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IN THE STATE OF SOUTH CAROLINA
In the Court of Appeals

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APPEAL FROM SUMTER COUNTY
Court of Commons Pleas

SC Court of Appeals

The Honorable W. Jeffrey Young, Circuit Court Judge

C.A. No.: 2013-CP-43-02284
Appellate Case No.: 2014-002215

Arrowpoint Capital Corporation/Arrowood Indemnity Co., Appellant,

v.

South Carolina Second Injury Fund, Respondent,

IN RE: Mary McConico, Employee,

v.

Yuasa-Exide, Inc., Employer, and Arrowpoint Capital Corp./Arrowood Indemnity Co., Carrier

**RECORD ON APPEAL
VOLUME II**

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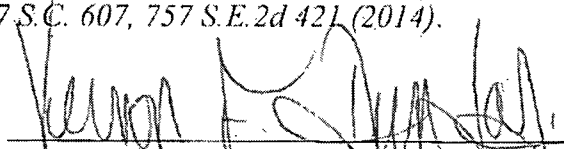
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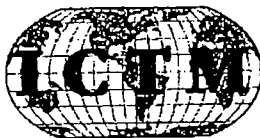
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The undersigned hereby certifies that the Record on Appeal contains all material proposed to be included by any of the parties and not any other material. The undersigned further certifies that the Record on Appeal filed in this matter complies with the South Carolina Supreme Court's Order and caption *In re: Advised Order Concerning Pers. Identifying Info. & Other Sensitive Info. In Appellate Court Filings*, 407 S.C. 607, 757 S.E.2d 421 (2014).


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INTERNATIONAL CENTER
FOR TOXICOLOGY AND MEDICINE

Literature Review

Lead and Health Effects

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November 27, 2009

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Lead is one of the most useful elements in industry;¹ it is resistant to corrosion, easily molded and shaped. "Lead and lead alloys are commonly found in pipes, storage batteries, weights, shots and ammunition, cable covers and sheets used to shield us from radiation. The largest use for lead is in storage batteries in cars and other vehicles."² The diversity of uses of lead has resulted in increased levels of lead in the environment. Human exposure to lead is common and results from the many uses of this metal due to its exceptional properties.² Prior to 1987 lead was used as a pigment in paints and was used as an additive in gasoline until 1995. In the general US population surveys, National Health and Nutrition Examination Surveys (NHANES) the mean blood lead level in 1976 was 15.8 µg/dL for US citizens.³

An enormous amount of information is available on the health effects of lead, many effects of lead have been known for centuries.² "Lead can induce a wide range of adverse effects in humans depending on the dose and duration of exposure."⁴ "Under conditions of extremely high respiratory exposure to lead, an acute encephalopathy can develop, accompanied by renal failure and severe gastrointestinal symptoms."⁵ In most cases, lead is absorbed more slowly and the clinical course is subacute or chronic. The adverse effects of chronically elevated blood lead levels range from the inhibition of enzymes associated in the synthesis of hemoglobin to severe pathology such as cerebral edema, usually associated with blood lead levels more than 150 µg/dL.⁵ The following table, from the toxicology textbook, *Clinical Environmental Health and Toxic Exposures*, highlights the range of adverse effects on adults based on blood lead levels.

TABLE 73-6. Summary of lowest observed effect levels for key lead-induced health effects in adults

Lowest observed effect level (blood lead (µg/dL))	Heme synthesis and hematologic effects	Neurologic effects	Renal effects	Reproductive effects	Cardiovascular effects
100-120	—	Encephalopathic signs and symptoms	—	—	—
80	Anemia	Encephalopathy symptoms	Chronic nephropathy	—	—
60	—	—	—	Reproductive effects in women	—
50	Reduced hemoglobin production	Overt subencephalopathic neurologic symptoms	—	—	—
40	Increased urinary ALA and elevated coproporphyrins	Peripheral nerve dysfunction (slowed nerve conduction)	—	—	—
30	—	—	—	Altered testicular function	Elevated blood pressure (white men, aged 40-59)
25-30	Erythrocyte protoporphyrin elevation in men	—	—	—	—
15-20	Erythrocyte protoporphyrin in women	—	—	—	—
<10	ALA-D inhibition	—	—	—	—

ALA, aminolevulinic acid; ALA-D, aminolevulinic acid dehydratase.

Table 73-6 Clinical Environmental Health and Toxic Exposure⁵



Encephalopathy, Dementia & Neurobehavioral Effects in Adults

Confusion, is a behavioral state of reduced mental clarity, coherence and capacity to reason. Inattention and disorientation are the main early signs, however, as the confusional state worsens there is deterioration in memory, perception, comprehension, problem solving, language and various aspects of emotional behavior.⁶ "When there is in addition to confusion an element of drowsiness, the patient is said to have an encephalopathy."⁷

"Dementia is a syndrome with many causes [and] affects more than 4 million Americans; 10% of persons over 70 and 20-40% of individuals over 85 have clinically identifiable memory loss."⁶ A simple definition of dementia is a deterioration in cognitive abilities that impairs successful performance of activities of daily living. Memory is the most common cognitive ability lost with dementia. "The most common causes of dementia are: Alzheimer's disease, Multi-infarct dementia, Alcoholism, Parkinson's disease and medication intoxication."⁶

The literature on an association between blood lead levels and neurobehavioral effects are inconsistent.^{8,9} The ATSDR's *Toxicological Profile for Lead* acknowledges that "the literature contains numerous case reports and small cohort studies that describe a higher incidence of these [non-specific] symptoms, including malaise, forgetfulness, irritability, lethargy, headache, fatigue, impotence, decreased libido, dizziness, weakness and paresthesia at blood leads that range from approximately 40 to 120 µg/dl following acute-, intermediate-, and chronic duration occupational exposure."²

In a meta-analysis in 2000, Meyer-Baron and Seeber found a statistically significant effect for 3 of 13 tests, Block Design, Logical Memory, and Santa Ana (dominant hand) tests.² A follow up meta-analysis in 2002 by Goodman et al found only 2 of 22 tests to have a statistical difference between exposed and unexposed workers, the Digit Symbol and D2 tests and no difference in the three tests found to be statistically different by Meyer-Baron and Seeber. Goodman et al concluded that "The data available to date are inconsistent and are unable to provide adequate information on the neurobehavioral effects of exposure to moderate blood concentrations of lead. A lack of consistency should caution against far reaching conclusions about the true biological effects of low to moderate lead concentrations."⁸

Despite the inconsistency in the literature, the ATSDR noted, "in studies where adults were exposed occupationally to lead, a number of neurobehavioral parameters were reportedly affected. Although as Goodman et al. (2002) pointed out, the lack of true measures of pre-morbid state, observer bias, and publication bias affect the overall assessment, the preponderance of the evidence indicates that lead is associated with neurobehavioral impairment in adult workers at blood leads below 70 µg/dl."² The abnormal neuropsychological test finding, even if truly associated with elevated blood lead levels, may be reversible and of no long-term significance.¹⁰ Lindgren et al found



"reversibility of function may occur when proximate blood lead level is maintained below 40 µg/dl."¹¹

Renal Effects

The kidney is a very complex organ, "One of the most highly differentiated organs in the body. Nearly 30 different cell types can be found. This panoply of cells modulates a variety of complex physiologic processes."⁶ The glomeruli are complex filters with fenestrated epithelia, the renal tubules are composed of highly differentiated cells that vary dramatically in function along the nephron allowing for selective transport of ions, molecules and fluids. Each of these specialized and unique cell types provide very unique functions. The complexity of the kidney with its many different cell types provides for a multitude of injuries each with different clinical effects and each with a multitude of different causes. "Well-described risk factors for the progressive loss of renal function include systemic hypertension, diabetes and activation of the renin-angiotensin-aldosterone system. Cigarettes smoking has been shown to either predispose or accelerate the progression of nephron loss. Lipid oxidation associated with obesity can accelerate progressive renal damage."⁶

Chronic kidney disease encompasses a spectrum of abnormal kidney function, and a progressive decline in glomerular filtration rate. Chronic renal failure typically corresponds to chronic kidney disease stages 3-5. End-stage renal disease represents a stage of chronic kidney disease where the accumulation of toxins, fluids and electrolytes that require removal by renal replacement therapy using dialysis or kidney transplant.⁶

Based on population survey data, "at least 6% of the adult population in the United States has chronic kidney disease at stages 1 or 2. An additional 4.5% of the U.S. population is estimated to have stage 3 and 4 chronic kidney disease."⁶ Diabetes mellitus and hypertension are the leading causes of chronic renal failure.

Acute exposure to high concentrations of lead can result in proximal tubular dysfunction, manifest by glycosuria and aminoaciduria. The renal manifestations of acute lead nephrotoxicity are usually reversible after treatment and removal from lead exposure.^{5,12}

Chronic exposure to high blood lead levels may cause chronic interstitial nephritis which may then progress to progressive nephron loss and renal failure, hypertension, hyperuricemia and gout.^{1,4,5} The dose and duration of lead exposure in relationship to renal dysfunction has been extensively studied. "Studies suggest that blood lead level of 60 µg/dl is the threshold for proximal tubular cell injury from lead. This finding is consistent with the observation of Buchet et al that workers who do not have a blood lead level over 62 or 63 µg/dl for up to 12 years do not have lead nephropathy."¹³ In a group of 53 workers with an average blood lead of 47 µg/dl, with 12 of the workers blood lead greater than 60 µg/dl and one with a blood lead over 70 µg/dl, the authors found "no adverse effects on kidney function were detected in the exposed

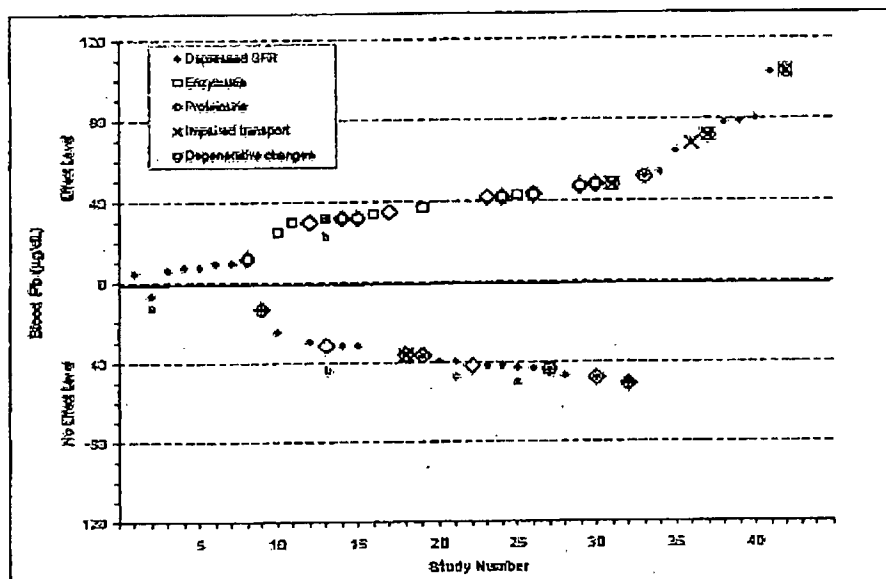


population."¹⁴ In his review, *Renal Effects of Environmental and Occupational Lead Exposure*, Loghman-Adham concluded, "Chronic lead poisoning may result in what has been termed lead nephropathy. The latter seems to develop when blood lead levels exceed a threshold of 60 µg/dl."¹⁵

The duration for significant exposure required to induce renal effects has been investigated in many studies. Chronic lead nephropathy may develop after "years of excessive exposure."¹⁵ In a study of smelter workers with an average of 18 years exposure (minimum 5 years) to continuously elevated blood lead levels (range 26 to 68 µg/dl) the authors found "that in adult male workers with long term moderate occupational exposure to lead, no significant change in the urinary excretion of low or high molecular weight proteins is detected and that serum concentrations of creatinine were not affected."¹⁶ They concluded that "adverse renal changes are unlikely to occur in most adult male lead workers when their blood lead concentration is regularly kept below 70 µg/dl."¹⁶ Another study of active (14.3 years of exposure) and retired (32.6 years of exposure) lead smelter workers, the authors concluded that "despite many years of moderate to heavy exposure to lead no signs of adverse effects on the kidney such as early tubular or glomerular malfunction were found."¹⁷

The Agency for Toxic Substances and Disease Registry's (ATSDR) *Toxicological Profile for Lead* describes the findings of a number of studies regarding the relationship between blood lead and qualitative aspects of kidney response. The studies reveal "Inconsistencies in the reported outcomes [including proteinuria and glomerular filtration rate (GFR)] across studies may derive from several causes. Epidemiological studies of general populations have shown a significant effect of age on the relationship between glomerular filtration rate and blood lead levels. Furthermore, hypertension can be both a confounder in studies of associations between lead exposure and creatinine clearance and a covariable."²

The ATSDR's review included the attached chart; "a cluster of observations of decrements in glomerular filtration rate appear at the low end of the blood lead range (<20 µg/dl range)."² The review contained seven studies that evaluated either serum creatine or creatine clearance in populations with blood lead levels below 10 µg/dl. The ATSDR indicates that the study authored by Dr. Hu¹⁸ found an "increase in creatinine clearance" associated with exposure to lead.² The remaining six studies had problems with results that conflicted with other studies of the same populations, made invalid conclusions or suffered from the lack of inclusion of covariants known to be strongly associated with the study outcome.



*Refer to Table 3-3 for study details (Indexed by study number)

a - Increase in creatinine clearance; b - >45 years of age; c - Decrease in serum creatinine; GFR - glomerular filtration rate

The first study cited in the above chart represents an analysis of the NHANES III data, the authors discuss finding of an association between higher blood lead levels and a higher prevalence of elevated serum creatinine; they do not indicate if this association was statistically significant. They compared persons with and without hypertension and found a significant difference, "mean blood lead and serum creatinine were higher and estimated GFR was lower for persons with hypertension. In contrast, no association was present between blood lead and either elevated serum creatine or chronic kidney disease among persons without hypertension"¹⁹ The association found in analysis of the NHANES III data was not found in a previous NHANES review, "No interaction between blood lead, blood pressure and kidney function was found from NHANES II."¹⁹

The third study in the low exposure group was undertaken "to determine whether long-term low-level environmental lead exposure plays a role in the progression of renal insufficiency in patients with chronic renal insufficiency."²⁰ The authors make an invalid conclusion; "long-term low-level lead exposure may subtly affect progressive renal insufficiency in the general population."²⁰ They attempt to generalize findings from persons with chronic renal insufficiency at the onset of their study to the general



population; any such attempt to generalize to non-similar populations is incorrect and such conclusions are invalid.

The next two^{21,22} studies involved evaluation of data from the Normative Aging Study, both analyses "considered the potential covariates known to be associated with blood lead and/or serum creatinine levels."²² While both of these studies considered age, body mass index, smoking status, alcohol intake, and hypertension, neither of these studies included diabetes which is the most frequent cause of chronic kidney disease.⁶ Payton et al noted the limitations of their study including the absence of sources of confounding could result in erroneous conclusions; "Renal function is determined by many factors, as in any epidemiologic study, there may be unknown sources of confounding, such as an unmeasured variable that could explain the relation of lead exposure to creatinine clearance."²¹ Kim et al "investigated the relationship between low-level lead exposure and renal function using longitudinal data from the Normative Aging Study."²² Without the inclusion of diabetes as a covariate both studies found that low level lead exposure correlates with impairment in renal function. However, in another evaluation of the same data from the Normative Aging Study, Wu et al "did not find any significant association between blood lead and impaired renal function as measured by serum creatinine or estimated creatinine clearance."²³

"Outcomes for the various renal toxicity end points are mixed over the blood lead range 20-50 µg/dl."² In contrast to the very low level studies, depressed glomerular filtration rate was, "with only one exception, not observed over this blood lead range."² The studies that produced the one exception in this range were analyses of South Korean lead workers in which the authors "found that lead has an adverse effect on renal function in the moderate dose range, particularly in older workers."^{24,25} The authors compared six renal outcomes with three hematological outcomes and four lead exposure markers, therefore they generated 42 model analyses. "When influential outliers were removed, higher lead measures remained associated with worse renal function in nine models. An additional five associations were in the opposite direction."²⁴ Therefore, 33 models found no association (including the measured creatine clearance) or actual improvement in outcomes with elevated blood lead. The findings were inconsistent across the age groups studied, "in several of the clinical outcome models, associations in the youngest age tertile were in the opposite direction of associations in the oldest age tertile. Higher lead measures were associated with worse renal function in older workers, but with lower BUN and serum creatinine in younger workers."²⁴

The authors of the South Korean studies noted the significance of covariates and confounding; "because non-lead related factors contribute to both renal dysfunction and elevated blood pressure, lack of adjustment for these variables in such models likely results in residual confounding."²⁵ Analysis of the covariates and confounders included in each of these studies revealed the likelihood of serious under estimation of "diabetes and hypertension the leading underlying etiology of chronic renal disease and end stage renal disease."²⁶ The authors state that covariates assessed "included diabetes and



hypertension (both based on participant report of physician diagnosis).^{24,25} Using self reported diagnoses revealed the prevalence of diabetes in the study population to be 0.8% and the prevalence of hypertension in the study population to be 7.2%. These prevalences are significantly less than the reported prevalences of the general South Korean population. In a population survey in 1999, the prevalence of diabetes was found to be 15.8%, although only 5.8% of the population had previously been diagnosed.²⁷ This was confirmed in the Korean Health and Genome Study which found the prevalence of diabetes to be 12.5% and hypertension to be 25.1% in Korean men.²⁸ The significant difference in the prevalence of diabetes and hypertension in these study populations and the general Korean population make any conclusions suspect as the "variables in such models likely result in residual confounding."²⁵ Support for a lack of association between blood lead and renal dysfunction when significant covariables are included in the analysis was evidenced by Dr. Hu et al in 2004, "In the absence of diabetes or hypertension, we did not see statistically significant associations of bone lead levels with either cross-sectional or longitudinal measures of renal function."²⁹

The ATSDR review concluded that "the overall dose-effect pattern suggests an increasing severity of nephrotoxicity associated with increasing blood lead with deficits in function and pathological changes occurring in association with blood leads exceeding 50 µg/dl."² This conclusion is supported by the consensus that long term exposure to blood lead levels in excess of 60 µg/dl may cause progressive lead induced renal disease.^{1,13-17,30}

Gout

Gout is a metabolic disease most often affecting middle-aged to elderly men. Acute arthritis is the most frequently early clinical manifestation of gout. Usually, only one joint is affected initially, but polyarticular acute gout can occur in subsequent episodes. The metatarsophalangeal joint of the first toe is often involved, but tarsal joints, ankles, and knees are commonly affected.

Gout is the result of an increased body pool of urate with hyperuricemia. Hyperuricemia may be related to the overproduction of uric acid, or the under excretion. Excretion of >800 mg of uric acid per 24 hours on a regular diet suggests that causes of overproduction of purine should be considered.⁵ A 24-hour urine collection for uric acid can, in some cases be useful in assessing the risk of stones, in elucidating overproduction or under excretion of uric acid. Serum uric acid levels can be normal or low at the time of the acute attack. This limits the value of serum uric acid determinations for the diagnosis of gout. Nevertheless, serum urate levels are almost always elevated at some time.

The relationship of gout and lead is due to the accumulation of lead in the proximal tubular cells which affect urate excretion.⁵ The basic toxicologic principle regarding dose and duration of exposure hold for lead and gout. Dr. Hu et al noted that chronically elevated "levels of lead (e.g., blood lead levels >60 µg/dL) are clearly



associated with gouty arthritis."³¹ Others have concluded that "Chronic high-dose lead exposure has been implicated in the development of gout."¹

Diabetes Mellitus

Diabetes mellitus (DM) is a common metabolic disorder resulting in hyperglycemia (high blood sugar). "The metabolic dysregulation associated with DM causes secondary changes in multiple organ systems that impose a tremendous burden on the individual with diabetes. In the United States, DM is the leading cause of end-state renal disease (ESRD). It also predisposes to cardiovascular disease."⁶

The prevalence of DM has risen dramatically over last 30 years. The rise in type 2 DM has been much more rapid because of increasing obesity and reduced activity levels. DM increases with age, in the US the prevalence of DM is about 10% in individuals aged 20-60, and 21% in persons over the age of 60. "DM prevalence also varies among different ethnic populations. In 2005, the Centers for Disease Control and Prevention (CDC) estimated that the prevalence of DM in the United States, (age > 20 years) was 13.3% in African Americans, 9.5% in Latinos and 8.7% in non-Hispanic whites."⁶

Type 2 DM has a strong genetic component, "individuals with a parent with type 2 DM have an increased risk of diabetes; if both parents have type 2 DM, the risk approaches 40%. The disease is polygenic and multifactorial, since in addition to genetic susceptibility, environmental factors (such as obesity, nutrition, and lack of physical activity) modulate the phenotype."⁶

The chronic complications of DM affect many organ systems and are responsible for the majority of morbidity and mortality associated with the disease. The chronic complications include, renal disease, retinopathy, neuropathy, coronary artery disease, peripheral arterial disease, and cerebrovascular disease. The risk of coronary heart disease events and mortality are two to four times greater in patients with type 2 DM.⁶

The ATSDR's Toxicological Profile for Lead² section on endocrine effects of lead does not consider lead to be associated with diabetes mellitus. There is no support to consider lead as a causal factor for the development of diabetes mellitus or any of its many chronic complications.



Coronary Heart Disease, Cardiomyopathy and Stroke

Atherosclerosis is a major cause of death and disability in developed societies. Atherosclerosis of the coronary arteries leads to Coronary Heart Disease (CHD) causing heart attacks and angina. Atherosclerosis of the arteries supplying the central nervous system frequently provokes strokes. Atherosclerosis can affect the renal vessels causing renal disease. The clinical expression of atherosclerosis may be chronic or a dramatic acute events such as a heart attack, stroke or sudden cardiac death.⁶

CHD is "the most common, serious, chronic, life-threatening illness in the United States."⁶ Obesity, type 2 diabetes mellitus, elevated cholesterol, hypertension and smoking are powerful risk factors for CHD. Other risk factors such as age and male gender are strong non-modifiable risk factors for disease.

The ATSDR's Toxicological Profile for Lead section on cardiovascular effects does not consider CHD or stroke to be potential effect of lead exposure.² Pocock et al found that after controlling for cigarette smoking there was no association between blood lead and heart attack or stroke. They concluded, "we see no convincing epidemiological evidence at present to support the claim that moderate elevations in body lead burden are of relevance to the risk of cardiovascular disease."³² There is no support to consider lead as a causal factor for the development of coronary heart disease, cardiomyopathy or stroke.

