Washington School District – In Service. October 24, 2008
2008	**"Vetting The Expert: Why, When and How" & "Bringing It All Home"** (Dr. Adler co-presented with Seattle attorney Jeff Robinson of Shroeter, Goldmark and Bender). TeamChild Benefit CLE" Collaboration Between Attorneys and Mental Health Experts: Beyond the Basics. Seattle, WA. October 3, 2008. www.teamchild.org.
2007	**"Parenting Evaluations – A Critical Review and Practice Skills"** Washington State Bar Association – Continuing Legal Education, Washington State Convention and Trade Center. Seattle, WA. August 8, 2007 and Doubletree Inn, Suite B, Spokane, WA, August 21, 2007. www.wsbacle.org
2004 & 2006	**"How to Help Your Client Survive a Parenting Evaluation in Washington"** National Business Institute Seminar, Washington State Convention and Trade Center, Seattle, WA. November 5, 2004; November 10, 2006. www.nbi-sems.com
2002	**"Child Abuse and Neglect"** Western State Hospital Forensic Seminar Series, Greg Gaggliardi, Ph.D., Seminar Director.
2000	**"Pediatric Psychopharmacology: The Cutting Edge"** Washington State and British Columbia Associations of School Psychologists Fall Conference, Vancouver, B.C.
1999	**"Pediatric Psychopharmacology: The Cutting Edge"** Child and Adolescent Neuropsychiatry Conference, Children's Hospital and Regional Medical Center, Seattle, WA.
1998	**"The Broad Bandwidth of Autism Spectrum Disorders"** Child and Adolescent Neuropsychiatry Conference, Children's Hospital and Regional Medical Center, Seattle, WA.

1997 **"No Need to Panic: Anxiety Disorders Update for the Clinician."** Child and Adolescent Neuropsychiatry Conference, Children's Hospital and Regional Medical Center, Seattle, WA.

1991 **"The Transition from Resident to Medical Director"**, American Psychiatric Annual Meeting, Course: Administration in Austerity. Paul Rodenhauser, M.D., Course Director.

1990 Adler RS, Jellinek MS. **Special Article: "After Teen Suicide: Issues for Pediatricians Who Are Asked to Consult to Schools".** Pediatrics. 86:6, 982 - 7.

1990 Adler RS. **Out-of-Control Behavior.** The Massachusetts General Hospital: Psychiatric Aspects of General Hospital Pediatrics. Jellinek MS, Herzog DB, Eds. Chicago: Year Book Medical Publishers, pp. 298-304.

1990 Adler RS, Jellinek M. **Suicide.** Ibid. pp. 324 - 30.

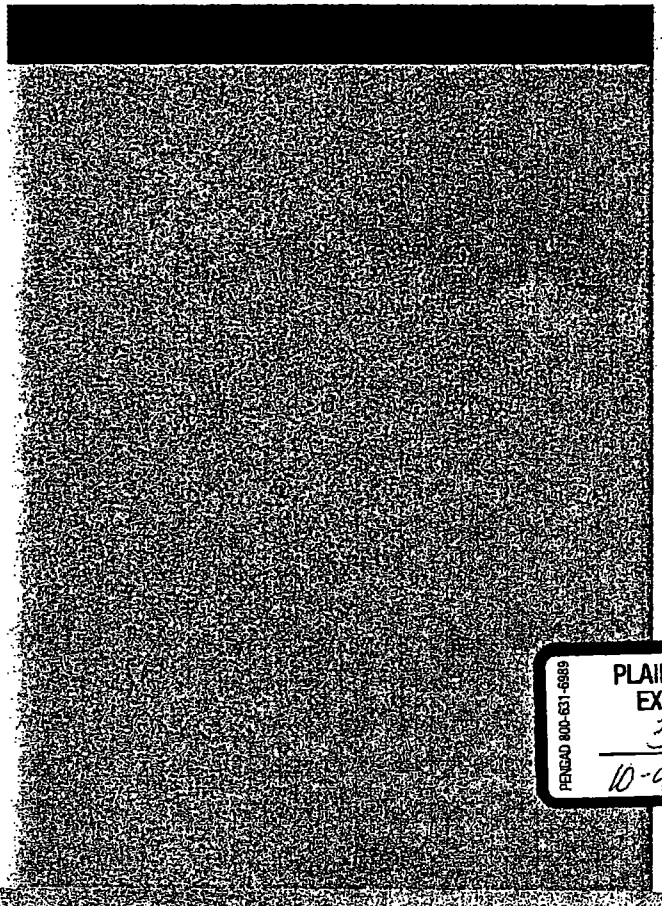
1985 Adler RS, Nafradi J, Szabo S. **"Cysteamine-induced Duodenal Ulcer is not Site-specific: Effect of Local Modulating Factors".** J. Experimental Pathology 2:2, 111-22.

1983 Adler RS, Gallagher GT, Szabo S. **"Duodenal Ulcerogens Cysteamine and Proprionitrile Decrease Duodenal Neutralization of Acid in the Rat".** Digestive Diseases and Sciences 28:8, 716-23.

Fetal Alcohol Syndrome

Diagnosis, Epidemiology, Prevention, and Treatment

Kathleen Stratton, Cynthia Howe, and Frederick Battaglia, *Editors*



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PLAINTIFF'S EXHIBIT
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What follows are pages 1, 63, and 74-78 of Plaintiff's Exhibit 31, Fetal Alcohol Syndrome: Diagnosis, Epidemiology, Prevention, and Treatment. Stratton K, Howe C, Battaglia F (editors). National Academy Press. (1996). The original text book is on file with the Greenville County Clerk of Court and an exact duplicate has been mailed to the South Carolina Supreme Court for reference to any other pages. These are the pages cited in petitioner's petition for certiorari.

Executive Summary

Fetal alcohol syndrome (FAS), first described in the published medical literature in 1968, refers to a constellation of physical abnormalities, most obvious in the features of the face and in the reduced size of the newborn, and problems of behavior and cognition, in children born to mothers who drank heavily during pregnancy. These latter features lead to the most concern. The costs of FAS and related conditions can be quite high—for the individual, for the family, and for society. Rates of FAS in several of the most complete studies are similar—on the order of 0.5 to 3 cases per 1,000 births. Assuming an annual birth cohort of approximately 4 million, this translates into 2 to 12 thousand FAS births per year in this country. These incidence figures are offered not as established facts but to emphasize the magnitude of a problem that has serious implications—for the individual and for society.

In recognition of the seriousness of this problem, the U.S. Congress mandated in Section 705 of Public Law 102-321, the ADAMHA Reorganization Act, that the Institute of Medicine (IOM) of the National Academy of Sciences conduct a study of FAS and related birth defects. This report is in response to that mandate.

The charge to the committee was to improve the understanding of available research knowledge and experience on:

- tools and approaches for diagnosing FAS and related disorders
- the prevalence of FAS and related disorders in the general population of the United States
- the effectiveness of surveillance systems, and

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Diagnosis and Clinical Evaluation of Fetal Alcohol Syndrome

Since the original description of fetal alcohol syndrome (FAS) approximately two decades ago, substantial progress has been made in developing specific criteria for delineating this syndrome. Nonetheless, a variety of key issues continue to sustain areas of controversy. The boundaries of the diagnosis, as well as the markers that should be used to delineate those boundaries, are perhaps the most vexing issues. Current discussions of FAS have also included discussions of a related condition known as "fetal alcohol effects." This latter condition has been variably defined, but often includes the concept that a subset of FAS features can occur and be related to a suspected maternal exposure to alcohol. Difficulties in obtaining an adequate history of alcohol intake, a widely recognized problem in medical evaluation, add to the complexity of this issue. The resolution of these issues is not trivial, for they have important implications for both patient care and social policy. Consequently, one charge of this committee was to evaluate existing diagnostic criteria and, in updating and expanding these, to consider the formulation of the best possible diagnostic guidelines, which could subsequently be used in epidemiologic, clinical, and basic research into this syndrome. In this context, it is helpful to step back and examine the process of diagnostic evaluation within its broadest context.

PURPOSE OF MEDICAL DIAGNOSES

A medical diagnosis serves several major purposes: to facilitate communication among clinicians; to facilitate communication between clinician and patient

and prepubertal photographs are often needed to establish an FAS diagnosis in these older patients.

The fact that this syndrome is most easily diagnosed in a specific period of life is not unique to FAS. Many syndromes become easier or more difficult to diagnose with increasing patient age. For example, the Wiedemann-Beckwith syndrome is defined by macrosomia at birth and large viscera. Typically the tongue is quite large and there is an omphalocele present in the newborn period. In infants this is a readily apparent disorder, but as the child ages organ size and overall height normalize. After puberty the syndrome is extremely subtle and would generally not be considered without the neonatal description. The Prader-Willi syndrome is defined by hypotonia in infancy, mental retardation, hypogonadism, and progressive obesity in childhood. While the hyoptonia, hypogonadism and the minor anomalies are present in infancy, the diagnosis (prior to the recent advent of chromosomal testing) was withheld until the obesity and developmental delay become apparent in the second or third year of life. Similarly, the hallmark of Fragile-X syndrome is testicular enlargement which is generally not found until after puberty. Prior to the discovery of the genetic marker, Fragile-X syndrome was rarely diagnosed until the second decade of life.

DIAGNOSTIC CATEGORIES

One of the key charges to the committee was to review and evaluate the diagnostic criteria for FAS and related conditions. The committee studied the previous diagnostic criteria and felt that some of the issues confusing the clinical and research communities could be resolved with fairly minor changes in the diagnostic categories and criteria. These new criteria can be found in Table 4-1.

The diagnostic criteria for FAS as described in the preceding section are found under Category 1, FAS with confirmed maternal alcohol exposure. A diagnosis is placed in this category when appropriate anomalies are found in face, brain, *and* growth, *and* a clear history of alcohol exposure is obtained. The committee currently defines the relevant history as one of a pattern of excessive intake characterized by substantial, regular intake or heavy episodic drinking. Evidence of this pattern may include: frequent episodes of intoxication, development of tolerance or withdrawal, social problems related to drinking, legal problems related to drinking, engaging in physically hazardous behavior while drinking, or alcohol-related medical problems such as hepatic disease. It is anticipated that patients in this category would remain the template for further delineation of the condition.

Category 2, FAS without confirmed maternal alcohol exposure, is assigned to patients with all the clear phenotypic features necessary for an FAS diagnosis but without a confirmed history of alcohol exposure. Many patients with FAS are in foster or adoptive placements and their prenatal exposure histories are unavailable. In other cases, the birth mother honestly cannot recall the specifics of her

alcohol use in gestation or remains in denial of her alcohol abuse. It is unfair to deny these patients the clinical benefits of a medical diagnosis, but it also seems imprudent to combine their cases with those in the first category. FAS with confirmed maternal alcohol exposure, when carrying out some forms of research. Therefore, it is recommended that they be grouped separately.

It also remains theoretically possible that individuals might be found with the full FAS phenotype and a confirmed negative history of gestational alcohol exposure. (Phenocopies do exist for nearly every condition.) Although we are not aware that this situation has arisen yet, such cases should not be considered in category 2. They do not have FAS.

Category 3, partial FAS with confirmed maternal alcohol exposure, is assigned to patients with a confirmed exposure to substantial amounts of alcohol in gestation, some components of the facial features of FAS, and any of the following: evidence of growth deficiency, CNS neurodevelopmental abnormalities, or a complex pattern of behavioral and cognitive abnormalities. This diagnostic category allows an FAS diagnosis to be given to someone who would not receive a Category 1 diagnosis, FAS with confirmed maternal alcohol exposure. This diagnosis could be particularly useful, for example, for some patients who present for diagnosis as an adult. The natural history of FAS is such that some of the "hallmark" indicators used in infancy or childhood are not maintained into adolescence or adulthood. For example, facial dysmorphia can become less distinct and some adolescents, particularly females, gain weight into or above normal ranges. Absent good medical records of growth retardation at birth and early infancy, an FAS diagnosis otherwise could not be given without Category 3. This diagnosis could also be given to those young children whose growth metrics are within normal ranges, which some studies suggest can occur if the mother cuts down her alcohol exposure in the third trimester. This diagnosis can also be used as a "holding" category as a means to defer a diagnosis of Category 1, FAS with confirmed maternal history of alcohol exposure, until more data collection or evaluation, including documentation as to whether behavioral and cognitive abnormalities persist over time, can support a more definitive diagnosis. In the newborn, for example, there is some controversy whether some behavioral abnormalities, such as abnormalities of state regulation, indicate or predict long-term dysfunction due to fetal alcohol exposure. In such cases, documentation of abnormalities over time would be important.

The naming of this diagnostic category was challenging for the committee, who found no perfect solution. Terms considered were probable, partial, and atypical. "Probable" was unsatisfactory, because it denotes uncertainty about the etiology of the condition and because of concerns that appropriate services or reimbursement for these services would not be made for a "probable" condition. The committee intends for this diagnostic category to include people with signs and symptoms attributable to significant prenatal alcohol exposure and who need medical, social services, and other attention. "Atypical" has recently been discarded

TABLE 4-1 Diagnostic Criteria for Fetal Alcohol Syndrome (FAS) and Alcohol-Related Effects

Fetal Alcohol Syndrome

1. FAS with confirmed maternal alcohol exposure[a]

A. Confirmed maternal alcohol exposure[a]

B. Evidence of a characteristic pattern of facial anomalies that includes features such as short palpebral fissures and abnormalities in the premaxillary zone (e.g., flat upper lip, flattened philtrum, and flat midface)

C. Evidence of growth retardation, as in at least one of the following:
- — low birth weight for gestational age
- — decelerating weight over time not due to nutrition
- — disproportional low weight to height

D. Evidence of CNS neurodevelopmental abnormalities, as in at least one of the following:
- — decreased cranial size at birth
- — structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia)
- — neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination

2. FAS without confirmed maternal alcohol exposure

B, C, and D as above

3. Partial FAS with confirmed maternal alcohol exposure

A. Confirmed maternal alcohol exposure[a]

B. Evidence of some components of the pattern of characteristic facial anomalies

Either C or D or E

C. Evidence of growth retardation, as in at least one of the following:
- —low birth weight for gestational age
- — decelerating weight over time not due to nutrition
- — disproportional low weight to height

D. Evidence of CNS neurodevelopmental abnormalities, as in:
- — decreased cranial size at birth
- — structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia)
- — neurological hard or soft signs (as age appropriate) such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination

E. Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties; deficits in school performance; poor impulse control; problems in social perception; deficits in higher level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention, or judgment

Alcohol-Related Effects

Clinical conditions in which there is a history of maternal alcohol exposure,[a,b] and where clinical or animal research has linked maternal alcohol ingestion to an observed outcome.

There are two categories, which may co-occur. If both diagnoses are present, then both diagnoses should be rendered:

4. Alcohol-related birth defects (ARBD)
List of congenital anomalies, including malformations and dysplasias

Cardiac	Atrial septal defects	Aberrant great vessels
	Ventricular septal defects	Tetralogy of Fallot
Skeletal	Hypoplastic nails	Clinodactyly
	Shortened fifth digits	Pectus excavatum and carinatum
	Radioulnar synostosis	Klippel-Feil syndrome
	Flexion contractures	Hemivertebrae
	Camptodactyly	Scoliosis
Renal	Aplastic, dysplastic, hypoplastic kidneys	Ureteral duplications
	Horseshoe kidneys	Hydronephrosis
Ocular	Strabismus	Refractive problems secondary to small globes
	Retinal vascular anomalies	
Auditory	Conductive hearing loss	Neurosensory hearing loss
Other	Virtually every malformation has been described in some patient with FAS. The etiologic specificity of most of these anomalies to alcohol teratogenesis remains uncertain.	

5. Alcohol-related neurodevelopmental disorder (ARND)
Presence of:
A. Evidence of CNS neurodevelopmental abnormalities, as in any one of the following:
— decreased cranial size at birth
— structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia)
— neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination

and/or:

B. Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties; deficits in school performance; poor impulse control; problems in social perception; deficits in higher level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention, or judgment

[a] A pattern of excessive intake characterized by substantial, regular intake or heavy episodic drinking. Evidence of this pattern may include frequent episodes of intoxication, development of tolerance or withdrawal, social problems related to drinking, legal problems related to drinking, engaging in physically hazardous behavior while drinking, or alcohol-related medical problems such as hepatic disease.
[b] As further research is completed and as, or if, lower quantities or variable patterns of alcohol use are associated with ARBD or ARND, these patterns of alcohol use should be incorporated into the diagnostic criteria.

by DSM-IV and the committee did not think that these patients were "not typical." In fact, all of their signs and symptoms are typical of FAS. "Partial" denotes, to some people, that the condition might not be as severe, which the committee did not wish to imply. The committee settled on the use of "partial" despite these reservations. This category, and its name, should remain flexible. As further research reveals the range of the manifestations of FAS, this diagnosis should evolve to be consonant with new data. This diagnosis can be used to categorize such patients for future use in studying and understanding the condition.

The term fetal alcohol effects (FAE) was initially proposed (Clarren and Smith, 1978) as a term for use when an adverse birth outcome could be proven to be related to alcohol exposure in utero. Generally, this term is properly used in animal models of alcohol teratogenesis and in large prospective group studies of humans exposed to alcohol prenatally. The term was not meant for use with individual patients. Terms such as "suspected fetal alcohol effect" and "possible fetal alcohol effect" were suggested as entries on differential diagnostic lists, but this approach has not been well understood. Later, the term alcohol-related birth defects was suggested for clinical use with this category of patient (Sokol and Clarren, 1989). This term presents clinical problems because most patients who seek diagnosis and do not have FAS, but were alcohol-exposed, do not have major malformations of organs; rather, they have evidence of CNS neurodevelopmental abnormality. The term "birth defects" generally is understood by most lay people to refer to gross structural anomalies, although the March of Dimes defines birth defects as abnormalities of either structure or function.

The committee believes that it may be helpful to subdivide this patient group of possible prenatal *alcohol-related effects* into two groups listed in Table 4-1 as categories 4 and 5. Category 4, "alcohol-related birth defects" (ARBD), is reserved for patients with physical anomalies, and category 5, "alcohol-related neurodevelopmental disorder" (ARND), is reserved for patients with neurodevelopmental problems. These diagnostic categories include clinical conditions for which clinical or animal research has linked maternal alcohol ingestion to an observed outcome. A history of confirmed maternal alcohol exposure is required for these diagnoses. The relevant history remains as defined for FAS, but the committee notes that as further research is completed and as, or if, lower quantities or variable patterns of alcohol use are associated with ARBD or ARND, these patterns of alcohol use should be incorporated into the diagnostic criteria. These final two diagnostic categories are intended to convey some degree of uncertainty whether prenatal alcohol exposure caused the adverse effects documented in an individual patient, or whether other factors were causative in this case. Whereas patients in categories 1, 2 and, 3 are mutually exclusive, patients could be in both categories 4 and 5. Because of the variability in the specific presentation of FAS, ARBD, or ARND, these diagnoses are most valuable clinically if accompanied

Fetal Alcohol Syndrome:



Guidelines for Referral and Diagnosis

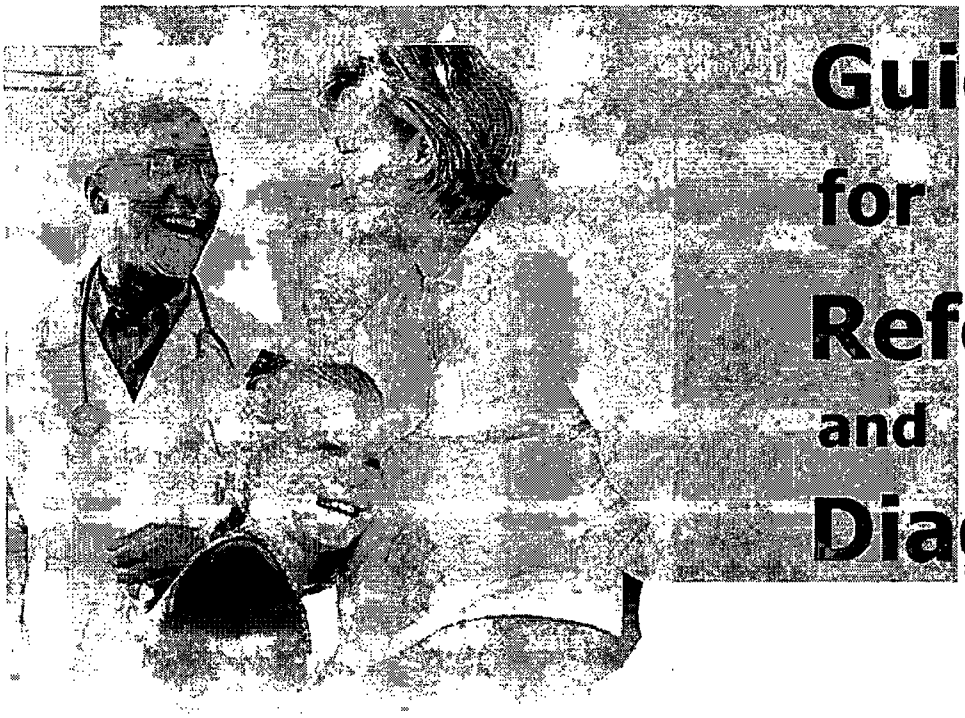


National Center on Birth Defects and Developmental Disabilities
Centers for Disease Control and Prevention
Department of Health and Human Services

in coordination with

National Task Force on
Fetal Alcohol Syndrome and Fetal Alcohol Effect

Fetal Alcohol Syndrome:



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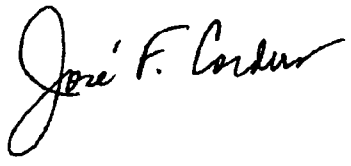
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Foreword

The National Center on Birth Defects and Developmental Disabilities at the Centers for Disease Control and Prevention, in collaboration with the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect, is pleased to present ***Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis.*** This document represents the deliberations of clinicians, researchers, parents, and representatives of governmental and non-governmental organizations, whose main goals were to increase the identification of individuals with fetal alcohol syndrome (FAS) using uniform criteria, and to improve the delivery of appropriate services to those individuals and their families. These new guidelines will help achieve those goals by educating medical and allied health professionals about FAS.

In 2003, we, in the FAS research and practice communities, celebrated the 30th anniversary of the first reports describing fetal alcohol syndrome. Since that time we have learned a great deal about this preventable condition. We now recognize that FAS represents the tip of the iceberg and that there is a continuum of outcomes associated with prenatal exposure to alcohol. These guidelines were undertaken, in part, as an effort to facilitate further identification, understanding, and study of all conditions resulting from prenatal exposure to alcohol. They build on previous work and incorporate important scientific and clinical knowledge that has been obtained in recent years. CDC is pleased to provide continuing support for the expansion and refinement of scientific descriptions for FAS and other disorders related to prenatal exposure to alcohol through its ongoing work with the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect and the federal Interagency Coordinating Committee on Fetal Alcohol Syndrome (ICCFAS).

Preventing all adverse outcomes associated with prenatal alcohol exposure remains a primary goal of CDC, as well as the entire U.S. Department of Health and Human Services. CDC is committed to working with other federal agencies, organizations in the private sector, relevant partners, and the public to achieve this goal. Similarly, CDC is committed to enhanced early identification of individuals with FAS and related disorders to ensure their access to appropriate services. These latest guidelines for referral and diagnosis are an important step towards that goal. Together we will ensure all persons with FAS and related disorders develop optimally and reach their full potential.



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Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis

EXECUTIVE SUMMARY

As part of the fiscal year 2002 appropriations funding legislation, the U.S. Congress mandated that the Centers for Disease Control and Prevention (CDC), acting through the National Center on Birth Defects and Developmental Disabilities (NCBDDD) Fetal Alcohol Syndrome (FAS) Prevention Team and in coordination with the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect (NTFFAS/FAE), other federally funded FAS programs, and appropriate non-governmental organizations, would:

- Develop guidelines for the diagnosis of FAS and other negative birth outcomes resulting from prenatal exposure to alcohol,
- Incorporate these guidelines into curricula for medical and allied health students and practitioners, and seek to have them fully recognized by professional organizations and accrediting boards, and
- Disseminate curricula to and provide training for medical and allied health students and practitioners regarding these guidelines.

Through the coordinated efforts of CDC, the NTFFAS/FAE, and a scientific working group (SWG) of experts in FAS research, diagnosis, and treatment, the following diagnostic criteria were developed over a 2-year period:

Facial dysmorphia

Based on racial norms, individual exhibits all three characteristic facial features:

- Smooth philtrum (University of Washington Lip-Philtrum Guide rank 4 or 5)
- Thin vermillion border (University of Washington Lip-Philtrum Guide rank 4 or 5)
- Small palpebral fissures (at or below 10th percentile)

Growth problems

Confirmed prenatal or postnatal height or weight, or both, at or below the 10th percentile, documented at any one point in time (adjusted for age, sex, gestational age, and race or ethnicity).

Central Nervous System Abnormalities

I. Structural

1) Head circumference (OFC) at or below the 10th percentile adjusted for age and sex.
2) Clinically significant brain abnormalities observable through imaging.

II. Neurological
Neurological problems not due to a postnatal insult or fever, or other soft neurological signs outside normal limits.

III. Functional
Performance substantially below that expected for an individual's age, schooling, or circumstances, as evidenced by:

1. Global cognitive or intellectual deficits representing multiple domains of deficit (or significant developmental delay in younger children) with performance below the 3rd percentile (2 standard deviations below the mean for standardized testing)

or

2. Functional deficits below the 16th percentile (1 standard deviation below the mean for standardized testing) in at least three of the following domains:

a) cognitive or developmental deficits or discrepancies
b) executive functioning deficits
c) motor functioning delays
d) problems with attention or hyperactivity
e) social skills
f) other, such as sensory problems, pragmatic language problems, memory deficits, etc.

Maternal Alcohol Exposure
I. Confirmed prenatal alcohol exposure
II. Unknown prenatal alcohol exposure

Criteria for FAS Diagnosis
Requires all three of the following findings:
1. Documentation of all three facial abnormalities (smooth philtrum, thin vermillion border, and small palpebral fissures);
2. Documentation of growth deficits
3. Documentation of CNS abnormality

A primary goal of these guidelines is to provide standard diagnostic criteria for FAS so that consistency in the diagnosis can be established for clinicians, scientists, and service providers. The guidelines are based on state-of-the-art scientific research, clinical expertise, and family input regarding the physical and neuropsychological features of FAS. The SWG sought to harmonize these guidelines with other diagnostic systems currently in use in this country and others (e.g., Canada). The SWG strove to provide a balance between conservative and overly inclusive diagnostic systems. Differential diagnosis from other genetic, teratological, and behavioral disorders was emphasized.