Peripheral Neuropathy

Peripheral neuropathy is a general term indicating peripheral nerve disorders of any cause. Peripheral neuropathies affect up to 8% of adults; the incidence increases with age.⁶ The multitude of causes and manifestations of such disorders make diagnosis bewildering.⁷ Evaluation begins with a history focusing on the time course of the illness, symptoms, medical conditions that predispose to neuropathy (e.g. diabetes mellitus, hypothyroidism, connective tissue disease, infection such as Lyme and HIV, and nutritional deficiency) toxic exposures (e.g., alcohol, arsenic, and lead) and family history. The distribution of sensory, motor and reflex changes determines whether the neuropathy is asymmetric or symmetric and whether the neuropathy is a polyneuropathy or a mononeuropathy. Electrodiagnostic studies help to classify the neuropathy into one of the three major categories, axonal, demyelinating, or neuronal.⁶

The ATSDR's *Toxicological Profile for Lead* section on neurologic effects of lead exposure reports that there are numerous studies available on peripheral nerve function.² Given the multitude of disease entities that are included under such a broad term, it is not surprising to find that the results of the studies are inconsistent. Ishida et al found no significant association between blood lead and median nerve conduction velocity.³³ Ehle reviewed many of the nerve conduction studies and "concluded that a



mild slowing of certain motor and sensory NCVs may occur at blood leads below 60 $\mu\text{g/dl}$, but that the majority of studies did not find correlations between blood lead and nerve conduction velocities below 70 $\mu\text{g/dl}$.³⁴ Several studies found improvement in NCV of the median (motor) NCV after decrease of blood lead levels. The conclusion of the ATSDR's review, "The results of these studies indicate that NCV effects occur in adults at blood leads <70 $\mu\text{g/dl}$, and possibly as low as 30 $\mu\text{g/dl}$."²

Anemia

"The anemia induced by lead is microcytic and hypochromic and results primarily from both the inhibition of heme synthesis and shortening of the erythrocyte lifespan. Lead interferes with heme synthesis by altering the activities of δ -aminolevulinic acid dehydratase (ALAD) and ferrochelatase. Correlations between blood lead and log erythrocyte protoporphyrin (EP) or zinc protoporphyrin (ZPP) indicate an apparent threshold for EP elevation in male workers at 25-35 $\mu\text{g/dl}$. In summary, of all the parameters examined, ALAD activity appears to be the most sensitive indicator of lead exposure. The threshold for blood lead increases in urinary ALA in adults is approximately 40 $\mu\text{g/dl}$; for increases in blood EP or ZPP, the threshold in adults is around 30 $\mu\text{g/dl}$."²

Erectile Dysfunction

Erectile dysfunction is associated with physiologic aging. In the Massachusetts Male Aging Study, a survey of men 40 to 70 years of age, 52% of respondents reported some degree of erectile dysfunction, 35% was of moderate or complete. The incidence of erectile dysfunction is also higher among men with certain medical disorders such as diabetes mellitus, obesity, lower urinary tract symptoms secondary to benign prostatic hyperplasia, heart disease, hypertension, and decreased HDL levels. Smoking is a significant risk factor in the development of erectile dysfunction. Medications used to treat diabetes or cardiovascular disease are additional risk factors.⁶

The ATSDR's Toxicological Profile for Lead section on reproductive effects does not even mention an erectile dysfunction as a potential reproductive effect of lead exposure.² The toxicologic profile does indicate that the literature supports lead induced changes in the quantity and quality of sperm in males with elevated blood lead levels. "Although the evidence for reduced fertility is not conclusive, it appears that a threshold for fertility effects in men could be in the blood lead range of 30-40 $\mu\text{g/dl}$."²



Hypertension

Hypertension is an extremely common chronic illness present in all populations. In the United States, based on results of National Health and Nutrition Examination Survey (NHANES), 33.5% of non-Hispanic blacks and 28.9% of non-Hispanic whites have hypertension. "Hypertension doubles the risk of cardiovascular diseases, including CHD, congestive heart failure, ischemic and hemorrhagic stroke, renal failure and peripheral arterial disease."⁶

There are many well recognized factors that contribute to blood pressure and idiopathic hypertension including age, obesity (elevated Body Mass Index – BMI), dietary salt intake, alcohol consumption, psychological stress, low levels of physical activity, and many medications. Hypertension increases with age; 65.4% of individuals 60 years of age or older have hypertension. In these hypertensive individuals, approximately 80-95% have primary or idiopathic hypertension. The prevalence of hypertension also varies with geographic region; "The prevalence of hypertension and stroke mortality rates are higher in the southeastern United States than in other regions."⁶ Family studies document a significant inheritable component to blood pressure and hypertension, in fact high blood pressure before age 55 occurs 3.8 times more frequently among persons with a family history of hypertension. "In African Americans, hypertension appears earlier, is generally more severe, and results in higher rates of morbidity and mortality from stroke, left ventricular hypertrophy, congestive heart failure and end-stage renal disease."⁶

In contrast to the many well known and accepted causal risk factors for the development of hypertension, "On balance across all human studies the relationship between blood pressure and blood lead concentration is inconsistent."³⁵ At first glance, meta-analysis of the literature may show a weak association between blood lead levels and blood pressure, however, further analysis of the literature provides no support for a causal relationship and raises significant questions regarding whether even an association exists.

The early controversy regarding a possible association relates to initial analysis of the NHANES II data.³⁶ Despite the inconsistent findings of the study, Harlan et al concluded that "analyses of the NHANES II data provide a link between blood lead levels and blood pressure." Their findings included "blood lead levels were significantly higher in younger men and women (aged 21 to 55 years) with high diastolic pressure than in those with normal blood pressure. However, in older persons (aged 56-74), blood lead levels were not significantly different for any of the blood pressure categories and there were no important trends."³⁷ They also found that a greater body mass index (weight/height²) was significantly associated with high diastolic blood pressure in men and women in both age groups. When the authors looked specifically at gender, they found that "for men, blood lead had a significant relationship to systolic and diastolic pressure, [but] for women, blood lead was not independently related to blood



pressure.³⁷ The authors did warn: "No causal inferences should be drawn from this cross-sectional survey about the blood pressure effects of lead, calcium, and zinc."³⁷

The NHANES II data has been subsequently analyzed many times with conflicting results. In 1988 Harlan re-analyzed the data and found a slightly different finding. In 1985 Harlan et al found that blood lead was only associated with elevated diastolic pressure, re-analysis in 1988 found an association with both diastolic and systolic pressure in men. He confirmed his earlier findings that "blood lead did not enter the final regression model for blood pressure in women."³⁸ In 1988 Gartside's analysis using a "more reliable forward stepwise regression"³⁹ found that "these results do not provide evidence of any reliability or of any consistency for the hypothesized association. The results obtained by this careful process lacked consistency, lacked reliability, and were weak or negligible. It is concluded, therefore, that there is no convincing evidence of an association between blood lead and blood pressure in NHANES II."³⁹ De Hond et al analyzed more recent NHANES data and found "there is no consistent relationship between blood pressure and blood lead in NHANES III dataset."⁴⁰

The contradictory results from many of the early studies investigating a possible association between blood lead levels and elevated blood pressures "are due to variation in criteria for including participants in the analysis (age groups, gender, race), weighting factors, covariates selected as confounders and statistical methods."⁴⁰ Harlan et al found thirty-four variables having significant correlations with blood pressure and that "age and body mass index were significant and independent predictors of blood pressure."³⁷ "Human population studies have not always been able to adequately address the impact of potentially confounding factors."⁴¹

In the most recent meta-analysis by Nawrot et al,³⁵ several of the early studies included in the analysis found very large changes in blood pressure associated with blood lead concentrations. Each of these early studies were significantly flawed in that they did not include many of the most relevant confounders. In the Kromhout⁴² study, with one of the largest reported associations, the only confounders included were smoking and Quetelet Index (weight/height² equivalent to BMI). In the discussion Kromhout noted that "additional analyses showed that this relation was influenced by one person with a high blood lead who also had severe hypertension. After exclusion of this person, the regression coefficient was reduced. The relation between blood lead and systolic blood pressure was no longer statistically significant after adjusting for age and Quetelet Index."⁴²

Orssaud et al⁴³ considered only age, BMI, and alcohol as confounders. They found two findings that argue against a dose-response that one would expect in a causally related interaction. Their results "indicate that an increase in blood lead value might result in an increase in systolic blood pressure up to a limiting value, while higher lead concentrations do not relate to blood pressure. In our study blood lead values are significantly related to blood pressure; this correlation, highly significant in young



subjects (24-34 years), decreases with age (not significant in 45-55 year olds).⁴³ The statistically significant finding would be attenuated with inclusion of the many additional significant influences on blood pressure, such as smoking and family history.

The study by Weiss et al⁴⁴ which found the highest association between blood lead levels and hypertension also had the largest variability in their results. The study began on 89 policemen; however, the final analysis was conducted on only 70 of the subjects. The authors indicate that they examined the relationship of blood lead to blood pressure adjusting for "other independent variables known to affect blood pressure." They specifically address age, body mass index and smoking as variables. They discuss alcohol, but note that they "could find no independent effect of alcohol consumption on blood pressure." This is in distinct contrast to other findings, "Alcohol intake was significantly associated with systolic and diastolic blood pressure in NHANES I. Alcohol intake is an independent cause of increased blood pressure."⁴⁵ Grandjean et al⁴⁵ found that "lead is related to increased blood pressure. However, the statistical significance of this association disappears when alcohol intake and hemoglobin are included in a multiple regression analysis." In the Weiss study, there is no discussion on how alcohol consumption was quantified in the study subjects; the lack of association may be related to recall bias on the part of the policemen. The initial results, without inclusion of many known confounders, was a statistically significant association between a high level of blood lead and elevation in systolic pressure. No significant association was found between blood lead and diastolic pressure. The authors then investigated "whether the observations were the product of a few influential points. For systolic pressure three individuals needed to be excluded." With the removal of these individuals, the association was only deemed "borderline significant"⁴⁴ as the authors chose to use a lower level of significance, not the usual 95% scientific confidence level. The Weiss et al analyses was significantly altered by the exclusion of three study participant outliers as can often be seen in small data sets. They did not find an association with alcohol consumption, a known confounder, and did not consider many other well known and important confounders. Inclusion of these confounders would have decreased the size of the finding and may have diminished the statistical significance even further.

The analysis by Kort and Zwennis⁴⁶ which contributed the fifth largest relationship between blood lead and blood pressure in Nawrot's³⁵ meta-analysis, was a very small study that did not include the most basic confounders. The authors stated, "From the study, which had its limitations (small groups, insufficient data on body mass index), it was concluded that a positive relationship may exist between blood lead level and blood pressure (notably systolic blood pressure)."⁴⁶ However, when the authors did analyze the data controlling for age, heart rate, and urinary cadmium they found "a nonsignificant result."⁴⁶ This small study provides evidence that inclusion of even minor confounders can alter the results of an analysis.

Neri et al³⁶ reported on two studies; an evaluation of Canadian population data and another study of a cohort of 500 foundry workers. The authors analysis of the Canadian population data of 2193 subjects including only 4 confounders; age, BMI, serum zinc



and hemoglobin level. In multiple regressions they found that "none of those employing control for zinc and hemoglobin attained significance."³⁵ They also noted that "body build other than slim entails a fourfold risk of antihypertensive medications and, in untreated subjects, a doubling of the prevalence of elevated blood pressure (over 90 mm Hg)."³⁵ Their analysis of the 500 foundry workers contributed the 3rd highest association between blood lead and blood pressure in the Nawrot et al³⁵ meta-analysis. The only confounders analyzed were age, weight (not BMI), and urinary cadmium levels. The authors found an association between diastolic blood pressure and blood lead, but the association with systolic blood pressure falls "short of conventional significance".³⁵ When the authors included urinary cadmium into the analysis they excluded weight "because of the limited number of data points per subject." With the inclusion of age, and urinary cadmium, no significant association was found between blood lead and blood pressure.

Moller and Kristensen's analysis of 1,052 subjects provided further evidence that consideration of confounders and influential variables alters the findings of a study. They concluded that there is a "weak positive association between blood lead and increased blood pressure, this association becomes weaker and statistically nonsignificant when changes in other risk factors are controlled."⁴⁷ They did make a very important clinical statement, "This association has little significance for the individual."⁴⁷

Current studies continue to exclude analyses of important confounders such as family history. The inclusion of confounders is highlighted by Hu et al⁴⁸, "the most significant factors related to the occurrence of hypertension were body mass index, family history of hypertension, and tibial lead level." A study participant was 2.329 times more likely to be hypertensive if he had a family history of hypertension. A study participant's odds of being hypertensive was only increased by 0.019 if he had an elevated tibial lead level.

Glenn et al⁴⁹ noted that "the few longitudinal studies that have examined the association of blood lead levels on changes in blood pressure have not consistently found lead to be a risk factor." While they concluded that blood lead was associated with systolic blood pressure, they "did not have data for family history of hypertension and therefore were not able to use this variable to analyze the genetic contribution to blood pressure change or its effect as a confounder." The failure to include factors related to the occurrence of hypertension, such as family history will cause overestimation of the relationship of evaluated factors.

In addition to the lack of inclusion of the multiple factors that significantly influence blood pressure, the results of the multitude of studies are inconsistent. For example, Harlan's analysis of the NHANES II data found that "blood lead levels were significantly related to systolic and diastolic pressure in males but not females."³⁷ Schwartz's⁵⁰ analysis on the NHANES II data was different; "this study confirms the association in males aged 20 to 74 and documents a significant, although weaker, association in females as well." A third analysis of the same NHANES II data reported still different findings. "A



statistically significant increase in blood pressure occurs in only 24 out of 156 analyses. The increase in blood pressure for the older age groups in white males is missing in white females and is contradicted in older blacks, where instead it decreases.³⁹ Sharp et al published exactly the opposite findings; "blood lead concentration plays a strong role as a determinant of blood pressure level among black males. An inverse relationship between systolic blood pressure and blood lead concentration was noted in nonblack males."⁴¹

Even some authors report findings in direct conflict with their earlier findings. In 1991 Joel Schwartz found in, "Lead has been shown to be associated with elevated blood pressure, this study confirms the association in males aged 20-74 and documents a significant, although weaker, association in females as well."⁵⁰ In 2001 Schwartz and colleagues assessed the association of hypertension and lead; "No association was found with blood lead."⁵¹

Parkinson et al evaluated the association of blood pressure and occupational lead exposure in workers in three battery plants. The author's analysis included several of the "known risk factors: age, alcohol use, a quantitative measure of current cigarette use, level of exercise and body mass index."⁵² They found that "blood pressure measurements were significantly correlated with age, body mass index and alcohol consumption. After controlling for other known risk factors, the associations between exposure and blood pressure were small and non-significant."⁵²

In another occupational study Maheswaran et al studied the association of blood pressure and occupational lead exposure in a lead battery manufacturer in the United Kingdom. In their article they discussed the weaknesses of several of the earlier occupational studies. "Three of these studies found a significant association between lead and blood pressure. In all three studies, however, alcohol consumption was either not taken into account or did not appear to influence blood pressure. De Kort et al did not measure body mass index in their study."⁵³ Maheswaran et al examined blood lead levels, zinc protoporphyrin and years of industrial exposure to lead. "Following adjustment for the confounding effects of age, body mass index and alcohol consumption, the effect of blood lead on systolic pressure was not significant. There was no association between diastolic blood pressure and blood lead. Zinc protoporphyrin levels and years of industrial lead exposure did not raise adjusted systolic or diastolic pressure."⁵³

In another occupational study, Glenn et al found that "change in systolic blood pressure during the study was associated with lead dose." A major weakness in this analysis of lead exposed workers was the lack of inclusion of two influential factors on blood pressure, alcohol and family history. "Very high alcohol consumption was associated with systolic blood pressure. We did not keep alcohol consumption in the final model because we did not have data on this variable for all subjects. We did not have data for family history of hypertension and therefore were not able to use this variable to analyze the genetic contribution to blood pressure change or its effect as a confounder."⁴⁹



The investigation of a possible association between blood lead and blood pressure has been ongoing for many years. There have been a multitude of studies including population and occupationally based; the results have been inconsistent and even contradictory with the majority of the studies failing to reach statistical significance.³⁵ Many of these studies have been included in the three meta-analyses discussed in the Agency for Toxic Substances and Disease Registry's (ATSDR) *Toxicologic Profile for Lead* which noted that "meta-analyses of the epidemiological findings have found a persistent trend in the data that supports a relatively weak, but significant association."² Even these three meta-analyses are inconsistent. Schwartz, in 1995 found "a highly significant and moderately consistent association was found, i.e., decrease of blood lead from 10 mg/dL to 5 mg/dL associated with a decrease of 1.25 mmHg."⁵⁴ Staessen et al (1994) found that "there was no relationship across studies between the strength of the blood pressure-blood lead relationship and the mean blood lead concentration. The published evidence suggests that there can only be a weak positive association between blood pressure and lead exposure. Any such relationship may not be causal."⁵⁵ Nawrot et al³⁵, authors of the most recent meta-analysis caution; "These overall results must be cautiously interpreted. In the majority of the population studies, the association between blood pressure and blood lead concentration did not reach statistical significance. The present meta-analysis shows an overall statistically significant positive relationship. On the other hand epidemiological studies can only show associations but not prove causation."³⁵

The overall evidence from a long search for a possible relationship between blood lead and blood pressure is inconsistent and contradictory. Many studies have found an association between blood lead and blood pressure, but the majority have not reached statistical significance. Statistical significance implies that the results are unlikely to have occurred by chance; therefore the results in the many studies that did not reach statistical significance may have occurred by chance alone. Associations that are found to be statistically significant are not necessarily important finding as was discussed by Moller et al,⁴⁷ "this association has little significance for the individual." Most importantly a statistically significant association does not prove causation. The literature raises significant questions regarding whether an association exists and clearly does not support a causal relationship between blood lead and blood pressure elevations.

A handwritten signature in black ink, appearing to read 'David Lukcs', is written over a horizontal line. Below the line, the text 'David Lukcs MD, MPH' is printed in a standard serif font.



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Ref Type: Internet Communication
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**Statistical Data Concerning Hypertension, Diabetes, Renal
Kidney Failure, and Memory Loss in Society**

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I. Hypertension

- ✚ **Source:** National Kidney Foundation,
http://www.kidney.org/news/newsroom/fs_new/10factsabtaframerkd.cfm (last visited Oct. 30, 2009).
- ✚ **Internal Citation for FN1:** National Center for Health Statistics. Health, United States, 2008, with Chartbook on the Health of Americans. Hyattsville, MD: 2008. Available at <http://www.cdc.gov/nchs/data/has/has08.pdf>
 - It is estimated that about 90% of middle-aged adults will develop high blood pressure in the remainder of their lifetime.
 - In the United States, the prevalence of hypertension is higher among blacks than whites.¹
 - The prevalence of high blood pressure is particularly high among black women at 44%.¹
 - **Age-adjusted Percent of Adults Aged 20 Years and Older With Hypertension and/or Taking Blood Pressure-Lowering Medication, 2003–2006**

Overall:	31.3%
Women:	
All Women	30.3%
White	28.3%
African American	44.1%
Mexican American	28.6%
Men:	
All Men	31.8%
White	31.2%
African American	42.2%
Mexican American	24.8%
Poverty Status:	
Poor	35.0%
Near Poor	34.1%
Non-Poor	30.3%

Internal Source: *Health, United States, 2008*¹

- ✚ **Source:** National Kidney Foundation,
<http://www.kidney.org/site/kidneyDisease/index.cfm?ch=209> (last visited Oct. 30, 2009).
- More than 40 percent of African Americans are believed to have high blood pressure, however.³ African-Americans also have higher death rates from causes related to high blood pressure. In 2004, for example, the death rates per 100,000 population from high blood pressure were 15.6 for white men, 49.9 for black men, 14.3 for white women and 40.6 for black women.³
- South Carolina is second in the nation in the prevalence of diabetes, and a leader in the prevalence of high blood pressure.

II. Diabetes

Source: American Diabetes Ass'n, <http://www.diabetes.org/diabetes-statistics.jsp> (last visited Oct. 30, 2009).

- 23.6 million people in the United States, or 8% of the population, have diabetes.
- **Total Prevalence of Diabetes**
 - **Age 20 years or older:** 23.5 million, or 10.7% of all people in this age group have diabetes.
 - **Non-Hispanic Whites:** 14.9 million, or 9.8% of all non-Hispanic whites aged 20 years or older have diabetes.
 - **Non-Hispanic Blacks:** 3.7 million, or 14.7% of all non-Hispanic blacks aged 20 years or older have diabetes.
 - The prevalence of diabetes is at least 2 to 4 times higher among non-Hispanic Black, Hispanic/Latino American, American Indian, and Asian/Pacific Islander women than among non-Hispanic white women.
- **Race and Ethnic Differences in Prevalence of Diagnosed Diabetes:** After adjusting for population age differences, 2004-2006 national survey data for people diagnosed with diabetes, aged 20 years or older include the following prevalence by race/ethnicity:
 - 6.6% of non-Hispanic whites
 - 7.5% of Asian Americans
 - 10.4% of Hispanics
 - 11.8% of non-Hispanic blacks

Source: National Kidney Foundation, http://www.kidney.org/news/newsroom/fs_new/10factsabtaframerkd.cfm (last visited Oct. 30, 2009).

- Diabetes is the leading cause of kidney failure in African Americans.
- The prevalence of diabetes in African Americans is much higher than in white Americans. Among those aged 40 to 74 years, the rate is 11.2 percent for white Americans, compared with 18.2 percent for African Americans.²
- Approximately 13.3 percent of all African Americans over 20 years of age (3.2 million) have diabetes.
- On average, African Americans are twice as likely to have diabetes as white Americans of similar age.
- The most common type of diabetes in African Americans is type 2 diabetes. The risk factors for this type of diabetes include: family history, impaired glucose tolerance, diabetes during pregnancy, high blood insulin levels and insulin resistance, obesity and physical inactivity.²
- African Americans with diabetes are more likely to develop complications of diabetes and to have greater disability from these complications than white Americans.
- African-Americans experience kidney failure about four times more often than white Americans with diabetes.

- African-Americans are also more likely to develop other serious complications such as heart disease and strokes and to experience greater disability than white Americans with diabetes.
- Death rates associated with diabetes are 20 percent higher for African American men and 40 percent higher for African American women, compared with their white counterparts.²

4 **Source:** Kaiser Family,
<http://www.statehealthfacts.org/profileind.jsp?ind=70&cat=2&rgn=42> (last visited, Oct. 30, 2009).

- **South Carolina: Percent of Adults Who Have Ever Been Told by a Doctor that They Have Diabetes, 2008**

Percent of Adults Who Have Ever Been Told by a Doctor that They Have Diabetes, 2008 Compare		
	10.1%	8.2%
	0.7%	0.8%
	87.4%	90.0%
	1.6%	1.0%

4 **Source:** South Carolina Department of Health and Environmental Control,
<http://www.scdhec.gov/health/chcdp/diabetes/statistics.htm> (last visited, Oct. 30, 2009).

- Diabetes is a major public health problem across the nation and the seventh leading cause of death in South Carolina (2005).
- According to the 2006 Behavior Risk Factor Surveillance Survey (BRFSS), South Carolina ranks seventh in diabetes prevalence, where an estimated 414,840 people in South Carolina are aware that they have diabetes.
- The prevalence of diabetes among adults in South Carolina was 9.6% in 2006 (BRFSS, SC DHEC).
- South Carolina is 8th in the nation for diabetes prevalence among African Americans (BFRSS, SC DHEC 2006).
- In South Carolina, African Americans of either gender are twice as likely to have diabetes as their white counterparts. About 13.8 % of African-Americans said that they had diabetes, while only 8.2% of whites reported having the disease.

III. Renal Kidney Failure

7 **Source:** National Institute of Diabetes and Digestive and Kidney Diseases, NIH, <http://kidney.niddk.nih.gov/kudiseases/pubs/kustats/index.htm> (last visited Oct. 30, 2009).

- **Prevalence for Kidney Disease:** (1999–2004): An estimated 11.5 percent of adults ages 20 or older (23 million adults) have physiological evidence of chronic kidney disease determined from data collected through the National Health and Nutrition Examination Survey.¹
- **End-stage Renal Disease (ESRD)**
 - **Prevalence (2006):** 506,256 U.S. residents were under treatment as of the end of the calendar year.²
 - **Resulting from these primary diseases:**
 - **Diabetes:** 188,381
 - **Hypertension:** 122,339
 - **Glomerulonephritis:** 80,164
 - **Cystic kidney:** 23,685
 - **Urologic disease:** 13,371
 - **All other:** 78,316

4 **Source:** National Kidney Foundation, http://www.kidney.org/news/newsroom/fs_new/10factsabtaframerkd.cfm (last visited Oct. 30, 2009).

4 **Internal Citations:**

1. US Renal Data System 2006 Annual Data Report (www.usrds.org)
2. "Diabetes in African Americans," from the National Kidney and Urologic Diseases Information Clearinghouse (www.niddk.nih.gov)
3. American Heart Association High Blood Pressure Statistics (www.americanheart.org)
4. "Early Identification of Renal Disease Among African Americans: A Continuing Problem," M Thornhill-Joyes and M. Moore, *Nephrology News & Issues*, November 1995, p.16-18.
 - Due to high rates of diabetes, high blood pressure and heart disease, African Americans have an increased risk of developing kidney failure, and requiring dialysis treatments or a kidney transplant to sustain life.
 - In 2004, the incidence of kidney failure per million population was 968 in African Americans, compared with 263 in white Americans.¹ African Americans constitute about 32 percent of all patients treated for kidney failure in the U.S., but only about 12 percent of the overall U.S. population.¹
 - African-Americans also develop kidney failure at an earlier age than white Americans. In 2006, the mean age for African-Americans at the start of treatment for kidney failure was 56.4 years, compared with 59.6 in white Americans.¹
 - Diabetes is the leading cause of kidney failure in African Americans. The prevalence of diabetes in African Americans is much higher than in white Americans. Among those aged 40 to 74 years, the rate is 11.2 percent for white Americans, compared with 18.2 percent for African Americans.²
 - High blood pressure is the second leading cause of kidney failure among African Americans, accounting for 34 percent of the new cases each year.¹ However, high

blood pressure remains the leading cause of death overall in African Americans because of its link with heart attacks and strokes.²

✦ **Source:** Office of Chronic Disease Epidemiology,
<http://www.scdhec.gov/health/epidata/docs/FACTckd09.pdf> (last visited Oct. 30, 2009).

➤ **WHO IS AT RISK FOR KIDNEY FAILURE?**

- Diabetes is the most common cause of kidney failure.
- High blood pressure is the second leading cause of kidney failure.
- African Americans are four times more likely to develop kidney failure than Whites.
- Diabetes and high blood pressure account for more than 70 percent of kidney failure in African Americans.

➤ **KIDNEY DISEASE IN SC**

- 1 in 8 South Carolinians have Chronic Kidney Disease (CKD).
- Approximately 460,000 people have CKD in South Carolina.
- South Carolina is, per capita, third in the nation with the number of patients on dialysis.
- 75% of kidney patients in South Carolina are African American.
- Three out of four of those waiting for a kidney are African American, and South Carolina ranks second in the nation in the need for organs among African Americans.

✦ **Source:** National Kidney Foundation,
<http://www.kidney.org/site/kidneyDisease/index.cfm?ch=209> (last visited Oct. 30, 2009).

- 1 in 8 South Carolinians has CKD.
- Approximately 546,000 people have CKD in South Carolina.
- South Carolina is, per capita, third in the nation with the number of patients on dialysis.
- Diabetes and high blood pressure are the leading causes of kidney disease
- South Carolina is second in the nation in the prevalence of diabetes, and a leader in the prevalence of high blood pressure.
- Kidney disease disproportionately affects African-Americans. 30% of South Carolinians are African-American, and nearly 75% of kidney patients in South Carolina are African-American.
- More than 600 South Carolinians are waiting for a kidney transplant.
- Three out of four of those waiting for a kidney are African-American, and South Carolina ranks second in the nation in the need for organs among African-Americans.

IV. Memory Loss & Aging

4 Source: Melissa Healy, Memory Loss: What's Normal What's Not, Los Angeles Times, Nov. 17, 2008.

- By about age 25, however, a human's memory has typically peaked and a long period of decline begins.
- Around about the half-century mark, cognitive performance starts to slide noticeably. Keys and glasses are forgotten. Names, places and words linger on the tip of the tongue.

4 Source: 3 Encyclopedia of Aging, 902, 905, & 914 (2002, David J. Ekerdt).

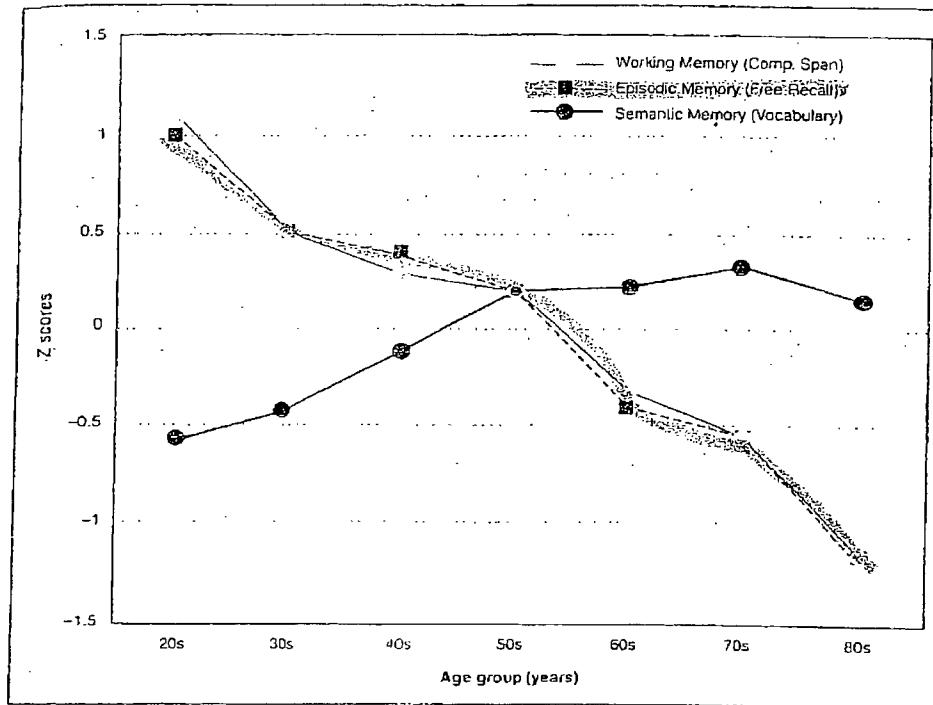
- Most people recognize that their memories are changing as they grow older. They have a hard time coming up with names; they have a harder time finding things they need; they have to rely more on external memory cues such as notes or calendars. P. 902
- Memory declines are experienced throughout the adult life span and not just in the older ages. P. 902
- Working memory and episodic memory decline significantly across the life span, while semantic memory increases significantly. P. 902
- **Episodic memories:** recollections that are actively retrieved as previous personal experiences. P. 905
 - The memory is a reconstruction of an earlier experience very much like looking something up in an internal cognitive diary.
 - Ex: "What did I have for dinner last night?" "Where did I park my car?" "Did I take my medicine this morning?" "Did you see Joan at the party last night?"
 - To answer each question, one attempts to reconstruct the original event.
 - Older adults do worse on most episodic memory tasks than do younger adults. (see chart saved in file 01464.04709)
 - Episodic memory requires one to put what one is trying to remember in a specific context.
- **Everyday memory-** refers to memory operations that routinely occur in one's daily environment. P. 914
 - Ex: remembering names, remembering plans for the day, recalling items that one needs to purchase at the store, remembering to take medications, remembering telephone numbers, directions, or recent newsworthy events.
 - Hallmark of everyday memory is that it involves the performance of tasks that occur naturalistically in the real world.
 - Laboratory studies generally provide evidence for a decline with age in everyday memory process. **See the West et al study (on order from library). These studies found evidence for age-related declines on a wide range of every-day memory lab tasks (remembering names, locations of objects, grocery lists, faces, telephone numbers, and news events)

every-day memory lab tasks (remembering names, locations of objects,
grocery lists, faces, telephone numbers, and news events)

- 4 Source: R.L.C. West et al, Everyday Memory Performance Across the Life Span: Effects of Age and Noncognitive Individual Differences, Psychology & Aging, 72-82 (1992).

- Source is on order from Greenville County Library System

Figure 1
Age group differences in working memory (as measured by computation span), episodic memory (as measured by free recall), and semantic memory (as measured by vocabulary).



NOTE: Adapted from Park, Lautenschlager, et al. in press. Copyright © 2002 by the American Psychological Association. Adapted with permission.

difficult to accomplish in aging research, however, because adults of different ages may process information differently at one of the earlier stages, thus violating the requirement that all be held constant until retrieval (Smith).

The major reason, however, for the loss of interest in identifying the stage at which aging had its effects is clear evidence that age has effects on all stages of memory: encoding, storage, and retrieval. For reasons not directly related to memory stage theory, however, memory research in the 1970s and early 1980s focused heavily on the encoding stage of memory. This focus on encoding was due to the development

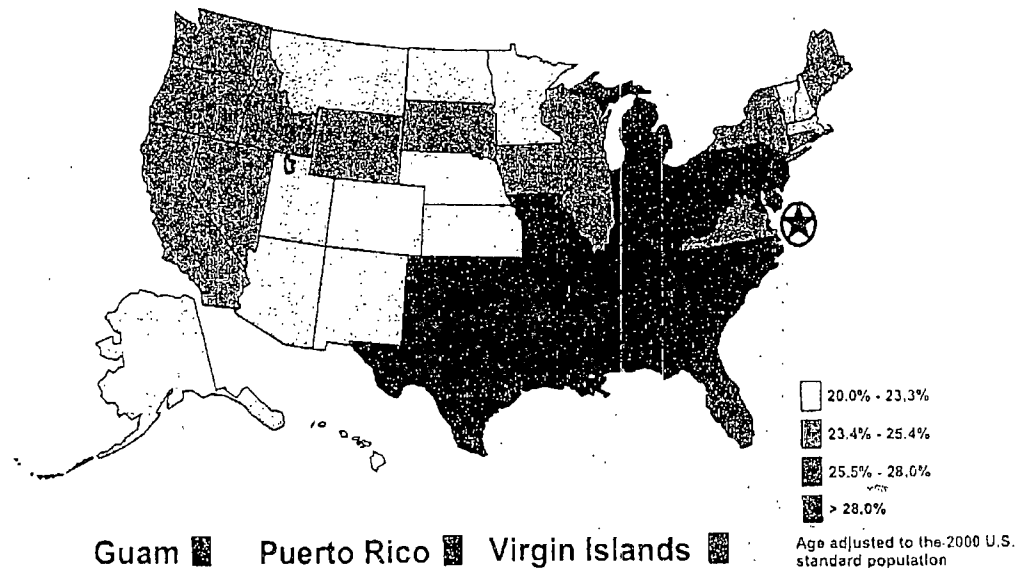
of a conceptual view of memory, the "levels of processing" framework, which proposed that the ability to remember was determined by the extent of semantic processing during the encoding of the to-be-remembered information (Craik and Lockhart). For this reason, much research on memory and aging during this period focused on the nature of encoding processes in different age groups. Even with the magnitude of the research effort, however, the relationship between aging and levels of processing is still unclear. The research did, however, suggest that encoding is especially affected by adult aging. For example, the memory performance of older adults, relative to younger adults, is more detrimentally affected by



Facts on High Blood Pressure



Percent of persons who were ever told they had high blood pressure,
Adults aged 20 years and older, 2003



20.0% - 23.3%: Alaska, Arizona, Colorado, Hawaii, Kansas, Massachusetts, Minnesota, Montana, Nebraska, New Hampshire, New Mexico, North Dakota, Utah, Vermont	25.5% - 28.0%: District of Columbia, Florida, Guam, Indiana, Maryland, Michigan, Missouri, New Jersey, Ohio, Oklahoma, Pennsylvania, Texas, Virgin Islands
23.4% - 25.4%: California, Connecticut, Idaho, Illinois, Iowa, Maine, Nevada, New York, Oregon, South Dakota, Virginia, Washington, Wisconsin, Wyoming	> 28.0%: Alabama, Arkansas, Delaware, Georgia, Kentucky, Louisiana, Mississippi, North Carolina, Puerto Rico, South Carolina, Rhode Island, Tennessee, West Virginia

Source: CDC, Behavioral Risk Factor Surveillance System

June 2005

Table 71 (page 1 of 2). Hypertension and elevated blood pressure among persons 20 years of age and over, by selected characteristics: United States, 1988-1994, 1999-2002, and 2003-2006

(Data are based on interviews and physical examinations of a sample of the civilian noninstitutionalized population)

[Click here for spreadsheet version](#)

Sex, age, race and Hispanic origin ¹ , and percent of poverty level	Hypertension ^{2,3} (elevated blood pressure and/or taking antihypertensive medication)			Elevated blood pressure ²		
	1988-1994	1999-2002	2003-2006	1988-1994	1999-2002	2003-2006
20 years and over, age-adjusted ⁴						
Both sexes ⁵	25.5	30.0	31.3	18.5	19.9	17.9
Male	26.4	28.8	31.8	20.6	19.1	18.2
Female	24.4	30.6	30.3	16.4	20.2	17.3
Not Hispanic or Latino:						
White only, male	25.6	27.6	31.2	19.7	17.6	17.4
White only, female	23.0	28.5	28.3	15.1	18.5	15.9
Black or African American only, male	37.5	40.6	42.2	30.3	28.2	26.5
Black or African American only, female	38.3	43.5	44.1	25.4	28.8	23.9
Mexican male	26.9	26.8	24.8	22.2	21.5	15.3
Mexican female	25.0	27.9	28.6	20.4	21.2	19.2
Percent of poverty level: ⁶						
Below 100%	31.7	33.9	35.0	22.5	23.3	22.6
100%-less than 200%	26.6	33.5	34.1	19.3	23.0	21.1
200% or more	23.9	28.2	30.3	17.5	18.2	16.6
20 years and over, crude						
Both sexes ⁶	24.1	30.2	32.1	17.6	19.9	18.2
Male	23.8	27.6	31.3	18.7	18.2	17.9
Female	24.4	32.7	32.9	16.5	21.6	18.6
Not Hispanic or Latino:						
White only, male	24.3	28.3	32.4	18.7	17.8	17.9
White only, female	24.6	32.8	33.4	16.4	21.6	18.8
Black or African American only, male	31.1	35.9	38.8	25.5	25.2	24.8
Black or African American only, female	32.5	41.9	42.8	22.2	27.2	22.4
Mexican male	16.4	16.5	16.6	13.9	14.1	10.9
Mexican female	15.9	18.8	20.0	12.7	13.8	13.0
Percent of poverty level: ⁶						
Below 100%	25.7	30.3	28.8	18.7	21.1	18.3
100%-less than 200%	28.7	34.8	35.8	19.8	24.1	22.5
200% or more	22.2	28.2	31.1	16.2	17.8	16.8
Male						
20-34 years	7.1	8.1	9.2	6.6	7.3	7.6
35-44 years	17.1	17.1	21.1	15.2	12.1	13.2
45-54 years	29.2	31.0	36.2	21.9	20.4	21.0
55-64 years	40.6	45.0	50.2	28.4	24.8	25.4
65-74 years	54.4	59.6	64.1	39.9	34.9	29.2
75 years and over	60.4	69.0	65.0	49.7	50.6	38.2
Female						
20-34 years	2.9	2.7	2.2	2.4	1.4	1.1
35-44 years	11.2	15.1	12.6	6.4	8.5	5.8
45-54 years	23.9	31.8	36.2	13.7	19.1	20.0
55-64 years	42.6	53.9	54.4	27.0	31.9	28.6
65-74 years	56.2	72.7	70.8	38.2	53.0	40.8
75 years and over	73.6	83.1	80.2	59.9	64.4	55.4

See footnotes at end of table.



Agency for Toxic Substances & Disease Registry

SEARCH

Environmental Medicine | CSEM | GREM | Continuing Education | Patient Education | PEHT | Community

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Printer-Friendly

Receive CE Credit

Grand Rounds

Community

Case Studies in Environmental Medicine (CSEM) Lead Toxicity

Full Screen

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What Are the Physiologic Effects of Lead Exposure?

Environmental Medicine
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Related Documents
CABS: Lead
Lead Screening
PHS: Lead
Pb/As/Cd/Cr Interaction
Pb/Mn/Zn/Cu Interaction
Pb/Chlorpyrifos/Hg/Me-Hg
ToxFAQs™: Lead
Toxguide: Lead 
Tox Profile: Lead

ATSDR Resources
ATSDR en Español
Case Studies (CSEM)
Exposure Pathways
Health Assessments
Health Statements
Interaction Profiles
Interactive Learning
Managing Incidents
Medical Guidelines
Minimal Risk Levels
Priority List
ToxFAQs™
ToxFAQs™ CABS
Toxicological Profiles
Toxicology Curriculum

External Resources

http://www.atsdr.cdc.gov/csem/lead/pbphysiologic_effects2.html

3/30/2011

CDC
Cancer
eLCOSH
EPA
Healthfinder®
Medline Plus
NCEH
NIEHS
NIOSH
OSHA

**Learning
Objectives**

Upon completion of this section, you will be able to

- describe how lead affects adults and children
- describe the major physiologic effects of chronic low- level lead exposure
- describe the major physiologic effects of acute high-level lead exposure.

Introduction

Lead serves no useful purpose in the human body, but its presence in the body can lead to toxic effects, regardless of exposure pathway.

- Lead toxicity can affect every organ system.
- On a molecular level, proposed mechanisms for toxicity involve fundamental biochemical processes. These include lead's ability to inhibit or mimic the actions of calcium (which can affect calcium-dependent or related processes) and to interact with proteins (including those with sulfhydryl, amine, phosphate and carboxyl groups) (ATSDR, 2005).

It must be emphasized that **there may be no threshold** for developmental effects on children.

- The practicing health care provider can distinguish overt clinical symptoms and health effects that come with high exposure levels on an individual basis.
- However, lack of overt symptoms does not mean "no lead poisoning."
- Lower levels of exposure have been shown to have many subtle health effects.
- Some researchers have suggested that lead continues to contribute significantly to socio-behavioral problems such as juvenile delinquency and violent crime (Needleman 2002, Nevin 2000).
- It is important to prevent all lead exposures.

While the immediate health effect of concern in children is typically

Neurological Effects	The nervous system is the most sensitive target of lead exposure. • There may be no lower threshold for some of the adverse neurological effects of lead in children. • Neurological effects of lead in children have been documented at exposure levels once thought to cause no harmful effects (<10 µg/dL) (Canfield 2003; CDC 1997a). • Because otherwise asymptomatic individuals may experience neurological effects from lead exposure, clinicians should have a high index of suspicion for lead exposure, especially in the case of children.
-----------------------------	--

Children

In children, acute exposure to very high levels of lead may produce encephalopathy and other accompanying signs of

- ataxia
- coma
- convulsions
- death
- hyperirritability
- stupor

The BLLs associated with encephalopathy in children vary from study to study, but BLLs of 70-80 µg/dL or greater appear to indicate a serious risk (ATSDR 2005).

- Even without encephalopathy symptoms, these levels are associated with increased incidences of lasting neurological and behavioral damage (ATSDR 2005).

Children suffer neurological effects at much lower exposure levels.

- Neurological effects may begin at low (and, relatively speaking, more widespread) BLLs, at or below 10 µg/dL in some cases, and it may not be possible to detect them on clinical examination.
- Some studies have found, for example, that for every 10 µg/dL increase in BLL, children's IQ was found to be lower by four to seven points (Yule *et al.*, 1981; Schroeder *et al.*, 1985; Fulton *et al.*, 1987; Landsdown *et al.* 1986; Hawk *et al.* 1986; Winneke *et al.* 1990 as cited in AAP 1993).
- There is a large body of evidence that associates decrement in IQ performance and other

Adults

There can be a difference in neurological effects between an adult exposed to lead as an adult, and an adult exposed as a child when the brain was developing.

- Childhood neurological effects, including ADHD, may persist into adulthood. Lead-exposed adults may also experience many of the neurological symptoms experienced by children, although the thresholds for adults tend to be higher.

Lead encephalopathy may occur at extremely high BLLs, e.g., 460 µg/dL. (Kehoe 1961 as cited in ATSDR 2005)

- Precursors of encephalopathy, such as dullness, irritability, poor attention span, muscular tremor, and loss of memory may occur at lower BLLs.

Less severe *neurological and behavioral* effects have been documented in lead-exposed workers with BLLs ranging from 40 to 120 µg/dL. (ATSDR 2005) These effects include

- decreased libido
- depression/mood changes, headache
- diminished cognitive performance
- diminished hand dexterity
- diminished reaction time
- diminished visual motor performance
- dizziness
- fatigue
- forgetfulness
- impaired concentration
- impotence
- increased nervousness
- irritability



Renal Effects

Many studies show a strong association between lead exposure and renal effects. (ATSDR 1999)

- Acute high dose lead-induced impairment of proximal tubular function manifests in aminoaciduria, glycosuria, and hyperphosphaturia (a Fanconi-like syndrome). These effects appear to be reversible (ATSDR 1999).
- However, continued or repetitive exposures can cause a toxic stress on the kidney, if unrelieved, may develop into chronic and often irreversible lead nephropathy (*i.e.*, chronic interstitial nephritis).

The lowest level at which lead has an adverse effect on the kidney remains unknown.

- Most documented renal effects for occupational workers have been observed in acute high-dose exposures and high-to-moderate chronic exposures (BLL > 60 µg/dL).
- Currently, there are no early and sensitive indicators (*e.g.*, biomarkers) considered predictive or indicative of renal damage from lead. (ATSDR 2000) Serum creatinine and creatinine clearance are used as later indicators.
- However, certain urinary biomarkers of the proximal tubule (*e.g.*, NAG) show elevations with current exposures, even at BLLs less than 60 µg/dL; and some population-based studies show accelerated increases in serum creatinine or decrements in creatinine clearance at BLLs below 60 µg/dL. (Staessen *et al.* 1992;



Hematological Effects

Lead inhibits the body's ability to make hemoglobin by interfering with several enzymatic steps in the heme pathway.

- Specifically, lead decreases heme biosynthesis by inhibiting *d*-aminolevulinic acid dehydratase (ALAD) and ferrochelatase activity.
- Ferrochelatase, which catalyzes the insertion of iron into protoporphyrin IX, is quite sensitive to lead.
- A decrease in the activity of this enzyme results in an increase of the substrate, erythrocyte protoporphyrin (EP), in the red blood cells (also found in the form of ZPP—bound to zinc rather than to iron).
- Also associated with lead exposure is an increase in blood and plasma *d*-aminolevulinic acid (ALA) and free erythrocyte protoporphyrins (FEP) (EPA 1986a as cited in ATSDR 1999).