In addition to diagnostic guidelines, guidance about medical, educational, social, and family services appropriate for individuals with FAS and their families are reviewed. Services that are applicable to all individuals with FAS and their families, as well as age-specific services, are included. Such services focus on increasing parent and professional knowledge of FAS, characteristics of the disor-

der, differences between FAS and other disorders, and appropriate techniques for parenting or educating affected individuals.

Prevention of FAS and related disorders is of tremendous public health importance. A large amount of research in recent years has enabled researchers and service providers to develop programs that are effective and targeted to specific populations for reducing the risk of an alcohol-exposed pregnancy, which prevents FAS. This research is reviewed herein and recommendations for identifying and intervening with women at risk for an alcohol-exposed pregnancy are provided.

Finally, these guidelines are not intended to be an endpoint in the discussion of diagnosing FAS. There is a great need to acquire science-based information that will facilitate diagnostic criteria for additional related disorders, such as Alcohol Related Neurodevelopmental Disorder (ARND). These guidelines conclude with a call for further research and continuous refinement of the diagnostic criteria for FAS and related conditions so that affected individuals and their families can receive important services that enable them to achieve healthy lives and reach their full potential.

Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis

Substantial empirical and clinical scientific evidence has shown that prenatal exposure to alcohol causes damage to the developing fetus. Such exposure is commonly cited as the leading preventable cause of birth defects and developmental disabilities (1-3). Children* exposed to alcohol during fetal development can suffer multiple effects. While the number and severity of negative effects can range from subtle to serious, the negative consequences are lifelong. The effects of prenatal exposure to alcohol and basic diagnostic features of fetal alcohol syndrome (FAS) were first described in the United States (U.S.) medical literature 30 years ago (4-8). In 1981, the U.S. Surgeon General issued a public health advisory warning that alcohol use during pregnancy could cause birth defects (9). Further, mandated labeling of alcohol products was established in 1989 (10). Despite the known adverse effects of prenatal exposure to alcohol, many children who experience these adverse effects do not receive proper diagnosis due to the absence of current diagnostic guidelines. These current guidelines, which were federally mandated of the Centers for Disease Control and Prevention (CDC) in the U.S. Department of Health and Human Services (DHHS) 2002 Appropriations Bill, seek to update and refine diagnostic and referral criteria in light of the scientific and clinical advances in the understanding of this disorder during the past 30 years.

These guidelines are organized into several sections. Background information and a history of the development of these guidelines are presented. Next, revised and refined diagnostic and referral criteria for FAS are described, including the empirical and clinical evidence that support each criterion. Comparison of these guidelines with other diagnostic methods currently in use is provided. Because diagnosis is not the endpoint for most clinicians who see children with FAS, a discussion of the essential services for affected individuals is included. Likewise, prevention of FAS by reducing the number of alcohol-exposed pregnancies is inherent in dealing with the disorder. Therefore, a discussion focused on identifying and intervening with women at risk for an alcohol-exposed pregnancy is provided. Finally, a discussion of future needs and efforts related to FAS and other prenatal alcohol-related disorders conclude this report.

BACKGROUND

Prevalence. Studies by CDC have reported FAS prevalence rates from 0.2 to 1.5 cases per 1,000 births across various populations (11-14). Other studies reflecting a variety of ascertainment methodologies have produced estimates ranging from 0.5 to 2.0 cases per 1,000 live births (15-16). Such rates are comparable with or above other common developmental disabilities such as Down syndrome or Spina Bifida (17). Using the CDC FAS estimates, among the approximately 4 million infants born each year, an estimated 1,000 to 6,000 will be born with FAS. Studies of particularly vulnerable populations yield prevalence estimates that far exceed those of other common disabilities. Disadvantaged groups, Native Americans, and other minorities have been documented to have

* Although referral and diagnosis for FAS can be made throughout the lifespan, the majority of individuals are referred and diagnosed in childhood. Thus, the terms "child" or "children" as used in these guidelines are not intended to preclude referral, assessment, and diagnosis of older individuals.

rates as high as three to five FAS affected children per 1,000 children (18-20). Available data also suggest that poverty is strongly associated with women's alcohol use before and during pregnancy, leading to an excess of children with FAS in impoverished groups (21-22).

The magnitude of the problem is even greater when the risk of FAS is considered by looking at the rate of alcohol-exposed pregnancies. In 1999, over half of all U.S. women of childbearing age reported alcohol consumption in the past month (23). The large majority of these women drank only occasionally, but 15% could have been classified as moderate or heavy drinkers (24-25). During that same period, 13% of women reported consuming five or more drinks on one occasion (binge drinking) in the past month (26). Given that nearly half of all U.S. pregnancies are unintended, and that millions of fertile women are sexually active while not using adequate contraception, an estimated 2% of women could be at risk for an alcohol-exposed pregnancy annually (27). More recently, higher rates have been found among subgroups of women, such as those treated for alcohol and drug problems, and women who have been incarcerated (28). Alcohol-related risk factors include drinking during pregnancy, pattern of alcohol use, alcohol dependence, use of multiple substances, having had a previous alcohol-exposed pregnancy, and having a partner or family member who drinks heavily (29-31). Women who receive little or no prenatal care, are unemployed, are socially transient, have lost children to foster or adoptive care because of neglect, abuse, or abandonment are more likely to have high alcohol use patterns that could affect a pregnancy (22,32). National survey data indicate that, while the percentage of women who abstain from alcohol use during pregnancy has increased slightly in recent years, 13% of women continue to use alcohol during pregnancy (26). Among pregnant women, approximately three percent report binge drinking (i.e., five or more drinks on any one occasion) or frequent drinking (i.e., seven or more drinks per week or five or more drinks on any one occasion) (1,33-34). Clearly, current prevalence rates of affected individuals and alcohol-exposed pregnancies indicate that the magnitude of the problem of FAS is a significant public health concern. However, because of the challenges of establishing accurate and timely prevalence information, the magnitude could be even greater than current data indicate.

Challenges in determining accurate prevalence. Despite the progress made over the past several decades to accurately establish and monitor the prevalence of FAS, the full magnitude of the problem is still not known. Primary care providers and others who care for children do not routinely or consistently identify individuals with FAS, which hinders efforts to account for these children in routine birth defects and developmental disabilities monitoring programs. Studies using multiple data sources (e.g., birth certificates, clinical charts, and medical records) show wide variations in identification of FAS cases depending on the population being surveyed (15,34). Four major factors lead to widespread failure to recognize FAS in primary pediatric care settings resulting in underestimates of the prevalence and impact of FAS (36-38):

- **No specific and uniformly accepted diagnostic criteria have been available.** The four broad areas of clinical features that constitute the diagnosis of FAS have remained essentially the same since first described in 1973: selected facial malformations, growth retardation, Central Nervous System (CNS) abnormalities, and maternal alcohol consumption during pregnancy. These four areas were reaffirmed in a 1996 report by the Institute of Medicine (IOM; 39-40). However, these broad areas of diagnostic criteria are not sufficiently specific to ensure diagnostic accuracy, consistency, and reliability . For example, clinicians do not have guidance about how many facial features must be present or the timing and severity of growth retardation needed to constitute

FAS diagnostic criteria. Thus, health providers are hampered in their efforts to screen and identify children with FAS.

- **FAS diagnosis is based on clinical examination of features, but not all children with FAS look or act the same.** Because each of the symptoms has a broad range of differential diagnoses, it is easy for a clinician to miss or misdiagnose FAS. Previous guidelines, including those included in the 1996 IOM report, did not account for children of different racial and ethnic groups or individuals of different ages . In addition, symptoms such as growth impairment, cognitive impairment, and learning disabilities can have a range of causes. Some of these causes or disorders have higher visibility and recognition than FAS, leading to misdiagnosis (or at least failure to include FAS in the total diagnosis). For instance, physicians are aware of the high prevalence of Attention Deficit, Hyperactivity Disorders (ADHD), but might not link attention problems to FAS. Without clear diagnostic criteria and instruction on their use, providers will continue to under-identify and under-diagnose FAS.

- **Lack of knowledge and misconceptions among primary care providers.** Many professionals believe that FAS can only occur if the mother is an alcoholic. Few know about the full range or progressive nature of the neurobehavioral symptoms that result from prenatal exposure to alcohol. Some incorrectly believe that FAS only occurs among low-income families or in Native American or other racial and ethnic minority groups (21). Better information on the impact of FAS among all populations and dissemination of race or ethnic variations in the diagnostic criteria can help clinicians understand the risk of prenatal alcohol exposure across populations. Knowledge about subpopulation variations in facial characteristics as well as growth curves for infants by gestational age also are important considerations (41).

- **Lack of diagnostic criteria to distinguish FAS from other alcohol-related conditions.** Creating and using diagnostic guidelines for FAS is a starting point for better defining the continuum of conditions related to prenatal alcohol exposure (40). FAS is a severe outcome of prenatal alcohol exposure. Other outcomes also occur and can result in major deficits. Terms such as Fetal Alcohol Effect (FAE), Alcohol-related Birth Defect (ARBD), and Alcohol-Related Neurodevelopmental Disorder (ARND) have been used to describe a spectrum of conditions related to prenatal alcohol exposure.

These four challenges indicate that what is urgently needed to advance the field of FAS diagnosis are current diagnostic guidelines based on empirical evidence as well as clinical experience. Such guidelines should be based on up-to-date scientific evidence and current clinical practices. Further, such guidelines would allow public health and service professionals to better determine the impact of FAS, and deliver needed services to affected children.

Congressional mandate. As part of the fiscal year 2002 appropriations funding legislation, Congress mandated that CDC, acting through the National Center on Birth Defects and Developmental Disabilities (NCBDDD) and in coordination with the National Task Force on Fetal Alcohol Syndrome and Fetal Alcohol Effect (NTFFAS/FAE), other federally funded FAS programs, and appropriate nongovernmental organizations, would:

- Develop guidelines for the diagnosis of FAS and other negative birth outcomes resulting from prenatal exposure to alcohol;

- Incorporate these guidelines into curricula for medical and allied health students and practitioners, and seek to have them fully recognized by professional organizations and accrediting boards; and
- Disseminate curricula to and provide training for medical and allied health students and practitioners regarding these guidelines.

Limit in scope. The mandate to CDC indicates that, in addition to guidelines for FAS, guidelines should be developed for other negative birth outcomes resulting from prenatal exposure to alcohol. However, it was subsequently determined through discussions with all interested stakeholders that the best course was to first develop guidelines for the full FAS diagnosis (see following). Then, in subsequent efforts, these guidelines could be expanded or refined to include other alcohol-related disorders. This approach was determined to provide the most timely and scientifically grounded guidelines at this time. However, this decision does not curtail ongoing efforts to define conditions beyond FAS or develop diagnostic guidelines for those conditions.

Note on terminology. Many terms are used to describe the continuum of effects that result from prenatal exposure to alcohol, including: Fetal Alcohol Effect, Alcohol-related Birth Defects, and Alcohol-Related Neurodevelopmental Disorder. A more recent term that has been introduced is Fetal Alcohol Spectrum Disorders (FASD). In April 2004, several federal agencies [(National Institutes of Health (NIH), CDC, & Substance Abuse and Mental Health Services Administration (SAMHSA)] along with experts in the field were convened at a summit sponsored by the National Organization on FAS (NOFAS) to develop a consensus definition of FASD. The resulting definition, adopted in these guidelines, is:

> *Fetal Alcohol Spectrum Disorders (FASD) is an umbrella term describing the range of effects that can occur in an individual whose mother drank alcohol during pregnancy. These effects may include physical, mental, behavioral, and/or learning disabilities with possible lifelong implications. The term FASD is not intended for use as a clinical diagnosis.*

APPROACH AND METHODS FOR DEVELOPMENT OF GUIDELINES

To meet its Congressional mandate, CDC convened an internal working group, led by staff from the FAS Prevention Team of NCBDDD, to conduct preplanning meetings to determine the best methods for development of all aspects of the guidelines: (a) general framework for referral and diagnosis, (b) development of guidelines for physical features (dysmorphia and growth) as well as exposure, and (c) development of guidance to clinicians concerning potential CNS abnormalities. Each of these aspects involved review of the literature, as well as discussions with consultants, clinicians, researchers, and parents of affected children. A description of general and specific methods of development of each aspect follows.

General review of literature. CDC staff identified various reports and documents to be used as the scientific basis for diagnostic guidelines. The science base for this work included, but was not limited to:

- Published scientific, peer-reviewed, literature on physical and neurodevelopmental effects of prenatal exposure to alcohol;
- The report of the IOM Committee to study FAS (40);
- Results from the work of the NTFFAS/FAE (42) ;
- Criteria from standard, widely used dysmorphology and neurodevelopmental textbooks or guides (40,43-44);
- Research on measuring the FAS facial phenotype (45-47);
- Reports on systems that operationally interpret the 1996 IOM criteria(48-50);
- Experience in developing a surveillance case definition for the Fetal Alcohol Syndrome Surveillance Network (FASSNET) (51);
- Ongoing state surveillance and research data, particularly work of Alaska (19,35,52), Colorado (53), New Jersey, Nevada, Washington, and the Four State FAS Consortium (consisting of North Dakota, Montana, Minnesota, and South Dakota);
- The American Academy of Pediatrics (AAP) August 2000 statements and recommendations on FAS and other effects related to maternal alcohol use (54); Position of the American Academy of Family Physicians, which refers to the AAP statement; and
- Canadian National Committee's efforts concerning standardization of guidelines for screening, diagnosis, and surveillance of FAS.

Framework. Before developing the specifics of the diagnostic and referral guidelines, CDC staff determined that it would be helpful to conceptualize the entire diagnostic and referral process encountered by affected individuals and their families. Such a framework also would assist professionals in understanding their specific role in the referral and diagnostic process, as well as guide them with next steps for each case. The framework was reviewed by health care policy consultants as well as members of the NTFFAS/FAE. Revisions were made based on their suggestions. This framework is presented in detail later in these guidelines.

Creation of the Scientific Working Group. The first step in development of the guidelines was to convene an internal CDC working group. The group consisted of members of the NCBDDD FAS prevention team, geneticists, developmental pediatricians, epidemiologists, and psychologists, as well as other allied health professionals. This internal working group developed a list of potential external experts who could be convened to develop the actual guidelines. The large external panel of experts was designated as a scientific advisory panel. From this panel, a subset of experts formed a Scientific Working Group (SWG) that delineated the specifics of the diagnostic criteria.

The external SWG convened by CDC included researchers, clinicians in general and specialty medicine, representatives from academic centers and state health agencies, as well as consumer representatives from the National Organization on FAS (NOFAS) and The Arc of the United States. The scientific advisory panel met in Atlanta, Georgia, on July 12, 2002, to begin deliberations on the proposed guidelines. At that meeting, four subgroups were created: FAS Referral and Diagnosis; ARND issues; Essential Services for Children with FAS/ARND; and Identifying and Intervening with Women at Risk for an Alcohol-Exposed Pregnancy. The subgroups met and began deliberations related to the guidelines in their respective topic areas.

A subsequent meeting of the SWG occurred on September 20, 2002, in conjunction with an NTFFAS/FAE meeting, also in Atlanta. This offered the opportunity for information sharing and feedback on progress made thus far from a range of stakeholders represented on the NTFFAS/FAE

(e.g., parents, providers, and researchers). The FAS Referral and Diagnostic subgroup and the Essential Services subgroup met to further deliberate on their recommendations. The recommendations from these two subgroups were then presented to the NTFFAS/FAE for review and input. The component of the criteria for FAS screening and diagnosis that presented the most difficulty to the FAS Referral and Diagnosis subgroup was the central nervous system (CNS)/neurobehavioral component. This group felt that members of the ARND subgroup were most qualified to develop that particular component of the criteria.

The ARND subgroup drew important information from a poll of the experts that was conducted to identify the CNS/neurobehavioral domains most affected by prenatal alcohol exposure (further description follows). Results of this poll were incorporated into the FAS Referral and Diagnosis Guidelines within the CNS/neurobehavioral component. The third meeting of the FAS Referral and Diagnosis subgroup was teleconferenced on March 11, 2003, and draft guidelines were reviewed and revised. At that time and based on review of the available scientific evidence concerning diagnosis of ARND, the scope of the guidelines was limited to FAS, with future efforts to be devoted to other prenatal alcohol-related disorders. This revised version of the diagnostic criteria was presented to and approved by the NTFFAS/FAE on March 13, 2003 with recommendations. Consensus among members of the external SWG and the NTFFAS/FAE was used to finalize each criterion of the guidelines for dysmorphology, growth, and prenatal exposure to alcohol. At the December 8 and 9, 2003, meeting of the NTFFAS/FAE, further discussions were held regarding the CNS/neurobehavioral criteria and subsequent revisions were made. Finalization of the FAS criteria was reached during a teleconference of the NTFFAS/FAE on May 13, 2004.

Highlights of deliberations of SWG: Physical criteria. The SWG concluded that a strict definition of FAS should be established first, not including diagnostic terms such as fetal alcohol effect (FAE), alcohol-related neurodevelopment disorder (ARND), and alcohol-related birth defect (ARBD) at this time because of insufficient scientific evidence on which to base diagnostic criteria for these related conditions. The SWG urged that CDC diagnostic guidelines for FAS use objective, quantitative measures to improve accuracy and reproducibility and apply specific case definitions guided by evidence-based knowledge and new technologies. The SWG also recommended use of racial and ethnic norms for facial features, head circumference (occipitofrontal circumference, OFC), and other key features when available.

During its deliberations, the SWG acknowledged the need to keep recommendations for the diagnosis of FAS relevant to practitioners working in clinical settings. Also, the SWG acknowledged differences between screening, diagnosis, surveillance, and research activities and the separate definitional needs of each of these activities. Effective tools and practical strategies for primary care settings were considered. The SWG encouraged CDC to call for action and collaboration among obstetricians, pediatricians, family practice physicians, and others providing primary care and screening services to children. The SWG also discussed the need for more data regarding the range of essential services for children who are diagnosed positive for FAS and stressed the importance of a multidisciplinary plan for care that links to the child's community healthcare provider or medical home.

Central nervous system abnormality criteria. Because the scientific evidence and professional consensus on CNS criteria are not yet at the level of specificity equal to that available for physical features, a different approach was used in developing CNS criteria. Further, it was decided that rather than

creating strict criteria for specific CNS domains and level of severity needed, more general guidelines should be described. Such general guidelines should assist the clinician in identifying areas of deficit most likely to be found with individuals who have FAS, as well as individuals with FAS who have less common types of deficits. This approach was considered optimal because a number of structures of the brain are affected versus a single, isolated structure. This generalized nature of damage from prenatal alcohol exposure can result in a wide array of neurodevelopmental outcomes.

To develop such neurodevelopmental guidelines for referral and diagnosis for FAS, the ARND/CNS subgroup polled clinicians and researchers who have extensive knowledge and experience with individuals who have FAS or other related diagnoses. These experts were individuals who specialize in neurobehavioral issues, have extensive research and clinical experience making the FAS/ARND diagnosis, and have contact with families and children with FAS. They were queried to find out what behavioral domains they encountered most frequently or were most essential for making an FAS/ARND diagnosis. Twenty-two clinicians were contacted and their responses were synthesized. The clinicians were asked to identify five areas of deficit they considered most important for diagnosis of FAS or related disorders. In addition, the clinicians were also asked to identify three to five specific behaviors that could be used as examples of each of the five areas of deficit.

The resulting guideline of neurodevelopmental features associated with FAS are presented as being as inclusive as possible, while understanding that certain areas of neurodevelopmental functions are more vulnerable to prenatal exposure to alcohol. Each domain is presented so as to include exemplars from direct observation or parent report that can be documented through standardized testing.

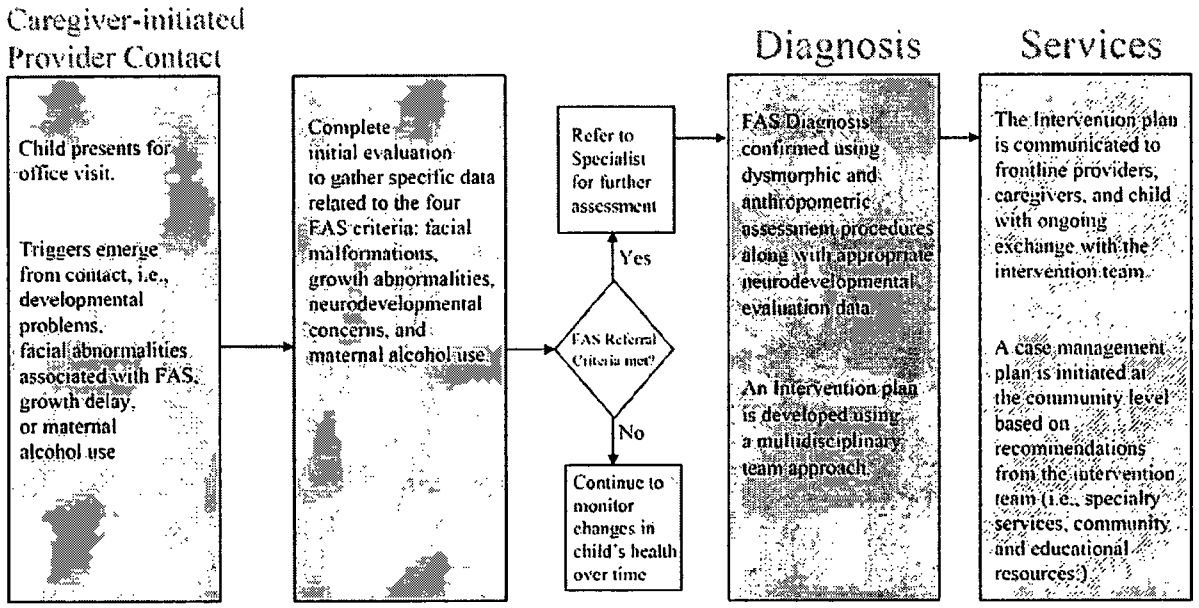
Medical diagnostic criteria are generally evaluated in two ways: (1) the criteria must be reliable and; (2) the criteria must be as valid as possible. The criteria that appear in these diagnostic guidelines meet both of these requirements. In developing these diagnostic guidelines, the SWG also considered the feasibility of applying the criteria in primary care practice and related settings where children are seen.

DIAGNOSTIC AND REFERRAL FRAMEWORK

The framework in Figure I was developed to help guide the discussions of the SWG as they deliberated on the guidelines for referral and diagnosis of FAS. The framework was developed to provide an overview of the entire identification, referral, diagnosis, and treatment process. This overview guided the SWG in identifying key points that needed to be addressed to develop specific guidelines. The framework reflects CDC's recommendation that developmental screening be implemented to improve children's health and help them reach their full potential. A discussion of the major points of the framework follows.

Initial identification. Initial recognition that a child or older individual has a potential problem can come from many sources. Often, parents notice differences between a child and his or her siblings. School systems, including Head Start and daycare staff, interact with a large number of children and often recognize when someone is having difficulty. Social service professionals, such as WIC clinic staff, social workers, and foster care agencies frequently recognize children and individuals having difficulty and needing evaluation. And finally, healthcare providers (particularly pediatricians) often are the first to screen for and detect problems; or obstetricians, who might be aware of

FIGURE 1. Framework for FAS Diagnosis and Services



a maternal substance abuse problem, might refer a newborn. Recognition of many of the problems associated with FAS is exactly the type of condition the "well child" visits to the doctor's office are meant to identify. It is assumed that triggers, such as facial abnormalities, growth delay, developmental problems, or maternal alcohol use, will emerge from the contact. Recognition of a potential problem should lead the provider, regardless of specific profession, to facilitate getting the person and his or her family to the appropriate next step.

Referral. The referral process is initiated at the point a clinician starts to have suspicions of an alcohol-related disorder for a child. This process is facilitated by thorough knowledge of the physical and neurodevelopmental domains affected in individuals with FAS, as well as characteristics that could trigger a referral. Examples of triggers are presented later, in the Referral section of these guidelines. In making a referral for a complete diagnostic evaluation for FAS, it is helpful for the referring provider to gather and document specific data related to the FAS criteria. These data will assist the provider in making the decision to diagnose the child or to refer the child to a multidisciplinary evaluation team for a confirmed diagnosis. In addition, these data could be forwarded to the multidisciplinary evaluation team to guide the diagnostic process. A complete review of systems, noting features consistent with FAS, would be most productive.

Diagnosis. At this stage, the child would be presented to a multidisciplinary team who would engage in a more thorough assessment of the child using FAS diagnostic procedures to evaluate dysmorphia and growth parameters, as well as obtain appropriate neurodevelopmental evaluation data. Once a diagnosis is made, an intervention plan would be developed using a multidisciplinary team approach. A variety of specialists could contribute to the multidisciplinary team, including dysmorphologists, developmental pediatricians, psychiatrists, psychologists, social workers, and educational specialists. Other clinicians, such as pediatricians and family practitioners, also might make the FAS diagnosis, with appropriate training in use of these guidelines. In many rural and less populated regions, these clinicians must make the diagnosis for many types of birth defects and developmental

disabilities. Many of these evaluation services are available within the community setting, for example school systems could provide neurocognitive assessments.

DIAGNOSTIC CRITERIA

Dysmorphia. Human congenital malformations are referred to as dysmorphic features or dysmorphia (55). Dysmorphia occurs when normal morphogenesis is interrupted, creating a particular feature which is shaped, sized, or positioned outside the normal range of development. Alcohol is a teratogen that results in dysmorphia through interference with nerve cell development and functioning, alterations in the ability of cells to grow and survive, increased formation of cell-damaging free radicals, altered pathways of biochemical signals within cells, and altered expression of certain genes and genetic information. In short, alcohol has been shown to interfere with fetal nerve cell development and function in a variety of ways (56-57).

In first describing the dysmorphic features of FAS, Jones and colleagues focused on short palpebral fissure, maxillary hypoplasia (with prognathism), and the presence of epicanthal folds that were observed for a majority of the children described. However, other features also were noted for some patients, including altered palmar fexional crease patterns (i.e., hockeystick crease), cardiac anomalies, joint disability, overlapping fingers, ear anomalies, hemangiomas, ptosis, hypoplastic nails, and pectus deformities (4,5). Over the next 30 years, additional features described included: microcephaly, short nose, smooth philtrum with thin vermillion border, cleft lip, micrognathia, protruding auricles, short or webbed neck, vertebra and rib anomalies, short metacarpal bones, menigomyelocele, hydrocephalus, and hypoplastic labia majora (43).