EPA estimated the threshold BLL for a decrease in hemoglobin to be 50 µg/dL for occupationally exposed adults and approximately 40 µg/dL for children, although other studies have indicated a lower threshold (e.g., 25 µg/dL) for children. (EPA 1986b as cited in ATSDR 1999; ATSDR 1999)

- Recent data indicate that the EP level, which has been used in the past to screen for lead toxicity, is not sufficiently sensitive at lower levels of blood lead and is therefore not as useful a screening test as previously thought (see the "Laboratory Evaluation" section for further discussion of EP testing.).

Lead can induce two types of anemia

Endocrine Effects	Studies of children with high lead exposure have found that a strong inverse correlation exists between BLLs and vitamin D levels. • Lead impedes vitamin D conversion into its hormonal form, 1, 25-dihydroxyvitamin D, which is largely responsible for the maintenance of extra- and intra-cellular calcium homeostasis. • Diminished 1, 25-dihydroxyvitamin D, in turn, may impair cell growth, maturation, and tooth and bone development. • In general, these adverse effects seem to be restricted to children with chronically high BLLs (most striking in children with BLLs > 62 µg/dL) and chronic nutritional deficiency, especially with regard to calcium, phosphorous, and vitamin D (Koo <i>et al.</i> 1991 as cited in ATSDR 1999). • However, Rosen <i>et al.</i> (1980) noted that in lead-exposed children with blood lead levels of 33-55 µg/dL, 1, 25-dihydroxyvitamin D levels were reduced to levels comparable to those observed in children with severe renal insufficiency. • Lead appears to have a minimal, if any, effect on thyroid function.
Gastrointestinal Effects	In severe cases of lead poisoning, children or adults may present with severe cramping abdominal pain, which may be mistaken for an acute abdomen or appendicitis.





Cardiovascular (Hypertension) Effects

Hypertension is a complex condition with many different causes and risk factors, including age, weight, diet, and exercise habits.

- Lead exposure is one factor of many that may contribute to the onset and development of hypertension.
- Although low to moderate lead level exposures (BLL < 30 µg/dL) show only a low degree of association with hypertension, higher exposures (primarily occupational) increase the risk for hypertensive heart disease and cerebrovascular disease as latent effects.
- One study found that adults who experienced lead poisoning as children had a significantly higher risk of hypertension 50 years later (relative to control adults without childhood lead exposure). (Hu, 1991, as cited in ATSDR 2000) The association has been shown in population-based studies with BLLs below 10 µg/dL. Data supports an association between lead exposure and elevations in blood pressure. (Victory *et al.* 1988; Schwartz 1995 as cited in ATSDR 2000; Korrick *et al.* 1999; Hu *et al.* 1996)
- It is estimated that, on a population basis, blood lead can account for a 1% to 2% variance in blood pressure. (ATSDR 2000) This could increase the incidence of hypertension a substantial amount, due to the high prevalence of hypertension of all causes in general populations.

Reproductive Effects

Reproductive effects examined in the literature include sperm count, fertility, and pregnancy outcomes. While several studies have implicated lead as contributing to reproductive and developmental effects, these effects have not been well-established at low exposure levels.

Male Reproductive Effects

Recent reproductive function studies in humans suggest that current occupational exposures decrease sperm count totals and increase abnormal sperm frequencies (Alexander *et al.* 1996; Gennart *et al.* 1992; Lerda 1992; and Lin *et al.* 1996 as cited in ATSDR 2000; Telisman *et al.* 2000).

- Effects may begin at BLLs of 40 µg/dL. (ATSDR 2005)
- Long-term lead exposure (Independent of current lead exposure levels) also may diminish sperm concentrations, total sperm counts, and total sperm motility (Alexander *et al.* 1996 as cited in ATSDR 2000).
- It is unclear how long these effects may last in humans after lead exposure ceases.

Fertility

It is not currently possible to predict fertility outcomes based on current BLLs or past lead exposure levels. (ATSDR 2000)

Pregnancy Outcomes

The effect of low-level lead exposures on pregnancy outcomes is not clear. Thus it appears that at higher (e.g., occupational) exposure levels, the evidence is clearer for an association

Developmental Effects

Developmental effects examined in the literature include pregnancy outcomes (e.g., premature births and low birth weights), congenital abnormalities, and post birth effects on growth or neurological development.

- Increasing evidence indicates that lead, which readily crosses the placenta, adversely affects fetus viability as well as fetal and early childhood development.
- Prenatal exposure to low lead levels (e.g., maternal BLLs of 14 µg/dL) may increase the risk of reduced birth weight and premature birth (ATSDR 1999).
- Although lead is an animal teratogen, most human studies have not shown a relationship between lead levels and congenital malformations.
- A study by Needleman *et al.* (1984) correlated increased prenatal lead exposure with increased risk for minor congenital abnormalities (e.g., minor skin abnormalities and undescended testicles).
- No association between prenatal lead exposure and major congenital abnormalities has been found (Ernhart *et al.* 1985, 1986; McMichael *et al.* 1986).
- In a retrospective study, a higher proportion of learning disabilities were found among school-aged children with biological parents who were lead poisoned as children 50 years previously (Hu 1991).

Other Potential Effects

Lead has been linked to problems with the development and health of bones. At high levels, lead can result in slowed growth in children.

- Studies have shown increased likelihood of osteoporosis (weakened bones later in life) in animals exposed to lead. A review of this issue can be found in Puzas (1992). Although this link has not been established in humans, it is likely that upon closer examination of lead-exposed individuals, lead will be shown to be a new risk factor for the disease.
- Research currently underway may provide more information about potential impacts of lead on osteoporosis (bone health) in the future.

Current available data are not sufficient to determine the carcinogenicity of lead in humans.

- EPA has classified elemental lead and inorganic lead compounds as Group 2B: probable human carcinogens. (ATSDR 1999) This classification is based in part on animal studies, which have been criticized because the doses of lead administered were extremely high (ATSDR 1999).
- The National Toxicology Program classifies lead and lead compounds as "reasonably anticipated to be a carcinogen" (NTP 2004).
- Information regarding the association of occupational exposure to lead with increased cancer risk is generally limited. This is because these occupational exposure studies, which primarily

Key Points

- Effects in children generally occur at lower BLLs than in adults.
- The developing nervous system of a child can be affected adversely at BLLs of less than 10 µg/dL. It is often impossible to determine these effects upon clinical examination.
- There is a wide range of neurological effects associated with lead exposure, some of which may likely be irreversible.
- Lead exposure can lead to renal effects such as Fanconi-like syndromes, chronic nephropathy, and gout.
- Most lead-associated renal effects or disease are a result of ongoing chronic or present high acute exposure or can be a latent effect of chronic past lead exposure.
- Lead inhibits several enzymes critical to the synthesis of heme, causing a decrease in blood hemoglobin.
- Today, lead exposure in children only rarely results in frank anemia.
- Lead's impairment of heme synthesis can affect other heme-dependent processes in the body outside of the hematopoietic system.
- Lead interferes with a hormonal form of vitamin D, which affects multiple processes in the body, including cell maturation and skeletal growth.
- Lead exposure may lead to increased risk for hypertension and its sequelae.
- Evidence suggests an association between lead exposure and certain reproductive and developmental

Progress Check

7. How do lead's effects **differ** in children and adults?

- ☐ A. Effects in children are more likely to be reversible.
- ☐ B. Adults suffer more neurological damage.
- ☐ C. Children are less likely to become anemic.
- ☐ D. Effects in adults tend to begin at higher exposure levels than in children.

Answer:

Clear

To review relevant content, see Adults in this section.

8. Lead toxicity can affect

- ☐ A. the kidneys and brain
- ☐ B. IQ and neurological development in children
- ☐ C. sperm count
- ☐ D. All of the above.

Answer:

Clear

To review relevant content, see Neurological Effects, Children, Renal Effects, and Reproductive Effects in this section.

Progress Check

7. How do lead's effects **differ** in children and adults?

- ☐ A. Effects in children are more likely to be reversible.
- ☐ B. Adults suffer more neurological damage.
- ☐ C. Children are less likely to become anemic.
- ☐ D. Effects in adults tend to begin at higher exposure levels than in children.

Answer:

Clear

To review relevant content, see Adults in this section.

8. Lead toxicity can affect

- ☐ A. the kidneys and brain
- ☐ B. IQ and neurological development in children
- ☐ C. sperm count
- ☐ D. All of the above.

Answer:

Clear

To review relevant content, see Neurological Effects, Children, Renal Effects, and Reproductive Effects in this section.

LEAD CONTROL PROGRAM

I. Blood Lead Monitoring

- | | |
|---------------------------------------|-----------------------|
| a. 0-19 ug/100 gm whole blood | Every 6 months |
| b. 20-29 ug/100 gm whole blood | Every 3 months |
| c. 30-34 ug/100 gm whole blood | Every 2 months |
| d. 35+ ug/100 gm whole blood | Every month |

II. Blood Lead of 35 and above:

- a. Employee will be considered on MRP if:
 - ❖ 6 month blood lead average (or last 3 samples) is 36 or above
 - ❖ One time blood lead reading of 44 or greater
 - MRP Paperwork
 - ✓ Perform detailed history
 - ✓ Check work practices
 - ✓ Personal health
 - ✓ Hygiene consultation
 - ✓ Supply respirator
 - ✓ Wipe samples for Pb analysis
 - Remove from work station, if (lead-in-air over 30 ug/m3) and refer to plant physician
- b. Employee will be removed from MRP (return regular job assignment) when two confirmed blood leads are below 33, and 6 month average is 36 or below.

***** Confirmed blood lead of 10 ug/100 gm or more on post-employment physical will be subject to disqualification of employment: !***

Signature: Mark M. J. Date: 1-25-2001

III. Workplace Safety

- a. Appropriate protective equipment, work apparel, and safety shoes are supplied for all employees
- b. Hair protection provided by the company to be worn by all employees in the manufacturing area
- c. At no time will work clothes nor safety shoes be taken outside of the plant. This includes
 - Lunch or break areas
 - Home for laundry

***** Uniform laundry service will be provided by plant*****

South Carolina Workers' Compensation Commission
P.O. Box 1715 • 1612 Marion Street
Columbia, South Carolina 29202-1715

WCC e# 7423889
Carrier File #
Carrier Code #
Employer FEIN

Lee Ernest Franklin
Claimant's Name
Address City State Zip
Home Phone #
Work Phone #
W. Hugh McAngus
Preparer's Name

Yuasa Exide, Inc.
Employer's Name
P.O. Box 1797, Sumter, SC 29150
Address City State Zip
Insurance Carrier
803-779-2300
Phone Number

Complete each information blank. To request a hearing, check box 13b., indicate the kinds of benefits claimed by checking the box(es) at lines 6, 7, 8, and 9, and file this form in duplicate.

A claim for worker's compensation benefits is made based on the following grounds:

- 1a. The claimant sustained an accidental injury to his entire body from 1974 to 1996 in Sumter County, State of South Carolina.
part of body hurt month day year
- 1b. Describe briefly how the accident occurred: Claimant was chronically exposed to lead in his employment, resulting in multiple illnesses and injuries.
2. Both the claimant and the employer were subject to the South Carolina Workers' Compensation Act at the time of injury.
3. The relationship of the employer and employee existed at the time of injury.
4. At the time of the injury the claimant was performing services arising out of and in the course of employment.
5. Notice of the accidental injury was given to the employer from 1974 forward in the following manner: Employer tested Claimant's blood lead levels and was aware of his ongoing injury. Written notice to Employer of this specific claim was given by letter dated March 20, 1997.
- ☒ 6. Due to injury, the claimant is in need of (check one):
☒ (a) medical examination and treatment for multiple illnesses and injuries as a result of exposure to lead.
☐ (b) additional medical examination and treatment for occupational disease to kidneys.
- ☒ 7. Due to injury, the claimant requests temporary total disability benefits because of lost compensable time from work and wages for the period of: From the date of last work ongoing.
- ☒ 8. Due to the injury, the claimant has permanent disability of the following nature and extent: (check one)
☒ (1) General Disability: ☒ Total ☐ Partial
☒ (2) Specific Disability: ☒ Total ☐ Partial
☒ (3) Permanent disability
Body part(s) affected are: loss of kidneys.
- ☐ 9. Due to the injury, the claimant has a serious bodily disfigurement consisting of N/A.
- 10a. At the time of the injury, the claimant was paid weekly wages of \$ Requested Form 20; and demands accounting of days worked and wages earned as provide by law.
- 10b. Give names and addresses of all employers from whom the claimant has worked since the date of the accident.
None.
- 11a. Further grounds of claim: Claimant requests benefits for total disability and possible lifetime benefits for brain damage as a result of exposure to lead.
- 11b. List names and addresses of all physicians or other medical specialists who have seen or treated the claimant as a result of the accident: Medical Department at Yuasa Exide; Dr. Pusadee Suchinda; Dr. A. W. Hursey; Dr. Wallace Cone; Dr. W. Bultman; Dr. Capers Hiott
- 11c. To the best of your knowledge, did you have any prior permanent disability?
If yes, describe: .
12. Appropriate benefits as provided in the ACT for the above grounds and other relief as the Worker' Compensation Commission may direct as just and proper.
- ☐ 13a. I am filing a claim. I am not requesting a hearing at this time.
- ☒ 13b. I am requesting a hearing.

Signature of Claimant/Representative

July 3, 2001
Date

Refer to R.67-205 and
R.67-206, R.67-207, and R.67-601 through R.67-615. Questions about the use of this form may be directed to the Commission's Judicial Department.

WCC Form #50. Rev. Date 9/90

50

Employee's Notice of Claim and/or Request for Hearing

STATE OF SOUTH CAROLINA
BEFORE THE WORKERS' COMPENSATION COMMISSION

WCC FILE NO. 7423889

LEE ERNEST FRANKLIN,)
)
 Claimant,)
)
 vs.)
)
 YUASA EXIDE, INC.)
)
 Employer,)
 and)
)
 AMERICAN NATIONAL FIRE)
 INSURANCE CO.,)
)
 Carrier,)
)
 Defendants.)
)

DECISION AND ORDER

COPY

DATE OF HEARING: Hearing held in Sumter, S.C. on September 30, 2004.

APPEARANCES: Claimant appeared and represented by W. Hugh McAngus, Esquire of McAngus, Goudelock & Courie, L.L.C. of Columbia, South Carolina and George C. James, Jr., Esquire, of Lee Law Firm of Sumter, South Carolina.

Defendants Yuasa Exide, Inc. and Great American Insurance Co. represented by Vernon F. Dunbar, Esquire, of Turner, Padgett, Graham & Laney, P.A. of Columbia, South Carolina.

PURPOSE OF THE HEARING: To determine issues as set forth on Forms 50 and 51 and any other issues that timely come before the Commission.

COMMISSIONER: George N. Funderburk, Commissioner

FILED: June 10, 2005

STIPULATIONS

The parties stipulated at the hearing to the following:

1. The purpose of the hearing was to determine the issues set forth on Forms 50 and 51, and any other issues that timely come before the Commission.
2. Venue was proper in Sumter County.
3. Notice of the hearing was timely and properly served on all parties of interest.
4. The Claimant seeks a determination of benefits under the South Carolina Workers' Compensation Act; therefore, the Commission has jurisdiction over this claim.
5. The Claimant's average weekly wage and compensation rate are \$602.63 and \$401.75, respectively.

APA SUBMISSIONS

Pursuant to the South Carolina Administrative Procedures Act and Regulations of the South Carolina Workers' Compensation Commission, the following records and documents were submitted into evidence.

Claimant's submissions:

- | | | | |
|----|--|----------|-------------|
| 1. | Article <i>Occupational Lead Poisoning</i> ,
<u>American Family Physician</u> | 02/15/98 | Pages 1-12 |
| 2. | Article <i>No Safe Level of Lead. Study</i>
<i>Suggests</i> , <u>Associated Press</u> | 04/30/01 | Pages 13-14 |
| 3. | Article <i>Assessment: Blood Lead Levels</i> | 06/01/99 | Pages 15-16 |
| 4. | Article <i>What Lead Does to Your Health</i> ,
<u>EPA</u> | 12/12/01 | Pages 17-20 |

5.	Article <i>Management Guidelines for Blood Lead Levels in Children and Adults</i>	11/27/00	Pages 21-22
6.	Article Isselbacher et al., <u>Harrison's Principles of Internal Medicine, Vol. 2, 13th Edition</u> , pp. 2463-2464; pp. 1315-1316		Pages 23-27
7.	Article Hu, Howard, M.D., ScD., et al., <i>The Relationship of Bone and Blood Lead to Hypertension</i> , <u>JAMA</u> , Col. 275, No. 15, PP. 1171-1181 8.	04/17/96	Pages 28-38
8.	Article <i>Information for Consumers: Lead Exposure in Adults - A Guide for Health Care Providers</i> , <u>New York State Department of Health</u>	01/2001	Pages 39-44
9.	Article <i>Studies Find Exposure to Lead More Hazardous Than Thought</i> , <u>U.S. Water News Online</u>	June 1996	Pages 45-46
10.	Letter from Hugh McAngus to Vernon Dunbar	1/15/02	Page 47
11.	Transcript of Hearing before S.C. Workers' Compensation Commission	02/04/02	Pages 48-72
12.	Brief and Memorandum of Law in Support of Defendants American National Fire Insurance Co. and Yuasa Exide, Inc., Appeal (with attachments)	12/29/03	Pages 73-95
13.	Deposition of Lee Ernest Franklin	01/14/99	Pages 96-140
14.	Deposition of Brian Hann	11/19/98	Pages 141-176
15.	Deposition of Dr. Eric Byrd	11/19/98	Pages 177-293
15A.	Deposition of Dr. Eugene Shippen	08/24/04	Pages A1-A60
16.	Records of Carroll H. Crawford, MEd, CRC ProRehab, Inc.	03/19/02	Pages 294-296

17.	Records of Howard Hu, M.D., MPH, ScD Harvard School of Public Health & Medicine dated	07/25/01 - 05/24/04	Pages 297-305
18.	Curriculum Vitae of Howard Hu, M.D., MPH, ScD	07/16/01	Pages 306-332
19.	Records of Richard P. Wedeen, M.D.	01/24/01	Pages 333-334
20.	Curriculum Vitae of Richard Peter Wedeen, M.D.	11/2000	Pages 335-359
21.	Letter from George C. James, Jr., to Richard P. Wedeen, M.D.	01/22/01	Pages 360-361
22.	Chain of Custody Form	01/23/01 - 01/24/01	Pages 362-363
22A.	Records of Michael E. Ullian, M.D. MUSC	09/01/04	Pages 22A1-22A3
22B.	Curriculum Vitae of Michael E. Ullian, M.D.	09/14/04	Pages 22B1-22B7
23.	Exide documentation concerning blood lead levels	08/05/74 - 11/07/95	Pages 364-378
24.	Exide Personal Employee Data Sheets	11/04/91 - 02/05/92	Pages 379-382
25.	Exide Employer Records provided to Attorney McAngus by Attorney Zeigler	08/05/74 - 10/18/96	Pages 383-673
26.	Medical Records from Exide	08/03/74 - 04/23/96	Pages 674-861
27.	Lab Reports from Exide	07/11/89 - 11/19/92	Pages 862-880
28.	Records of Pasadee Suchinda, M.D. Sumter Medical Specialists	04/29/85 - 05/29/91	Pages 881-899
29.	Records of Wallis D. Cone, M.D. Sumter Urological Associates, PA dated	03/10/81 - 05/29/85	Pages 900-904
30.	Tuomey Regional Medical Center	03/10/81 - 05/01/96	Pages 905-1646

31. Deposition of Howard Hu, M.D., MPH, ScD 9/20/04

Defendant submissions:

- | | | | |
|----|--|-----------------|-------------|
| 1. | OSHA Regulations (Substance Date Sheet for Occupational Exposure to Lead 1910.1025 etc.) | 5/31/99-6/24/93 | Pages 1-41 |
| 2. | Claimant's Blood Lead Levels reports | 7/14/76-7/23/79 | Page 45 |
| 3. | Records of S.C. Workers Compensation Commission Coverage & Compliance | | Pages 46-70 |

Exhibits:

- | | | |
|----|---------------------------|------------------|
| 4. | Records of Exide | 12/09/83-10/4/85 |
| 5. | Record of Tuomey Hospital | 5/21/88 |

STATEMENT OF THE CASE

This hearing comes before the undersigned Commissioner based on the Form 50 filed by Claimant alleging occupational disease as a result of lead exposure during his employment with Yuasa Exide, Inc. Claimant alleges that from 1974 through 1996 while working for Employer, he was continuously exposed to lead which resulted in high blood pressure, kidney disease and failure, and ultimately to carcinoma of the kidneys and removal of his kidneys. Claimant alleges he is permanently and totally disabled as a result of his kidney disease. Claimant contends that Defendants, the carrier on the risk at the date of last injurious exposure, should be liable for this claim. Claimant requests that Utica-Mohawk language be incorporated in the Order.

Defendants contend any responsibility for this claim should lie with the workers' compensation insurance carriers who insured Exide during the earlier parts of his employment and

that Claimant's last injurious exposure occurred during the period of 1974-1985. Defendants contend that any lead exposure during its coverage period (1991-1996) was within OSHA permissible exposure limits and therefore not injurious. Defendants further deny this claim asserting it should be barred by the statute of limitations. Defendants admit Claimant is permanently and totally disabled as a result of his kidney disease.

EVIDENCE OF THE CASE

Claimant testified that he worked for Employer for more than 23 years and had no health problems when his employment began. He initially worked in the assembly area which was considered a high lead area. He first worked for 2 years as a stacking machine operator. This job consisted of working with battery plates stacked on a rack, and Claimant testified that there was lead dust on the plates, which fell as the plates were unloaded. He then worked in pasting operation for 6-7 years mixing lead powder with acid. He testified that in both jobs there was dust in the air and on the floor. He also worked as a winkle burner, welding lead plates with a torch. He then worked as a material handler for 7 years. In this job, he drove a forklift and moved battery plates and elements around the assembly area. He testified that dust blew off the material and back towards him while driving the forklift. At one point, Employer suggested that Claimant try driving his forklift backwards so that the lead dust wouldn't fly in his face. There was a ventilation system installed at some point, but most of the time this did not work right. When it wasn't working, production continued.

Claimant testified that when his employment began, he was provided a paper mask. This changed to a "v-shaped" mask and eventually to a respirator. Regardless of the mask, dust got in on his face and in his nose. The respirator did not stay sealed and would loosen with sweat. At the end

of the day, he would shower in the locker room with special soap to get the lead dust off of his skin and blow his nose to get the dust out. At some point, Claimant's blood pressure and lead levels were high and he was moved to the shipping department, the only low lead area in the plant. Claimant testified that the shipping area was at the back of the plant and he walked through the manufacturing area about 8 times a day to get to and from shipping. The areas were not closed from each other and were only separated by a driveway, with forklifts traveling between the two areas. There was dust in the shipping area which continued to get in his nose.

Claimant testified he first had blood pressure problems in the eighties which continued until he left Employer. He took medication except for periods when his monthly supply ran out before he obtained the next prescription. Claimant began dialysis three times per week for three and a half hours per day in 1993, which continued until his kidneys were removed in 1996. He continued to work during dialysis. He hasn't had any blood pressure problems since his kidneys were removed.