Despite the heterogeneity of expression for dysmorphic features related to prenatal exposure to alcohol, core facial dysmorphia have emerged through human and animal studies. Experimental studies with a mouse model and primates indicate that the facial dysmorphia observed for individuals with FAS are the result of disturbances of cellular migration during organogenesis along the midline of the face (58). Using anthropomorphic measurements of all facial features, clinical researchers have confirmed the midline feature abnormalities (59). Studies of clinic-referred samples also support these features as discriminant for FAS (60-61). Based on these scientific findings and the extensive clinical experience of the SWG, the following facial dysmorphic features were determined to meet the dysmorphia criteria essential for FAS (based on racial norms):

- Smooth philtrum (measured as 4 or 5 on Lip-Philtrum Guide*)
- Thin vermillion border (measured as 4 or 5 on Lip-Philtrum Guide) (i.e., upper lip)
- Small palpebral fissures (measured as ≤10th percentile according to age and racial norms)

The individual must exhibit all three characteristic facial features; however, additional features also can be present. For example, maxillary hypoplasia is often noted for individuals with FAS as well as those associated features described previously. Cross-sectional and longitudinal studies indicate that many features can change with age or development. After puberty, the characteristic facial features associated with FAS can become more difficult to detect (62). However, recent findings indicate that these three key features remain for the majority of individuals with FAS (47,50).

* University of Washington Lip-Philtrum Guide (49-50).

Use of these three cardinal features (smooth philtrum, thin vermillion border, and small palpebral fissures) to assess whether an individual's dysmorphia is consistent with FAS, is compatible with the IOM report and other diagnostic systems currently in use. Specific criteria were chosen by the SWG to maximize inclusiveness of potential cases on this diagnostic parameter and, therefore, might differ somewhat from other systems currently in use. For example, the 1999 version of University of Washington 4-digit code system uses the same philtrum and vermillion border criteria (as noted by reference to its Lip-Philtrum Guide), but uses a cutoff of the 3rd percentile (2 or more standard deviations below the norm) for palpebral fissures , which is a more conservative cutoff (50). This more conservative approach results in fewer individuals meeting the dysmorphia criteria for an FAS diagnosis but also reduces potential false positives for the diagnosis. Other ways to assess dysmorphia for the FAS diagnosis include checklists or weighted checklists (63-65). Of those checklists reviewed by the SWG, all designated the philtrum, vermillion border, and palpebral fissures as the cardinal facial features of FAS (either by higher weighting or explicit notation). However, because of the cumulative nature of some such lists, an individual who has several of the associated features but not the cardinal features could still be given the FAS diagnosis. Thus, the checklists tend to be more inclusive than the current guidelines, with greater potential for false-positive diagnoses. Review of available diagnostic systems seems to indicate that the dysmorphic criteria agreed upon by the SWG provide a balance between conservative and overly inclusive diagnostic systems.

Differential diagnosis of dysmorphia. Individual dysmorphic features are not unique to any particular syndrome. Even rare defects or certain clusters of dysmorphic features can appear in a variety of syndromes. Therefore, a process of differential diagnosis is essential in making an accurate FAS diagnosis. Following, in Table 1 is a list of syndromes with dysmorphic features that overlap with the primary features of the FAS diagnosis (43). As can be seen from Table 1, none of the syndromes with single overlapping features (except for Toluene embryopathy) have the full constellation of small palpebral fissures, thin vermillion border, and smooth philtrum. However, there are some syndromes in which the constellation of features (primary, occasional features, or both) give a "gestalt" that is similar to the "gestalt" of FAS. These syndromes should be considered in particular when completing the differential diagnosis. Table 2 lists these syndromes, along with the overlapping and differentiating features.

Growth problems. Growth retardation, variably defined, has been documented consistently in individuals with FAS. However, these observations used a variety of parameters (e.g., height, weight, and head circumference), severity levels (below 25th percentile, below 10th percentile, or below 3rd or 2nd percentile), and timing of growth problems (current, birth or present at any point during life). The SWG reviewed available literature, clinical expertise, and practical issues to arrive at benchmarks for each of these three aspects of growth abnormalities.

The primary parameters of growth that need to be impaired to meet the growth retardation criteria of FAS are height, weight, head circumference, or a combination thereof. Anecdotally, a small number of children with FAS have been found to have disproportionate height in relation to weight. However, because multiple organic factors can lead to growth deficiencies (e.g.,. brain structure abnormalities leading to poor skeletal growth or disruption of endocrine function leading to poor weight gain), and because most children with FAS are symmetrical for height and weight (66), it was determined that deficiencies in *either* height or weight, but not height for weight, should be included as growth parameters that might be affected by FAS. Thus, children with growth retarda-

Table 1. Differential diagnosis of individual features associated with FAS

Feature	Syndromes
Smooth philtrum	Cornelia de Lange syndrome
	Floating-Harbor syndrome
	Geleophysic dysplasia
	Opitz syndrome
	Toluene embryopathy
Thin Vermillion border	Miller-Dieker (Lissencephaly) syndrome
	Fetal Valproate syndrome
	Geleophysic dysplasia
	Cornelia de Lange syndrome
	Toluene embryopathy
Small palpebral fissures	Campomelic dysplasia
	DiGeorge sequence
	Dubowitz syndrome
	Duplication 10q sequence
	Duplication 15q sequence
	FG syndrome
	Maternal phenylketonuria (PKU) fetal effects
	Oculodentodigital syndrome
	Opitz syndrome
	Trisomy 18 syndrome
	Williams syndrome
	Velocardiofacial syndrome
	Toluene embryopathy

Note: Features that discriminate these disorders from FAS can be found in Jones, 1997.

tion in height or weight would meet the growth retardation criteria for the FAS diagnosis. Consistent with poor brain development, it was determined that head circumference should be included as a CNS parameter rather than a growth parameter (67-68).

Severity of growth retardation has been defined for each parameter as at or below the 10th percentile or at or below the 3rd percentile by a majority of FAS studies. The primary issue for severity is inclusion or exclusion of children for the FAS diagnosis. Use of the 10th percentile would result in more false-positive FAS diagnoses; while use of the 3rd percentile would result in a greater number of false-negatives. For public health reasons of capturing the largest number of children who might need services, the 10th percentile was chosen by the SWG. As noted previously in the dysmorphia section, use of the 10th percentile strikes a balance among criteria used in other diagnostic systems. Again, the 1999 edition of the University of Washington 4-digit code takes a conservative approach, using the 3rd percentile as the cutoff. Checklist systems often do not specify a particular level of growth retardation and some do not specify which growth parameters should be considered (i.e., height, weight, or height relative to weight). This lack of specificity could lead to inconsistency in diagnostic method, which in turn, could lead to inconsistent application of the FAS diagnosis.

Table 2. Differential diagnosis of syndromes similar to FAS

Syndrome	Overlapping Features	Differentiating Features
Aarskog syndrome	Small nose with anteverted nares, broad philtrum, maxillary hypoplasia, and wide-spaced eyes	Rounded face, down-slant to palpebral fissures, widow's peak, crease below lower lip, incomplete out folding of upper helices, and dental eruption problems.
Williams syndrome	Short palpebral fissures, anteverted nares, long philtrum, depressed nasal bridge, and epicanthal folds	Wide mouth with full lips, stellate pattern of the iris, periorbital fullness, and connective tissue disorders.
Noonan's syndrome	Low nasal bridge, wide-spaced eyes, and epicanthal folds	Down-slant to palpebral fissures, keratoconus, wide mouth, and protruding upper lip
Dubowitz syndrome	Short palpebral fissures, wide spaced eyes, and epicanthal folds	Shallow supraorbital ridge with nasal bridge near the level of the forehead, and broad nasal tip
Brachmann-DeLange syndrome	Long philtrum, thin vermillion border, anteverted nares, and depressed nasal bridge	Single, bushy eyebrow extending across forehead, long eyelashes, downturned mouth, high arched palate, and short limbs (yielding short stature)
Toluene embryopathy	Short palpebral fissures, midface hypoplasia, smooth philtrum, and thin vermillion border	Micrognathia, large anterior fontanel, down-turned mouth corners, hair patterning abnormalities, bifrontal narrowing, and ear abnormalities
Fetal hydantoin syndrome (Fetal dilantin syndrome)	Wide-spaced eyes and depressed nasal bridge	Short nose with bowed upper lip
Fetal valproate syndrome	Epicanthal folds, anteverted nares, long philtrum with thin vermilion border, and wide-spaced eyes	High forehead, infraorbital crease or groove, and small mouth
Maternal PKU fetal effects	Epicanthal folds, short palpebral fissures, long underdeveloped philtrum, and thin vermillion border	Small upturned nose, round facies, and prominent glabella

The current guidelines provide consistent growth parameters that should be assessed, as well as a more inclusive level of the 10th percentile.

The primary issue that emerged in the discussion of timing of growth retardation was whether growth retardation needs to be present at the time of the diagnosis, or whether it could have occurred previously and been resolved. This is particularly important when including prenatal growth retardation or early growth problems due to failure to thrive. Because a great number of treatments exist for growth problems (e.g. feeding tubes or hormone therapy), the SWG adopted the criteria that any history of growth retardation, including prenatal growth deficiencies, should be allowed within the diagnostic criteria (61).

Thus, the growth retardation criteria adopted by the SWG are: confirmed prenatal or postnatal height, weight, or both at or below the 10th percentile documented at any one point in time (adjusted for age, sex, gestational age, and race or ethnicity). The committee noted that the examiner should make sure that the single point in time when the growth deficit was present does not correlate with a point in time when the child was nutritionally deprived.

Differential diagnosis of growth problems. Growth retardation and growth deficiencies occur in children, adolescents, and adults for a great many reasons. Some of the most obvious reasons have to do with insufficient nutrition. This could be a particular problem for infants with poor sucking responses who experience failure to thrive. In addition, several genetic disorders result in specific growth deficiencies (e.g., dwarfism). Prenatal growth retardation can be due to a variety of factors, including maternal smoking or other behaviors leading to hypoxia, poor maternal nutrition, or genetic disorders. Both environmental and genetic bases for growth retardation should be considered for differential diagnosis when considering the FAS diagnosis.

Central nervous system abnormalities (CNS). More than 2,000 scientific papers regarding the teratogenic effects of alcohol exposure for CNS have been published over the past 30 years (69-71). Studies of the impact of fetal exposure to alcohol show a range of short- and long-term cognitive and behavioral outcomes resulting from these CNS abnormalities. Complicating detection of these abnormalities is that FAS neurobehavioral presentation changes across the lifespan (4,61,72-79). Despite this developmental process, CNS deficits generally persist throughout the lifespan (80). Longitudinal studies have found that many adults affected by FAS have complex mental health disorders, are affected by the consequences of neurobehavioral deficits, and are unable to sustain independent living (81).Prenatal exposure to alcohol can result in an array of structural, functional, neurological problems, or a combination of these, as well as abnormalities of the CNS (40). To meet the FAS diagnostic criteria for CNS abnormality, structural, neurological, or functional deficits, or a combination thereof, must be documented. Note that it is also possible for an individual to present with more than one CNS structural, neurological, functional deficit or abnormality. Guidelines for each type of CNS abnormality follow.

I. Structural

1. Documented small or diminished overall head circumference (OFC at or below the10th percentile) adjusted for age and gender (including head circumference at birth; 4,68). For children who have overall growth deficiency (i.e., height and weight below the 10th percentile) to meet this criteria for CNS abnormality, the child's head circumference should be disproportionately small to his or her overall size (i.e., OFC at or below the 3rd percentile).

2. Clinically significant brain abnormalities observable through imaging techniques (e.g. reduction in size, or change in shape of the corpus callosum, cerebellum, or basal ganglia) as assessed by an appropriately trained professional (4,58,82-88).

   Note: An individual could meet the CNS abnormality criteria for the FAS diagnosis through a structural abnormality, yet not demonstrate detectable functional deficits.

II. Neurological

Documented evidence of neurological damage to the CNS. Neurological problems of CNS can include seizures not due to a postnatal insult or fever or other soft neurological signs outside normal limits (e.g., in coordination, visual motor difficulties, nystagmus, or difficulty with motor control; 89-92). As with head circumference, abnormal neurological findings can be most predictive of underlying CNS abnormality due to prenatal alcohol exposure, rather than later environmental factors, in younger children. The use of norm-referenced measures of neurological functioning is recommended.

III. Functional

Assessment findings that indicate deficits, problems, or abnormalities in functional skills of the CNS. Early brain damage is usually generalized rather than specific, with increased specificity of abnormalities revealed as development progresses. The functional abilities affected by prenatal exposure to alcohol vary greatly from person to person, depending on the amount of alcohol exposure, timing of exposure, and pattern of exposure (e.g., chronic exposure versus binge episodes). Despite this inherent variation in effects, several areas of significant functional vulnerability have been observed consistently by clinicians and clinical researchers with particular damage to corresponding structures reported (e.g., corpus callosum, cerebellum, or basal ganglia). Variability in exposure impact results in variability of structural, neurological, or functional deficits, or a combination thereof in affected individuals.

For functional deficits, it is generally accepted that multiple locations in the brain (and corresponding functional capability) are affected by prenatal exposure to alcohol. To address this issue, functional deficits that fulfill the CNS abnormality criteria can be met in two ways:

(1) Global cognitive deficit (e.g., decreased IQ)
or significant developmental delay in children too young for an IQ assessment;

OR

(2) Deficits in THREE or more specific functional domains

These two ways of meeting the criteria for a functional CNS abnormality were adopted because of the composite nature of cognitive/intellectual and developmental measures (93-94). Decreased performance on a standardized measure of cognition/intelligence or development assumes deficits in multiple domains. In the absence of such a measure, several specific domains need to be assessed individually to determine that multiple functional domains have been affected. The specific domains most often cited as areas of deficit or concern for individuals with FAS are described below, although other domains and abilities can be affected and this list is not exhaustive. It should

be noted that for each of the following specific domains described, other agents and environmental factors can produce deficits or outcomes similar to prenatal alcohol exposure, making care differential diagnosis essential. Finally, these descriptions are intended to be suggestive and examples of likely and possible problems a clinician might encounter then need to assess using psychometric instruments. The exemplars are not intended to be exhaustive, or to present a necessary list of behaviors to be used as a checklist without reliable and valid assessment.

a) **Cognitive deficits or significant developmental discrepancies.** It is important to note that global deficits or delays can leave the child scoring in the normal range of development, but below what would be expected for his or her environment and background (61,95-99). In addition to formal testing (either through records or current testing), behaviors that may be observed (or reported) in the clinical setting that suggest cognitive deficits or developmental delays that should be assessed by standardized testing include but are not limited to **specific learning disabilities (especially math and/or visual-spatial deficits); uneven profile of cognitive skills; poor academic achievement; discrepancy between verbal and nonverbal skills; and slowed movements or reaction to people and stimuli (e.g., poor information processing).** (75,100-103)

b) **Executive functioning deficits.** Executive functioning (EF) is defined as the ability to maintain an appropriate problem solving set for attainment of a future goal and that this ability includes the more specific skills of inhibition, planning, and mental representation (104). Behaviors that can be observed (or reported) in the clinical setting that might indicate an EF deficit that should be assessed by standardized testing include, but are not limited to **poor organization, planning, or strategy use; concrete thinking; lack of inhibition; difficulty grasping cause and effect; inability to delay gratification; difficulty following multistep directions; difficulty changing strategies or thinking of things in a different way (i.e., perseveration); poor judgment; and inability to apply knowledge to new situations.** (105-108)

c) **Motor functioning delays or deficits.** Both gross motor and fine motor skills can be impaired for individuals with FAS (109-111). Visual-motor/visual-spatial coordination is a particularly vulnerable area of functioning (99,112-113). Behaviors that can be seen (or reported) in the clinical setting that indicate motor problems that should be assessed by standardized testing include, but are not limited to **delayed motor milestones; difficulty with writing or drawing; clumsiness; balance problems; tremors; and poor dexterity. For infants, a poor suck is often observed.** (61,114-116)

d) **Attention and hyperactivity problems.** Attention problems are often noted for children with FAS, with many children receiving a diagnosis of attention-deficit hyperactivity disorder (AD HD; 117). Although such a diagnosis can be applied, some research has shown that the attention problems for children with FAS do not seem to fit the classic pattern of ADHD. Individuals with FAS tend to have difficulty with the encoding of information and flexibility (shifting) aspects of attention; whereas children with ADHD typically display problems with focus and sustaining attention (118-119). Individuals with FAS also can appear to display hyperactivity because their impulsivity may lead to increased activity levels. Behaviors that may be observed (or reported) in the clinical setting that suggest attention problems related to FAS that should be assessed by standardized testing include, but are not limited to **described by adult as "busy"; inattentive; easily distracted; difficulty calming down; overly active; difficulty completing tasks; and/or trouble with transitions. Parents might report inconsistency in attention from day to day (e.g., "on"**

days and "off" days). (79,120-125)

e) **Social skills problems.** The executive, attention, and developmental problems described previously often lead to clinically significant difficulty for people with FAS when interacting with peers and others. Because of the mental representation problems, individuals with FAS often have social perception or social communication problems that make it difficult for them to grasp the subtler aspects of human interactions (107,126-127). Consistent difficulty understanding the consequence of behavior or inappropriate behavior is frequently described for individuals with FAS (62,80). Behaviors that can be observed (or reported) in the clinical setting that indicate these types of social difficulties that should be assessed by standardized testing include, but are not limited to **lack of stranger fear; often scape-goated; naiveté and gullibility; easily taken advantage of; inappropriate choice of friends; preferring younger friends; immaturity; superficial interactions; adaptive skills significantly below cognitive potential; inappropriate sexual behaviors; difficulty understanding the perspective of others; poor social cognition; and clinically significant inappropriate initiations or interactions.** (128-130) It should be noted that standardized assessment of social problems can be quite difficult. Social functioning is a multifaceted domain that can require several areas of assessment.

f) **Other potential domains that can be affected.** In addition to these five most often cited problem areas, deficits and problems to be assessed by standardized testing can present in several other areas, including **sensory problems (e.g., tactile defensiveness and oral sensitivity); pragmatic language problems (e.g., difficulty reading facial expression; poor ability to understand the perspectives of others); memory deficits (e.g., forgetting well-learned material, and needing many trials to remember); and difficulty responding appropriately to common parenting practices (e.g., not understanding cause-and-effect discipline).** While abnormalities in these "other" areas have been reported for some individuals with FAS, expert consensus suggests deficits in these areas present at a lower frequency than do those in the other five specific domains (62).

THESE GUIDELINES STRONGLY RECOMMEND THAT FUNCTIONAL DOMAINS BE ASSESSED USING NORM-REFERENCED STANDARDIZED MEASURES. DOMAINS SHOULD BE ASSESSED BY APPROPRIATE PROFESSIONALS USING RELIABLE AND VALIDATED INSTRUMENTS.

Level of functional deficit. In these guidelines, global cognitive deficits and developmental delay, or deficits in three or more specific domains, are defined as performance substantially below that expected for an individual's age, schooling, or circumstances. Several statistical thresholds have been suggested to operationally define performance substantially below expected levels. Previous research indicates that approximately only one-quarter of individuals diagnosed with FAS perform at the most conservative level of below the 3rd percentile (2 standard deviations below the mean) on standardized measures (95). In keeping with this finding, and to adequately capture the full spectrum of effects, the SWG adopted two levels of functional deficits that would meet the criteria for a CNS abnormality: (1) for significant global cognitive deficit performance below the 3rd percentile (i.e., 2 standard deviations below the mean); and (2) for three or more specific domains performance below the 16th percentile (i.e., 1 standard deviation below the mean) on standardized measu res of individual domains. Thus, individuals scoring below the normal range on a global measure of IQ or development and individuals scoring in the below average range on standardized measures of three

specific functional domains would meet the criteria for functional CNS abnormality for diagnostic purposes.

Ideally, functional deficits should be established through appropriate standardized neuropsychological testing by a trained professional. The SWG recognized that such standardized testing might not be readily available in all diagnostic settings. Clinicians are strongly encouraged to supplement their observations by obtaining standardized testing through early intervention programs, public schools, and psychologists in private practice. The SWG emphasizes the need for psychometric testing when evaluating an individual for the FAS diagnosis, and use of clinical judgment alone could veer away from the goal of implementing standard diagnostic criteria for FAS. In addition, such testing will facilitate the development of individualized and appropriate treatment plans for diagnosed individuals. These guidelines are intended to provide information concerning the types of CNS abnormalities that might be observed, as well as the level of deficit that can be expected. In addition, they seek to support the need for quality assessments administered by trained professionals when establishing CNS abnormalities associated with the FAS diagnosis.

These guidelines for assessment of CNS abnormalities for making the FAS diagnosis is in harmony with, although not duplicative of, other diagnostic guidelines and systems. The 1999 version of the University of Washington 4-digit code and the guidelines developed by Health Canada requires performance below the 3rd percentile (2 standard deviations below the mean on standardized testing) in three separate domains in which global deficits count as one domain. Some health systems might find this approach useful in situations in which resources for standardized testing are readily available.

Mental health problems and lifelong consequence. Difficulty in any of the functional CNS areas described above can lead to maladaptive behavior and mental health problems with lifelong consequences. Commonly co-occurring mental health issues (excluding attention problems) reported by clinicians and cited in the scientific literature to date, include ***conduct disorders, oppositional defiant disorders, anxiety disorders, adjustment disorders, sleep disorders, and depression*** *(81,95,131-134).* Although attention problems can be classified as a mental health issue or psychiatric condition, in these guidelines they are treated as a primary deficit resulting from alcohol-related CNS damage, rather than a secondary mental health issue. There are considerable animal, human, and clinical studies that document attention deficits for many individuals with prenatal exposure to alcohol (117). In addition, decreased adaptive skills and increased problems with daily living abilities have been consistently documented, although further research is needed . Such problems include dependent living conditions, disrupted school experiences, poor employment records and encounters with law enforcement (including incarceration; 95). Although these mental health and very debilitating lifelong consequences should not be used for the purpose of diagnosis, it should be noted that they are very prevalent among individuals with FAS and are very likely to be the presenting conditions that should trigger a referral and comprehensive diagnostic evaluation.

Differential diagnosis of CNS abnormalities. Differential diagnosis of CNS abnormities involves not only ruling out other disorders but also specifying co-occurring disorders. The CNS deficits associated with FAS, in particular functional deficits, can be produced by many different factors in addition to prenatal alcohol exposure. It is important to determine that the observed functional deficits are not better explained by other causes. In addition to other organic syndromes that produce deficits in one or more of the previously cited domains (e.g., Williams syndrome and Down syn-

drome), significantly disrupted home environments or other external factors can produce functional deficits in multiple domains that overlap with the domains that are affected by FAS. In making the differential diagnosis of FAS by ruling out other syndromes, CNS abnormalities should be evaluated in conjunction with dysmorphia and laboratory findings. The more difficult differentiation is for CNS abnormalities resulting from environmental influences (e.g., abuse or neglect, disruptive homes, and lack of opportunities). ***To assist with differential diagnosis between FAS and environmental causes for CNS abnormalities it is important to obtain a complete and detailed history for the individual and his or her family.***

In addition to ruling out other causes for CNS abnormalities, a complete diagnosis should identify and specify other disorders that can co-exist with FAS (e.g., autism, conduct disorder, and oppositional defiant disorder). ***It is very important to note that a particular individual might have a conduct disorder in addition to FAS, but that not all persons with a conduct disorder have FAS and not all individuals with FAS will have a conduct disorder.*** Thus, organic causes, environmental contributions, and comorbidity should all be considered for both inclusive and exclusive purposes when evaluating someone for the FAS diagnosis (62,135). Finally, differential diagnosis for the CNS abnormalities within the FAS diagnosis is extremely difficult and should be conducted by professionals trained not only in the features of FAS, but also in the features of a broad array of birth defects and developmental disabilities so as to understand the distinguishing characteristics.

Maternal alcohol exposure. Documentation and confirmation of prenatal alcohol exposure can be extremely challenging. For birth mothers, admission of alcohol use during pregnancy can be very stigmatizing. The situation can be further complicated if the woman is still using alcohol, especially at high consumption rates. In this situation, information about alcohol use might need to be obtained from other reliable informants, such as a relative. However, the overwhelming situation encountered in the clinical setting is when a child or adult is being evaluated for FAS and little or no information about the index pregnancy is available. This frequently occurs for children in foster and adoptive homes. In this situation, every effort should be made to obtain the necessary information, but lack of confirmation of alcohol use during pregnancy should not preclude an FAS diagnosis if all other criteria are present. This would be considered "unknown prenatal alcohol exposure". In very rare instances, there will be confirmed absence of exposure. Documentation that the birth mother did not drink any amount of alcohol from conception through birth would indicate that the FAS diagnosis is not appropriate. This typically implies that the birth mother knew the date of conception (e.g., a planned pregnancy) and did not consume alcohol from that day forward, or she was prevented from drinking for some reason (e.g., incarceration). It must be noted that simple denials of alcohol use might or might not be credible and corroborating evidence should be obtained whenever possible. However, given the imprecise nature of exposure information, the following two qualifiers for prenatal alcohol exposure were suggested by the SWG:

I. **Confirmed prenatal alcohol exposure** requires documentation of the alcohol consumption patterns of the birth mother during the index pregnancy based on clinical observation; self-report; reports of heavy alcohol use during pregnancy by a reliable informant; medical records documenting positive blood alcohol levels, or alcohol treatment; or other social, legal, or medical problems related to drinking during the index pregnancy.

II. **Unknown prenatal alcohol exposure** indicates that there is neither a confirmed presence nor a confirmed absence of exposure. Examples include: the child is adopted and prenatal exposure(s) is unknown; the birth mother is an alcoholic, but confirmed evidence of exposure during pregnancy does not exist; and conflicting reports about exposure exist that cannot be reliably resolved.

CRITERIA FOR FAS DIAGNOSIS

A diagnosis of FAS requires the presence of all three of the following findings:

1. Documentation of all three facial abnormalities (smooth philtrum, thin vermillion border, and small palpebral fissures);

2. Documentation of growth deficits; and

3. Documentation of CNS abnormalities (structural, neurological or functional, or combination thereof).

It should be noted that confirmed prenatal alcohol use can strengthen the evidence for diagnosis, but is not necessary in the presence of all the previous findings. Confirmed absence of alcohol exposure would rule out the FAS diagnosis. The FAS diagnosis should be made only after ruling out other possible diagnoses associated with each criterion. A brief outline of the diagnostic criteria for FAS is presented in Table 3.