Claimant denied having any history drinking problems. The only moonshine he has ever had was given to him by a supervisor at a company picnic. Claimant also testified he did work around leaded gasoline helping a friend replace engines, but this was not everyday. Four or Five other co-workers were going through dialysis at the same time as Claimant. On cross-examination, Claimant was shown three medical reports: (1) 1983 report which stated he told the doctor about stump hole liquor; (2) 1985 report which states he told the doctor he stopped drinking stump hole liquor in April; and (3) 1988 report where Claimant was told to stop alcohol. Claimant responded that he never denied drinking alcohol, he just denied drinking it on a regular basis, and he only drank stump hole liquor one time.

On cross-examination, Claimant testified that his brother and some cousins have also undergone dialysis, and the cousins also had hypertension and diabetes. His mother has hypertension and diabetes. Claimant took his blood pressure medications except when they ran out, which didn't happen routinely. Employer would tell him during physicals when his blood pressure was high, and his doctors always told him to take his medicine. In the early eighties Claimant was aware that he was loosing protein which was a sign of kidney failure. Initially when Claimant's blood lead levels were high, he was not removed from his work area. Eventually he was removed until his lead levels came back down, then he was put back in his regular job. When his blood lead levels began staying high, he was moved to material handler and eventually to shipping. Claimant testified that his blood pressure was continuously rising and his kidney problems progressively worsening. Once someone is moved to shipping, there is no place left to move. Once in shipping, he continued to be monitored and remembers being told that his blood levels were 50 or above. When he began dialysis in 1993, Claimant was aware Employer didn't want him around high amounts of lead because there were high amounts in his body, but no one told him this was because of his kidney problems. Employer was telling everyone that high lead amounts were not good.

Claimant submitted medical opinions from Dr. Howard Hu and Dr. Richard P. Wedeen. Both confirmed Claimant's disease was causally related to his exposure with Employer. Dr. Howard Hu is a Professor of Occupational and Environmental Medicine who also continues to practice occupational and environmental medicine. In his reports, Dr. Howard Hu opined that Claimant's occupational lead exposure at the Yuasa Exide Battery plant was the major contributing factor towards the causation of his chronic renal failure, end-stage renal failure, need for dialysis, bilateral renal cell carcinomas, and need for bilateral nephrectomies. Dr. Hu concluded that although

Claimant's early employment exposed him to higher levels of lead, Claimant's continued exposure from 1991-1996 more likely than not resulted in additional lead-induced injuries to his kidneys and the onset of renal carcinoma requiring removal of his kidneys in January 1996.

In his deposition, Dr. Hu confirmed that Claimant's condition resulted from his continuous exposure to lead. After Claimant's initial diagnosis of renal disease in 1981, Claimant's exposure lasting until 1996 aggravated or combined with the disease causing renal cell carcinoma. Claimant's high blood pressure and renal failure were first noted in 1981. Claimant's lead exposure directly effected his kidneys and blood pressure, and the high blood pressure in turn continued to contribute to the kidney disease. Claimant's continued exposure to lead until 1996 continued to have an adverse affect on his kidneys and blood pressure.

Dr. Hu testified that the preponderance of the evidence and literature suggest that even a low to moderate lead exposure (exposure causing blood levels of 10-40) will negatively affect renal function, and he cited 9 articles to support this conclusion. Dr. Hu conceded that poorly controlled blood pressure probably exacerbated Claimant's kidney problems, and that African-Americans have a higher incidence of hypertension. However, Dr. Hu asserted that African-Americans also have higher incidences of lead exposure than whites, so he could not conclude that the higher incidences were actually due to genetics versus environmental exposure. When questioned about the "Gerharsson's study", Dr. Hu admitted that the study showed no adverse effects on the kidneys of lead workers with a blood level of up to 60-70. However, Dr. Hu testified that the study was flawed for two reasons. One, the study is 12 years old and more studies have come out since which do show a connection of low to moderate exposure and renal function. Two, the study suffers from healthy worker bias, meaning only the working population is studied, ignoring those already sick who have

left the workforce.

Dr. Richard P. Wedeen is a Professor in Environmental and Community Medicine and board certified in Internal Medicine with a subspecialty in Nephrology. In his report, Dr. Wedeen concluded that Claimant's lead exposure and medical history were consistent with lead nephropathy as the major cause of his hypertension and progressive renal failure.

Defendants obtained a medical opinion from Dr. Michael E. Ullian, a Professor of Medicine in the Division of Nephrology who is board certified in Internal Medicine with a subspecialty in Nephrology. In his report, Dr. Ullian concluded that lead exposure did initiate Claimant's kidney disease. Dr. Ullian specifically pointed out Claimant's significant lead exposures at work, the "almost simultaneous" beginnings of both high blood pressure and kidney insufficiency, and the fact that no other causes of Claimant's kidney disease had been found. Dr. Ullian concluded that Claimant's kidney cancer was not directly related to lead exposure, stating that "[i]t is fairly common for kidney cancer to develop in patients whose kidneys have failed."

Defendants also relied on the opinion of Dr. Eugene Shippen, an Exide consultant who is board certified in family practice. Dr. Shippen testified that he worked as a consultant for Exide as well as other companies in the lead industry. Claimant objected to Defendants attempt to qualify Dr. Shippen as an expert in lead toxicology. Dr. Shippen admitted that the majority of seminars he has attended were sponsored by the lead industry and that he has no formal medical education in nephrology, environmental medicine, occupational medicine, or epidemiology. Dr. Shippen also admitted he has never done any independent medical or peer-reviewed research on lead toxicity.

In his deposition, Dr. Shippen testified that he examined Claimant once in 1989 during a visit to the Sumter plant, when one of the medical staff pointed out Claimant had elevated blood pressure

and some early renal problems. At the time, Claimant's lead level was around 34 and Dr. Shippen felt that the minor renal dysfunction was not related to his hypertension. He testified that Claimant's early exposure to lead prior to the implementation of OSHA standards aggravated the Claimant's pre-existing hypertension. Although Claimant continued to be exposed to lead after the OSHA standards were implemented in 1979 through 1996, these levels were not enough to cause further damage. The additional damage to Claimant's kidneys was caused by Claimant's noncompliance with treatment for hypertension.

On cross-examination, Dr. Shippen admitted there was no evidence of hypertension, kidney disease, or renal failure when Claimant began working for Exide in 1974. He also testified that Claimant was exposed to high levels of lead at the beginning of his employment. Dr. Shippen testified that lead exposure can cause kidney dysfunction and admitted that Claimant began having renal dysfunction in the 1980's. Although he disagrees with the literature, he admitted that some current literature supports a connection between lead exposure and high blood pressure. He agrees that Claimant is disabled and requires dialysis. He also agrees that some individuals may differ in their susceptibility to lead damage.

Dr. Eric Byrd has been the plant physician for Employer since 1988. In his deposition, he testified that he is also plant manager for 4 other companies and practices family medicine. Dr. Byrd testified that lead exposure can cause, aggravate or accelerate kidney disease. In his opinion, a normal blood level is below 10, and levels below 60-70 would not cause significant medical problems. He admitted that he relies on internal industry literature exclusively as the basis for these opinions. He concluded that Claimant's early exposure did affect his kidney function. Dr. Byrd testified that Claimant's continued exposure was not high enough to cause additional damage, but

did testify that exposure levels of 50 could aggravate or accelerate existing kidney disease. Dr. Byrd testified that he believes Claimant was noncompliant with his high blood pressure medication and that his kidney disease would not be as severe had he been compliant.

Dr. Byrd testified that in preparation for his deposition, he spoke with Dr. Shippen who provided him with an article concluding low level lead exposure did not have any affect on renal dysfunction. Dr. Byrd testified he also relied on Harrison's Book of Internal Medicine. He testified that kidney disease can increase blood pressure, that high blood pressure can cause kidney damage, but that lead exposure does not significantly increase blood pressure. However, Dr. Byrd admitted that Harrison's Principles of Internal Medicine, which he believes is an authoritative source, reports an association between hypertension and blood lead levels of 30, and that Claimant's blood levels were consistently above 30 during his employment.

FINDINGS OF FACT

Based on the stipulations of the parties, testimony and evidence received and produced at this hearing, the undersigned Commissioner finds the following facts based upon a preponderance of the evidence:

1. The parties to this proceeding are subject to and bound by the terms and provisions of the South Carolina Workers' Compensation Act. This finding is based upon the stipulations and admissions of the parties.
2. The Claimant's average weekly wage is \$602.63 with a compensation rate of \$401.75.
3. The date of Claimant's last injurious exposure was his last day of work in January 1996. It is undisputed that Mr. Franklin was exposed to lead up until the last date of his employment.

4. The Claimant suffered a compensable occupational disease as a result of chronic lead exposure, ultimately leading to cancer, removal of Claimant's kidneys and ongoing dialysis. Every medical provider in evidence concluded that Claimant's kidney disease was either caused by, accelerated, or aggravated by his exposure to lead on the job.

5. Claimant is entitled to, and Defendants shall pay, causally related medical treatment, both past and future for Claimant's lifetime.

6. Claimant is permanently and totally disabled as a result of his kidney disease.

7. Claimant shall receive, and Defendants shall pay, 500 weeks of total and permanent disability compensation.

8. Claimant shall receive compensation in a lump sum payment. However, Claimant's request for Utica-Mohawk language is denied. This finding is based on the lack of Commission authority to order such allocation of a lump sum award.

9. Claimant timely filed his claim for occupational disease satisfying the statute of limitations.

CONCLUSIONS OF LAW

Under the foregoing findings of fact, and accordingly, as provided in the South Carolina Code of Laws, as amended, it is concluded as follows:

1. Pursuant to South Carolina Code Ann. §42-11-10 and other applicable law, Claimant's average weekly wage is \$602.63 with a compensation rate of \$401.75.

2. Pursuant to South Carolina Code Ann. §42-11-10 and other applicable law, Claimant sustained compensable occupational disease as a result of chronic lead exposure during his employment with Employer and its predecessor companies.

3. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985) and Glenn v. Columbia Silica Sand Company, 112 S.E.2d 711, 236 S.C. 13 (1960) and other applicable law, Claimant's last injurious exposure was on his last day of employment in January 1996. There is no dispute that Claimant was exposed to lead until his last day of work.

4. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985) and Glenn v. Columbia Silica Sand Company, 112 S.E.2d 711, 236 S.C. 13 (1960) and other applicable law, Defendants are liable for this claim in its entirety as the carrier at risk on the date of Claimant's last injurious exposure. Where the employment at the time of disability is of a kind that contributes to the disease, liability for the entire compensation is that of the employer and the carrier at the time of the last exposure causally related to the disease.

5. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985), §42-15-40 and other applicable law, Claimant's claim is not barred by the statute of limitations.

6. Pursuant to South Carolina Code Ann. §42-9-10, §42-11-40, §42-9-10 and other applicable law, Claimant is permanently and totally disabled. Defendants shall pay 500 weeks of total and permanent disability compensation.

7. Pursuant to South Carolina Code Ann. §42-15-60 and other applicable law, Defendants shall pay all causally related medical treatment, both past and future for Claimant's lifetime.

8. Pursuant to South Carolina Code Ann. §42-9-301 and other applicable law, Defendants shall pay Claimant's award in a lump sum.

9. Claimant's request for Utica-Mohawk language is denied. Although the Commission may approve settlement with this language, the Commission does not have the authority to "order" this language anymore than it can order settlements themselves.

ORDER

IT IS ORDERED that the Claimant suffered a compensable occupational disease as a result of lead exposure during his employment with Employer.

IT IS FURTHER ORDERED that Claimant is permanently and totally disabled due to his kidney disease and entitled to 500 weeks of disability payments at the rate of \$401.75 per week. Payment shall be made in lump sum.

IT IS FURTHER ORDERED that Claimant is entitled to, and Defendants shall pay, causally related medical treatment, past and future for the period of Claimant's lifetime.

AND IT IS SO ORDERED.


George N. Funderburk, Commissioner

South Carolina Workers' Compensation Commission

6/3, 2005

CERTIFICATE OF SERVICE

This is to certify that the undersigned has this date served this order in the above entitled action upon all parties to this cause by depositing a copy hereof, postage paid, in the United States mail addressed to the attorney or attorneys for said parties.

W H M
Y F D

This 10 day of June, 2005

By Wayne H. Martin
Administrative Assistant to the Commissioner

APPELLATE PANEL DECISION AND ORDER
OF THE
SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION
W.C.C. FILE NO. 7423889

LEE ERNEST FRANKLIN,

EMPLOYEE,
CLAIMANT,

-VS-

YUASA EXIDE, INC.,

AFFIRM IN PART/
REVERSE IN PART

EMPLOYER,

AND

AMERICAN NATIONAL FIRE INSURANCE COMPANY,

INSURER,
DEFENDANTS.

Appellate Panel Review held in Columbia, South Carolina on November 22, 2005, timely
and properly served upon all parties of interest.

Appellate Panel Decision and Order filed

May 3, 2006

APPEARANCES:

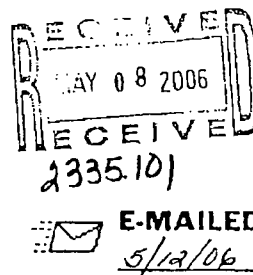
Claimant represented by:

MCANGUS, GOUDELOCK & COURIE, LLC with
W. Hugh McAngus appearing

Defendants represented by:

Vernon F. Dunbar

Appeal Due 6/2/06



STATEMENT OF THE CASE

The parties were heard by Commissioner George N. Funderburk on September 30, 2004 in Sumter, South Carolina. On June 10, 2005, the Hearing Commissioner issued the following Order:

"IT IS ORDERED that the Claimant suffered a compensable occupational disease as a result of lead exposure during his employment with Employer.

IT IS FURTHER ORDERED that Claimant is permanently and totally disabled due to his kidney disease and entitled to 500 weeks of disability payments at the rate of \$401.75 per week. Payment shall be made in lump sum.

IT IS FURTHER ORDERED that Claimant is entitled to, and Defendants shall pay, causally related medical treatment, past and future for the period of Claimant's lifetime.

AND IT IS SO ORDERED."

Within the statutory period, counsel for Defendants filed an Application for Review, setting forth grounds for appeal, copies of which were furnished to all interested parties prior to oral argument.

All proper testimony has been taken. Such, together with all documentary evidence has been delivered by argument to the individual members of the Panel and have since been under study and consideration.

By appeal, it is respectfully submitted that the Hearing Commissioner erred in the following:

1. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the date of Claimant's last injurious exposure to lead was on the last day of work in January 1996, when the reliable, probative and substantial evidence of the case does not support such a finding of fact or conclusion of law, the error being that the Claimant did not work in January 1996 and that the last

date of injurious exposure, if any, occurred from 1974 to 1981 or from 1981 through 1989.

2. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the Claimant suffered an occupational disease as a result of chronic lead exposure, the error being that the Claimant failed to prove that he was exposed to lead during the coverage period provided by Great American Insurance Company/American National Fire Insurance Company, which could have resulted in the kidney disease and kidney cancer.
3. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the Defendants are responsible for payment of all causally related medical treatment, both past and future for the Claimant's lifetime, the error being that §42-15-20 states that no compensation shall be payable, which may have accrued to notice being given to an employer.
4. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the Claimant timely filed his claim for occupational disease within two years, when the reliable, probative and substantial evidence of the case clearly reveals that notice was not given within two years from the date of accident nor within two years from the date of disablement or diagnosis.
5. The Hearing Commissioner erred in finding as fact and concluding as a matter of law that the Claimant is entitled to payment of the award in a lump sum, the error being that the Claimant never filed a Form 24 pursuant to Reg. 67-1605 requesting a lump sum nor did the Claimant obtain the Defendants' consent to payment of any award in lump sum pursuant to Reg. 67-1605, the error being that benefits are not owed and due until the promulgation of an award and that such is to be paid in weekly allocations unless a Form 24 has been filed and approved by the Commission; or, there is a finding regarding the date of disability.
6. The Hearing Commissioner erred in failing to find as a fact that the Claimant was continuously exposed to dangerously high levels of lead from the period 1974 through 1983, which exceeded current municipal exposure limits as established by OSHA, the error being that the Claimant's exposure to lead during this period constitutes the last injurious exposure and is the proximate cause of the Claimant's current disability.
7. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the proximate cause or etiology of the Claimant's renal kidney disease is as a consequence of chronic lead exposure, when the reliable, probative and substantial evidence of the case reflects that genetics, medical history, failure to comply with medical treatment regimens and hypertension are proximate causes of Claimant's kidney disease.

8. The Hearing Commissioner erred in failing to consider OSHA Standards and Federal Regulations regarding the permissible exposure limits to lead in his findings and conclusions with respect to what amount of exposure to lead constituted the last injurious exposure during the Claimant's employment.
9. The Hearing Commissioner failed to address the amount of exposure to lead, which can be deemed injurious as opposed to non-injurious, the error being that the Commissioner's finding of compensability is predicated upon speculation, conjecture and surmise.
10. The Hearing Commissioner erred in failing to find the date of Claimant's disability, the error being the date upon which the Claimant became disabled after having been made aware of the possible causal relationship between his employment activities and his kidney disease, the two statute of limitations bars this claim.
11. The Hearing Commissioner erred in finding as a fact and concluding as a matter of law that the last injurious exposure occurred during the time that Claimant was listed as an employee, as opposed to the time in which the Claimant last worked, with respect to designating Great American Insurance Company/American National Fire Insurance Company as the carrier on risk for payment of the Claimant's disability benefits, the error being that the Claimant's injurious exposure during the time that the Employer's Insurance of Wausau Insurance Company provided coverage during the period from 1974 through 1989, reflecting the period of an injurious exposure to lead.
12. The South Carolina Commissioner legally erred and abused its discretion in preventing the Defendants from adding additional parties to the case with respect to prior insurance companies, the error being that because the last injurious exposure occurred when other insurance carriers were on risk, the Defendant insurance carrier has been unduly prejudiced and ordered to pay benefits when the injury did not occur during its coverage period as a consequence of an injury by accident or occupational disease.
13. The Hearing Commissioner erred in failing to find that the Claimant's disability began in 1992/1993 when he began his dialysis treatment, the error being that prior to beginning dialysis treatment, the Claimant was made aware of the effect of exposure by lead on his kidneys.
14. The Hearing Commissioner erred in failing to place weight upon testimony that the Claimant was provided with safety equipment to minimize his exposure to lead resulting from his employment beginning in 1989 until he last physically worked in 1995, the error being that utilization of recommended OSHA and NIOSH equipment diminishes the possibility that the Claimant suffered an injurious exposure as a consequence of exposure to lead subsequent to 1989.

15. The Hearing Commissioner erred in failing to consider or place any weight upon the purchase agreement of Yuasa Exide, which states that the Exide Corporation and its insurance carrier, the Employer's Insurance of Wausau, will not be responsible for any claims regarding workers' compensation benefits filed by Exide employees, the error being that the Claimant was an Exide employee and has filed a workers' compensation claim.

Pursuant to S.C. Code Ann. §42-17-50 (1985), we, the Appellate Panel, have reviewed the award, weighed the evidence and considered all issues raised on appeal. The Panel may make its own findings of fact and reach its own conclusions of law. Based upon a review of the foregoing, the Panel affirms in part and reverses in part the Order of June 10, 2005 and enters the following findings of fact and conclusions of law.

FINDINGS OF FACT

1. The parties to this proceeding are subject to and bound by the terms and provisions of the South Carolina Workers' Compensation Act. This finding is based on the stipulations and admissions of the parties.
2. The Claimant's average weekly wage is \$602.63 with a compensation rate of \$401.75. This finding is based on the stipulations and admissions of the parties.
3. Claimant was employed by Yuasa Exide, Inc., a lead battery plant, from 1974 until 1996. American National Insurance Fire Insurance Co. provided workers' compensation coverage to Employer from 1991 through 1998.
4. Claimant suffered a compensable occupational disease as a result of chronic lead exposure, ultimately leading to cancer, removal of Claimant's kidneys and ongoing dialysis. Every medical opinion in evidence concluded that Claimant's kidney disease was either caused, accelerated, or aggravated by his exposure to lead on the job.
5. It is undisputed that Claimant was continuously exposed to lead during his employment up until his last day of work with Employer. The date of last injurious exposure was Claimant's last day of work in January 1996.
6. Claimant became disabled in January 1996, when he left work and underwent the removal of both kidneys for end-stage renal failure.
7. Defendants assertion that consideration must be given to Employer provided, safety equipment, compliance with OSHA guidelines, and overall decrease in lead exposure levels to determine injurious vs. non-injurious exposure is not persuasive. Such a determination is contrary to the rule of Glenn v. Columbia.

Silica Sand Company, 236 S.C. 13, 112 S.E.2d 711 (1960), providing that in cases of occupational disease, where the employment is of a kind contributing to the disease, liability is assigned to the carrier who was on the risk when the disease resulted in disability.

8. Claimant's employment was of a kind contributing to kidney disease. Every medical opinion in evidence concludes that lead exposure can cause kidney disease. Claimant was exposed to lead throughout his employment. Therefore, liability for the entire claim falls on Carrier, who was on the risk when Claimant became disabled in January 1996.
9. Claimant's kidney disease was a direct result of chronic lead exposure, a hazard greater than that ordinarily incident to normal employment and peculiar to the lead battery industry.
10. The first definitive diagnosis as to the cause of the Claimant's Kidney disease was made on January 24, 2001, by Dr. Richard Wedeen, who concluded that Claimant's "loss of kidney function and the development of end-stage renal disease was, in my opinion, due to lead nephropathy." (APA p.334). Thus, the statute of limitations would not have begun to run until this date of definitive diagnosis.
11. Claimant did not become aware of any definitive diagnosis until January 24, 2001. In his 1999 deposition, Claimant testified that no physician had informed him that his kidney failure or hypertension were a result of his employment, and that when he asked Dr. Suchinda if his condition was work-related she said "there was no medical proof." (APA p 126, lines 17-21).
12. The requirement of notice has been met. The first definitive diagnosis as to the cause of the Claimant's Kidney disease was January 24, 2001. Claimant provided notice well before the definitive diagnosis by filing his initial Form 50 in 1997.
13. Defendant's Motion to Add Parties was correctly denied. Liability in cases of occupational disease is not apportioned among successive carriers. As Carrier was on the risk when Claimant's disease resulted in disability, Carrier is liable for the entire claim.
14. Claimant is permanently and totally disabled as a result of his kidney disease. This finding is based on the stipulations and admissions of the parties.
15. Claimant's kidney disease was not accelerated or aggravated by non-compensable causes which would entitle Defendants to any apportionment of the award under §42-11-90.
16. Claimant shall receive, and Defendants shall pay, 500 weeks of total and permanent disability compensation, paid in weekly allocations. Benefits accrued to date are due and payable immediately. Claimant's request for payment of

compensation in lump sum is denied as it was not properly pled in Claimant's Form 50 or raised before the Hearing Commissioner.

17. The purchase agreement introduced by Defendant is irrelevant in a determination of Carrier's liability under the Act. Any cause of action for alleged indemnification would lie in civil court, and the Commission lacks jurisdiction to address the purchase agreement.