Changes in presentation of criteria across development. As would be expected for any congenital syndrome, presentation of the diagnostic features can change over development. With regards to facial features (small palpebral fissures, smooth philtrum, and thin vermillion border), it has generally been accepted that these features are most evident during infancy and the early preschool years. However, longitudinal data that specifically address this issue are not available currently. One also might expect growth parameters to change (and generally normalize) with development. This might especially be the case for children who receive nutritional or other interventions. Perhaps the criterion for which the most change over development is observed is CNS abnormalities. While structural abnormalities would remain consistent, neurological and functional deficits could resolve or change presentation at various stages of development. During the newborn and infancy periods, difficulty with arousal or behavior regulation might be observed. Slightly older infants could display delayed or abnormal motor skills. During late infancy approaching the preschool period, general cognitive developmental delay is generally reflected through delayed milestones, especially early language acquisition (note, however, that basic language skills of vocabulary and syntax generally are not areas of deficit for children with FAS). During the preschool period, attention problems and hyperactivity can emerge. In typically developing children, the late preschool through early school age (e.g., 4 through 7 years of age) is when many executive functioning and social perception skills are acquired. For example, simple planning or organization skills are learned, as well as understanding the physical and mental perspective of others. Preschoolers and school-aged children often do not fully acquire these skills. Throughout the school-age period, children with FAS can exhibit deficits or difficulty with any of the CNS domains listed in the diagnostic criteria. However, each child's pattern of strengths and weaknesses is likely to be very individualized. During adolescence and through adulthood, the pattern of deficits continues to be present and in addition, lifelong conse-

Table 3: Brief Outline of Diagnostic criteria for Fetal Alcohol Syndrome

Facial dysmorphia

Based on racial norms, individual exhibits all three characteristic facial features:

- Smooth philtrum (University of Washington Lip-Philtrum Guide rank 4 or 5)
- Thin vermillion border (University of Washington Lip-Philtrum Guide rank 4 or 5)
- Small palpebral fissures (at or below 10th percentile)

Growth problems

Confirmed prenatal or postnatal height or weight, or both, at or below the 10th percentile, documented at any one point in time (adjusted for age, sex, gestational age, and race or ethnicity).

Central Nervous System Abnormalities

I. Structural

1) Head circumference (OFC) at or below the 10th percentile adjusted for age and sex.
2) Clinically significant brain abnormalities observable through imaging.

II. Neurological

Neurological problems not due to a postnatal insult or fever, or other soft neurological signs outside normal limits.

III. Functional

Performance substantially below that expected for an individual's age, schooling, or circumstances, as evidenced by:

1. *Global cognitive or intellectual deficits representing multiple domains of deficit (or significant developmental delay in younger children) with performance below the 3rd percentile (2 standard deviations below the mean for standardized testing)*
   or
2. *Functional deficits below the 16th percentile (1 standard deviation below the mean for standardized testing) in at least three of the following domains:*
   a) cognitive or developmental deficits or discrepancies
   b) executive functioning deficits
   c) motor functioning delays
   d) problems with attention or hyperactivity
   e) social skills
   f) other, such as sensory problems, pragmatic language problems, memory deficits, etc.

Maternal Alcohol Exposure

I. Confirmed prenatal alcohol exposure
II. Unknown prenatal alcohol exposure

Criteria for FAS Diagnosis

Requires all three of the following findings:

1. Documentation of all three facial abnormalities (smooth philtrum, thin vermillion border, and small palpebral fissures);
2. Documentation of growth deficits
3. Documentation of CNS abnormality

quences of those deficits emerge, including mental health problems, inability to achieve independent living, and criminal activity (62,95,126-137).

Individuals who do not meet the full diagnostic criteria for FAS. The 1996 IOM report noted that most individuals with deficits resulting from prenatal exposure to alcohol do not express all of the features necessary to meet the criteria for an FAS diagnosis. This was confirmed in the comments of researchers, clinicians, and members of the NTFFAS/FAE during the deliberations of the SWG. Consensus has not been reached by either of these groups, or the scientific and clinical community at large regarding evidenced-based diagnostic criteria for any prenatal alcohol-related condition other than FAS. However, there is grave concern that individuals who present with the same neurodevelopmental deficits as individuals diagnosed with FAS, but who do not present with the full facial features or growth deficits, are not being afforded services because they are not given a diagnosis of FAS. Ongoing funding has been provided by the National Institute on Alcohol Abuse and Alcoholism (NIAAA) to conduct research that might result in evidence-based diagnostic criteria for individuals with other conditions caused by prenatal alcohol use. Currently, CDC is using a collaborative database of neurodevelopmental data from five intervention studies to explore the nature of individuals who could be considered in the diagnostic category of ARND, as well as data from a prospective cohort study of 5-year-olds in Denmark. However, at this time, the only diagnostic category with scientific evidence to support clinical criteria is FAS. As future data and science are available, these guidelines can be refined and expanded to delineate other conditions resulting from prenatal alcohol exposure.

CONSIDERATIONS FOR A REFERRAL FOR AN FAS DIAGNOSTIC EVALUATION

Very often the front-line providers of services (medical, educational, or social) are faced with making the decision of whether or not to refer a child, individual, or family for a full FAS diagnostic evaluation. The SWG recognizes that this may be a difficult decision. For biological families, there may be social stigma associated with any evaluation concerning prenatal alcohol exposure. In other families, direct information about alcohol use during pregnancy may not be available or only suspected. Thus, the following guidelines were developed to provide assistance in making the referral decision, although, it is recognized that each case must be evaluated individually. Further, these guidelines were developed with the idea that when in doubt, it was preferable to refer for full evaluation by a multidisciplinary team with experience in evaluating prenatal alcohol exposure.

- For situations with known prenatal alcohol exposure: A child or individual should be referred for full FAS evaluation when there is confirmed significant prenatal alcohol use (i.e., 7 or more drinks per week or 3 or more drinks on multiple occasions, or both). If prenatal alcohol exposure in the high risk range is known in the absence of any other positive screening criteria, the primary healthcare provider should document this exposure and closely monitor the childs ongoing growth and development.

- For situations with unknown prenatal alcohol exposure: A child or individual should be referred for full FAS evaluation when:
  - there is any report of concern by a parent or caregiver (foster or adoptive parent) that his or her child has or might possibly have FAS.
  - all three facial features are present (smooth philtrum, thin vermillion border, and small palpebral fissures).

- one or more facial features are present in addition to growth deficits in height or weight, or both.
- one or more facial features are present, along with one or more CNS abnormalities.
- one or more facial features are present, along with growth deficits and one or more CNS abnormalities.

SERVICES APPROPRIATE FOR AFFECTED INDIVIDUALS AND THEIR FAMILIES

Diagnosis is never an endpoint for any individual with a developmental disability and his or her family. This is particularly true for individuals with FAS, their families, and their community. As described in the framework section, the FAS diagnosis and the diagnostic process (especially the neuropsychological assessment) are part of a continuum of care that identifies and facilitates appropriate health care, education, and community services. The learning and life skills affected by prenatal exposure vary greatly among individuals, depending on the amount of alcohol exposure and the timing and pattern of exposure, as well as each individual's current and past environment (138-139). As a result, the services needed for individuals with FAS and their families vary according to what parts of the brain have been affected, the age or level of maturation of the person, the health or functioning of the family, and the overall environment in which the person is living. Thus, service needs for any particular individual and his or her family can be quite individualistic (140).

Despite the required individualization in service needs, some general areas of service and specific services have been identified as helpful to people with FAS and their families (62). While the ideal circumstance are services and interventions that have been specifically developed for individuals with FAS and their effectiveness has been established through rigorous scientific evaluation, such programs are only now being researched and developed. Thus, most evidence for the benefit of services has been gleaned from research with other populations, clinical wisdom, and family experiences. These three sources have drawn heavily from information obtained concerning risk and protective factors that have been found through systematic research, using natural history methodology, to promote positive development or reduce the incidence of negative long-term consequences of FAS (i.e., reduce secondary conditions; 141). First, these factors will be reviewed. Then, services that are applicable to all individuals with FAS, regardless of life stage will be presented. Finally, essential services appropriate for individual life stages will be presented.

Risk and Protective factors. In a landmark study of secondary conditions for individuals with FAS, Dr. Ann Streissguth and her colleagues delineated not only the lifelong consequences of FAS, but also several basic factors that are protective or increase the risk of negative outcomes (95). This study identified many lifelong negative consequences of FAS, including disrupted school experiences, legal problems, incarceration, mental health problems, substance abuse problems, inappropriate sexual behavior, dependent living, and poor employment history. Importantly, these risks are not mutually exclusive. Any individual can (and most likely will) experience multiple risk factors that can have a cumulative effect (141). Moreover, these risks can be exacerbated by family and community expectations. Research has shown a relation between the number of risk factors encountered by an individual and poor outcomes. Reducing the number and severity of risk factors is an important step in providing services to individuals with FAS and their families. The disorder of FAS has often been described as a "hidden" disability because of the subtlety of the dysmorphia and good basic language skills (e.g., vocabulary and syntax) of many affected individuals. These factors lead to individuals with FAS being treated inappropriately because of caregivers' unrealistic

developmental expectations. FAS might be either not recognized or mislabeled as stubbornness or "bad" behavior by a caregiver, or others who encounter the affected individual (e.g., teachers, extended family, and friends). Families raising children with developmental disabilities typically report considerable parental stress related to this aspect, particularly for parents of children with FAS (62).

Several factors have been identified that can potentially reduce the odds of long-term negative outcomes in children with FAS (protective factors), including a stable and nurturing home environment during the school years, early diagnosis (before 6 years of age), absence of exposure to violence, few changes in caretaking placements, and eligibility for social and educational services. Interventions and services that maximize these protective factors while reducing risk factors will provide the best benefit to anyone with FAS and improve their chances for achieving their developmental potential (95,138).

General Needs. Helpful interventions should include those that stabilize home placement and improve parent-child interaction (138). One method for accomplishing this goal is to increase the understanding of the disorder by parents, teachers, law enforcement personnel, and other professionals who might become involved with the affected individual. Children with FAS often need unique parenting because of their difficulty with cause and effect reasoning and other executive functioning skills. Caregiver education should highlight and explain differences in the thought processes of children with FAS from typically developing children and children with other developmental disabilities. This would enable parent s to avoid potentially difficult situations (e.g., avoiding overly stimulating environments) and better manage problems when they do arise. Overall, a better functioning family that results from caregiver education promotes the stable, nurturing home that has been shown to be a positive protective factor for children with FAS (142).

Beyond the home environment, other professionals also need increased education and information concerning FAS (135). Parents can facilitate this understanding by learning to become advocates for their child. Such advocacy includes both linking families with needed community resources and making sure that the child receives maximum benefit from that service. Because the myriad of service systems is confusing and inconsistent across states, families must be educated about them at the local level. The world of social and educational services can be overwhelming, confusing, and inconsistent, and usually has a unique vocabulary that must be learned. Thus, it is important that along with a diagnosis, clinicians need to help caregivers in learning about available services, how to determine which services are appropriate for their child, and how to work productively with service providers (62).

Many prenatally exposed infants and children enter the foster or adoptive care system at an early age. A recent study estimated that the prevalence of children with FAS (or a related disorder) in the foster care system is 10 times that of the general population (143). However, while protective service agencies (PSAs) might have information about a child's prenatal history, staff members are generally not knowledgeable about FAS, do not understand the impact of the child's having FAS, or do not communicate the child's FAS status to other service systems. As a result, foster and adoptive families are most often not educated about the long-term effects and are unprepared to meet their child's needs. However, most PSAs require foster parents to take a specified number of educational courses annually. These courses should include education about the effects and developmental needs

of the child with FAS since the majority of foster parents will encounter at least one child with FAS or a related disorder during their time as a foster parent.

The assessment process is integral to a well-developed treatment plan. As has been emphasized in these guidelines, part of the diagnostic process is a comprehensive neuropsychological assessment, not only to establish CNS abnormities for the diagnosis, but also to develop the best treatment plan possible. Such a treatment plan minimizes risk factors for lifelong negative consequences and promotes protective factors that maximize developmental potential. Clinicians and s ervice providers must ensure that assessments include communication and social skills; emotional maturity; verbal and comprehension abilities; language usage; and, if appropriate, referral for medication assessments. Finally, it is the responsibility of the community at large to ensure that children with disabilities, including children with FAS, have access to and are assimilated into school, recreational, and social activities.

Age-specific services. Basic child development informs clinicians and service providers that the abilities of any one individual change dramatically at different stages of development. Intervention research informs providers that the most effective programs are those that are geared towards an individual's developmental level. There are specific "turning points" during which children demonstrate rapid and fundamental changes in their understanding of the world and in their problem-solving skills (e.g., development of object permanence or acquisition of formal operational thought). The services identified in the following sections include those for both the child and the family, grouped broadly by developmental stage. It is recognized that many of these services span beyond a single age category with considerable overlap, especially for the family.

Prenatal services. Significant development of all major organ systems occurs throughout gestation; thereby, making it imperative that women who drink during pregnancy be identified by the medical community as early as possible and be provided intervention services. Findings have indicated that children born to women who stop drinking at any point during their pregnancy have better outcomes than those who continue to drink throughout pregnancy (73). To ensure this happens, physicians, nurses, and other allied health professionals need to be trained to screen patients for possible alcohol use, to understand the detrimental effects of prenatal exposure to alcohol, to understand alcoholism as a treatable disorder, and to be familiar with treatment services. Many women who drink during pregnancy and their partners are not educated about FAS or the harmful effects of drinking during pregnancy. These prospective parents might not be prepared to address their shame or the needs of their children. Some women who experience an alcohol-exposed pregnancy might have been exposed themselves prenatally to alcohol. Therefore, treatment personnel need to be educated about both FAS and needed services to provide more appropriate treatment, as well as provide ongoing support and monitor these families closely.

Services for birth to 3 years of age. The first years of life are an important time for physical, cognitive, and emotional development. Decades of research have consistently shown the benefits of early intervention for children with developmental disabilities. Clinicians working with this age group need to familiarize themselves with the state systems that service this population. In particular, Part C of the Individuals with Disabilities Education Act (IDEA) provides early intervention for children 0 to 3 years of age . In many states, this program is administered through local health departments. A particularly appealing aspect of the Part C portion of IDEA is that FAS is considered a "presumptive eligibility" diagnosis. Presumptive diagnoses allow children "at risk" of later develop-

mental delay to be served without meeting particular eligibility criteria. That is, children who are at risk for later developmental problems can receive services, even if they test in the normal range or do not meet other eligibility criteria. This is very important for children with FAS because only about 25% score in the significantly developmentally delayed range (or the range of mental retardation for older children; 95). Referring children who have the FAS diagnosis as well as children who are exposed but do not meet the full diagnostic criteria for FAS, ensures that these children are monitored and, at appropriate ages, referred to appropriate ancillary services as needed. Many states maintain birth defects registries that track the ICD-9 code encompassing FAS (i.e., 760.71 Fetal Alcohol Syndrome) which also can facilitate such monitoring. All infants who are known to have been exposed prenatally included in these registries can be referred to the state's special child health services (note: each state will have its own name for this service) that are part of all states' maternal and child health systems. Special child health services programs provide case management and referral services for children with birth defects or developmental disabilities as well as facilitate evaluation for early intervention programs.

As noted previously, a stable and nurturing caregiving environment is a protective factor for children with FAS. Child development literature states that stable and nurturing environments promote secure attachments between infants and caregivers (144). Secure attachment facilitates emotional, social, and personality development in positive ways. Insecure or negative attachment can lead to inappropriate development in these areas, and at the most severe end of the continuum, the psychopathology of reactive attachment disorder (RAD). The time between birth and 3 years of age has been shown to be a particularly salient time for development of child and caregiver attachment. Disruption in the caregiving environment can lead to poor or negative attachment between infant and caregiver (145). Because many children with FAS are in the foster care system and experience multiple placements (due both to the nature of the system and the difficulty in parenting a child with FAS), they are at tremendous risk for negative attachment including RAD.

Services for children 3 to 6 years of age and school age. It is often during the toddler period that children with FAS will be identified and can be diagnosed. It is essential that states establish FAS diagnostic centers or ensure that their child evaluation centers have clinicians who are trained in the dysmorphic and other diagnostic criteria associated with prenatal exposure.

Following the families themselves, the educational system serves as the most constant service provider for individuals with FAS from early childhood through adolescence. Therefore, beginning with preschool programs and through secondary education, generalized essential services can be delineated. When a child reaches three years of age, early intervention services (IDEA Part C) end and families are referred to preschool handicapped programs (sometimes called special needs preschool) that are administered through IDEA Part B. Part B of IDEA differs from Part C in that there are no presumptive eligibility diagnoses. Eligibility for educational services under this program is related entirely to functional criteria and their relation to educability. As noted previously, this can make it difficult for children with FAS to qualify for these special education services because only a quarter of the children with FAS achieve standardized test scores in the range of significant developmental delay (or mental retardation), the usual eligibility threshold. However, within the IDEA framework, there are a few categories that allow children with FAS who score above the eligibility threshold to still qualify. A few children with FAS will qualify in the category of learning disabled (LD). This category is for children who demonstrate a statistically significant discrepancy (2 standard deviations or more) between overall IQ and one or more academic skills (e.g., math).

Another eligibility category that has been helpful in qualifying children with FAS is behavior disorder. However, this category should be used with care because children with FAS can learn negative behaviors from other children without receiving the benefits of a structured environment. Finally, the category of other health impaired (OHI) can be used at the discretion of the individual education plan (IEP) committee, which includes parents as well as school personnel. Services available through the school system go beyond classroom settings. Children can receive various therapies, including physical therapy (usually most appropriate for very young children), speech and language therapy, occupational therapy, or social skills training. These last two areas are particularly helpful to children with FAS because of the visual-motor deficits and problems in social interactions they encounter.

Training parents to be effective educational advocates is essential to maximize the benefits of their child's special education and to understand their child's rights. The purposes of the IEP are to insure access to appropriate habilitation and rehabilitation services (physical, occupational, speech, behavioral, mental health, and other related services) and to ensure that academic curricula are balanced with vocational training and skills of daily living (e.g., personal hygiene, money management, and family life education), when appropriate. In addition to training parents about the educational system, the preschool period, as well as elementary school years, are times when parents become more acutely aware of their child's imitations. Reinforcement and updating of information learned in early parent education settings will benefit both the child and the parents. Reviewing lessons learned will help parents adjust their expectations for the child's current functioning, as well as his or her future possibilities.

As noted previously, it is important that school staff be trained to recognize possible characteristics associated with FAS, as well as appropriate techniques for instructing students with FAS. Irrespective of whether alcohol-exposed children are in regular or handicapped preschool programs, educational methodologies need to be developed that best address their learning styles and that appropriate behavioral or mental health services are available and initiated.

Beyond services available through the educational system, families raising preschool and school-aged children continue to need services to promote positive family functioning. Such services might include behavior management training, family (or child) counseling, parenting workshops that focus on the unique aspects of parenting a child with FAS, or other types of continuing education. One service that becomes very important during these years is respite care. Such care allows a trained individual to stay with the affected child while caregivers or other family members take advantage of some time away from the child with FAS. Respite care has been shown to significantly reduce family stress and improve family functioning (146-147). Unfortunately, respite care, especially formalized and high-quality respite care, is not readily available in most communities. A clinician can help in this situation by working with the family to develop informal respite care situations, such as help from an extended family member working with the family and to provide the necessary education about FAS to such a respite care provider.

Services for adolescents. Adolescence, and even preadolescence, is one of the major turning points in the life of an individual with FAS. His or her body is changing, cognitive abilities are changing, peer groups are changing, and community expectations are changing. Because of the confusing nature of all these changes, adolescence often is the period when behavioral and mental health problems become more pronounced. Depression or anxiety, or both, can set in as the individual

struggles to cope with these changes. Increased opportunities to experience alcohol and/or drugs can lead to substance abuse problems. Families could become involved with juvenile or criminal justice systems. These are some of the most serious secondary conditions that research has identified for individuals with conditions associated with prenatal alcohol exposure (95).

It is often during adolescence that families experience high levels of stress and tension. As such, the need for individual counseling (for both child and parent), family counseling, and a strong support network becomes more crucial. However, this can be the exact stage at which agencies are reluctant to provide such services, especially if the FAS-related disability factor is not recognized. Because some amount of rebellion is expected during adolescence, the challenging behaviors of the teenager with FAS might be dismissed as transient. At the other end of the spectrum, the challenging behaviors of the adolescent with FAS can be so severe as to involve the criminal justice system.

Because adolescents will soon be leaving the safety and structure of the educational system, vocational and transitional services become essential during this stage. These services often represent a shift from academic skills and achievements to daily living skills, including employment skills. It is very important that these services be started in early adolescen ce, and not left until the individual is about to age-out of the educational system. In addition, beyond teaching the specific skills that go with a particular job, it might be necessary to explicitly teach those skills related to being a good employee (e.g., punctuality and minimized socializing). Most individuals will learn these skills through basic maturity and observational learning. Individuals with FAS often need explicit instruction as well as lifestyle supports (e.g., a job coach).

As for all adolescents, sexual behavior often becomes a critical issue during this stage. The boundaries for appropriate interaction with the opposite sex, the subtle nature of social cues, and impulse issues are difficult for any adolescent, but more so for the adolescent with FAS. Close supervision is the first line of defense during the adolescent years. However, such supervision often conflicts with the adolescent's growing desire for independence. This must be navigated with care to avoid alienating the adolescent. Also, it is probably best to be open and explicit with the teenager with FAS concerning the issues of contraception, sexually transmitted diseases, and sexual harassment. Failure to address these issues can have serious, and possibly life-threatening consequences for the affected individual, his or her family, and any children resulting from unintended pregnancies.

As mentioned previously, individuals with FAS are at high risk for involvement with the juvenile and criminal justice systems. Their lack of executive functioning skills (i.e., poor judgment), fluid language skills, and naïve social skills make them particularly vulnerable to participating in criminal activity. However, these same deficits demand that when they do encounter the justice system, their deficits should be taken into account during all aspects of justice proceedings (i.e., charges, process, punishment, and rehabilitation). As such the juvenile and criminal justice systems are major social systems in need of education regarding FAS. Special rehabilitation programs with staff that are trained to work with adolescents and young adults with FAS should be established. Such programs should be based on scientifically-based research findings that evaluate practicality as well as effectiveness.

Services for adults. In addition to all the services mentioned for the preceding age groups, adults with FAS often need support in every area of their lives. Everyday needs such as transportation issues, job assistance, housing assistance, medication reminders, money assistance, and support and

assistance when unpredicted issues arise should continue to be monitored and supported. Although not consistently available, clearly a system needs to be established that assists people with FAS in living as independently in the community as possible and includes support for housing, healthcare, and employment.

Because of federal, and often state legislation, it is very difficult for people with FAS to receive services from state developmental disability agencies, unless exposed individuals have met the eligibility criteria for services before 22 years of age. Eligibility criteria are generally based on levels of intelligence, as well as functional limitations in at least three areas associated with skills of daily living. As a result, many exposed individuals will not be eligible for services that often include an individual service plan (ISP), case management, residential and employment assistance, and possibly social security disability payments.

People with FAS might be eligible for federal assistance, such as, Medicaid, Supplemental Security Income (SSI), and Section 8 Housing subsidies, not because of their disabilities but because of their low income status. However, obtaining these services can be difficult. For example, most states have long waiting lists for Section 8 housing because of both the high demand and great need often leading to a shortage of rental units. Eligibility criteria for these services, even when based on income, should include a priority category for persons with FAS. Unfortunately, many of these housing options do not offer the support and structure that adults with FAS often need. Housing remains one of the main issues in supporting individuals with FAS for which there are no appropriate services developed or identified.

IDENTIFYING AND INTERVENING WITH WOMEN AT RISK FOR AN ALCOHOL-EXPOSED PREGNANCY

Despite ongoing efforts to inform childbearing-aged women of the risk associated with alcohol use during pregnancy, surveys conducted by the CDC and SAMHSA indicate that 9% to 12% of pregnant women in the United States report consuming alcohol and that approximately 3% report drinking at levels than have been consistently associated with adverse effects on the fetus (148-149). Also of concern are the high proportions of non pregnant childbearing-aged women whose drinking patterns exceed safe levels as defined by public health agencies (150-151). Currently more that half of all women of childbearing age (18 through 44 years of age) report alcohol use, and one in eight report binge drinking in the past month. Many of these women are sexually active and are not taking effective measures to prevent pregnancy. These women are at risk for an alcohol-exposed pregnancy in that they could have an unrecognized pregnancy and continue drinking early in pregnancy at levels that are harmful to the fetus.

One public health strategy for preventing alcohol-exposed pregnancies is to identify characteristics of women at greatest risk of having a child affected by prenatal alcohol exposure and implement prevention programs in subpopulations with higher proportions of these identified risk factors. Over the past 20 years, concerted efforts have been made to identify factors among childbearing-aged women associated with harmful patterns of alcohol consumption. One extensive review of studies reporting characteristics of women giving birth to a child with FAS found that low socioeconomic status (SES), African-American and American-Indian/Alaska-Native ethnicity, and being a smoker were characteristics commonly found among women in this group (152). Additional studies using cross-sectional survey data and special populations have extended our understanding of char-

acteristics associated with childbearing-aged women at high risk for having an alcohol-exposed pregnancy based on current drinking patterns. Factors associated with risk include being a smoker, having a low SES, being unmarried, having a history of previous or current illicit drug use, having a history of physical or sexual abuse, having psychological stress, and having mental health disorders (153-156). In an attempt to identify community-based settings that have higher proportions of preconceptional women at increased risk for an alcohol-exposed pregnancy, CDC sponsored an epidemiological study of special populations that included: women in alcohol and drug treatment centers, a large urban jail, publicly funded primary care clinics, a university-based gynecology clinic in a large urban hospital, and a cohort of women responding to a newspaper solicitation (27). Being at risk for an alcohol-exposed pregnancy correlated significantly with being (or having ever been) a smoker, having a history of inpatient treatment for drugs or alcohol, having a history of inpatient mental health treatment, having multiple sex partners, and having experienced recent physical abuse.

Primary prevention of alcohol-exposed pregnancies requires the accurate identification of women who are drinking at thresholds that have been associated with adverse pregnancy and infant outcomes before pregnancy occurs. Dietary guidelines from the U.S. Department of Health and Human Services recommend that women drink no more than one drink per day to avoid increased risk for adverse health conditions and that women who are pregnant, planning a pregnancy, or at risk of pregnancy abstain from alcohol use altogether. Research findings support these recommendations with evidence of increased risk for birth defects, spontaneous abortions, and deficits in neurocognitive development and growth at levels of seven or fewer drinks per week in some studies (157-162).