CONCLUSIONS OF LAW

Under the foregoing findings of fact, and accordingly, as provided in the South Carolina Code of Laws, as amended, it is concluded as follows:

1. Pursuant to South Carolina Code Ann. § 42-17-50 and other applicable law, the Commission has the power to affirm, amend, or reverse the decision of a Single Commissioner.
2. Pursuant to South Carolina Code Ann. §42-1-40 and other applicable law, Claimant's average weekly wage is \$602.63 with a compensation rate of \$401.75.
3. Pursuant to South Carolina Code Ann. §42-1-160 and other applicable law, Claimant sustained compensable occupational disease as a result of chronic lead exposure during his employment with Employer and its predecessor companies.
4. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985) and Glenn v. Columbia Silica Sand Company, 112 S.E.2d 711, 236 S.C. 13 (1960) and other applicable law, Claimant's last injurious exposure was on his last day of employment in January 1996.
5. Pursuant to Hanks v. Blair Mills, Inc. 335 S.E.2d 91, 286 S.C. 378 (Ct. App. 1985) and Glenn v. Columbia Silica Sand Company, 112 S.E.2d 711, 236 S.C. 13 (1960) and other applicable law, Defendants are liable for this claim in its entirety as the carrier at risk on the date of Claimant's last injurious exposure.
6. Pursuant to South Carolina Code Ann. §42-15-40 and other applicable law, Claimant filed his claim within the two-year statute of limitations.
7. Pursuant to South Carolina Code Ann. §42-15-20 and other applicable law, Claimant provided timely notice to his employer.
8. Pursuant to South Carolina Code Ann. §42-9-10 and other applicable law, Claimant is permanently and totally disabled. Defendants shall pay 500 weeks of total and permanent disability compensation.

9. Pursuant to South Carolina Code Ann. §42-15-60 and other applicable law, Defendants shall pay all causally related medical treatment, both past and future for Claimant's lifetime.
10. Claimant's request for payment of compensation in lump sum is denied as it was not properly pled in Claimant's Form 50 or raised before the Hearing Commissioner.
11. Pursuant to South Carolina Code Ann. §42-11-90 and other applicable law, Defendants are not entitled to any apportionment of the award.

ORDER

IT IS, THEREFORE, ORDERED the Decision and Order of the Single Commissioner is hereby affirmed in part and reversed in part.

IT IS ORDERED that the Claimant suffered a compensable occupational disease as a result of lead exposure during his employment with Employer.

IT IS FURTHER ORDERED that Claimant is permanently and totally disabled due to his kidney disease and entitled to, and Defendants shall pay, 500 weeks of disability payments at the rate of \$401.75 per week.

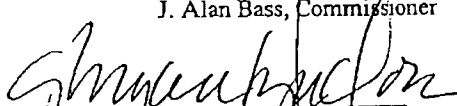
IT IS FURTHER ORDERED that Claimant is entitled to, and Defendants shall pay, causally related medical treatment, past and future for the period of Claimant's lifetime.

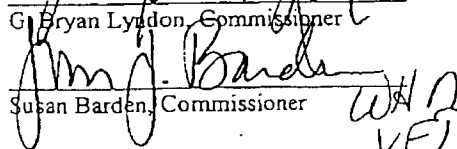
AND IT IS SO ORDERED.

S.C. WORKERS' COMPENSATION COMMISSION

J. Alan Bass, Commissioner

CONCUR:


G. Bryan Lyndon, Commissioner


Susan Barden, Commissioner

CERTIFICATE OF SERVICE

This is to certify that the undersigned has this date served this order in the electronic fashion upon all parties to this case by depositing a copy hereof, postage paid, in the State of South Carolina, to the attorney of record for each party.

This 24th day of May, 2006
By C. Madison
Administrative Assistant to the Commissioner

SOUTH CAROLINA WORKERS' COMPENSATION COMMISSION

W.C.C. FILE NO: 9643549

YUASA EXIDE, INC. AND GREAT
AMERICAN INSURANCE COMPANY,

Plaintiffs/Employer and Carrier,

vs.

SOUTH CAROLINA SECOND INJURY FUND

Defendant.

IN RE:

LEE ERNEST FRANKLIN,

Employee/Claimant,

v.

YUASA EXIDE, INC.,

Employer.

DECISION AND ORDER

HEARING:

Held in Sumter, South Carolina on November 27, 2007.

APPEARANCES:

Plaintiff/Employer and Carrier were represented by
Vernon F. Dunbar, Esquire, of TURNER, PADGET,
GRAHAM & LANEY, P.A.

Defendant, South Carolina Second Injury Fund was
represented by Latonya D. Edwards, Esquire.

PURPOSE OF THE HEARING:

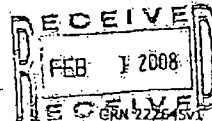
To determine issues as set forth on the Forms 54 and
55.

HEARD BY:

The Honorable George N. Funderburk, Commissioner

FILED:

January 31, 2008



STIPULATIONS

1. The purpose of the hearing is to determine the issues as set forth on the Forms 54 and 55. Also, whether the claim filed by Great American Insurance Company a/k/a American National Fire Insurance Company is barred by Section 15-3-600, which provides that any action for relief must be commenced within ten years after the cause of action has accrued.
2. Notice of the hearing was timely and properly provided to all parties of interest.
3. Proper venue is in Sumter County, South Carolina as agreed upon by all parties.
4. The South Carolina Worker's Compensation Commission has subject matter jurisdiction over this proceeding.
5. Without objection, the commission file was made a part of the record excluding any unstipulated medical reports, self-serving statements and hearsay testimony.

APA SUBMISSIONS

APA#	NAME OF PROVIDER/OTHER	DATE(S) OF RECORD(S)	PAGE NUMBERS
1	Letters from Dr. Hu	05/24/04 & 07/25/01	1 - 9
2	Dr. Wedeen Report	01/24/2001	10 - 11
3	Portion of Claimant's Employment file	1983 - 1985	12 - 20
4	Industrial Hygiene Laboratory Studies	1974 - 1985	21 - 25
5	Report of Dr. ER Shippen	09/14/2004	26 - 30
6	Report of Carroll Crawford	03/19/2002	31 - 33

STATEMENT OF THE CASE

Plaintiffs, Yuasa Exide, Inc., the employer, and Great American Insurance Company, the worker's compensation insurance carrier, by and through its attorney argued that the worker's compensation claim of Lee Ernest Franklin with respect to lead exposure resulting in renal kidney failure was subject to Second Injury Fund reimbursement. The plaintiffs contend that because the South Carolina Worker's Compensation Commission had ruled as a matter of law Lee Ernest Franklin's subsequent exposures to lead below OSHA's permissible exposure guidelines constituted subsequent accidents, Lee Ernest Franklin's last accidental injury occurred on his last date of employment in November 1995.

Lee Ernest Franklin, the claimant, had been exposed to high levels of lead that exceeded OSHA's permissible exposure guidelines between the years 1974 and 1985. During the 1980's, Mr. Franklin was diagnosed with Stage I renal kidney disease that continuously progressed to Stage V. The claimant's kidney disease was as a consequence of his continuous exposure to lead. The plaintiffs argued that had the claimant ceased working at Yuasa Exide at the time of his first diagnosis around 1982, the preexisting Stage I renal kidney disease would not have been aggravated by the claimant's subsequent exposures to lead.

The plaintiffs further argue that as a result of the claimant's exposure to lead, he developed hypertension. The claimant's hypertension was certainly aggravated throughout his employment with the employer because of his continuous exposure to lead. Again, relying upon the expert testimonies of Dr. Howard Hu, Associate Professor

of Harvard Schools of Public Health & Medicine, Richard P. Wedeen, and Eugene Shippen, M.D., neither the claimant's hypertension nor chronic renal disease would have been continuously aggravated but for the claimant's continuous exposure to lead.

Accordingly, the plaintiffs assert that because the disease or condition of heavy metal poisoning is listed as a condition that is presumed to be an impairment that results in 78 weeks or more of disability and constitutes a hindrance and obstacle to employment pursuant to Section 42-9-400 (d)(23), the claim is subject to reimbursement.

In the alternative, the plaintiffs assert that Dr. Eugene Shippen's testimony and his medical questionnaire corroborate that this claim is reimbursable for both heavy lead metal poisoning and the preexisting condition of hypertension. Dr. Howard Hu's testimony corroborated this conclusion. It is clear that Mr. Franklin's hypertensive state preexisted his last accidental injury or exposure in 1995; and that such was constantly aggravated as a result of subsequent exposures to lead. Moreover, Mr. Franklin's hypertensive state constituted a serious condition and a hindrance or obstacle to employment and reemployment. Because the claimant's hypertensive condition results in 78 weeks or more disability and combined with the renal kidney failure caused by heavy metal poisoning, the claim is also subject to reimbursement as a consequence of the diagnosis of hypertension.

The South Carolina Second Injury Fund, by and through its attorney, Latonya D. Edwards, first contended that the plaintiff's claim for reimbursement was barred by the ten year statute of limitations as set forth in Section 15-3-600 S. C. Code Annot.

§15-3-600 (2007). Specifically, the Fund avers that because an action for relief was not commenced within ten years after Lee Ernest Franklin's injury or contraction of an occupational disease, the plaintiff's claim must be barred as a matter of law. The Fund does not dispute that the plaintiffs placed it on notice of a potential claim for reimbursement in 1998. The Fund also concedes that the claim was not deemed compensable until after all appeals had been exhausted and abandoned by the parties in 2006.

Nonetheless, the Fund argues that the ten year statute of limitations should be applied despite the fact that Title 42 does not expressly set forth a statute of limitations on Second Injury Fund claims. The Fund's avers a delay in timely filing reimbursement claims often creates administrative confusion and unnecessary costs.

Last, Attorney Edwards contends that the claim is not subject to reimbursement due to the fact that Mr. Franklin suffered with an occupational disease, which is simply a progression of the initial or Stage I of the chronic renal disease that first began in the 1980's. Therefore, the Fund contends that only one injury occurred during the 1980's. There was no second accident, but merely a progression of the renal kidney disease caused by heavy metal poisoning.

In response to the Fund's arguments, the plaintiffs contend that Section 15-3-600 is not applicable and Section 42-9-400 does not set forth a statute of limitations by which to commence an action against the South Carolina Second Injury Fund. In short, the plaintiffs assert that because it properly and timely placed the Fund on notice of a claim while litigation involving compensability was still ongoing, it had

complied with the requirements as set forth in Section 42-9-400 S.C. Code Annot.
§42-9-400 (2007).

Additionally, the plaintiffs noted that the Appellate Panel Decision and Order filed on May 3, 2006 found as a fact and concluded as a matter of law that the claimant was not aware of the compensability of this claim until January 24, 2001, when he was definitively diagnosed as having an occupational disease or suffered from an accidental injury as a consequence of his employment duties. Dr. Howard Hu provided the definitive diagnosis. Therefore, the plaintiffs argued that even if a ten year statute of limitations statute is applicable in this case pursuant to Section 15-3-600, such did not begin to run until January 24, 2001, which is the earliest date the parties became aware of a potential compensable claim.

Finally, with respect to the Fund's assertion that the claimant did not suffer subsequent injuries by accident, but rather his disability was as a result of a progression of a progressive occupational disease, it was noted that the Hearing Commissioner and Appellate Panel concluded as a matter of law that the claimant's disability was clearly as a consequence of continuous exposure to lead or from heavy metal lead poisoning, which "either caused, accelerated, or aggravated" the claimant's kidney disease.

EVIDENCE OF THE CASE

Lee Ernest Franklin, the claimant, is a 54-year-old married male, who graduated from high school. Mr. Franklin also attended technical college for four years. Mr. Franklin testified that he was employed at Yuasa Exide, Inc. for 23 years and nine

months. Prior to his employment at Yuasa Exide, Inc., the claimant had worked at V. B. Williams Furniture Company.

The claimant testified that when he began working at Yuasa Exide, Inc., formerly known as The Exide Plant and Exide Battery in 1974, he was given a physical. The claimant stated that he no health problems when he began his work at Exide with respect to high blood pressure or kidney problems.

Mr. Franklin stated that he first began working as a stacking machine operator, which required him to stack negative and positive plates in the battery manufacturing process. The claimant testified that he was commonly exposed to lead dust when he began his work at The Exide Plant as a stacking machine operator in 1974 (Hrg. Tr. pp. 14-15).

Subsequently, the claimant was assigned to work in the pasting operation department. The pasting operation required Mr. Franklin to mix powdered lead with a formula of acid into a paste. The paste was then compressed into the grids, which in turn were utilized to manufacture a plate (Hrg. Tr. p. 15, ll. 3-11). The claimant further testified that the lead powder was stored in drums, which was dumped into a large mixer. According to the claimant, when the drums of lead were dumped, lead dust was in the environment (Hrg. Tr. p. 15, ll. 12-25). The claimant stated that during this operation, dust would collect on him and on the floor. Mr. Franklin testified that he worked as a pasting operator for approximately seven years (Hrg. Tr. p. 15-16).

The claimant was later transferred to the material handlers operation (Hrg. Tr. p. 16). In this manufacturing process, the claimant welded lead plates with a torch. The

claimant indicated that when welding lead plates, smoke and dust was created (Hrg. Tr. p. 17, ll. 2-12). Furthermore, the claimant indicated that as a material handler, he was required to transport material to other departments (Hrg. Tr. p. 17, ll. 13-25). During the transporting of various materials, he was exposed to dust. The claimant worked in the materials handling department for approximately seven years.

The claimant testified that his employment in the stacking, pasting and materials handlers departments were all considered high lead levels. The claimant last worked in the shipping department, which is a low lead area (Hrg. Tr. p. 21, ll. 22-25; p. 22, l. 1).

The claimant testified that he began having kidney problems in the early 1980's (Hrg. Tr. p. 32, ll. 21-25). As a matter of fact, the claimant admitted on cross-examination that when he was informed that he was losing protein, he knew it was a sign of kidney failure (Hrg. Tr. p. 32, ll. 21-25; p. 33, ll. 1-7). Upon being told that the claimant was having kidney failure, the claimant was moved from the pasting department to a less lead-laden environment (Hrg. Tr. p. 33, ll. 2-7). The claimant further admitted that he did not have to be moved while he was employed in the stacking department due to high lead levels, but was first moved once he began working in the pasting department during the late 1970's through 1983 (Hrg. Tr. p. 33).

The claimant testified that over the course of his employment at Yuasa Exide, Inc., he was removed from his employment duties in a particular department and reassigned to another department until his lead levels returned to an acceptable level (Hrg. Tr. p. 34-36).

MEDICAL EVIDENCE

Dr. Eric Byrd is an occupational medical physician, who provided medical services for Yuasa Exide's employees. Dr. Byrd testified via deposition that he has attended seminars concerning lead and lead exposure. Dr. Byrd further testified that he has reviewed studies regarding lead exposure and kidney function as a consequence of his work duties as an occupational medicine physician (Depo. Tr. pp. 17-19).

Dr. Byrd testified that over a three-year period from July 1976 through July 1979, the claimant's blood lead levels range from 60 to 91 micrograms per deciliter of blood (Depo. Tr. p. 23, ll. 9-17). According to Dr. Byrd, during this three year period of time, Mr. Franklin's blood lead levels generally were above 70 micrograms per deciliter of blood (Depo. Tr. p. 23, ll. 18-19).

Dr. Byrd also testified that from May 1980 through August 1984, Mr. Franklin's blood lead levels ranged from 40 to 60 micrograms per deciliter of blood (Depo. Tr. p. 23, ll. 20-25 and p. 24, ll. 1-8). Finally, from November 1983 through January 1989, the claimant's blood lead levels range from 31 to 57 micrograms per deciliter of blood.

Dr. Byrd testified that Yuasa Exide, Inc. monitored the employees' blood lead levels to prevent any type of illnesses that result from lead exposure. The removal process begins when an individual's blood lead levels reach 50 micrograms per deciliter of blood or greater (Depo. Tr. p. 28).

Dr. Byrd testified that beginning in 1981, it was discovered that Mr. Franklin had abnormalities of BUN or creatinine with respect a potential problem with his kidneys (Depo. Tr. p. 43). The claimant was requested to make an appointment with an

urologist, Dr. Cone, to discuss the elevation of BUN. Dr. Byrd testified that he was concerned that the claimant's kidney problems were related to lead exposure and discussed such with him and made an appointment for him to see Dr. Pusadee Suchinda, a nephrologist (Depo. Tr. p. 43 and 44, ll. 1-25; p. 45, ll. 1-10).

Dr. Byrd further testified that in June 1985, Mr. Franklin had stated in a roundtable discussion with former plant physician, Dr. Hursey. Dr. Hursey told the claimant to seek other employment because of his chronic renal condition and continuous lead exposure. Dr. Byrd testified from his review of the notes that Dr. Hursey had indicated that he had discussed the claimant's employment duties and his kidney condition on numerous occasions and that the claimant should not work around lead (Depo. Tr. p. 47, ll. 2-25). Dr. Byrd went on to explain that he was of the opinion that Mr. Franklin's exposure to lead in the early years of employment first caused or contributed renal problems. However, he was of the opinion that Mr. Franklin's subsequent employment duties, which did not result in his blood lead levels exceeding 50 or 60 micrograms per deciliter of blood, had little affect upon the claimant's kidney function (Depo. Tr. p. 50, ll. 3-18). According to Dr. Byrd, there is no doubt that the claimant's exposure to high levels of lead in the 1970's and early 1980's caused damage to the claimant's kidneys (Depo. Tr. p. 54, ll. 16-23).¹

Next, Dr. Eugene R. Shippen, a board certified physician and expert in lead toxicology, had testified in the merits hearing on behalf of the defendants. According to Dr. Shippen's testimony, he has approximately 25 years of practical experience in

¹ By 1988, Mr. Franklin had already been removed to a low lead level area wherein the lead in the air is less than 30 micrograms per cubic meter.

dealing with lead toxicology. Dr. Shippen is a member of the American Occupational Society Environmental Clinic Panel, which consists of nationally known American lead experts. Dr. Shippen completed a medical questionnaire about the aggravating effect of lead upon the claimant's health maladies.

The claimant retained Dr. Howard Hu, a Professor of Occupational and Environmental Medicine at the Harvard School of Public Health. In a report dated July 25, 2001, Dr. Hu noted that the claimant began with a pre-employment blood lead level of 28 ug/dL. When the claimant's blood lead level was next measured on February 11, 1975, it was 50 ug/dL. Dr. Hu noted that this substantial increase resulted from the claimant's occupational lead exposure. Dr. Hu noted that the claimant's blood lead levels fluctuated between 36 and 69 ug/dL until March 22, 1978, when it increased to 80 ug/dL. A peak blood lead level of 91 ug/dL was observed on August 21, 1978. Subsequently, the blood lead levels of Mr. Franklin fell to 70 ug/dL and below.

Dr. Hu opined that Mr. Franklin's occupational lead level exposure at Yuasa Exide, Inc. was a major contributing factor towards the causation of his renal failure. In particular, Dr. Hu opined that the claimant was exposed to lead at a level that exceeded OSHA Regulations and is associated with a toxic effect on the kidney. According to Dr. Hu, the OSHA standard requiring medical removal protection is 50 ug/dL. Dr. Hu noted that the claimant's blood lead levels exceeded the OSHA Regulations for most of the time that he worked at Yuasa up until the point where the first elevated serum creatinine was detected in 1981. Dr. Hu noted that this excessive exposure to lead in violation of OSHA Regulations continued during the 1980's; and that this "high

occupational exposure over 10+ years that result in body burdens typically associated with chronic renal failure..."

Dr. Hu later opined that subsequent exposures to lead at work aggravated the renal kidney disease and accelerated kidney failure.

Based upon the foregoing arguments, a review of the deposition testimonies, and the entire record, the following findings of fact are made:

FINDINGS OF FACT

1. Yuasa Exide, Inc. and Great American Insurance Company, the plaintiffs, clearly proved by a preponderance of the evidence that it is subject to reimbursement from the South Carolina Second Injury Fund with respect to the accidental injury to the kidneys or occupational disease suffered by Lee Ernest Franklin during the course and scope of his employment with Yuasa Exide, Inc. from 1974 through 1996.

2. The plaintiffs' claim is not barred by Section 15-3-600. First, there is no case law or any statutory law reflecting that a ten year statute of limitation is applicable in claims for reimbursement against the South Carolina Second Injury Fund. The only requirement in order to obtain funds from the Second Injury Fund with respect to a time limitation is set forth in Section 42-9-400. Specifically, if an employer and carrier does not file a claim prior to paying 78 weeks of benefits, the claim is deemed barred.

3. Assuming *arguendo* that a ten year statute of limitations defense is applicable with respect to Second Injury Fund claims, the instant case would not be subject to the ten year statute of limitations for the following reasons. The employer, carrier and claimant did not know whether the claim would be deemed compensable

until after all appeals had been exhausted. A notice of intent to appeal was filed with the South Carolina Court of Appeals but was subsequently withdrawn in 2007. Therefore, once the plaintiffs withdrew its appeal, the Order of the Circuit Court affirming the Commission's finding of compensability became the law of the case. The Circuit Court's Decision was promulgated on August 21, 2006. It appears that at the latest the parties recognized that the claim was, indeed, compensable and subject to reimbursement occurred on August 21, 2006. Therefore, the ten year statute of limitations would not begin to run until the date of the Circuit Court Order.

On the other hand, the earliest date upon which the ten year statute of limitations could potentially begin to run would be when the claimant first became aware as a matter of law and from a medical standpoint that he had sustained a compensable injury or malady as a consequence of his employment duties at Yuasa Exide, Inc. According to the Orders of the Commission and the Court, Lee Ernest Franklin was unaware of a compensable malady until January 24, 2001. The Full Commission Appellate Panel concluded that January 24, 2001 constituted the date of a definitive diagnosis. Prior to January 24, 2001, there was absolutely no evidence of a compensable accidental injury or occupational disease. Therefore, there was no basis for the plaintiff's to have filed a claim for reimbursement. Accordingly, the earliest date by which a theoretical ten year statute of limitations could be applied would be from January 24, 2001. Clearly, the plaintiffs filed a claim for reimbursement well before the expiration of a ten year statute of limitations if either the dates of 2001 and 2006 is utilized.

3. The plaintiffs proved by a preponderance of the evidence that it is entitled to reimbursement from the Second Injury Fund for benefits paid and payable to Lee Ernest Franklin as a consequence of heavy metal poisoning resulting from continuous lead exposure from 1974 through 1995. These exposures resulted in continuous and aggressive injuries. The continued exposure to lead accelerated the claimant's kidney failure. According to the Full Commission Appellate Panel's Decision and Order the subsequent exposures to lead accelerated and aggravated the claimant's renal kidney disease and the damage resulting from renal kidney disease. The subsequent exposures to lead was a direct cause of the renal kidney disease progressing from Stage I to Stage V. Absent the claimant's employment duties effect on his hypertension and renal kidney disease, the claimant's disease would not have progressed in the manner which it did as a result of his continuous exposure to lead.

4. Because the claimant suffered from renal kidney failure caused by heavy metal (lead) poisoning, the claim is subject to reimbursement. Because heavy metal poisoning is a listed disease, it is presumed that it constitutes a hindrance and obstacle to employment, and that such a condition leads to substantially higher medical costs, and disability in excess of 78 weeks.

5. The claim for reimbursement from the Second Injury Fund can also be predicated upon the claimant's preexisting disease of hypertension, which was clearly aggravated and accelerated by his constant subsequent exposures to lead until he last worked at Yuasa Exide, Inc. The hypertensive condition clearly qualifies this claim for

reimbursement as is evidenced by the Second Injury Fund medical questionnaire completed by Dr. Eugene Shippen; and through his testimony.