Of further concern is the pattern in which alcohol is consumed. Heavy episodic drinking, as in binge drinking, can result in increased severity of the teratogenic exposure effects because of the higher peak blood alcohol levels achieved in this pattern of consumption as opposed to lower level daily consumption (40,163). Historically, a binge episode has been considered to be five or more drinks on any one occasion, but evidence of the presence of gender effects in alcohol metabolism and higher morbidity and mortality among women than men with similar consumption patterns has prompted recommendations for defining a binge episode for women as four or more drinks on any one occasion or on any one day. Current recommendations on clinical thresholds published by the NIAAA are that women drinking more than seven drinks per week or more than three drinks on any given day in the past month be further assessed for risk of developing alcohol-related problems. As stated earlier, pregnant women are advised to abstain from alcohol use. This is a long-standing federal advisory and one supported by major professional societies as well (9,164).

Measurement of current alcohol consumption can be enhanced through the use of reliable screening and assessment methods. A number of screening instruments have been developed that offer the practitioner options for clinical assessment of childbearing-aged women. When selecting a screening tool for routine implementation, healthcare professionals should consider factors such as the goals of the screening process, the target population, and the ease of administration. All women of childbearing age should be screened for alcohol use, including women who are pregnant or nursing, women who are planning a pregnancy, and women who are sexually active and not using contraception (such as teens and college-aged women).

Methods and Considerations for Establishing Reliable Estimates of Alcohol Use. Measures most commonly used in alcohol assessment include items that address quantity, frequency, and pattern (variability) of drinking. Quantity-frequency measures (QF) inquire about average or typical consumption patterns. The simplest measures assess the amount of drinking on average drinking days (Q), and the average number of days on which alcohol is consumed (F). QF measures can be used to estimate a woman's average number of drinks per day or the amount of absolute alcohol consumed per day (AA score). To assess for binge drinking, some investigators have recommended that screening questions should include measures of maximum quantity consumption and frequency of maximum quantity consumption (165). *Helping Patients with Alcohol Problems: A Practioner's Guide,* developed by the NIAAA, (166) recommends quantity-frequency and maximum quantity questions as the primary screening test. These questions have been shown to have relatively high sensitivity and specificity, are easy to use, and can be incorporated into a health practice with minimal cost and effort (167).

Additional considerations for alcohol screening include measurement of the types of alcoholic beverages the woman consumes. The development of new ways of marketing wine and beer, including higher alcohol concentration malt liquors and beer in 20- to 45-ounce containers, has increased the need to provide women who drink with more specific standards to estimate their consumption. Studies show that reliance on standard drink measurements when assessing drinking practices of heavier drinkers or those consuming higher alcohol content beverages can result in considerable underestimation of alcohol consumption (168-169). Moreover, beverage-specific questions have been shown to be more accurate than grouped beverage questions (170).

One of the most reliable assessment tools for gathering accurate self-reported alcohol use is the timeline follow back (TLFB) measure (171). This method of alcohol use assessment typically asks respondents to think back over the past 90 days and report daily drinking amounts during that period of time. Respondents are given a calendar and asked to identify special events that occurred during that time period, such as parties, birthdays, or holidays that might serve to trigger recall of drinking occasions. A major strength of the TLFB measure is its ability to capture both average daily drinking and sporadic drinking that might entail drinking at higher levels of consumption. Because of the time requirements necessary for gathering information using the TLFB approach, it is not easy to use in primary care settings but it has been used in clinical research to establish the reliability and validity of other briefer screening and assessment tools (172).

Screening Tools. Several brief questionnaires have been developed over time to screen for problematic alcohol use in adults in diverse populations and settings. Screening tools currently in use include the CAGE, AUDIT, T-ACE, TWEAK, MAST, S-MAST, NET, RAPS4, and RAPS4-QF (173). The CAGE (174)is a four-item alcohol use measure designed to detect alcoholism and is one of the most commonly used screening instruments for men and women. The four questions ask the respondent: (C) Have you ever felt you should CUT down on your drinking? (A) Have people ever ANNOYED you by criticizing your drinking? (G) Have you ever felt bad or GUILTY about your drinking? and (E) Have you ever had a drink first thing in the morning to steady your nerves or to get rid of a hangover (EYE OPENER)? Each item is scored as a 0 (no) or 1 (yes) and scores of 2 or greater are considered clinically significant.

The AUDIT is a 10-item self-report questionnaire developed by the world health organization (WHO) for identifying individuals whose alcohol consumption has become hazardous or harmful

to their health (175). The items on the AUDIT are constructed to measure frequency of alcohol consumption, dependence symptoms, and the personal and social consequences of drinking. The first three questions of the AUDIT address the quantity, frequency, and maximum amount of alcohol consumed. The remainder of the questionnaire has two items from the CAGE, (feelings of guilt about drinking and the need for a drink first thing in the morning after a heavy drinking session—eye-opener) in addition to questions on the frequency of being unable to stop drinking once drinking starts, frequency of being unable to do what is normally expected because of alcohol use, frequency of memory loss of the previous night due to drinking, frequency of injury to self or others as a result of drinking, and frequency of others expressing concern over the individual's drinking (a relative, friend, or doctor). A score of eight or more is considered significant for high-risk consumption. The measure has good psychometric properties and has an advantage over other screening tools in that it measures not only experienced consequences of drinking, but also whether an individual is currently drinking at levels likely to eventually result in problems (176). The instrument has been used and validated in cross-cultural populations (177). One review of 38 studies on screening for alcohol problems in women and men in primary care settings found the AUDIT was more effective in identifying individuals with at-risk, hazardous, or harmful drinking patterns, while the CAGE proved superior in detecting alcohol abuse and dependence (178). Another study of women who were receiving outpatient care through the Veterans' Administration found that the AUDIT-C (comprised of the first three items of the AUDIT on quantity, frequency, and maximum drinks per drinking occasion) proved to be more sensitive than the full 10-item AUDIT (179). Such findings suggest that when time of administration is a consideration, the shorter AUDIT-C might be used as the screener of choice.

Two screening tools that have been specifically developed and used with pregnant women are the T-ACE and the TWEAK (180-183). The T-ACE has four questions that take less than a minute to ask. The questions are: (T) TOLERANCE, how many drinks does it take to make you feel high? (A) Have people ANNOYED you by criticizing your drinking? (C) Have you ever felt you ought to CUT DOWN on your drinking? (E) EYE OPENER, Have you ever had a drink first thing in the morning to steady your nerves or get rid of a hangover? One point is given for each affirmative answer to the A, C, E questions, two points when a pregnant woman reports a tolerance of more than two drinks to feel high. A positive screen is a score of two or more points. The T-ACE has been shown to be an efficient screen for risk drinking in pregnant women and out-performs medical staff assessment alone (184). Its brevity (four-items) is an important asset and increases its utility for clinical practice.

Like the T-ACE, the TWEAK attempts to elicit information on the following: (T) TOLERANCE for alcohol; (W) WORRY or concern by family or friends about drinking behavior; (E) EYE OPENER, the need to have a drink in the morning; (A) "blackouts" or AMNESIA while drinking; and (K) the self-perception of the need to CUT DOWN on alcohol use. Scores range from zero to seven. The tolerance and worry questions each contribute two points and the other three questions contribute one point each. Any endorsement of the worry question is scored a two. On the tolerance question, if three or more drinks are needed to feel high, the question is scored as a two. Other versions of the tolerance question ask: How many drinks does it take before the alcohol makes you fall asleep or pass out? Or, if you never drink till you pass out, what is the largest number of drinks you have or can hold? These questions are scored as a two if the woman answers five or more drinks. Using these questions, however, results in lower sensitivity and specificity. A total score of three or more on the TWEAK is suggestive of harmful drinking pat terns (185).

Studies assessing the relative effectiveness of various screening tools have yielded different findings depending on the populations studied. Bradley et al. (186) summarized data from 13 published, peer-reviewed articles that contained information on the performance of alcohol screening questionnaires with non pregnant women and with obstetric patients during the periconceptional period. The results revealed that, for non pregnant women, the CAGE had low sensitivity in predominantly White female populations, but was a reasonable choice for identifying past year or lifetime alcohol dependence in predominantly African American female populations. Overall, the five-item TWEAK appeared to be the optimal screening questionnaire for identifying heavy drinking or alcohol abuse and dependence in racially mixed populations of non pregnant and pregnant women.

Some clinicians have suggested that the use of screeners best suited for identifying high-risk heavy drinkers might not be optimal for identifying pregnant women with irregular or lighter patterns of consumption that might still pose a risk for fetal development. Using confidential reporting procedures, one study examined the effectiveness of the TWEAK for assessing any report of drinking following pregnancy recognition in a group of low-income pregnant women participating in WIC (187). With a cut point score of two or greater, the specificity of the TWEAK was high for all ethnic groups studied; however sensitivity, while high for White non-Hispanic women, was low for African-American non-Hispanic and Hispanic women. Because any endorsement of alcohol use was sufficient for classification as a drinker in this sample, low sensitivity on the TWEAK might have been due to the fact that women were drinking at levels that were too low to result in alcohol-related problems but, were still high enough to be detrimental to the developing fetus. From a practical standpoint, the high specificity of the TWEAK supports its utility in busy clinic settings. High specificity suggests the TWEAK is effective in screening women who are not high risk, and as shown in the study cited previously, women who report not drinking at all. Nevertheless, the fact that the TWEAK had variable sensitivity for women from different ethnic backgrounds suggests that additional methods of screening should be employed in order to increase the detection of women who might need intervention.

Drinking among adolescents and college students has long been recognized as a significant problem with far-reaching public health implications (188-189). Although the most problematic drinking in adolescence has been documented among males when compared to females, alcohol use in females has been associated with decreased use of contraception (increasing the likelihood of an unintended pregnancy), increased sexual assault, and more sexually transmitted diseases. Because of these significant negative health consequences, the American Medical Association Guidelines for Adolescent Preventive Services recommend screening of adolescents for alcohol and other drug use as part of routine medical care (190). To effectively screen adolescents, the ideal screening tool must be developmentally appropriate and practical for use in busy medical offices or clinics. Although the CAGE and the AUDIT are relatively brief, their developmental appropriateness is questionable. Indeed, current research suggests that the CAGE is not appropriate for screening adolescents and that a much lower cut point of two (rather than the eight recommended for adults) on the AUDIT is optimal for identifying alcohol use problems in this population (191).

Several measures have been developed specifically for use with adolescents, such as the Rutgers Alcohol Problem Index (192) and the College Alcohol Problem Scale (193); however, these tools might not be practical for universal screening. One brief screening device, the CRAFFT, developed for adolescents, is simple to score, inquires about alcohol and drug use, and was found to have good psychometric properties in a sample of predominately female youths 14 through 18 years of age

(194). CRAFFT is an acronym for the first letters of key words in the test's six questions: (C) Have you ever ridden in a CAR driven by someone (including yourself) who was high or had been using alcohol or drugs? (R) Do you ever use alcohol or drugs to RELAX, feel better about yourself, or fit in? (A) Do you ever use alcohol or drugs while you are by yourself, ALONE? (F) Do you ever FORGET things you did while using alcohol or drugs? (F) Does your family or FRIENDS ever tell you that you should cut down on your drinking or drug use? (T) Have you ever gotten into TROUBLE while you were using alcohol or drugs? Each question on the CRAFFT is given a score of one and a cut point of two provides moderate sensitivity (.70) and excellent specificity (.94) for identifying alcohol use disorders in adolescents. The authors CRAFFT recommend that any positive answer on this measure be followed by further assessment of quantity, frequency, and pattern of use to increase sensitivity and to guide decisions about the need for intervention.

A number of studies have shown a strong association between alcohol intoxication and trauma (195). The Trauma Questionnaire was developed to screen patients in an unobtrusive way without asking them directly about their alcohol use (196). The questionnaire consists of four questions asking about injuries in the last five years (fractures or dislocations of bones or joints, traffic accidents, head injury, or injury during a fight or assault). The questionnaire has been shown to have high sensitivity and specificity for identifying high-risk alcohol use and to be acceptable to respondents and physicians.

Computer-Assisted Interviews and Laboratory Screening Measures. The method of delivery of questions about alcohol use has been shown to influence truthful reporting. Clinical interviews might not be as effective in eliciting truthful responses from women because practitioners are not always comfortable asking these questions in a face-to-face interview. Moreover, women could underestimate their alcohol use because of reluctance to discuss this potentially sensitive subject. For this reason, self-administered questionnaires might improve the validity of self-report.

The Audio Computerized Self-Report Interview has been successfully used in prenatal clinics serving disadvantaged low-literacy minority women (197). Questions asked by a recorded voice through earphones are drawn from the TWEAK with quantity and frequency questions pertaining to three months before and during pregnancy are included. Acceptability studies have revealed that patients liked this method of screening.

Laboratory screening measures offer promise for obtaining objective evidence of problem drinking (198-199). The most common biomarkers are gamma glutamyltransferase (GGT) and carbohydrate-deficient transferring (CDT). Fatty acid ethyl esters synthase (FAEE) can be found in the hair of alcohol using women. This biomarker could hold promise for screening for alcohol use in pregnant women, although the dynamics of enzyme expression appear to be complex and changes occur only at high alcohol doses. Low sensitivity in non alcoholic women and the high cost of laboratory analysis make these measures less feasible for use in more universal screening.

Brief Intervention. Brief intervention (BI) has been shown to be a low-cost, effective treatment alternative for alcohol problems that uses time-limited, self-help, and preventative strategies to promote reductions in alcohol use in nondependent individuals, and in the case of dependent people, to facilitate their referral to specialized treatment programs (200-203). Overall, BI for alcohol problems has been shown to be more effective than no intervention and often as effective as more extensive intervention (204). Effective prevention programs frequently employ a multicomponent

approach combining cognitive-behavioral techniques with norms clarification, education, and motivational enhancement interventions. BI is usually restricted to fewer than four sessions and is typically performed in a treatment setting that is not specific for alcoholism. It is often performed by personnel who are not specialists in the treatment of alcohol use and abuse, and is provided to individuals at varying levels of risk for negative consequences because of drinking, rather than those who are considered alcohol dependent (205).

Six elements characterize the key ingredients of standardized brief intervention summarized by the acronym FRAMES (206). These are: Feedback of personal risk, Responsibility for personal control, Advice to change, Menu of ways to reduce or stop drinking, Empathetic counseling style, and Self-efficacy or optimism about cutting down or stopping drinking. The intervention also involves establishing a drinking goal in the form of a signed contract and follow up of progress with ongoing support. Studies reveal that supportive, nonjudgmental techniques in which trained personnel counsel women can lead to decreased alcohol consumption during pregnancy. The most effective intervention approaches avoid the use of moral or volitional injunctions and instead focus on reduction of alcohol use without criticism or provocation of guilt (205). Effective interviewers have been found to have a thorough knowledge of the intervention technique, an optimistic attitude about change, a compassionate style, genuineness and respect for clients, an ability to avoid arguments that evoke patient defensiveness, and comfort discussing alcohol problems (207-208).

Motivational Interviewing. A technique often used in brief interventions is motivational interviewing (MI). MI uses an empathic , client-centered counseling approach to increase readiness for change by resolving ambivalence about behavior change (207). The process explores the client's ambivalence in an atmosphere of acceptance, warmth, and positive regard. Although the session is structured and to the point, direct persuasion and coercion are avoided. The goal is to enhance the discrepancy between the reasons for changing versus staying the same. More than 24 studies of MI have found beneficial effects in decreasing problem drinking and other health-related problem behaviors (209).

Manualized Brief Intervention. Although the findings of the previously cited studies provide the foundation for intervention and prevention efforts, the usefulness of MI in a busy clinic or medical practice might be limited in that it requires additional training and clinical skills development. Recently, standardized manualized BI techniques to reduce alcohol consumption have been developed. One such intervention is Project CHOICES (Changing High-risk alcohol use and Increasing Contraception Effectiveness Study), which is funded by CDC. Project CHOICES is an example of a brief intervention using motivational interviewing techniques aimed at preventing alcohol-exposed pregnancies among high-risk women in various community settings (210). This project focused on both alcohol risk reduction and pregnancy postponement until alcohol use was decreased in a group of non pregnant women who were of childbearing age, fertile, sexually active, and using ineffective or no contraception. Phase I results revealed that at six-month follow up, 68.5% of the women were no longer at risk of having an alcohol-exposed pregnancy, 12.6% had reduced their drinking only, 23.1% reported using effective contraception only, and 32.9% reported doing both. A Phase II randomized controlled trial is currently underway testing the efficacy of this approach and is slated for completion in fall 2004. Another recent CDC-funded study still underway is Project Balance, which uses a briefer adaptation of Project CHOICES and is aimed at female college students, encouraging them to abstain from alcohol or to use contraception if they drink.

Four randomized controlled studies, funded by NIAAA, serve as further examples of the utility and effectiveness of this approach. These studies included women of different socioeconomic and cultural backgrounds and were conducted in doctors' offices and community settings. Two of the studies found that manualized BI was successful in decreasing alcohol use during subsequent pregnancies in high-risk women, thus preventing possible negative developmental sequelae in their offspring (211). The other two studies, one working with high-risk, White, middle class pregnant women in physicians' offices and one working in WIC community clinics with primarily low risk, low-income Hispanic clients, also found manualized BI to be a promising approach (212-214). In the WIC study, better infant outcomes were reported, including longer birth lengths and a lower infant mortality rate. Significantly, in both studies, the control conditions that included an assessment of alcohol consumption and simple advice to stop or cut down on drinking were almost as effective as the manualized BI itself. The success of these projects in reducing alcohol consumption in both experimental and control groups was postulated to be due to the desire of pregnant women to have healthy pregnancies and to the time and attention that interventionists provided for women in both groups.

Computerized Brief Intervention One recently developed study is investigating the use of a computerized method of a BI that incorporates the use of vessel size and normative education that allows a woman to evaluate her own consumption levels (169). Once a woman has been educated about her consumption levels, she then participates in a standardized BI. This approach is promising in that it incorporates many recommended aspects of screening, educating, and assisting women in recognizing that they might have a drinking problem, and then providing a brief intervention. The automated computerized assessment is designed for use in prenatal clinics and is currently being tested at a large HMO through an NIAAA grant. Although the effectiveness of this intervention has yet to be validated, it offers a potentially useful method that could be expanded nationally.

Improving Use of Screening and Brief Intervention Technology by Clinicians. Research devoted to finding ways to encourage clinicians to use brief interventions indicates that routine educational approaches might not be effective. Effective strategies include (1) conducting educational programs at the intervention site; (2) using specific step -by-step, evidence-based clinical protocols; (3) using skills-based role playing; (4) holding peer group discussions; and (5) using a credible expert trainer or educator. Brevity, repetition, and reinforcement of recommended practices are also key program elements (215). In an effort to enhance physician uptake of current screening and intervention approaches for preventing alcohol-exposed pregnancies, the NIAAA and the Office of Research on Minority Health recently collaborated in the development of a guide for primary care providers for screening pregnant and non pregnant women on selected health behaviors, including alcohol use, and recommendations for appropriate advice depending on the level of alcohol use and consequences (166). More recently, CDC funded four regional training centers on FAS to provide education and training to medical and allied health professionals and students about identification and diagnosis of children affected by prenatal alcohol exposure and effective approaches for intervening with and preventing these conditions. More information about these and other resources can be found by accessing the websites provided at the end of this section.

Universal screening for alcohol use should be conducted among all women of childbearing age. Screening can be done in both physicians' offices and in community health settings. Simple screening techniques that include measures of quantity, frequency, and heavy episodic drinking, as well as

behavioral manifestations of risk drinking, have proven to be most beneficial. For non pregnant women, the AUDIT, TWEAK, and CAGE have been found to be useful screening tools depending on the population characteristics of the community as described in the studies reviewed earlier. However, studies on gender-specific modifications of these screening tools have been recommended because women report increased psychosocial problems at lower levels of consumption than men. Based on some empirical evidence, reduced cut points for women have been recommended, including a cut point of 4 or more for the AUDIT, 2 or more for the TWEAK, and 1 or more for the CAGE (186). For pregnant women, the T-ACE and the TWEAK are the recommended screening tools of choice. The CRAFFT shows promise as an alcohol and other drug screener for female adolescents. Findings support better identification when screening instruments are used versus clinical assessment alone. Brief interventions administered by physicians and allied health professionals in medical and non medical settings are effective in bringing about reductions in drinking.

Women who are pregnant, planning a pregnancy, or at risk of pregnancy should be advised not to drink, as no safe threshold of alcohol use during pregnancy has been established. Non pregnant childbearing-aged women should be advised to drink no more than seven drinks per week and no more than three drinks on any one occasion.

Both BI approaches and MI techniques have been found to be efficacious with pregnant and non pregnant women in promoting reduction in alcohol consumption. Furthermore, some evidence suggests pregnant women are motivated to stop drinking even if the intervention includes only an assessment of alcohol use with simple advice to stop or reduce drinking. Research also indicates that interventions are effective with pregnant women who are light as well as high-risk drinkers. Moreover, preconceptional counseling of women of childbearing age who are at risk for an alcohol-exposed pregnancy and who are not using effective contraception has been demonstrated as a promising method of prevention as well as computerized versions of standardized methods of intervention. This year the U.S. Preventive Services Task Force released recommendations calling for use of the screening and behavioral counseling interventions for adults in primary care setting, including pregnant women (216-217). These evidence-based recommendations mirror the content of these guidelines with respect to the specific types of screening tools recommended and the components of effective brief interventions to be used. The recommendations of the U.S. Preventive Services Task Force, which is supported by the federal Agency for Healthcare Research and Quality, provide further support for the need for widespread public health implementations of these prevention technologies to reduce the harmful consequences of alcohol misuse, including FAS and other prenatal alcohol-related conditions. Information about reported and other federally sponsored studies in FAS and prenatal alcohol screening and intervention can be obtained at the following websites: www.nih.gov; www.cdc.gov; www.samhsa.gov; and www.preventiveservices.ahrq.gov.

SUMMARY AND FUTURE STEPS

In 2002, CDC was congressionally mandated to develop diagnostic guidelines for FAS and other prenatal alcohol-related disorders and integrate them into medical and allied health education.

With input from a SWG composed of clinicians, experts, and families, and from the NTFFAS/FAE, scientific and clinical evidence was reviewed to develop guidelines that offer a balance between conservative and overly inclusive definitions of FAS. Criteria for conditions not meeting the clinical definition of FAS (e.g., ARND) were not established because scientific evidence is insufficient at this time. Clinical and scientific research on FAS and those conditions resulting from prenatal alcohol exposure that do not meet the criteria for an FAS diagnosis is currently underway. These findings and advances will contribute to further refinement of the FAS criteria, and could potentially delineate additional diagnostic categories and criteria for conditions other than FAS. The development of these FAS guidelines is a continuous process. Efforts to develop and refine other diagnostic categories to identify FAS and related conditions need to continue.

During this guidelines development process, several key issues emerged that deserve mention.

1. More information on the neurodevelopmental effects of prenatal exposure to alcohol is needed. Particular emphasis should be placed on finding the unique aspects of FAS that will help differentiate it from other birth defects or developmental disabilities, or both.

2. Efforts to improve the clinical assessment tools (e.g., facial and growth measures) used to diagnose FAS should continue, particularly in terms of racial and ethnic variations and age.

3. All children should be screened for the possibility of an FAS diagnosis. As physicians and other allied health professionals become educated about this disorder, screening for FAS should become routine.

4. Better communication between obstetricians, gynecologists, and pediatricians is needed to improve documentation on prenatal alcohol use. This would help with the diagnosis of prenatal alcohol exposure in the child and could help identify women at risk for future alcohol-exposed pregnancies.

5. Service agencies must provide a way to qualify children with FAS and related disorders who do not meet their traditional eligibility requirements.

6. Further research and resources are needed to identify and treat women at risk for an alcohol-exposed pregnancy.

7. Awareness, both in the public and professional arenas, about the dangers of drinking alcohol during pregnancy and about FAS and how the condition affects children and their families is essential. A key avenue to avoiding FAS is active promotion of programs to increase awareness of the dangers of drinking alcohol during pregnancy and promotion of prevention activities that increase understanding of the risks of alcohol as well as the risks for an alcohol-exposed pregnancy.

Over 30 years ago, researchers first described FAS . Much has been learned about the disorder since that time, as is reflected in these guidelines. However, there is still much more to learn about the entire spectrum of effects from prenatal alcohol exposure. Future work will address these gaps. To reduce FAS and other prenatal alcohol-related conditions, the key is prevention. Federal, state, and local agencies; clinicians and researchers; educational and social service professionals; and families

need to work together to educate women of childbearing age and communities across the country about the risks of drinking alcohol during pregnancy.

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Citation: Bertrand J, Floyd RL, Weber MK, O'Connor M, Riley EP, Johnson KA, Cohen DE, National Task Force on FAS/FAE. Fetal Alcohol Syndrome: Guidelines for Referral and Diagnosis. Atlanta, GA: Centers for Disease Control and Prevention; 2004.

Notes

Notes



CENTERS FOR DISEASE CONTROL AND PREVENTION
NATIONAL CENTER ON BIRTH DEFECTS AND DEVELOPMENTAL DISABILITIES

To learn more about Fetal Alcohol Syndrome, contact:

FAS Inquiries

Web site:
Http://www.cdc.gov/ncbddd/fas

Order materials:
Http://www.cdc.gov/ncbddd/fas/faspub.htm

E-mail: FASInquiries@cdc.gov
Phone: 404-498-3947
Fax: 404-498-3040
Health Line: 1-888-232-5929

Department of Health and Human Services

FAS Facial Photographic Analysis Report

IDENTIFICATION

Name: Charles (First) _____ (Middle) Williams (Last)

Subject I.D.: None

Source of Photo: RSA

Gender: Male

Race: African American/Caucasian

Birth Date: [redacted]

* Normal PFL Charts: Iosub Lip-Philtrum Guide: African American

PHOTO ASSESSMENT

	Frontal	¾ View	Lateral
File Name	DSC01330.JPG		
Date of Photo	11/15/2012		
Age (yrs) in photo	29.93		
Date of Photo Assessment	11/19/2012		
Photo Assessor	RSA		

Length of Real Internal Measure of Scale(sticker) placed on forehead (mm): 19.05

Length of Internal Measure of Scale in Frontal Photo (pixels): 168.0

Left Palpebral Fissure Length:	In photo (pixels) 289.8	True Length (mm) 36.1	*Z-score	1.03
Right Palpebral Fissure Length:	In photo (pixels) 284.2	True Length (mm) 35.4	Z-score	0.80
Mean Palpebral Fissure Length:	In photo (pixels) 287.0	True Length (mm) 35.8	Z-score	0.92
Inner Canthal Distance:	In photo (pixels) 294.0	True Distance (mm) 33.3	Z-score	

Flat Philtrum (5-point rank): In Frontal Photo 4 In ¾ Photo 4

Thin Upper Lip: Circularity (perimeter²/area) 40.5 5-Point rank (Circ) 3 5-Point rank (Scale) 3

☐ clown eyebrows ☐ ptosis ☐ strabismus ☐ epicanthal folds

☐ flat midface ☐ protruding ears ☐ flat nasal bridge ☐ hypertelorism

Other anomalies present: None reported

Comments:

Other syndromes present: None reported

PHOTO QUALITY

	Frontal	¾ View	Lateral
Side showing			
Head rotation (5-point rank/degrees) to subject's Right (+) or Left (-)			
Head tilt (5-point rank) toward subject's Right (+) or Left (-) shoulder			
Head tip (degrees) Up (+) or Down (-) from Frankfort Horizontal Plane			
Exposure (3-point rank)			
Focus (3-point rank)			
Facial Expression (3-point rank)			
Reliability of ABC-Score for palpebral fissure length (5-point rank)			
Reliability of ABC-Score for philtrum (5-point rank)			
Reliability of ABC-Score for upper lip (5-point rank)			

PLAINTIFF'S EXHIBIT 33 10-9-12



No Picture Available

No Picture Available

DSC01330.JPG

OUTCOME

	A (PFL)	C (Philtrum)	B (Lip)
ABC-Score			
Data Used	mean	3/4 View	circularity

4-Digit Diagnostic Code for Face: 2: FAS features mild

E4-1 Diagnostic Criteria for Fetal Alcohol Syndrome (FAS) and Alcohol-Related Effects

Fetal Alcohol Syndrome

1. FAS with confirmed maternal alcohol exposure[a]
   A. Confirmed maternal alcohol exposure[a]
   B. Evidence of a characteristic pattern of facial anomalies that includes features such as short palpebral fissures and abnormalities in the premaxillary zone (e.g., flat upper lip, flattened philtrum, and flat midface)
   C. Evidence of growth retardation, as in at least one of the following:
      — low birth weight for gestational age
      — decelerating weight over time not due to nutrition
      — disproportional low weight to height
   D. Evidence of CNS neurodevelopmental abnormalities, as in at least one of the following:
      — decreased cranial size at birth
      — structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia)
      — neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination

2. FAS without confirmed maternal alcohol exposure
   B, C, and D as above

3. Partial FAS with confirmed maternal alcohol exposure
   A. Confirmed maternal alcohol exposure[a]
   B. Evidence of some components of the pattern of characteristic facial anomalies
   C, D, and E
   Either C and D or E

C. Evidence of growth retardation, as in at least one of the following:
   — low birth weight for gestational age
   — decelerating weight over time not due to nutrition
   — disproportional low weight to height
D. Evidence of CNS neurodevelopmental abnormalities, as in:
   — decreased cranial size at birth
   — structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia)
   — neurological hard or soft signs (as age appropriate) such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination
E. Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties; deficits in school performance; poor impulse control; problems in social perception; deficits in higher level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention, or judgment

Alcohol-Related Effects

Clinical conditions in which there is a history of maternal alcohol exposure,[a,b] and where clinical or animal research has linked maternal alcohol ingestion to an observed outcome.

There are two categories, which may co-occur. If both diagnoses are present, then both diagnoses should be rendered:

4. Alcohol-related birth defects (ARBD)
List of congenital anomalies, including malformations and dysplasias

Cardiac	Atrial septal defects Ventricular septal defects	Aberrant great vessels Tetralogy of Fallot
Skeletal	Hypoplastic nails Shortened fifth digits Radioulnar synostosis Flexion contractures Camptodactyly	Clinodactyly Pectus excavatum and carinatum Klippel-Feil syndrome Hemivertebrae Scoliosis
Renal	Aplastic, dysplastic, hypoplastic kidneys Horseshoe kidneys	Ureteral duplications Hydronephrosis
Ocular	Strabismus Retinal vascular anomalies	Refractive problems secondary to small globes
Auditory	Conductive hearing loss	Neurosensory hearing loss
Other	Virtually every malformation has been described in some patient with FAS. The etiologic specificity of most of these anomalies to alcohol teratogenesis remains uncertain.	

5. Alcohol-related neurodevelopmental disorder (ARND)
Presence of:
A. Evidence of CNS neurodevelopmental abnormalities, as in any one of the following:
   — decreased cranial size at birth
   — structural brain abnormalities (e.g., microcephaly, partial or complete agenesis of the corpus callosum, cerebellar hypoplasia)
   — neurological hard or soft signs (as age appropriate), such as impaired fine motor skills, neurosensory hearing loss, poor tandem gait, poor eye-hand coordination

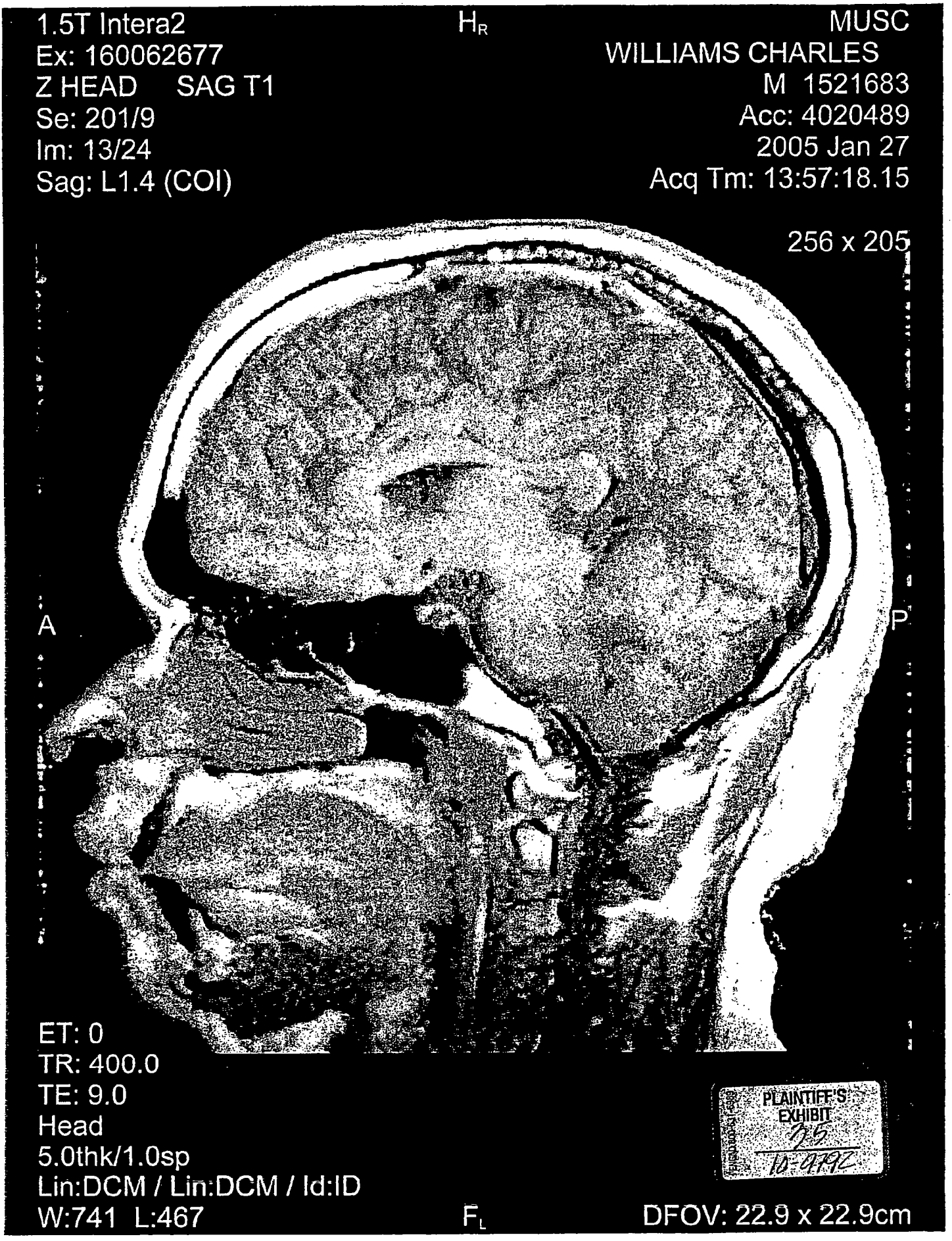
and/or:

B. Evidence of a complex pattern of behavior or cognitive abnormalities that are inconsistent with developmental level and cannot be explained by familial background or environment alone, such as learning difficulties; deficits in school performance; poor impulse control; problems in social perception; deficits in higher level receptive and expressive language; poor capacity for abstraction or metacognition; specific deficits in mathematical skills; or problems in memory, attention, or judgment

[a] A pattern of excessive intake characterized by substantial, regular intake or heavy episodic drinking. Evidence of this pattern may include frequent episodes of intoxication, development of tolerance or withdrawal, social problems related to drinking, legal problems related to drinking, engaging in physically hazardous behavior while drinking, or alcohol-related medical problems such as hepatic disease.

[b] As further research is completed and as, or if, lower quantities or variable patterns of alcohol use are associated with ARBD or ARND, these patterns of alcohol use should be incorporated into the diagnostic criteria.

PENCAD 800-631-6989
PLAINTIFF'S EXHIBIT
34
10-9792


1.5T Intera2
Ex: 160062677
Z HEAD SAG T1
Se: 201/9
Im: 13/24
Sag: L1.4 (COI)
H_R
MUSC
WILLIAMS CHARLES
M 1521683
Acc: 4020489
2005 Jan 27
Acq Tm: 13:57:18.15
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TR: 400.0
TE: 9.0
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Lin:DCM / Lin:DCM / Id:ID
W:741 L:467
F_L
DFOV: 22.9 x 22.9cm
PLAINTIFF'S EXHIBIT
25
10-4-12

Applied Neuroscience, Inc.

228 176th Terrace Drive
St. Petersburg
Florida, 33708
qeeg@appliedneuroscience.com

Phone 727-244-0240
Fax 727-392-1436
email

PLAINTIFF'S EXHIBIT 36 10-9792

Quantitative EEG Analyses

PATIENT INFORMATION
Name: Mr. Williams
Exam#: CHWIEC1
Age: 21.12 years
Sex: Male
Handedness: Right

Medication: None.

RECORDING
Date: January 24, 2004
Ref. By: Jim Evans
Test Site:
Analysis Length: 2:00 Minutes
Ave. EEG Reliability: 0.97 (SH)
Ave. EEG Reliability: 0.92 (TRT)

HISTORY: The client was retained in first grade, and dropped out of school when 17 and in the ninth grade. He reported some head injuries. One of these occurred when he was 5 or 6 and was hit on the left side of his forehead.The other occurred at about the same age when his sister hit him in the head with a hammer. There now is an indentation in his skull (approximatedly at electrode site PZ). He was in an auto accident at 17, but denies any head injury at that time. He reported that he had overdosed on his Mother's "nerve pills" a couple of months before the crime for which he is incarcerated, and implied that this may have caused him to "lose it mentally" at the time of the crime. He denied any significant history of alcohol or illegal substance use, and said that the only illegal substance he ever used was marijuana. There was a report that his Mother is an alcoholic. He reported a history of childhood sexual abuse by an uncle

SUMMARY OF EEG ANALYSES:

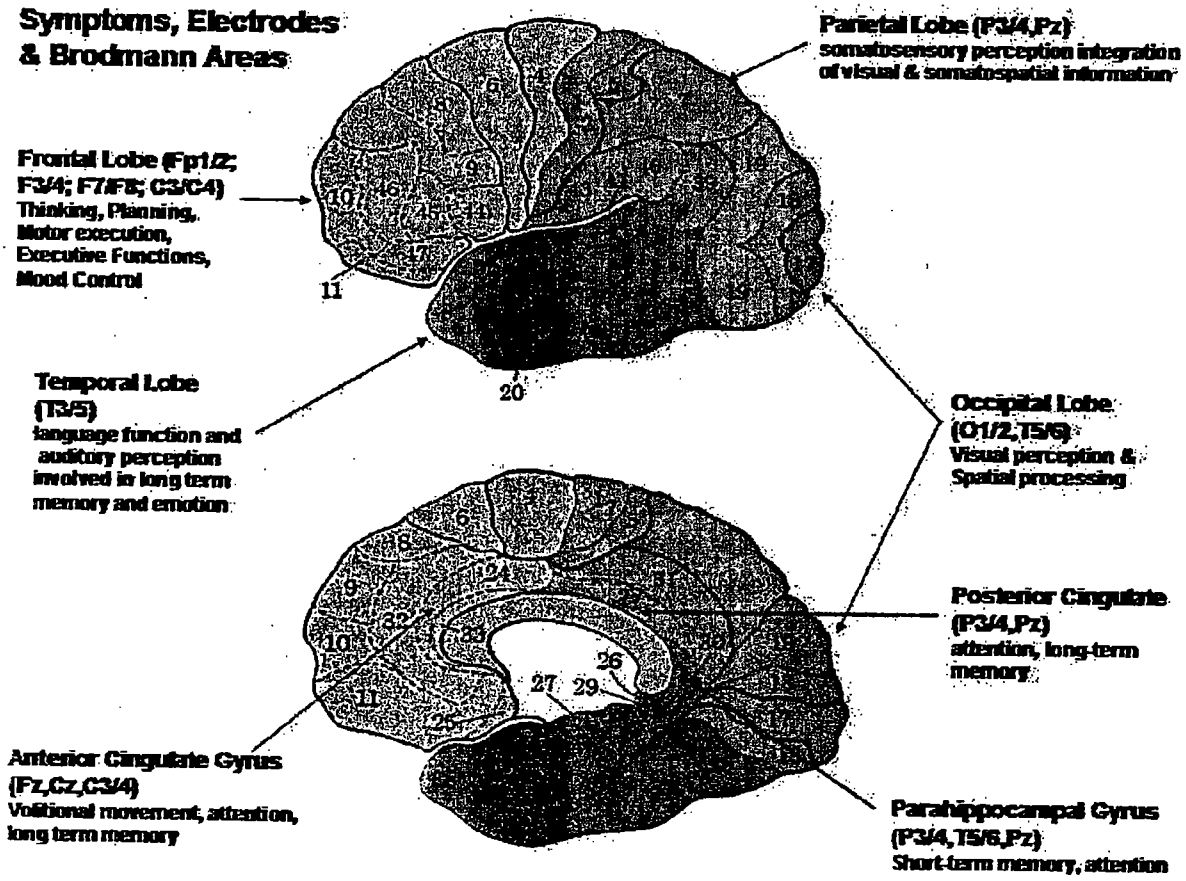
The power spectral analyses were deviant from normal with excessive power in midline posterior regions and especially in the left hemisphere over a wide frequency range. Elevated power was also present in right occipital-parietal regions at 26 - 30 Hz. LORETA 3-dimensional source analyses were consistent with the surface EEG and showed excessive current sources in the left supramarginal gyrus of the parietal lobe and the left superior temporal gyrus with a maximum at 6 Hz (Brodmann areas 13, 39 & 40). Elevated LORETA current sources were present in the middle cingulate gyrus and the mal frontal gyrus with a maximum at 8 Hz (Brodmann areas 6, 24 & 32). Elevated LORETA current sources were

also present in the right inferior and fusiform occipital gyri with a maximum at 28 Hz (Brodmann areas 17, 18 & 19). EEG amplitude asymmetry, EEG coherence and EEG phase were abnormal, especially in bilateral frontal, temporal and parietal-occipital relations. Reduced coherence was present in the bilateral frontal, temporal and parietal-occipital relations which indicates reduced functional connectivity. This condition is often related to reduced speed and efficiency of information processing. The mild traumatic brain injury discriminant function detected a pattern in the EEG that is commonly present in individuals with a history of traumatic brain injury. In summary, the qEEG analyses were deviant from normal and showed de-regulation of the medial frontal lobes, left parietal and bilateral temporal lobes, middle cingulate gyrus and the right occipital lobe. The temporal lobes are involved in auditory information processing, short-term memory, receptive language on the left and face recognition on the right. The left parietal lobe is involved in visual-spatial information processing, short-term memory and receptive language. The cingulate gyrus is involved in volitional motor control and attention control by regulation of limbic emotional and memory input to the cortex. The occipital gyri are involved in visual-spatial information processing and match mismatch of frontal generated expectations and sensory inputs. The frontal regions are involved in mood control, executive functioning, abstract thinking and social skills. To the extent there is deviation from normal electrical patterns in these structures, then sub-optimal functioning is expected.

Robert W. Thatcher, Ph.D., QEEG-D, BCIA, ECNS

Electrical NeuroImaging

Linking a patient's symptoms and complaints to functional systems in the brain is important in evaluating the health and efficiency of cognitive and perceptual functions. The electrical rhythms in the EEG arise from many sources but approximately 50% of the power arises directly beneath each recording electrode. Electrical NeuroImaging uses a mathematical method called an "Inverse Solution" to accurately estimate the sources of the scalp EEG (Pascual-Marqui et al, 1994; Pascual-Marqui, 1999). Below is a Brodmann map of anatomical brain regions that lie near to each 10/20 scalp electrode with associated functions as evidenced by fMRI, EEG/MEG and PET NeuroImaging methods.



Conventional EEG Samples and Quantitative EEG Analyses

Fig. 1 - Example of EEG and Absolute Power Z scores – Linked Ears eyes closed condition

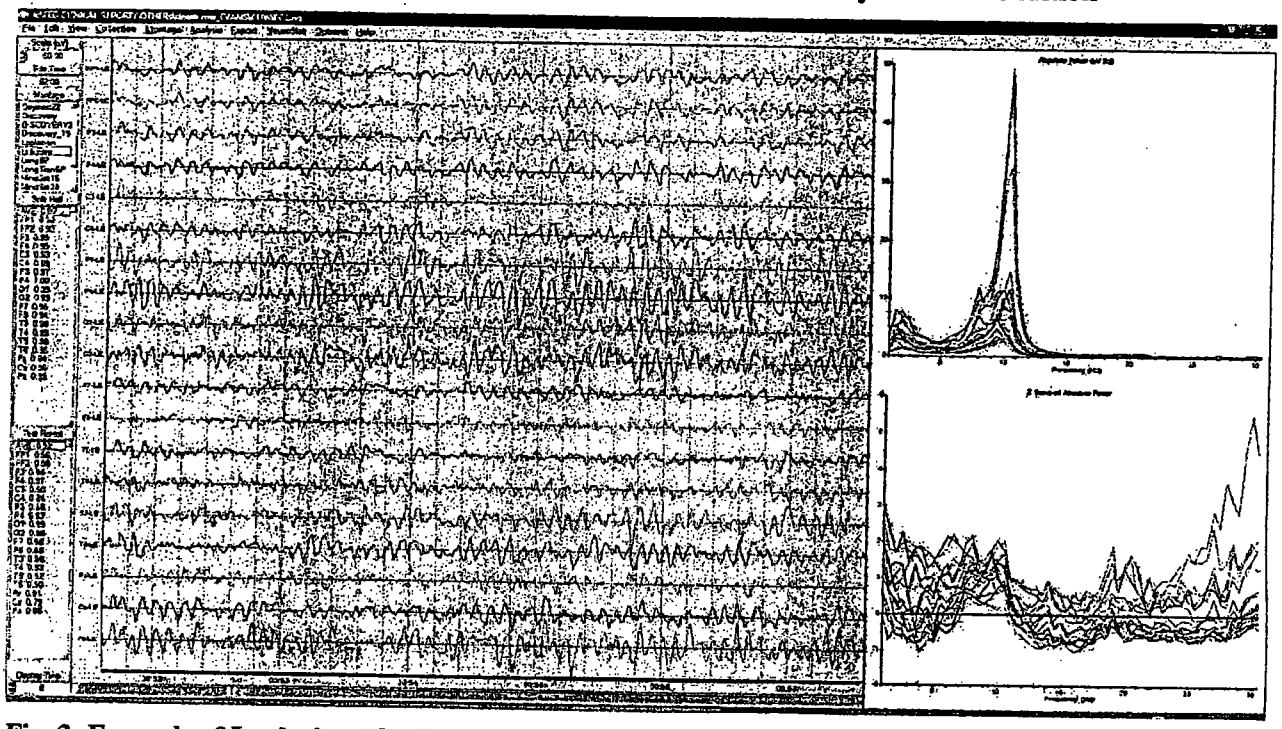


Fig. 2- Example of Laplacian Absolute Power – eyes closed condition

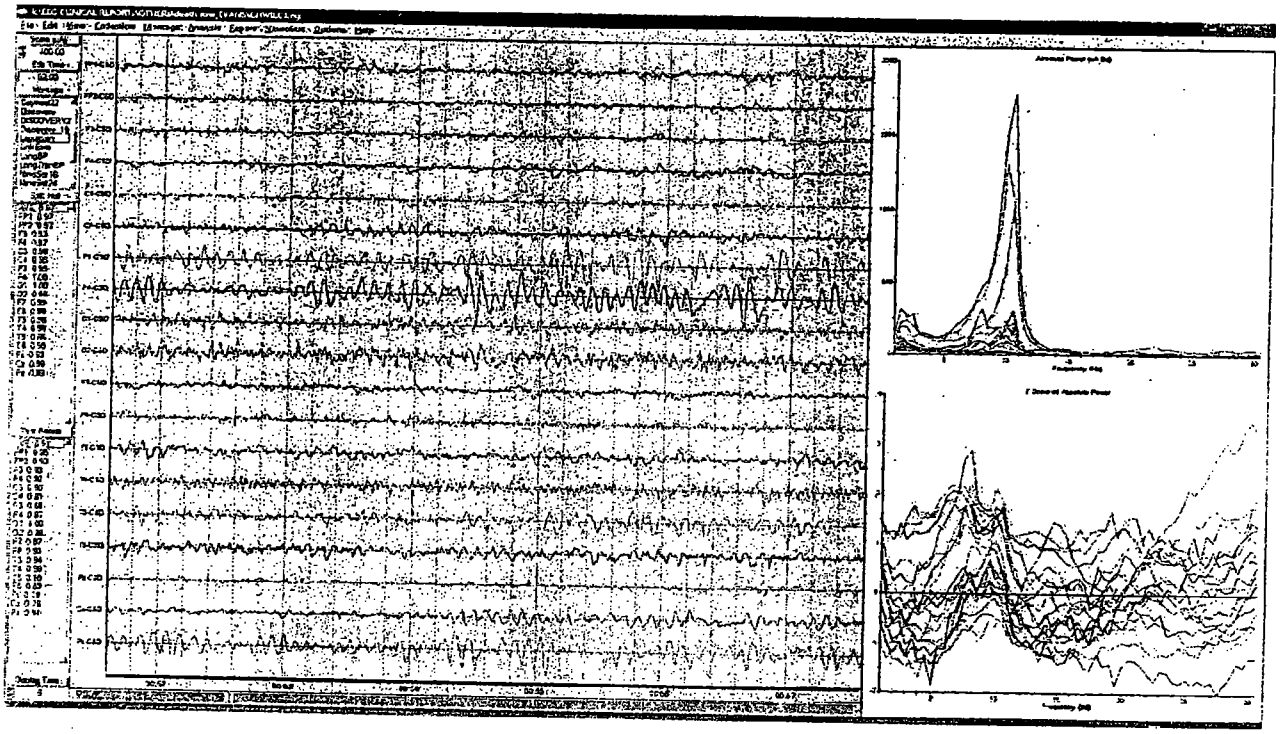


Fig. 3 – Example of LORETA Z Scores at 6 Hz. (Brodmann Area: 13, 39 & 40)

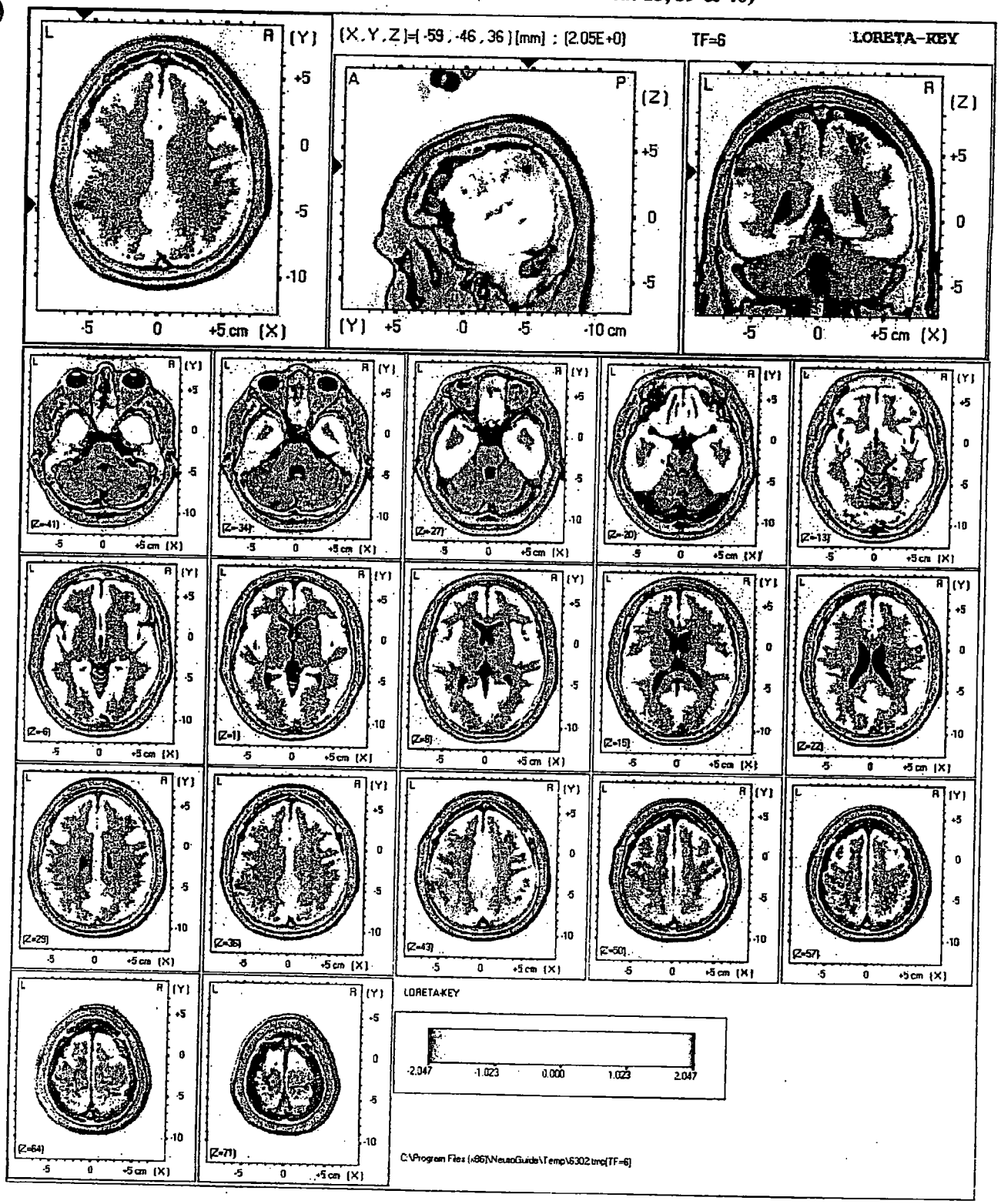


Fig. 4 – Example of LORETA Z Scores at 8 Hz. (Brodmann Area: 6, 24 & 32)