6. The Second Injury Fund's claim that there was no subsequent accidents which aggravated or accelerated the claimant's renal kidney disease is not supported by the evidence or the law. The Fund introduced no evidence to contradict the opinions of Drs. Richard Wedeen, Shippen, Hu, and Byrd. Additionally, in the case of the State Worker's Compensation Fund v. The South Carolina Second Injury Fund in RE: Hunt v. S.C. State Forestry Commission, 313 S.C. 536, 443 S.E.2d 546 (1994), the Court noted that a claimant's arteriosclerosis and cardiac disease qualified as a subsequent disability when the worker became disabled as a consequence of the preexisting malady. In short, the Court rejected the Fund's argument that an employer and carrier are only entitled to reimbursement if a subsequent injury takes the form of some observable event producing an injury. 313 S. C. 536, 539, 443 S.E.2d 546, 548.

7. The record is replete with evidence that the goals of the Second Injury Fund were met in this case. Yuasa Exide retained Lee Ernest Franklin in employment after knowledge of his prior impairments caused by renal kidney failure beginning in Stage I and continuing through Stage V. The claimant constantly was moved to various departments to minimize his exposure to lead. Moreover, the employer provided additional safety measures to reduce the claimant's exposure to lead and to minimize or eliminate subsequent accidental injuries.

8. During the claimant's continued employment he incurred a subsequent disability from the injury by accident and/or by occupational disease arising out of and

in the course of his employment, which ultimately resulted in end stage renal kidney failure.

9. The claimant's last injury resulting from lead exposure combined with the last stage of renal kidney disease prior to Stage V and/or aggravated the preexisting condition to cause a substantially greater disability and would have been caused from the subsequent injury alone; which was as a result of being exposed to lead within permissible guidelines as established by OSHA.

10. The end stage renal kidney disease most probably would not have occurred but for the preexisting hypertensive conditions and the preexisting first four stages of renal kidney disease.

11. The claimant's extensive hypertension would qualify for payment of 78 weeks or more in disability benefits.

CONCLUSIONS OF LAW

1. S.C. Code Annot. §42-1-160 (2007) governs accidental injuries.
2. S.C. Code Annot. §42-11-90 (2007) governs occupational diseases.
3. S.C. Code Annot. §42-9-400 (2007) governs South Carolina Second Injury Fund claims.

ORDER

IT IS HEREBY ORDERED that the South Carolina Second Injury Fund shall reimburse Yuasa Exide, Inc. and Great American Insurance Company and its assigns or

agents for all benefits paid to and payable to Lee Ernest Franklin in accordance with the statute.

AND IT IS SO ORDERED.

**SOUTH CAROLINA WORKERS'
COMPENSATION COMMISSION**

GNL
George N. Funderburk, Commissioner

Columbia, South Carolina

January 29, 2008

CERTIFICATE OF SERVICE

This is to certify that the undersigned has this date served this order in the above entitled action upon all parties to this cause by depositing a copy hereof, postage paid, in the United States mail addressed to the attorney or attorneys for said parties.

This 31 day of January, 2008.
Elaine M. Boyd
Administrative Assistant to the Commissioner

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389 S.C. 422, *; 699 S.E.2d 687, **;
2010 S.C. LEXIS 320, ***

Transportation Insurance Company and Flagstar Corporation, Carrier/Respondent, v. South Carolina Second Injury Fund, Appellant. Travelers Insurance Company and Barclays American, Carrier/Respondent, v. South Carolina Second Injury Fund, Appellant. Travelers Insurance Company and It's Fashion, Carrier/Respondent, v. South Carolina Second Injury Fund, Appellant. Great American Insurance Co. and Yuasa Exide, Inc., Carrier/Respondent, v. South Carolina Second Injury Fund, Appellant. Travelers Insurance Company and Marc Knapp DBA, Carrier/Respondent, v. South Carolina Second Injury Fund, Appellant. Travelers Insurance Company and All American Termite & Pest, Carrier/Respondent, v. South Carolina Second Injury Fund, Appellant.

Opinion No. 26880

SUPREME COURT OF SOUTH CAROLINA

389 S.C. 422; 699 S.E.2d 687; 2010 S.C. LEXIS 320

May 12, 2010, Heard
September 13, 2010, Filed

SUBSEQUENT HISTORY: Rehearing denied by Transp. Ins. v. SC Second Injury Fund, 2010 S.C. LEXIS 337 (S.C., Oct. 6, 2010)

DISPOSITION: [***1] REVERSED.**CASE SUMMARY**

PROCEDURAL POSTURE: Respondent insurers brought proceedings against appellant Second Injury Fund (fund), seeking reimbursement in cases in which more than ten years passed since claimants' date of injury. The South Carolina Workers' Compensation Commission determined that the statute of limitations found in S.C. Code Ann. § 15-3-600 did not apply to reimbursement cases. The fund appealed.

OVERVIEW: The fund argued that the ten-year limitations period of § 15-3-600 applied to reimbursement cases brought pursuant to S.C. Code Ann. § 42-9-400. The appellate court agreed. S.C. Code Ann. §§ 15-3-20(A), 15-3-600 mandated that reimbursement actions had to commence within ten years after the cause of action accrued. Because a reimbursement action was an action for relief not provided for in the chapter and there was no statute of limitations in S.C. Code Ann. tit. 42 that applied to claims for reimbursement, § 15-3-600 applied to claims for reimbursement. The fund also argued that a cause of action for

reimbursement accrued on the date of the claimant's injury. The appellate court disagreed. The proper time for accrual to begin was the date notice is given to the **fund**. The equitable doctrine of laches did not bar reimbursement in this case. The **fund's** laches arguments were not properly before the appellate court either because the issue was not preserved or the issue was unappealed and was now the law of the case.

OUTCOME: The decisions of the Workers' Compensation Commission were reversed.

CORE TERMS: reimbursement, statute of limitations, laches, carrier, citation omitted, civil actions, claimant's, notice, cause of action, preserved, date of injury, full commission, accrued, default, accrual, accrue, Reform Act, proper notice, entitled to reimbursement, abandoned, pursued, notice requirement, whole record, express terms, unambiguous, prescribed, unappealed, insured, insures, notify

LEXISNEXIS® HEADNOTES

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Governments > Legislation > Statutes of Limitations > Time Limitations 

Workers' Compensation & SSDI > Benefit Determinations > Cumulative & Successive Disabilities 

HN1  See S.C. Code Ann. § 15-3-600 (2005).

Civil Procedure > Appeals > Standards of Review > De Novo Review 

Governments > Legislation > Interpretation 

HN2  Statutory interpretation is a question of law subject to de novo review. More Like This Headnote | *Shepardize*: Restrict By Headnote

Administrative Law > Judicial Review > Standards of Review > Statutory Interpretation 

Governments > Legislation > Interpretation 

HN3  The construction of a statute by the agency charged with its administration will be accorded the most respectful consideration and will not be overruled absent compelling reasons. More Like This Headnote

Administrative Law > Judicial Review > General Overview 

Workers' Compensation & SSDI > Administrative Proceedings > Judicial Review > General Overview 

HN4  The Administrative Procedures Act governs judicial review of the Workers' Compensation Commission's decisions. More Like This Headnote | *Shepardize*: Restrict By Headnote

Administrative Law > Judicial Review > Standards of Review > Substantial Evidence 

Workers' Compensation & SSDI > Administrative Proceedings > Judicial Review > Standards of Review > Substantial Evidence 

HN5  Substantial evidence is not a mere scintilla of evidence nor evidence viewed from one side, but such evidence, when the whole record is considered, as would allow reasonable minds to reach the conclusion the Full Commission

reached. [More Like This Headnote](#) | [Shepardize: Restrict By Headnote](#)

[Governments > Legislation > Statutes of Limitations > General Overview](#) 

HN6  Statutes of limitations are not simply technicalities. Statutes of limitations embody important public policy considerations in that they stimulate activity, punish negligence, and promote repose by giving security and stability to human affairs. Statutes of limitations relieve courts of the burden of trying stale claims of those who have slept on their rights. The purpose of statutes of limitation is to ensure litigation is brought within a reasonable time in order that evidence be reasonably available and there be some end to litigation. [More Like This Headnote](#)

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HN7  See S.C. Code Ann. § 15-3-600.

[Governments > Legislation > Statutes of Limitations > General Overview](#) 

[Governments > Legislation > Statutes of Limitations > Time Limitations](#) 

HN8  So that other unnamed civil actions were not excluded from having a limitations period, the legislature created the S.C. Code Ann. § 15-3-600 ten-year default statute of limitations provision for other causes of action not specifically enumerated in S.C. Code Ann. tit. 15. [More Like This Headnote](#)

[Governments > Legislation > Statutes of Limitations > General Overview](#) 

HN9  See S.C. Code Ann. § 15-3-20(A) (2005).

[Governments > Legislation > Interpretation](#) 

[Workers' Compensation & SSDI > Administrative Proceedings > Judicial Review > General Overview](#) 

HN10  When reading a workers' compensation statute, the Supreme Court of South Carolina will strictly construe its terms, leaving it to the legislature to amend and define any ambiguities. [More Like This Headnote](#) | [Shepardize: Restrict By Headnote](#)

[Governments > Legislation > Interpretation](#) 

HN11  The cardinal rule of statutory construction is to ascertain and effectuate the intent of the legislature. The text of a statute as drafted by the legislature is considered the best evidence of the legislative intent or will. If a statute's language is plain, unambiguous, and conveys a clear meaning, then the rules of statutory interpretation are not needed and a court has no right to impose another meaning. A court will give words their plain and ordinary meaning, and will not resort to a subtle or forced construction that would limit or expand the statute's operation. [More Like This Headnote](#) | [Shepardize: Restrict By Headnote](#)

[Workers' Compensation & SSDI > Benefit Determinations > Cumulative & Successive Disabilities](#) 

HN12  See S.C. Code Ann. § 42-9-400(f).

Governments > Legislation > Statutes of Limitations > Time Limitations 

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > General Overview 

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > Notice Periods 

Workers' Compensation & SSDI > Benefit Determinations > Cumulative & Successive Disabilities 

HN13  S.C. Code Ann. § 42-9-400(f) is not a statute of limitations, but a notice requirement. Moreover, there is no statute of limitations in S.C. Code Ann. tit. 42 that applies to claims for reimbursement. Reimbursement actions are actions for relief not provided for in the chapter. S.C. Code Ann. § 15-3-600 (2005). Hence, S.C. Code Ann. §§ 15-3-20(A), 15-3-600 mandate that reimbursement actions must commence within ten years after the cause of action shall have accrued. [More Like This Headnote](#)

Governments > Legislation > Statutes of Limitations > General Overview 

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > General Overview 

HN14  The Court of South Carolina has rejected the contention that the two-year statute of limitations for employees' workers' compensation claims contained in S.C. Code Ann. § 42-15-40 (Supp. 2009) also governed claims for reimbursement. [More Like This Headnote](#)

Governments > Legislation > Statutes of Limitations > General Overview 

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > General Overview 

HN15  Because a reimbursement action is an action for relief not provided for in the chapter and there is no statute of limitations in S.C. Code Ann. tit. 42 that applies to claims for reimbursement, S.C. Code Ann. § 15-3-600 applies to claims for reimbursement. For these reasons, § 15-3-600 applies to claims for reimbursement. [More Like This Headnote](#)

Governments > Legislation > Statutes of Limitations > Time Limitations 

Workers' Compensation & SSDI > Administrative Proceedings > Claims > Time Limitations > General Overview 

Workers' Compensation & SSDI > Benefit Determinations > Cumulative & Successive Disabilities 

HN16  The proper time for accrual of a claim for reimbursement begins is the date notice is given to the Second Injury Fund. [More Like This Headnote](#)

Civil Procedure > Appeals > Reviewability > Preservation for Review 

HN17  Issues and arguments are preserved for appellate review only when they are raised to and ruled on by the lower court. An unappealed ruling is the law of the case and requires affirmance. [More Like This Headnote](#) | [Shepardize: Restrict By Headnote](#)

Civil Procedure > Appeals > General Overview 

Civil Procedure > Appeals > Briefs 

Civil Procedure > Appeals > Reviewability > Preservation for Review 

HN18  An issue is deemed abandoned where the appellant fails to provide arguments or supporting authority for his assertion. Short, conclusory statements made without supporting authority are deemed abandoned on appeal and therefore not preserved for review. More Like This Headnote | *Shepardize*: Restrict By Headnote

COUNSEL: Latonya Dilligard Edwards , of Columbia, for Appellant.

Deborah Casey Brown  and Kyle A. Hougham, both of Gallivan, White & Boyd, Jason Alexander, Griggs, of Wilson, Jones, Carter & Baxley, and Vernon F. Dunbar , of Turner, Padgett, Graham & Laney, all of Greenville; Stephen L. Brown , William L. Howard , and Russell G. Hines , all of Young, Clement & Rivers, of Charleston, for Respondents.

JUDGES: CHIEF JUSTICE TOAL , KITTREDGE , and HEARN , JJ., concur. PLEICONES , J., concurring in part and dissenting in part in a separate opinion in which Acting Justice James E. Moore , concurs.

OPINION BY: TOAL 

OPINION

[*425] [688] CHIEF JUSTICE TOAL :** We granted South Carolina Second Injury Fund's (Fund) motion to transfer several appeals from the circuit court to this Court. We reverse the decisions of the South Carolina Workers' Compensation Commission.

[*426] [689] FACTS/PROCEDURAL HISTORY**

On June 20, 2007, the South Carolina General Assembly passed the Workers' Compensation Reform Act (Reform Act), which provides for the winding down of the Fund by June 30, 2013. See S.C. Code Ann. § 42-7-320(A) (Supp. 2009). **[***2]** Subsequent to the passage of the Reform Act, several carriers requested reimbursement for claims in which more than ten years had passed since the claimant was injured. The Fund denied reimbursement in those cases based on the general ten-year statute of limitations, which provides **HN17**  "[a]n action for relief not provided for in this chapter must be commenced within ten years after the cause of action shall have accrued." S.C. Code Ann. § 15-3-600 (2005). Carriers in the present action pursued reimbursement in cases in which more than ten years passed since claimant's date of injury.

Travelers Insurance Company  (Travelers) insured four of the six employers involved in this action. The four employers insured by Travelers are Barclay's American, It's Fashions, All American Termite & Pest, and Marc Knapp. ¹ Travelers is requesting reimbursement for claimants with the following dates of injury: December 19, 1989; July 18, 1994; August 19, 1996; and August 8, 1997. Travelers provided timely and proper notice to the Fund pursuant to South Carolina Code Ann. § 42-9-400 (Supp. 2009) in all four of these claims.

FOOTNOTES

¹ The Fund believes Marc Knapp, It's Fashions, and All American Termite & Pest **[***3]** are no longer in business because no information was discovered on the Secretary of State's database for these businesses.

Transportation Insurance Company (Transportation) insures Flagstar Corporation and is seeking reimbursement for a claimant with a March 19, 1993 date of injury. Transportation provided **[*427]** proper notice of a possible claim to the **Fund** pursuant to section 42-9-400.

Great American Insurance Company (Great American) insures **Yuasa Exide, Inc.**, which is now known as EnerSys Corporation. Great American is seeking reimbursement for a January 1, 1996 date of injury. ² Great American provided proper notice of a possible claim to the **Fund** pursuant to section 42-9-400. ⁷

FOOTNOTES

² The commission found the date of injury to be January 24, 2001. January 1, 1996 is the **Fund's** position for the date of injury.

The cases referenced above have been adjudicated by the Workers' Compensation Commission, which ultimately determined the statute of limitations found in section 15-3-600 did not apply to reimbursement cases. The **Fund** appealed these cases to the circuit court. However, while the appeals were pending, the **Fund** sought review by this Court and we transferred the appeals from the circuit court **[***4]** to this Court.

ISSUES

- I. Does the statute of limitations provision in section 15-3-600 apply to reimbursement cases pursued against the **Fund**?
- II. When does time begin to accrue for claims brought under section 42-9-400?
- III. Does laches apply such that the carriers outlined above are not entitled to reimbursement?
- IV. Under section 42-9-400 is **Yuasa Exide** entitled to reimbursement?

STANDARD OF REVIEW

HN2 Statutory interpretation is a question of law subject to de novo review. *Town of Summerville v. City of N. Charleston*, 378 S.C. 107, 110, 662 S.E.2d 40, 41 (2008) (citation omitted). **HN3** "The construction of a statute by the agency charged with its administration will be accorded the most respectful consideration and will not be overruled absent compelling reasons." *Dunton v. S.C. Bd. of Exam'rs In Optometry*, 291 S.C. 221, 223, 353 S.E.2d 132, 133 (1987) (citations omitted). **HN4** The Administrative Procedures Act governs judicial review of the Workers' Compensation Commission's decisions. See *Lark v. Bl-Lo, Inc.*, 276 S.C. 130, 276 S.E.2d 304 (1981). This Court can modify the commission's decision in this case only if the **Fund's** substantial rights have been prejudiced because the decision is affected by **[***5]** an **[**690]** error of law or is clearly erroneous in view of the reliable, probative, and substantial evidence on the whole record. See S.C. Code Ann. § 1-23-380(5)(d), (e) (Supp. 2009); see also **[*428]** *Shealy v. Aiken County*, 341 S.C. 448, 454-55, 535 S.E.2d 438, 442 (2000). **HN5** "Substantial evidence is not a mere scintilla of evidence nor evidence viewed from one side, but such evidence, when the whole record is considered, as would allow reasonable minds to reach the conclusion the Full Commission reached." *Shealy*, 341 S.C. at 455, 535 S.E.2d at 442 (citation omitted).

LAW/ANALYSIS

I. Statute of Limitations

The **Fund** argues the ten-year statute of limitations period outlined in section 15-3-600 applies to reimbursement cases brought pursuant to section 42-9-400. We agree.

HN6 "Statutes of limitations are not simply technicalities." *Moates v. Bobb*, 322 S.C. 172, 176, 470 S.E.2d 402, 404 (Ct. App. 1996). "Statutes of limitations embody important public policy considerations in that they stimulate activity, punish negligence, and promote repose by giving security and stability to human affairs." *Id.* (citation omitted). Statutes of limitations relieve courts of the burden of trying stale claims of those who have *****6** slept on their rights. See *McKinney v. CSX Transp., Inc.*, 298 S.C. 47, 49-50, 378 S.E.2d 69, 70 (Ct. App. 1989). "The purpose of statutes of limitation is to ensure litigation is 'brought within a reasonable time in order that evidence be reasonably available and there be some end to litigation.'" *Hooper v. Ebenezer Senior Servs. & Rehab. Ctr.*, 377 S.C. 217, 227, 659 S.E.2d 213, 218 (Ct. App. 2008) (quoting *City of North Myrtle Beach v. Lewis-Davis*, 360 S.C. 225, 231, 599 S.E.2d 462, 464 (Ct. App. 2004)).

Section 15-3-600 is found in chapter 3 of title 15 which addresses "Limitations of Civil Actions." Section 15-3-600 states, **HN7** "An action for relief not provided for in this chapter must be commenced within ten years after the cause of action shall have accrued." **HN8** So that other unnamed civil actions were not excluded from having a limitations period, the legislature created this ten-year default statute of limitations provision for other causes of action not specifically enumerated in title 15. South Carolina Code Ann. § 15-3-20(A) (2005) provides, **HN9** "Civil actions may only be commenced within the periods prescribed in this title after the cause of action has accrued, **[*429]** except when, in special *****7** cases, a different limitation is prescribed by statute."

HN10 When reading a workers' compensation statute this Court will strictly construe its terms, leaving it to the legislature to amend and define any ambiguities. *Wigfall v. Tideland Utils., Inc.*, 354 S.C. 100, 110, 580 S.E.2d 100, 105 (2003). **HN11** "The cardinal rule of statutory construction is to ascertain and effectuate the intent of the legislature." *Hodges v. Rainey*, 341 S.C. 79, 85, 533 S.E.2d 578, 581 (2000) (citation omitted). The text of a statute as drafted by the legislature is considered the best evidence of the legislative intent or will. See *id.* "If a statute's language is plain, unambiguous, and conveys a clear meaning, then the rules of statutory interpretation are not needed and a court has no right to impose another meaning." *Strickland v. Strickland*, 375 S.C. 76, 88, 650 S.E.2d 465, 472 (2007) (citation omitted). "The Court will give words their plain and ordinary meaning, and will not resort to a subtle or forced construction that would limit or expand the statute's operation." *Harris v. Anderson County Sheriff's Office*, 381 S.C. 357, 362, 673 S.E.2d 423, 425 (2009) (citation omitted).

Section 42-9-400 is plain and unambiguous. *****8** Section 42-9-400(f) states in pertinent part:

HN12 "An employer or his carrier must notify the Workers' Compensation Commission and the Director of the Second Injury **Fund** in writing of any possible claim against the **fund** as soon as practicable but in no event later than after the payment of the first seventy-eight weeks of compensation. . . . Failure to comply with the provisions of this subsection shall bar an employer or his carrier from recovery from the **fund**."

HN13 This is not a statute of limitations, but a notice requirement. Moreover, there is no statute of limitations in title 42 that applies to claims for reimbursement. Reimbursement

[**691] actions are actions "for relief not provided for in this chapter." S.C. Code Ann. § 15-3-600 (2005). Hence, sections 15-3-20(A) and 15-3-600 mandate that reimbursement actions must commence within ten years after the cause of action shall have accrued. If this were not so, then so long as the notice requirement of 42-9-400(f) was met, these actions could theoretically [**430] extend endlessly into the future, thwarting the intent of the legislature's passing of section 15-3-600.

In *Greenwood Mills, Inc. v. Second Injury Fund*, 315 S.C. 256, 433 S.E.2d 846 (1993), ^{HN14} this [***9] Court rejected the contention that the two-year statute of limitations for employees' workers' compensation claims contained in S.C. Code Ann. § 42-15-40 (Supp. 2009) also governed claims for reimbursement. *Greenwood Mills*, 315 S.C. at 259, 433 S.E.2d at 848. The question presented in this case was not before the Court in *Greenwood Mills*. *Greenwood Mills* merely addressed whether section 42-15-40 applied to claims for reimbursement. We found that, on its face, section 42-15-40 was inapplicable to claims for reimbursement. *Id.* at 259, 433 S.E.2d at 847. The Court noted that section 42-15-40 "applies to claims for compensation, not reimbursement; it governs claims addressed to the Commission as opposed to those addressed to the Fund; it speaks to an employee's injury, not to an employer's notice of injury." *Id.* (emphasis in original). Like this Court did in *Greenwood Mills*, we must apply the default statute of limitations according to its express terms. ^{HN15} Because a reimbursement action is an "action for relief not provided for in this chapter" and there is no statute of limitations in title 42 that applies to claims for reimbursement, section 15-3-600 applies to claims for reimbursement. [***10] For these reasons, section 15-3-600 applies to claims for reimbursement, and the decisions of the Workers' Compensation Commission are reversed.

II. Accrual of Time

The Fund argues a cause of action for reimbursement accrues on the date of claimant's injury. We disagree.