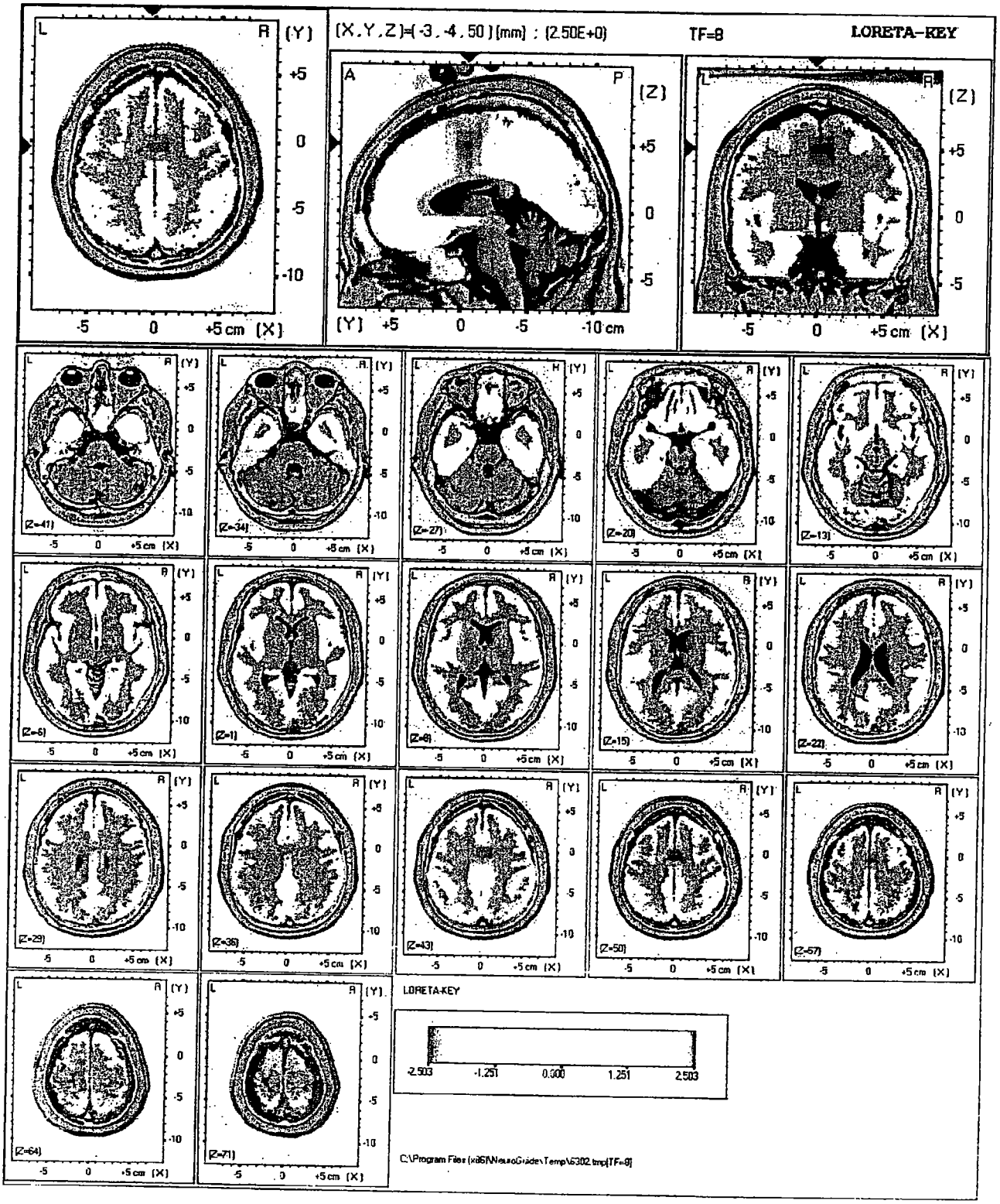
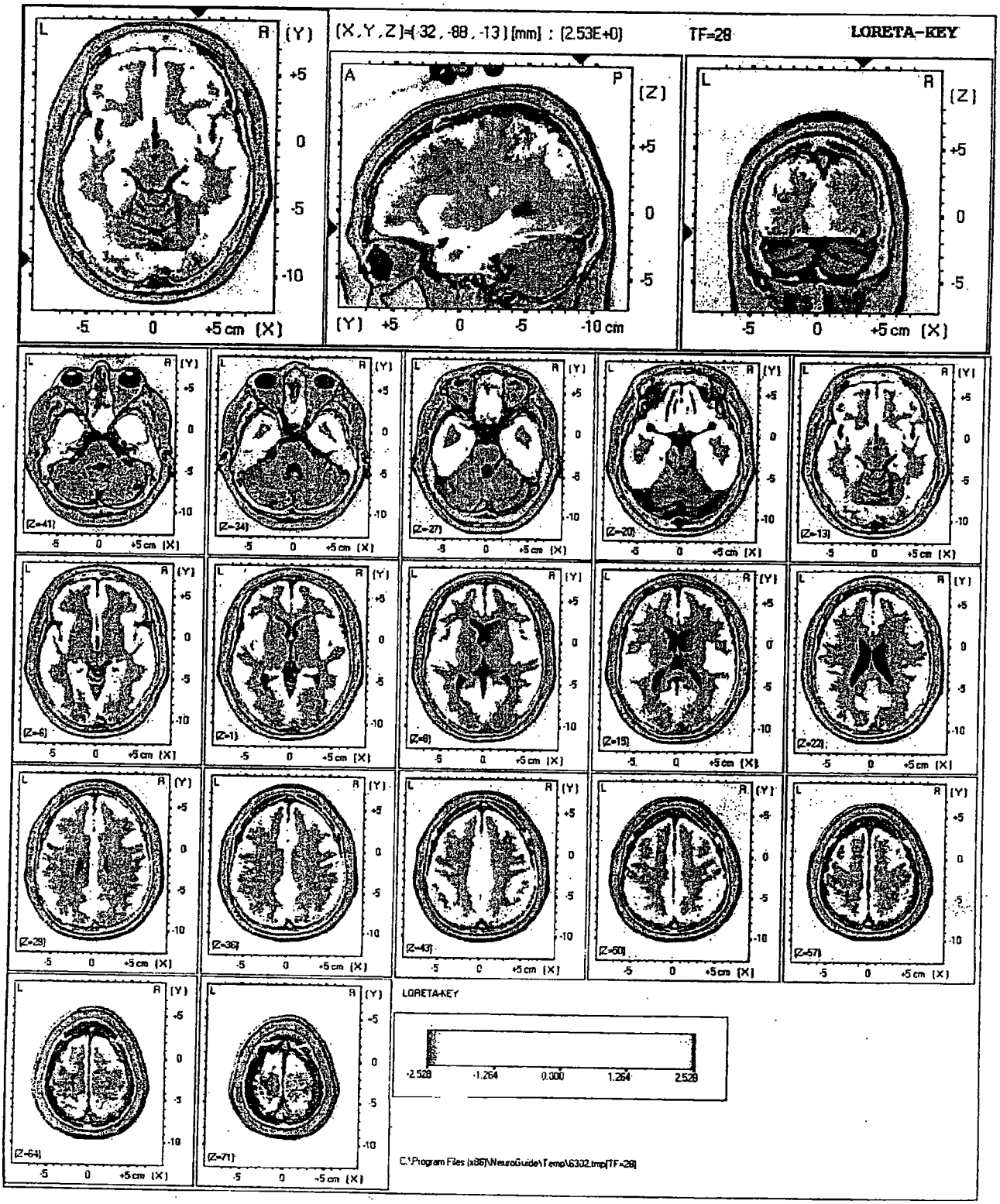


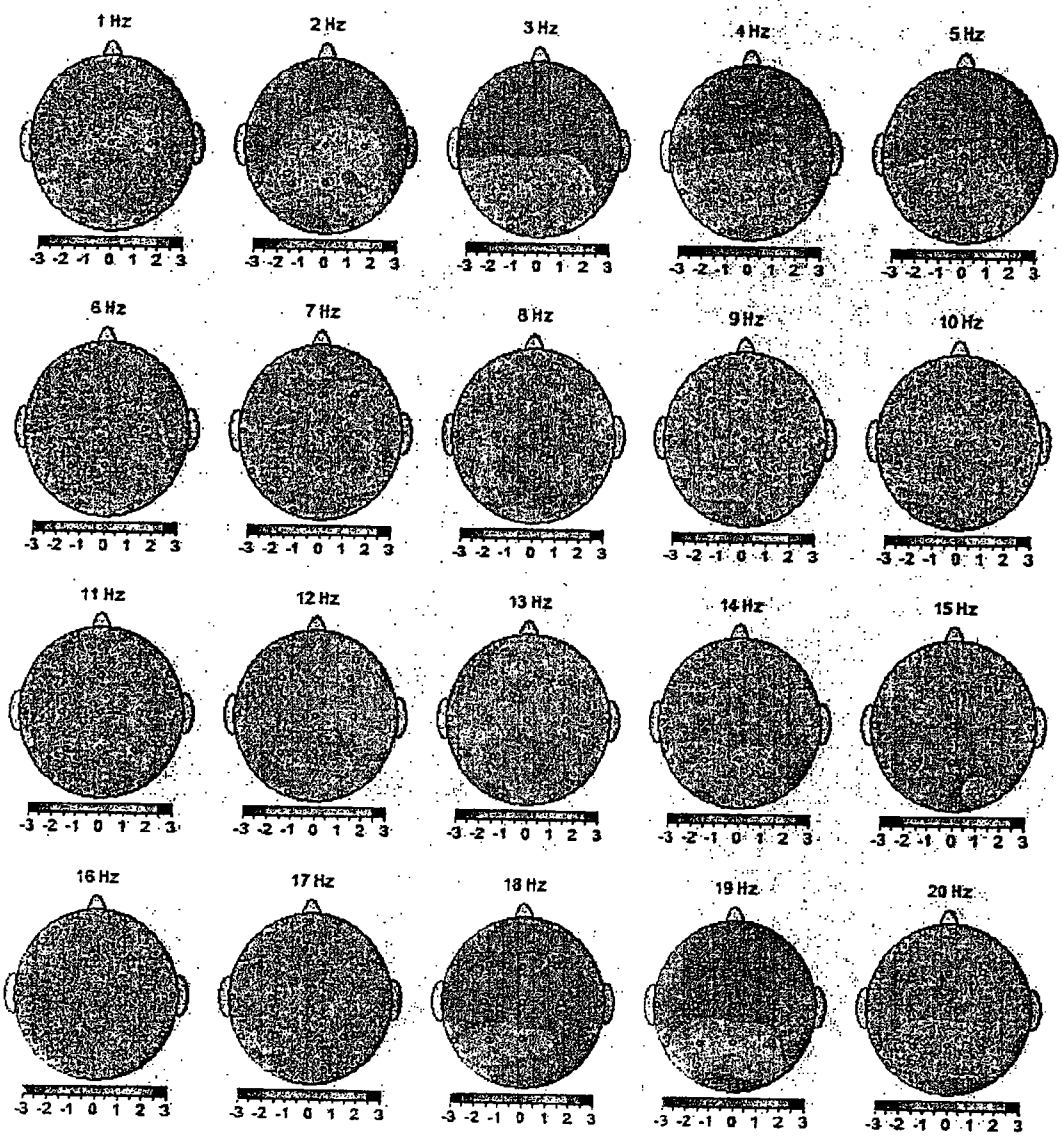
Fig. 5 – Example of LORETA Z Scores at 28 Hz. (Brodmann Area: 17, 18 & 19)



Montage: LinkEars

EEG ID: CHWIEC1

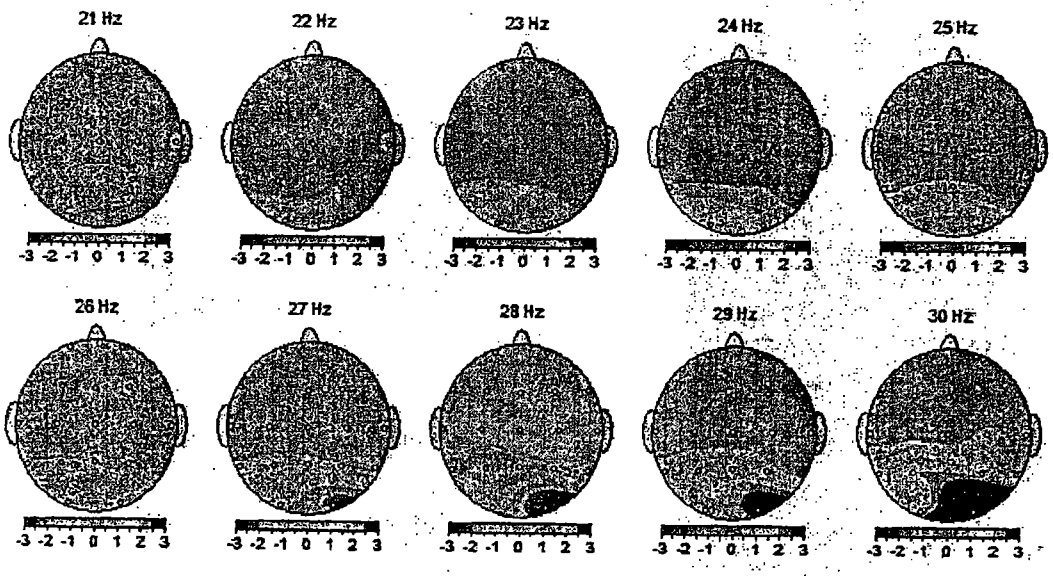
Z Scored FFT Absolute Power



Montage: LinkEars

EEG ID: CHWIEC1

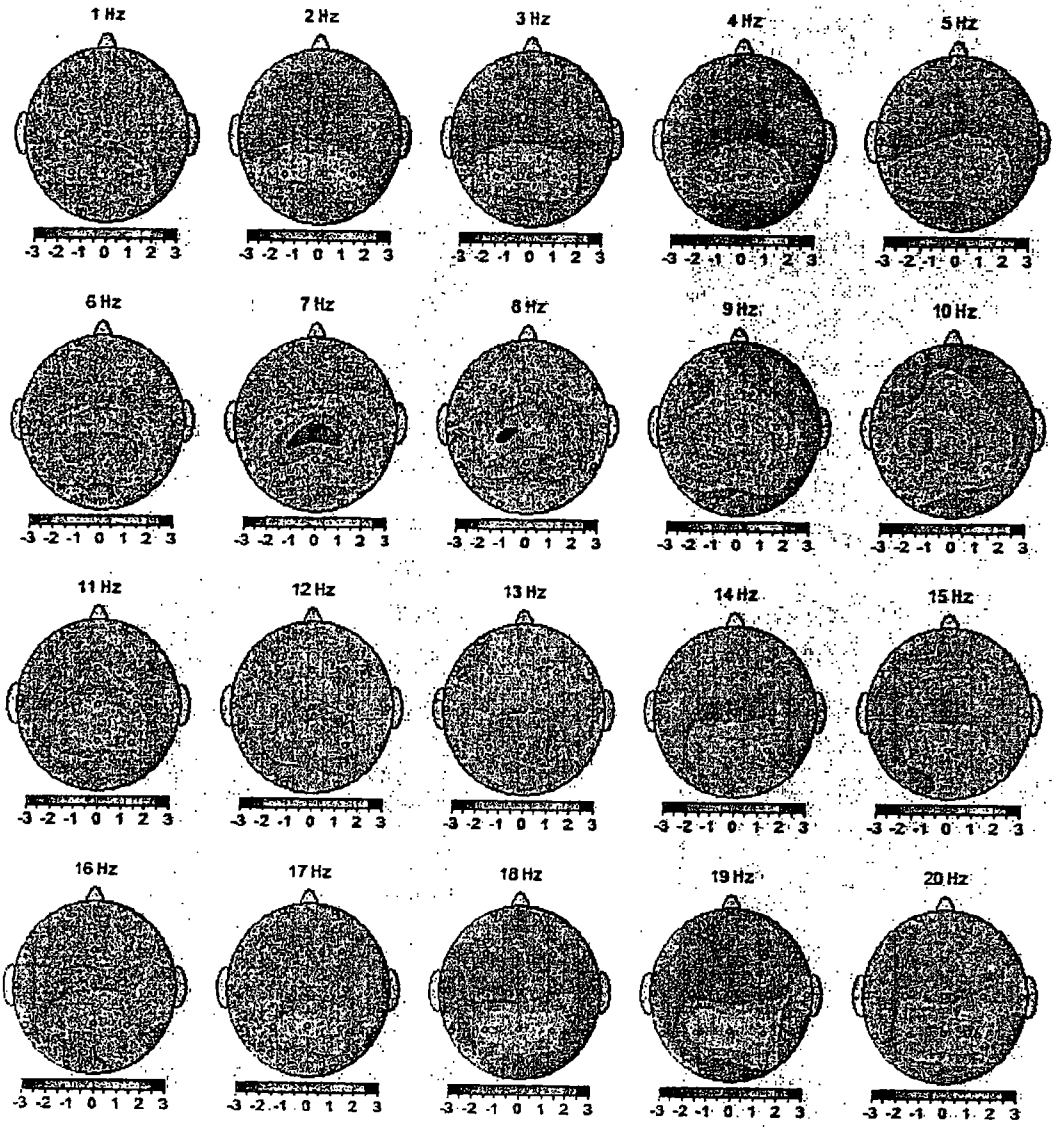
Z Scored FFT Absolute Power



Montage: Laplacian

EEG ID: CHWIEC1

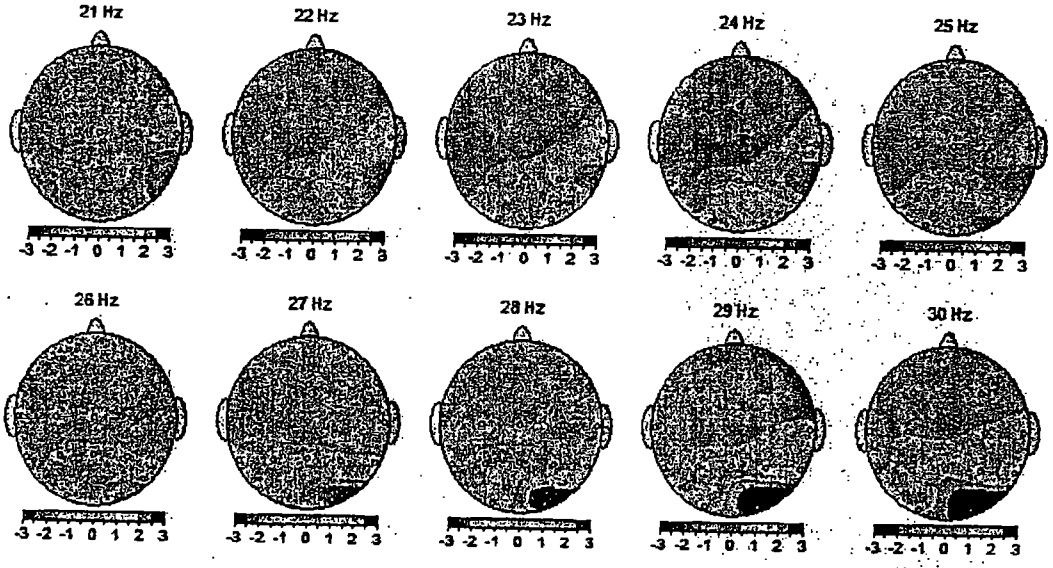
Z Scored FFT Absolute Power



Montage: Laplacian

EEG ID: CHWIEC1

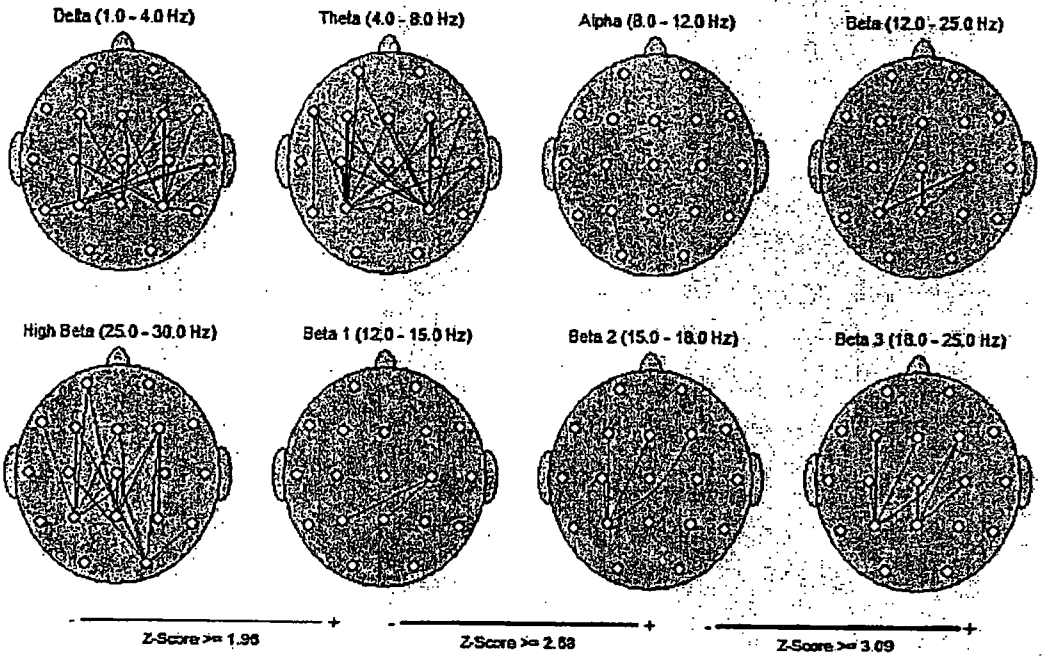
Z Scored FFT Absolute Power



Montage: LinkEars

EEG ID: CHWIEC1

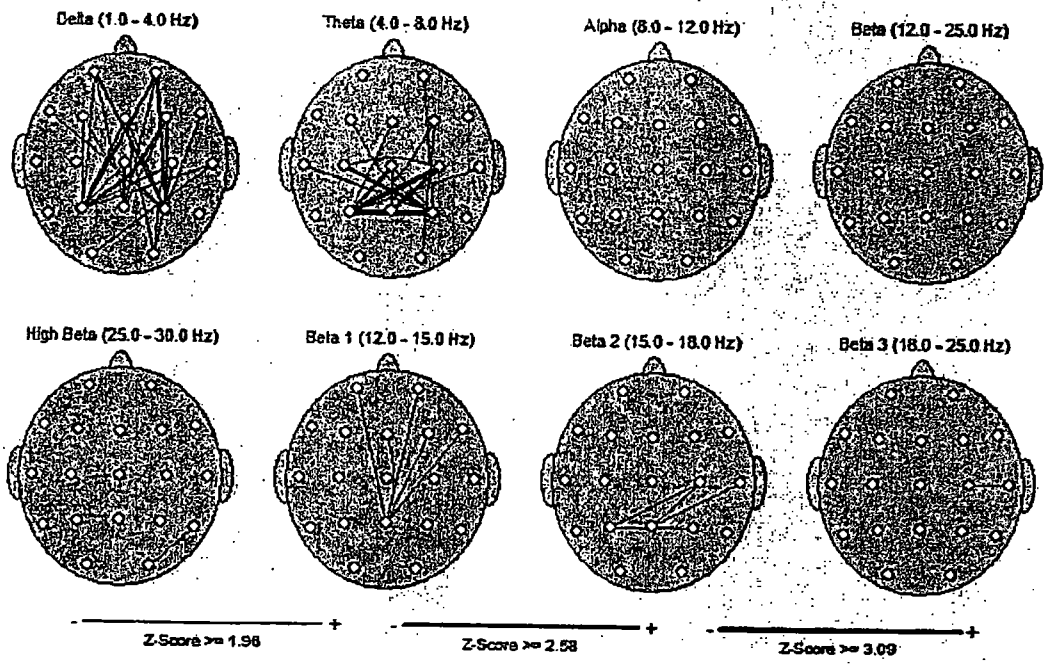
Z Scored FFT Amplitude Asymmetry



Montage: LinkEars

EEG ID: CHWIEC1

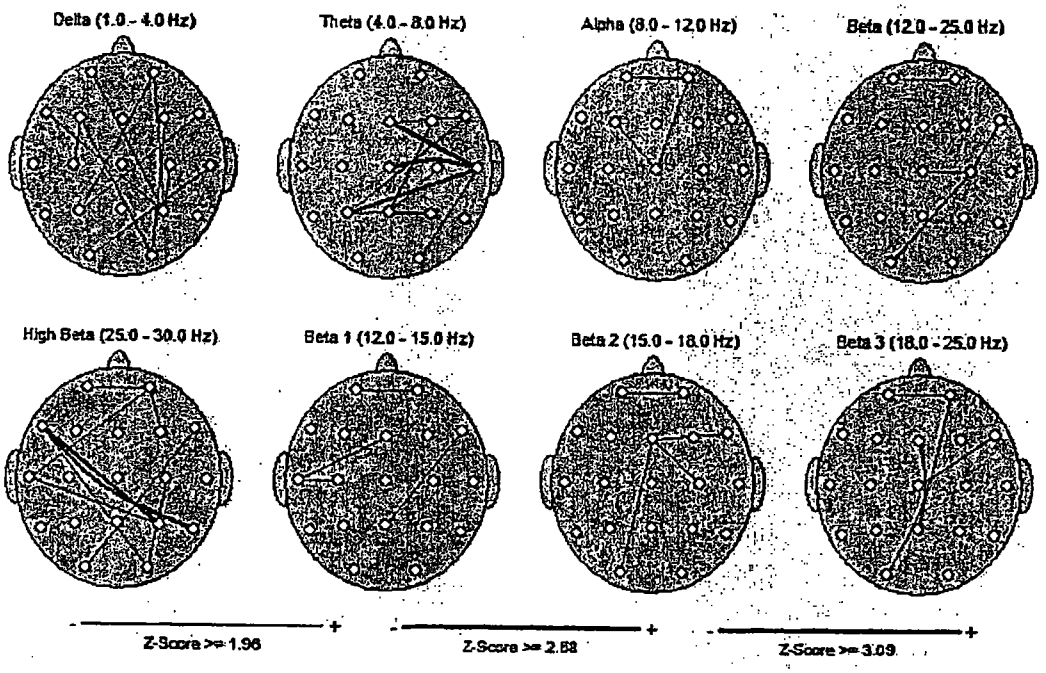
Z Scored FFT Coherence



Montage: LinkEars

EEG ID: CHWIEC1

Z Scored FFT Phase Lag



Montage: LinkEars

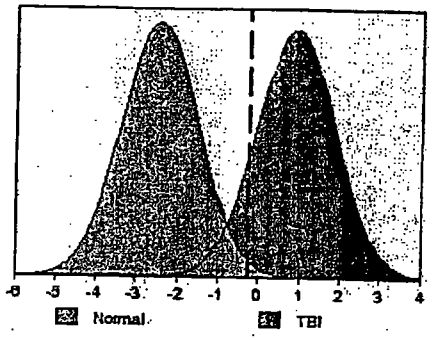
EEG ID: CHWIEC1

Traumatic Brain Injury Discriminant Analysis*

TBI DISCRIMINANT SCORE = -0.28

TBI PROBABILITY INDEX = 85.0%

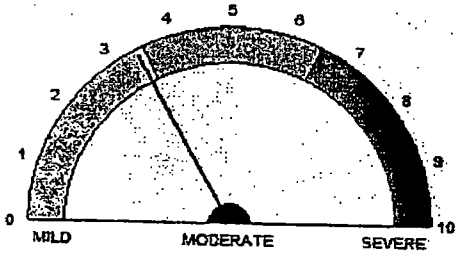
The TBI Probability Index is the subject's probability of membership in the mild traumatic brain injury population. (see Thatcher et al, EEG and Clin. Neurophysiol., 73: 93-106, 1989.)