The Fund contends there are three possible dates on which time starts to accrue: (1) date of claimant's injury; (2) date carrier provides notice to the Fund; and (3) when the underlying claims conclude. The date the claimant is injured is not the proper time because at that time, in most instances, carriers would not be aware of a possible claim to the Fund. We find ^{HN16} the proper time for accrual to begin is the date notice is given to the Fund. By the time notice is given, carriers are aware of a possible claim and have up to 78 weeks after paying benefits to notify the Fund of a claim. Hence, a cause of [**431] action for reimbursement begins to accrue on the date a carrier provides notice to the Fund. ³

FOOTNOTES

³ The result in this case of accrual beginning when notice is provided is that Great American's claim is the only one that is not precluded by the running of the ten-year statute of limitations.

III. Laches

The Fund argues that if [***11] the ten-year statute of limitation does not apply, the equitable doctrine of laches bars reimbursement in this case. We disagree.

^{HN17} "Issues and arguments are preserved for appellate review only when they are raised to and ruled on by the lower court." *Elam v. S.C. Dep't of Transp.*, 361 S.C. 9, 23, 602 S.E.2d 772, 779-80 (2004) (citations omitted). An unappealed ruling is the law of the case and requires affirmance. See *Charleston Lumber Co., Inc. v. Miller Housing Corp.*, 338 S.C. 171, 175, 525 S.E.2d 869, 871 (2000) (citations omitted).

The **Fund's** laches claim against Flagstar is not preserved because it failed to plead laches and the full commission did not rule on it. The **Fund's** laches arguments against Barclay's American, Marc Knapp, and It's Fashions are not preserved because it did not raise laches in the courts below. The **Fund's** laches argument regarding **Yuasa Exide** is not preserved because the issue was not pled below and neither the single commissioner nor the full commission ruled on laches. The single commissioner made the following finding of fact in All American Termite & Pest's case, "The SIF waived any defense based on the equitable doctrine of laches by failing to assert [***12] the affirmative defense on their Form 55 or Form 58." The [***692] **Fund** failed to appeal the single commissioner's finding to the full commission; thus, it is the law of the case. In summary, the **Fund's** laches arguments are not properly before this Court either because the issue is not preserved or the issue was unappealed and is now the law of the case. ⁴

FOOTNOTES

⁴ At oral argument there was much discussion regarding laches. While laches does not apply to any of the cases in this appeal, laches could certainly be applicable to cases where carriers seek reimbursement from the **Fund**.

[*432] IV. Yuasa Exide

The **Fund** contends Great American is not entitled to reimbursement because claimant's, hypertension and liver failure did not preexist claimant's occupational exposure. We disagree.

The **Fund** failed to cite any authority for its position. Moreover, the half-page argument made by the **Fund** falls far short of overcoming the substantial evidence standard of review. Hence, the **fund** has abandoned this issue. See *First Sav. Bank v. McLean*, 314 S.C. 361, 363, 444 S.E.2d 513, 514 (1994) (*HN18*) (issue deemed abandoned where appellant failed to provide arguments or supporting authority for his assertion); *Eaddy v. Smurfit-Stone Container Corp.*, 355 S.C. 154, 164, 584 S.E.2d 390, 396 (Ct. App. 2003) [***13] ("[S]hort, conclusory, statements made without supporting authority are deemed abandoned on appeal and therefore not preserved for our review.2).

CONCLUSION

For the foregoing reasons, the decisions of the Workers' Compensation Commission are reversed.

KITTREDGE and **HEARN**, JJ., concur. **PLEICONES**, J., concurring in part and dissenting in part in a separate opinion in which Acting Justice James E. Moore concurs.

CONCUR BY: PLEICONES (In Part)

DISSENT BY: PLEICONES (In Part)

DISSENT

JUSTICE PLEICONES: I dissent in part and concur in part, and would affirm the decisions of the Workers' Compensation Commission (Commission) holding that S.C. Code Ann. § 15-3-600 (2005) is inapplicable to these reimbursement requests.

Chapter 3 of Title 15, entitled "Limitation of Civil Actions," is concerned with the time periods,

i.e. statutes of limitation, within which a 'civil action' can be commenced. A "civil action" within the meaning of Title 15 is brought in a court, and is "commenced when the summons and complaint are filed with the clerk of court..." S.C. Code Ann. § 15-3-20(B) (2005). **[*433]** See also Rule 2, SCRPC; "There shall be one form of action to be known as civil action"; compare *McDowell v. S.C. Dep't of Soc. Serv.*, 304 S.C. 539, 405 S.E.2d 830 (1991)

[*14]** (administrative proceeding is a civil action for purposes of the attorney's fees statute once it reaches circuit court, but not while before agency). On the other hand, a claim for reimbursement from the **Fund** is made by written notice to the Commission and to the Director of the **Fund**. S.C. Code Ann. § 42-9-400(f) (Supp. 2009). I agree with the Commission that S.C. Code Ann. § 15-3-600, the default statute of limitations provision for civil actions, is irrelevant to the question whether carriers timely pursued their reimbursement claims from the **Fund**.

The majority also holds that it is following *Greenwood Mills, Inc. v. Second Injury Fund*, 315 S.C. 256, 433 S.E.2d 846 (1993) by applying "the default statute of limitations [i.e. § 15-3-600] according to its express terms." As I read *Greenwood Mills*, it holds that the statute of limitations found in S.C. Code Ann. § 42-15-40 did not apply to **Fund** reimbursement claims, but that those claims were timely so long as they are made within the period provided by S.C. Code Ann. § 42-9-400(f) (Supp. 2009). Here, all the carriers gave proper and timely notice as required by § 42-9-400. In my opinion, *Greenwood Mills* does not support the majority's **[***15]** conclusion that a **Fund** reimbursement request is a "civil action" within the meaning of Title 15.

In light of my view that the Commission correctly found § 15-3-600 inapplicable, I would not reach the accrual issue. Moreover, I agree with the majority that neither **[**693]** the laches question nor the **Yuasa Exide** issue is preserved for our appellate review.

I concur in part and dissent in part.

Acting Justice James E. Moore Jr., concurs.

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* Click on any *Shepard's* signal to *Shepardize*® that case.

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S.C. SECOND INJURY FUND REIMBURSEMENT REQUEST FORM

Mail To:

SECOND INJURY FUND OPERATIONS
POST OFFICE BOX 12349
COLUMBIA, S.C. 29211

Second Injury Fund No.
Carrier No.
Date of Accident
Compensation Rate

PLEASE REVIEW INSTRUCTIONS ON REVERSE SIDE

Employee:		Employer:		Carrier	
Date of first payment of compensation:			Date for 100% Medical Reimbursement: (1st Day of 79th Week after accident)		
Type of Comp Paid	Date Beginning	Date To	Weeks	Amount	
TEMPORARY TOTAL					
TEMPORARY PARTIAL					
PERMANENT PARTIAL					
PERMANENT TOTAL					
DEATH OR OTHER (SPECIFY)					
TOTAL INDEMNITY TO DATE					
WEEKS AMOUNT					
LESS 78 WEEKS	Wks at Full Compensation Rate				
	Wks at Temporary Partial Rate				
NET REIMBURSABLE INDEMNITY REIMBURSEMENT					
LESS ANY PRIOR INDEMNITY REIMBURSEMENT					
TOTAL INDEMNITY REIMBURSEMENT REQUESTED					
TOTAL MEDICAL REIMBURSEMENT REQUESTED (ENCLOSED ITEMIZED LIST - SEE INSTRUCTIONS)					
TOTAL REIMBURSEMENT REQUESTED					

I hereby certify, that to the best of my knowledge and belief, the information as indicated above is true and correct as of this date: _____

MAIL REIMBURSEMENT TO:

	NAME
	ADDRESS

		NAME OF CARRIER OR EMPLOYER, IF SELF-INSURED
		SIGNED BY
		TITLE DATE

I. MEDICAL REIMBURSEMENT:

- A. Reimbursement for all medical bills incurred during the first 78 weeks will be 50% of any amount over \$3,000.
(Example: \$5,000 less \$3,000 = \$2,000 x 50% = \$1,000 Reimbursement)
- B. Reimbursement for all medical bills incurred after the first 78 weeks will be 100% of any amount.
(Example: \$1,000 x 100% = \$1,000 Reimbursement)
- C. Recommend you request reimbursement when it reaches \$5,000 and quarterly thereafter until the 78th week.
- D. Or, file on completion of the 78th week.
- E. Medical after 78 weeks will be reimbursed quarterly beginning 91st week.

II. REIMBURSEMENT REQUESTS:

- A. The Second Injury Fund will provide Reimbursement Request forms when the case is accepted for reimbursement:
 - 1. A completed Reimbursement Request form will be returned to the Second Injury Fund for each request. Both indemnity and medical may be included on the same form.
 - 2. The Reimbursement Request form must be accompanied by an itemized list of all medical expenses, if medical reimbursement is requested.
- B. The itemized list must be in the following format:

	<u>DESCRIPTION</u>	<u>DATE INCURRED</u>	<u>AMOUNT</u>
1.	General Hosp.	1-14-75 to 1-24-75	\$3,470.14
2.	Dr. Hanson	1-24-75	\$384.20
3.	Jones Boot Store Orthopedic Boots	3-6-75	\$47.20
4.	Drug Bills*	1-4-75 to 4-4-76	\$327.84

NOTE: Copies of all medical bills attached and numbered to correspond with itemized list. Also use the date incurred to place these bills in chronological order.

- * One entry may be made for all drug bills incurred during 1st 78 weeks and one entry after the 78th week, **BUT DO NOT** combine the two entries.

CASE NOTES

Where a settlement has been entered into, it is a duty to file a copy of this settlement agreement with the Industrial Commission. *Lowther v Standard Oil Co.*, 206 SC 286, 31 SE2d 889 (1945).

Subject to the conditions in this section [Code 1962 § 72-191] and Code 1962 § 72-351 the Industrial Commission has the power to approve a lump sum settlement, and make it final and binding and not subject to review un-

der any conditions. *Atkins v Charleston Shipbuilding & Drydock Co.*, 206 SC 63, 33 SE2d 46 (1945).

Workers' compensation settlement agreement was not binding until it had been approved by Industrial Commission, and thus, prior to such approval, claimant could unilaterally repudiate offer of settlement which had been previously accepted by his attorney. *Mackey v Kerr-McGee Chemical Co.*, 312 SE2d 585 (1984).

§ 42-9-400. Manner in which employer or insurance carrier shall be reimbursed from Second Injury Fund when disability results from preexisting impairment and subsequent injury.

(a) If an employee who has a permanent physical impairment from any cause or origin incurs a subsequent disability from injury by accident arising out of and in the course of his employment, resulting in compensation and medical payments liability or either, for disability that is substantially greater, by reason of the combined effects of the preexisting impairment and subsequent injury or by reason of the aggravation of the preexisting impairment, than that which would have resulted from the subsequent injury alone, the employer or his insurance carrier shall in the first instance pay all awards of compensation and medical benefits provided by this Title; but such employer or his insurance carrier shall be reimbursed from the Second Injury Fund as created by § 42-7-310 for compensation and medical benefits in the following manner:

(1) Reimbursement of all compensation benefit payments payable subsequent to those payable for the first seventy-eight weeks following the injury.

(2) Reimbursement of fifty percent of medical payments in excess of three thousand dollars during the first seventy-eight weeks following the injury and then reimbursement of all medical benefit payments payable subsequent to the first seventy-eight weeks following the injury; *provided, however*, in order to obtain reimbursement for medical expense during the first seventy-eight weeks following the subsequent injury, an employer or carrier must establish that his liability for medical payments is substantially greater, by reason of the combined effects of the preexisting impairment and subsequent injury or by reason of the aggravation

of the preexisting impairment, than that which would have resulted from the subsequent injury alone.

(b) If the subsequent injury of such an employee shall result in the death of the employee, and it shall be determined that the death would not have occurred except for such preexisting permanent physical impairment, the employer or his insurance carrier shall in the first instance pay the compensation prescribed by this Title; but he or his insurance carrier shall be reimbursed from the Second Injury Fund created by § 42-7-310, for all compensation payable in excess of seventy-eight weeks.

(c) In order to qualify under this section for reimbursement from the Second Injury Fund, the employer must establish ("by written records which shall be filed with the Commission and the fund" was deleted April 13, 1982.) when claim is made for reimbursement thereunder, that the employer had knowledge of the permanent physical impairment at the time that the employee was hired, or at the time the employee was retained in employment after the employer acquired such knowledge. *Provided, however,* the employer may qualify for reimbursement hereunder upon proof that he did not have prior knowledge of the employee's preexisting physical impairment because the existence of such condition was concealed by the employee or was unknown to the employee.

(d) As used in this section, "*permanent physical impairment*" means any permanent condition, whether congenital or due to injury or disease, of such seriousness as to constitute a hindrance or obstacle to obtaining employment or to obtaining reemployment if the employee should become unemployed.

When an employer establishes his prior knowledge of the permanent impairment, then there shall be a presumption that the condition is permanent and that a hindrance or obstacle to employment or reemployment exists when the condition is one of the following impairments:

- (1) Epilepsy
- (2) Diabetes
- (3) Cardiac disease
- (4) Arthritis
- (5) Amputated foot, leg, arm or hand
- (6) Loss of sight of one or both eyes or a partial loss of uncorrected vision of more than seventy-five percent bilateral
- (7) Residual disability from poliomyelitis
- (8) Cerebral palsy

- (9) Multiple sclerosis
- (10) Parkinson's disease
- (11) Cerebral vascular accident
- (12) Tuberculosis
- (13) Silicosis
- (14) Psychoneurotic disability following treatment in a recognized medical or mental institution
- (15) Hemophilia
- (16) Chronic osteomyelitis
- (17) Ankylosis of joints
- (18) Hyperinsulinism
- (19) Muscular dystrophy
- (20) Arteriosclerosis
- (21) Thrombophlebitis
- (22) Varicose veins
- (23) Heavy metal poisoning
- (24) Ionizing radiation injury
- (25) Compressed air sequelae
- (26) Ruptured intervertebral disc
- (27) Hodgkins disease
- (28) Brain damage
- (29) Deafness
- (30) Cancer (EFFECTIVE June 8, 1982: not retroactive.)
- (31) Sickle-cell anemia (EFFECTIVE June 8, 1982: not retroactive.)
- (32) Pulmonary disease (EFFECTIVE June 8, 1982: not retroactive.)
- (33) Mental retardation provided the employee's intelligence quotient is such that he falls within the lowest percentile of the general population. However, it shall not be necessary for the employer to know the employee's actual intelligence quotient or actual relative ranking in relation to the intelligence quotient of the general population.
- (34) Any other pre-existing disease, condition or impairment which is permanent in nature and which:
 - (a) Would qualify for payment of weekly disability benefits of seventy-eight weeks or more under Section 42-9-30 exclusive of benefits payable for disfigurement or
 - (b) Would support a rating of seventy-eight or more weeks of weekly disability benefits when evaluated according to the standards

applied to Workers' Compensation claims in South Carolina, or combines with a subsequent injury to cause a permanent impairment rated at seventy-eight weeks or more under Section 42-9-30.

(e) The Second Injury Fund shall not be bound as to any question of law or fact by reason of any compensation agreement, settlement, award, and adjudication to which it was not a party, or in relation to which it was not notified at least twenty days prior to a hearing on liability that it might be subject to liability for the injury or death.

(f) An employer or his carrier shall notify the Industrial Commission and the Director of the Second Injury Fund in writing of any possible claim against the fund as soon as practicable but in no event later than after the payment of the first seventy-eight weeks of compensation.

Failure to comply with the provisions of this subsection shall bar an employer or his carrier from recovery from the fund. (EFFECTIVE May 4, 1976.)

(g) If the employee has a permanent physical impairment, as defined in this section and the prerequisites for reimbursement have been met, and if it can be shown that the subsequent injury most probably would not have occurred "but for" the presence of the prior impairment, then reimbursement will be granted as provided in this section even if the combined effects or the aggravation of the preexisting condition by the subsequent injury does not cause the employer's liability for compensation and medical benefits to be substantially greater than that which would have resulted from the subsequent injury alone.

(h) When a third party is deemed to be an employer for the purposes of paying workers' compensation benefits that third party will be entitled to reimbursement from the Second Injury Fund if either he or the employer of record have met the knowledge requirements outlined in this section, as well as all other requirements.

(i) The Second Injury Fund is entitled to a credit for sums recovered by the employer or his workers' compensation carrier from third parties, after the employer or his workers' compensation carrier have been reimbursed for the moneys paid out by them and not reimbursed by the fund.

(j) The Second Injury Fund can enter into compromise settlements at the discretion of the director with approval of a majority of the Industrial Commission, provided a bona fide dispute exists.

HISTORY: 1962 Code § 72-601; 1972 (57) 2578; 1974 (58) 2237; 1976 Act No. 560 § 1, May 4, 1976; 1982 Act No. 314, eff April 13, 1982; 1982 Act No. 438, eff June 8, 1982.

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JUL 17 2015

SC Court of Appeals

IN THE STATE OF SOUTH CAROLINA
In the Court of Appeals

APPEAL FROM SUMTER COUNTY
Court of Commons Pleas

The Honorable W. Jeffrey Young, Circuit Court Judge

C.A. No.: 2013-CP-43-02284
Appellate Case No.: 2014-002215

Arrowpoint Capital Corporation/Arrowwood Indemnity Co., Appellant,

v.

South Carolina Second Injury Fund, Respondent,

IN RE: Mary McConico, Employee,

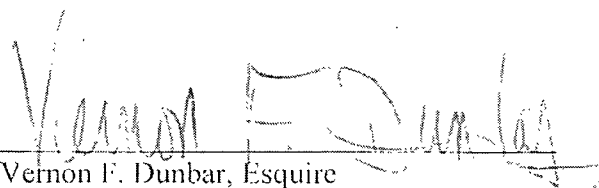
v.

Yuasa-Exide, Inc., Employer, and Arrowpoint Capital Corp./Arrowwood Indemnity Co., Carrier

CERTIFICATE OF COMPLIANCE

The undersigned hereby certifies that the Record on Appeal filed in this matter contains all the material proposed to be included by the parties to this matter and does not include any other material. The undersigned further certifies that the Record on Appeal filed in this matter complies with the South Carolina Supreme Court's Order and caption *In re: Advised Order Concerning Pers. Identifying Info. & Other Sensitive Info. In Appellate Court Filings*, 407 S.C. 607, 757 S.E.2d 421 (2014).

June 25, 2015


Vernon F. Dunbar, Esquire
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Post Office Box 2980
Greenville, South Carolina 29602
(864) 239-4000
Attorneys for Appellants

IN THE STATE OF SOUTH CAROLINA
In the Court of Appeals

APPEAL FROM SUMTER COUNTY
Court of Common Pleas

The Honorable W. Jeffrey Young, Circuit Court Judge

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JUL 17 2015

SC Court of Appeals

C.A. No.: 2013-CP-43-02284
Appellate Case No.: 2014-002215

Arrowpoint Capital Corporation/Arrowood Indemnity Co., Carrier, Appellant,
v.
South Carolina Second Injury Fund, Carrier, Respondent,

In re:

Mary McConico, Employee/Claimant

v.

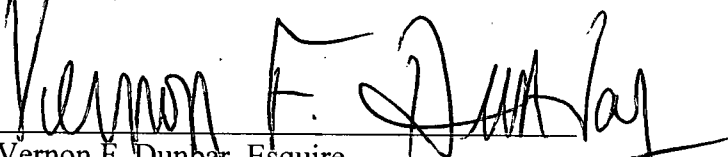
Yuasa-Exide, Inc., Employer and Arrowpoint Capital Corp./Arrowood Indemnity Co., rier.

PROOF OF SERVICE

I certify that I have served the Record on Appeal, Final Brief and Final Reply Brief of Appellant by depositing a copy of them in the United States Mail, postage prepaid, on the 15th day of July, 2015 addressed to:

The Honorable Jenny Abbott Kitchings
Clerk of Court
South Carolina Court of Appeal
1015 Sumter Street
Columbia, South Carolina 29211

Timothy Killen (Final Brief and Final Reply Brief only)
Wilson, Jones, Carter & Baxley
4500 Fort Jackson Boulevard
Columbia, South Carolina 29209



Vernon F. Dunbar, Esquire
McAngus, Goudelock & Courie LLC
Post Office Box 2980
Greenville, South Carolina 29602
(864) 239-6735
Attorney for Appellants

REPLY TO

VERNON F. DUNBAR
Direct Dial: (864) 239-6735
vernon.dunbar@mgclaw.com

July 15, 2015

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JUL 17 2015

SC Court of Appeals

The Honorable Jenny Abbott Kitchings
Clerk of Court
South Carolina Court of Appeals
1015 Sumter Street
Columbia, South Carolina 29211

RE: Arrowpoint Capital Corporation/Arrowood Indemnity Co. v. South Carolina
Second Injury Fund
In Re: Mary McConico v. Yuasa-Exide, Inc.
Appellate Court Tracking No.: 2014-002215
Civil Action No.: 2013-CP-43-02284

Dear Ms. Kitchings:

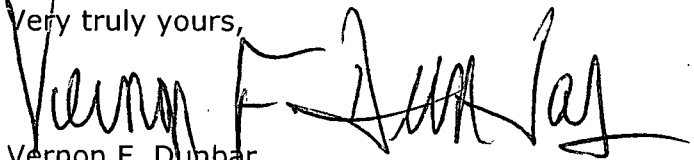
Enclosed for filing in the above referenced matter are the following:

1. An unbound copy and 9 bound copies of the Record on Appeal;
2. An unbound copy and 9 bound copies of Appellant's Final Brief;
3. An unbound copy and 9 bound copies of Appellant's Final Reply Brief;
4. An original and one copy of the Proof of Service reflecting service on the South Carolina Court of Appeals for the Record on Appeal, Final Brief and Final Reply Brief as well as reflecting service of the Final Brief and Final Reply Brief to Timothy Killen, attorney for the South Carolina Second Injury Fund, as the Record on Appeal was previously served.

I would appreciate it if you would return a clocked copy of the Proof of Service in the envelope provided for your convenience.

Please call me if you have any additional questions regarding the enclosed information. With kindest regards, I remain/

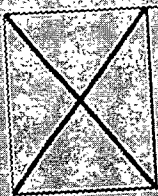
Very truly yours,


Vernon F. Dunbar

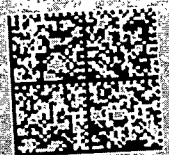
VFD/rhd

Enclosures

cc: Timothy B. Killen, Esquire (w/copy of Final Brief and Final Reply Brief
of Appellant)
Eric Rowell, Arrowpoint Capital Corp.



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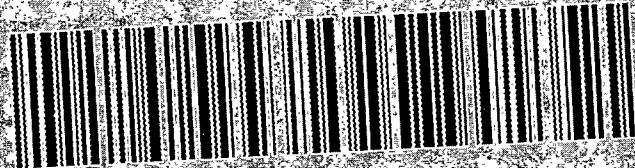
Rosie Devoy
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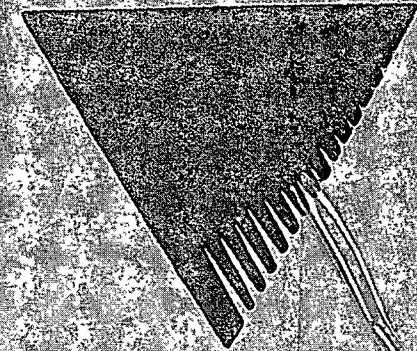
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Columbia SC 29201-3726

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FROM



Letter