			RAW	Z
FP1-F3	COH	Theta	87.28	0.96
T3-T5	COH	Beta	71.70	1.65
C3-P3	COH	Beta	73.70	0.37
FP2-F4	PHA	Beta	0.04	-1.35
F3-F4	PHA	Beta	-0.82	0.01
F4-T8	AMP	Alpha	-45.40	0.21
F8-T6	AMP	Alpha	-103.80	0.08
F4-T8	AMP	Beta	-31.47	-0.51
F8-T6	AMP	Beta	-72.36	-0.26
F3-O1	AMP	Alpha	-30.36	1.11
F4-O2	AMP	Alpha	-69.65	0.43
F7-O1	AMP	Alpha	85.03	-1.13
F4-O2	AMP	Beta	-92.08	-0.91
P3	RP	Alpha	70.14	1.09
P4	RP	Alpha	72.41	-1.18
O1	RP	Alpha	55.69	-0.26
O2	RP	Alpha	64.20	0.24
T4	RP	Alpha	52.37	1.26
T5	RP	Alpha	59.35	0.53
T6	RP	Alpha	57.83	0.88

TBI SEVERITY INDEX = 3.39

This severity score places the patient in the MILD range of severity.



			RAW	Z
FP1-C3	COH	Delta	48.63	0.15
FP1-FP2	COH	Theta	94.27	1.45
O1-F7	COH	Alpha	35.73	0.17
O2-T6	COH	Alpha	89.66	0.77
P3-O1	COH	Beta	72.62	-0.04
FP1-T3	PHA	Theta	0.21	-1.84
T3-T4	PHA	Theta	78.84	1.07
O1-F7	PHA	Alpha	-39.09	0.47
F7-F8	PHA	Alpha	-0.07	-1.62
T5-T6	PHA	Beta	3.08	-0.04
C3-F7	AMP	Delta	71.09	0.59
FP2-F4	AMP	Delta	-16.26	0.09
C4-F8	AMP	Delta	67.92	0.62
O1-O2	AMP	Theta	-8.91	-0.27
P3-F7	AMP	Alpha	157.89	0.79
FP2-P4	AMP	Alpha	-151.10	-1.00

The TBI Severity Index is an estimate of the neurological severity of injury. (see Thatcher et al, J. Neuropsychiatry and Clinical Neuroscience, 13(1): 77-87, 2001.)

*Statement of Indications of Use:

The Discriminant Analysis and Severity Index are to be used by experienced and qualified professionals for the post-hoc statistical evaluation of the human electroencephalogram (EEG). The Discriminant Analysis and Severity Index are to be viewed as an adjunct to the evaluation of the patient, and they do not serve as a primary basis for a diagnosis. Warning: Inclusion criteria of a history of traumatic brain injury and greater than 13 years of age must be adhered to.

Montage: LinkEars

EEG ID: CHWIEC1

Technical Information

Record Length: 04:24

Edit Length: 02:00

Reliability:

	Split Half	Test Retest
Average	0.97	0.92
FP1	0.92	0.96
FP2	0.92	0.96
F3	0.95	0.94
F4	0.95	0.91
C3	0.98	0.90
C4	0.99	0.84
P3	0.97	0.88
P4	1.00	0.87
O1	0.99	0.99
O2	0.99	0.98
F7	0.96	0.98
F8	0.94	0.98
T3	0.96	0.98
T4	0.99	0.93
T5	0.98	0.92
T6	0.98	0.90
Fz	0.94	0.91
Cz	0.96	0.79
Pz	0.99	0.86

Sampling Rate: 128

Collection Hardware: Lexicor (High Pass Off)

An Addendum to Neuroguide QEEG Report

Important disclaimer - QEEG tests are ancillary tests that are not intended to provide a diagnosis by themselves, but are used to evaluate the nature and severity of deregulation in the brain such as in mild traumatic brain injury. The QEEG tests provide a quantitative assessment of areas of brain dysfunction and information on impaired conduction and connectivity between different regional neural networks in the brain. The assessment of impaired connectivity is based on abnormal measurements of Coherence and Phase.

The TBI Discriminant does not provide a diagnosis for MTBI but only information on the presence of a pattern in the EEG that is often found in patients with a history of mild traumatic brain injury. The TBI discriminant also provides information about connectivity and excitability of brain regions.

The TBI discriminant is to be used only on patients with a clinical history and symptoms of a Traumatic Brain Injury and Post Concussion syndrome.

The diagnosis of MTBI is a clinical one and is not based on any one test. A diagnosis is performed by the clinician, who integrates the medical history, clinical symptoms, neurocognitive tests with the above mentioned brain function tests as well as other information to render a diagnosis.

The information on impaired brain connectivity is derived primarily from abnormal measurements of Coherence and Phase. Assessments of regional abnormality rely also on abnormal amplitude (power) distribution across the spectrum of EEG frequencies as compared to the normative database.

Description of the Neuroguide Normative Database:

The NeuroGuide normative database in versions 1.0 to 2.4.6 included a total of 625 carefully screened individual subjects ranging in age from 2 months to 82 years. NG 2.5.1 (6/12/2008) involved the addition of 53 adult subjects ranging in age from 18.3 years to 72.6 years resulting in a normative database of 678 subjects. The inclusion/exclusion criteria, demographics, neuropsychological tests, Gaussian distribution tests and cross-validation tests are described in several peer reviewed publications (Thatcher et al, 1983; 1987; 2003). Two year means were computed using a sliding average with 6 month overlap of subjects. This produced a stable and higher age resolution normative database with a total of 21 different age groups. The 21 age groups and age ranges and number of subjects per age group is shown in the bar graph in Appendix F figure 2 in the NeuroGuide Manual (click Help > NeuroGuide Help).

The individuals used to create the normative database met specific clinical standards of no history of neurological disorders, no history of behavioral disorders, performed at grade level in school, etc. Most of the subjects in the normative database were given extensive neuropsychological tests. Details of the normative database are published at: Thatcher, R.W., Walker, R.A. and Guidice, S. Human cerebral hemispheres develop at different rates and ages. Science, 236: 1110-1113, 1987 and Thatcher R.W., Biver, C.L., North, D., Curtin, R. and Walker, R.W. Quantitative

EEG Normative Databases: Validation and Clinical Correlation. Journal of Neurotherapy, 2003, 7(3/4): 87-121. You can download a description of the normative database by going to www.appliedneuroscience.com and clicking on the webpage Articles & Links > Articles > Article #5.

Is there a normative database for different montages including bipolar montages?

Yes. The raw digital data from the same group of normal subjects is analyzed using different montages such as Average Reference, Laplacian current source density, a common reference based on all

19 channels of the 10/20 system and standard clinical bipolar montages (e.g., longitudinal, circular, transverse). Users can create any montage that they wish and there will be a normative reference database comparison available for both eyes closed and eyes open conditions

Age range of the LORETA current density and Source Correlation normative databases

The LORETA current density and source correlation norms use the same subjects as are used for the surface EEG norms and the age range is 2 months to 82 years. The computational details of the LORETA current density norms are published at: Thatcher, R.W., North, D., Biver, C. EEG inverse solutions and parametric vs. non-parametric statistics of Low Resolution Electromagnetic Tomography (LORETA). Clin. EEG and Neuroscience, Clin. EEG and Neuroscience, 36(1), 1 – 9, 2005 and Thatcher, R.W., North, D., Biver, C. Evaluation and Validity of a LORETA normative EEG database. Clin. EEG and Neuroscience, 2005, 36(2): 116-122. Copies of these publications are available to download from www.appliedneuroscience.com by clicking Articles & Links > Articles > Numbers 11 and 12. The computational details of the LORETA source correlation norms are in the NeuroGuide manual, click Help > NeuroGuide help > Appendix-G

Implementation of LORETA measurement in Neuroguide

The Key Institute's LORETA equations and the LORETA viewer (Pacual-Marqui et al, 1994; Pascual-Marqui, 1999) can be launched by a single mouse click in the NeuroGuide window. NeuroGuide Deluxe exports frequency domain and time domain edits of 19 channel x 256 point digital EEG in microvolts (or uv^2) in the Lexicor electrode order as the standard input to the Key Institute T-Matrix. Rows are 256 microvolt time points and the columns are 19 channels at a sample rate of 128 thus producing 0.5 Hz resolution from 1 to 30 Hz. 1 Hz increments in the LORETA viewer are computed as the sum of adjacent 0.5 Hz bins and thus the 'Time Frame' control in the LORETA Viewer is frequency from 1 to 30 Hz. (see Pascual-Marqui RD, Michel CM, Lehmann D., 1994. Low resolution electromagnetic tomography: a new method for localizing electrical activity in the brain. International Journal of Psychophysiology 18:49-65. For computational details see: Pascual-Marqui. R.D., 1999. Review of Methods for Solving the EEG Inverse Problem. International Journal of Bioelectromagnetism, Volume 1, Number 1, pp:75-86. Pascual-Margui, R.D., 2004. Free software and documentation from the Key Institute that was downloaded from http://www.unizh.ch/keyinst/NewLORETA/Software/Software.htm.)

There is a need to match different amplifiers to the amplifier on which the Neuroguide amplifier that acquired the original database information.

This stems from the fact that amplifiers have different frequency gain characteristics. The matching of amplifiers to the Neuroguide database amplifier was done by injecting microvolt calibration signals of different amplitudes and frequencies into the input of the respective EEG machines and then computing correction curves to exactly match the amplifier characteristics of the norms and discriminant functions. The units of comparison are in microvolts and a match within 3% is generally achieved. The Neuroguide research team double checked the amplifier match by computing FFT and digital spectral analyses on calibration signals used to acquire the norms with the calibration signals used to evaluate a given manufacturers amplifiers.

History of the Scientific Standards of QEEG Normative Databases

A review of the history of QEEG normative databases was published in Thatcher, R.W. and Lubar, J.F. History of the scientific standards of QEEG normative databases. In: Introduction to QEEG and Neurofeedback: Advanced Theory and Applications, T. Budzinsky, H. Budzinsky, J. Evans and A. Abarbanel (eds)., Academic Press, San Diego, CA, 2008. A copy of the publication can be downloaded at: http://www.appliedneuroscience.com/HistoryofQEEG%20Databases.pdf

QEEG Normative Database Publications and Validations:

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Neurotherapy Center of Dallas, Inc.

Jonathan E. Walker, M.D.
Neurology
Clinical Neurophysiology
EEG Biofeedback

Chris Williams

PATIENT NAME: C. H. Wiec
REFERRING PHYSICIAN: Dr. James Evans@eva328927@charter.net

RAW EEG REPORT

EYES CLOSED EEG: There is some well-developed posterior dominant 9 to 10 Hz activity with some independent frontal 8 to 9 Hz activity seen intermittently fairly frequently. There is also some theta activity at 6 to 7 Hz seen in frontal, central and temporal regions. No persistent background asymmetries are evident and no paroxysmal discharges are seen.

EYES OPEN: There is some persistent low amplitude 10 to 11 Hz activity and, again, there is independent intermittent frontal alpha activity at about 10 Hz which is often present when the occipital alpha is not as prominent. No persistent background asymmetries are evident and no paroxysmal discharges are seen.

IMPRESSION: Abnormal because of the prominent intermittent independent frontal alpha activity. This is consistent with the neuropsychological testing suggesting some frontal lobe dysfunction and probable attentional difficulty and memory problems. I did not see any abnormalities in the parietal regions on the EEG. The low coherence in the parietal on the QEEG and the abnormal phase at P4 and F8 certainly would be suggestive of residual effects from a closed head injury. I am not aware that abnormalities in the raw EEG have been described in schizophrenia. However, there is a schizophrenia-discriminate in the E. Roy John database. I do not know if you can run your data through that database but we do have the E. Roy John database if you wanted us to do that. I talked with Robert Lawson about doing that if you wish. In addition to EEG there have been abnormalities of the topography 300 response in schizophrenia. This was described by Duffy Archives General Psychiatry, 4728 to 4734, 1983. A type paper describing EEG characteristics and subtyping of schizophrenia was given by E. Roy John and his associates in Biological Psychiatry, 1994, Volume 36, Pages 801 through 25. They also found differences in the QERP profiles. Unfortunately, the QERP

ABSTRACTION - ROBERTS

PLAINTIFF'S EXHIBIT 37 10-9-97

12870 Hillcrest Road • Suite 201 • Dallas, Texas 75230

database is no longer available. PET and MRI abnormalities have been described in schizophrenia with decreased activity in the frontal lobes and caudate. The reference is CNS Spectrums, Volume 2, No. 4, April 1997, by Monty Buchsbaum and colleagues. Dr. Grizelure did a review of abnormalities in schizophrenia and schizotypy in Clinical EEG, Volume 31, Pages 23 to 29. He felt that the operant conditioning of cortical slow potentials and known as the contingent negative variation might be useful in training more normal brain activity in schizophrenics. I have not personally treated schizophrenia but my recollection is that in the John database there is an increase in the relative power of theta in both temporal lobes in most schizophrenics. If that is not what the QEEG showed then I probably would not train theta in the temporal lobes. But if it is elevated that would be worth trying.

Sincerely,

J.E. Walker, MD

Jonathan E. Walker, M.D.
Board Certified in Neurology and EEG

JW/ems
Dictated but not read. Subject to transcription error.

[redacted]

HOSPITAL TIMELINE

CONFIDENTIAL ATTORNEY WORK PRODUCT

January 19th 2003

6/13/03

GHS POINT OF ENTRY HISTORY

CW is admitted to the ER at 4:50 am

Pt. overdosed on Klonopin and Estradiol. Told Ems he took 40 pills at about 4:00am

Triaged to CC, Room 3

No Respiratory distress

Medications: crack

Cannot read physician assessment handwriting.

Under physician orders, records shows charcoal administered and then OD, Depression, and non-existent coping skills

Disposition shows him improved at discharge, mental information given, can't read rest or signature

The case information sheet shows the admitting physician to be Karl D. Kelley

GHS Emergency Department Sheet with Timeline

Shows allergy to crack

4:50am Pt to CC 3 no stretcher per EMS. EMS reports pt called for help stating "I took 40 pills." Pt presents to ER drowsy-does respond with occasional one word answers. Neg. assessment completed-see flow sheet

4:55am 16 Fr Foley inserted-clear yellow urine return

5:15am IB FR NG tube to R nose. X-ray ordered

5:35am Rad techs @ hs for pcsr.

6:00am X-ray OK per Mr. Hoffert. Activated charcoal. Pt continues sleeping and unresponsive. Resp. spontaneious. VSS

6:35am Pt's mother and father @ hospital Family updated on POC

7:15am Report to Suzy RN. Pt. asleep, no change

7:30 am Pt asleep-Resp. even unlabored-family at bs

8:30am Pupil unreactive-total 350cc urine ouptut noted-gag reflex present-suction mouth with some secretions-some charcoal noted

Discharge time 1800, ambulatory, resp unlabored

PHYSICIAN RECORD

TIME SEEN 5:10am, room CC 3 EMS arrival

Historian: patient and ems

Suicidal Thoughts Moderate to severe
Suicide Attempt Intentional Drug Overdose

Context:
Situational Problems/significant other

Current associated complaints

depressed, angry, frustrated

Suicidal thoughts (Can't read doctor note)

Other problems-Unknown

Allergies Crack?

Smoker, drugs, single

Doctor conducts physical exam

disconjugate gaze, no response to commands, withdraws to pain

Unable to assess future suicidal behavior

orientation uncooperative

Abnormal gait (Can't read doctor notes)

Procedures

Restraints, Charcoal

Drug Screen Positive for Tricyclics

NURSING ASSESSMENT

Clean appearance, drowsy, oriented to person, Speech slurred, no sensory deficit, no difficulty hearing, response to pain, Airway clear, respirations spontaneious, Breath sounds clear, Heartbeat regular, cap refill less than three seconds, heart sounds, S1 and S 2, neck veins flat, edema absent, pulse radial R +2, L +2, DPR +2 L+2, Monitor SR, Bowel sounds present, abdomen soft, Foley, Skin Elastic, Temp normal, musculo, intact

Nurses social history indicates, smokes, drinks occ/not at all, under domestic violence and abuse, unable to assess, problem overdose

SOCIAL WORK ASSESSMENT

SW. note 6/13/03

Pt is a 20 year old SBM seen in ER after pt treated for apparent OD on Klonopin. Pt told EMS he took Klonopin but later told SW he could not remember what he took. ER staff told SW pt's UDS positive on for tricyclic antidepressants, not Klonopin. Pt. said he had an argument with a girlfriend and got upset and then took OD, per pt. Pt denied using ETOH or illegal drugs. Pt has no hx of suicide attempt, no psych tx inpast and no drug tx in past, per pt. Pt said he did not know if he was feeling suicidal or homicidal at time of interview. Pt denies visual or auditory hallucinations. SW. to consult with ER Dr. re disposition DS Klasing.
Per ER Dr. request, contract for safety written to be signed by patient and pt's mother Daisy Williams, Sw. waiting on Mrs. Williams to arrive in ER to sign safty contract. DS Klasing, LMSW

Progress Notes

Referred to Piedmont Mental Health
20 Powderhorn Road, Simpsonville
233-8175 or 963-3421

open mon-fri 8:30-5 pm

Contract For Safety by Responsible person

Chris contracts not to attempt to harm himself or others and to follow up with outpatient counseling on 6/16. If thought of harming others or myself occur, I will call 911 or seek assistance immediately at the nearest emergency department. Signed by Chris and Klasing and Physician

Daisy then signs statement that she fully understands the need to supervise Chris and all possible means of harm will be carefully monitored. Signed by Daisy and Klasing and Physician, Dr. S. Kilgore.

GHS SYSTEM Emergency Department Sheet 6/13/03

Shows drugs given and dosages, temp, blood pressure

8:50 Medicated as ordered-Will continue to assess for alertlessness

9:05 Dr. Kilgor at bs to talk with pt-able to open his eyes to questions

9:50 Pt waking some-pulled NB tube-Pulled Foley

10:00 Ammonia inhalant at the face, some awake noted will continue to assess

10:30 Pt still sleeping at intervals-able to wake at times -moving extremeties

10:50 Dr. Kilgore at bedside-talking with pt

11:08 EKG-f hr cardicu ez-draw to lab

11;40 Dr. Kilgore @bs talking with pt's family

12:00Family at bs-pt waking some

1:30 _____on S. worker to talk with family

2:10 Pt asleep-moved to hallway to evaluate

3:10 Pt asleep-will wake will _____ continue to assess

4:25 Report from Suzy RN and pt asleep no distress and easily awakene

5:00 SW _____for evaluation

6:00 Discharge instruction given-verbalize understanding, some numbers, family at bs-escorted pt out _____

Interdisciplinanary Patient/family education flowsheet

6:25

It looks as though a nurse did this assessment

Learning needs home care needs, patient safety and Patient rights and responsibilities

Person taught-patient

Beadiness to Learn No limitations/barriers

Learning Preferences Written

Department Nursing

Teaching Method Care Notes, Written

Otcome Able to verbalize or demonstrate understanding of content. No review necessary

Cannot read Topic Discussed

CONFIDENTIAL ATTORNEY WORK PRODUCT

CHRIS WILLIAMS SECOND ADMISSION TO GHS ER FOR OD

January 15, 2003

GHS EMERGENCY DEPARTMENT POINT OF ENTRY HISTORY

Arrival date 6/15/03 Time 3:40am

Triaged at 3:40, triage level III

Chief complaint: Tried to kill self on Wednesday night with OD; warned he might try to kill himself again.

This time under allergic to medictions it reads N/CA

Signed by RN Wellborn

There is a strange square drawn under physician assessment

Physicians orders reads depression, social services, and a few unreadable marks.

Disposition reads MHC Monday

Condition at discharge stable

GREENVILLE HOSPITAL SYSTEM EMERGENCY DEPARTMENT

3:45 am Amb to _____ with SI
security watch called for to chg nurse

4:15 am Moved to 10B-placed in four _____

4:17 am Security _____

5:00 am _____

6:15 am Pt sitting up in chair_____

7:18 am D/C instructions provided to pt-stated understanding. no further c/o voiced

Evaluation Yes

Discharge time 7:18
To home
ambulatory
Accompanied by self
Neuro status A and O
Resp: unlabored

EMERGENCY PHYSICIAN RECORD

Historian: patient

Context: SI ___with OD now_____
_____to go home _____

related to parent, significant other, lost job

afraid he might hurt himself

Current associated complaints

depressed
(then marks through other choices that are hard to decipher)

Arrived by private car
all systems neg. except as marked

Past History

prior suicide attempt
psychiatric problems-depression

Nursing assessment reviewed

Neuro/psych modd/affect, depressed affect

Orientation normalx3

NURSING ASSESSMENT

Appearance clean
Behavior cooperative
Orientation Person , Place, Time
Speech clear and appropriate
Sensory no deficit
Hearing no difficulty
Response to Stimuli Spontaneously
Respirations spontaneous
Cardio edema
gastro no complaint
Urinary pressure
Temp normal
musculo intact

SOCIAL HISTORY

Smokes half pack per day
Drinks occasionally
No to family abuse
Education 12 years, able to read and write

Problem SI

GREENVILLE HOSPITAL SYSTEM 24 HOUR BEHAVIORAL RESTRAINT DEVICE RECORD

6/15/03 3:45 am Physician signs restraint order

Reason for restraint SI-flight risk

open door

Restraint Use Discussed with Patient/Family Date: 6/14/03 Time 3:45

signed by nurse

It looks as though he was restrained for three hours

GREENVILLE HOSPITAL SYSTEM MENTAL HEALTH REFERRAL FORM

Please follow up with Greenville MHC Present date 6-15-03 Patients name: Chris Williams
DOB [redacted] SS# [redacted] Home phone 288-05-5
Address 706 Waxford Way, Simpsonville, 29681

Reason for Referral: Pt seen in ER on this date with c/o tried to kill self on Wednesday. Worried he might try again. Pt. reports lots of stress-job, parents, girlfriend. Pt. too OD on Wednesday. At present pt has no suicide plan and does not want to kill self. Pt denies psych hx, denies HI, denies hallucinations, denies delusions, denies ETOH, drugs. No hx of suicide attempts. Please assess. Polles Edwards (She is a social worker who is no longer there. I don't know what her credentials were as the hospital hires "social workers" from a number of backgrounds)

PROGRESS NOTE BY SOCIAL WORKER
6/15/03

Consulted by ER RN to see pt who is a 20 BM who presents alone with c/o tried to kill self on Wednesday by OD. Worried he might try again. Spoke with pt who reports a lot of stress and things. Pt very depressed on Wednesday, took OD on multiple pills belonging to his parents. App 45 pills. Pt. came to ER, was d/cd the next day. Pt denies hx of suicide attempts, denies psych tx hx. Denies hx of depression. Pt denies suicide plan tonight-just fells it could happen again. Pt. does not want to hurt himself, wants to prevent another suicide attempt. Pt would not kill himself due to his family=doesn't want to hurt them. Pt. is stressed due to family problems-parents fight daily, job problems, girlfriend, problems. Pt feels he can't handle it alone. Pt not sleeping well, decreased appetite, denies etoh, denies, drugs, denies hallucinations. Pt states he would consider outpt tx. Pt works at BiLo _____..

Polles Edwards

INTERDISCIPLINARY PATIENT/FAMILY EDUCATION FLOWSHEET

6/15 Learning needs patient safety, diagnosis, community resources
Person Taught patient
Readiness to Learn No limitations/barriers
Learning Preferences no preference
Department Nursing
Teaching Method Written, verbal
Outcome Able to verbalize or demonstrate understanding of content. No review necessary

Looks as though signed by nurse

DISCHARGE INSTRUCTIONS

You need to make a follow-up appointment with mental health center to make sure your problem is taken care of. The care you were given in the ER was for your emergency only. Your appointment should e made for Monday. If your insurance says you can only go to certain doctors, please check with them before making this appointment. They may give you a different doctor to see.

You should return to the ER if you fell worsening symptoms develop new problems.

6/17 dictation shows positive for tricyclics from first admission