

THE WELDING ROD PRIMER

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Introduction

This article is designed to provide risk managers and insurance claims professionals with a brief overview of the factual background of welding rod litigation and its evolving issues. It is hoped that it will assist them in better understanding these hotly debated and complex issues.

Manganese

(L. magnes: magnet, from magnetic properties of pyrolusite; Itl. manganese, corrupt form of magnesia)

Manganese is the twelfth most common element in the earth's crust and is the fourth most widely used metal in the world. The ancient Egyptians and Romans used it in the manufacture of glass. It was recognized by Scheele, Bergman, and others as an element and isolated by Gahn in 1774 by reduction of the dioxide with carbon.

Nearly 90 percent of all manganese produced each year is used in the production of steel. It is a gray-white color, resembling iron, but is harder and very brittle. The metal is reactive chemically, and decomposes in cold water slowly. Manganese is used to form many important alloys. In steel, manganese improves: rolling and forging qualities, strength, toughness, stiffness, wear resistance, hardness, and hardenability.

The dioxide (pyrolusite) is used as a depolarizer in dry cells, and is used to "decolorize" glass that is colored green by impurities of iron. Manganese by itself colors glass an amethyst color, and is responsible for the color of true amethyst. The dioxide is also used in the preparation of oxygen and chlorine, and in drying black paints. The permanganate is a powerful oxidizing agent and is used in quantitative analysis and in medicine. Manganese has also been used in fertilizer production, the manufacture of fireworks, matches, and as a catalyst in animal feed supplements.

Manganese is an essential trace element found in the cells of virtually all-living organisms and may be essential for utilization of B1. Manganese deficiency has been observed to affect the skin, produce changes in cholesterol and glucose metabolism, as well as affect growth, and cause reproductive failure and depression.

Manganese gains entry into the body through both inhalation and ingestion. It is found in the air, water and soil. In non-occupationally exposed individuals, the major contributor is food, including: blueberries, nuts and seeds, seaweed, egg yolks, whole grains, legumes and tea. Approximately 3-5 percent of ingested manganese is absorbed through the gastrointestinal system while airborne manganese, especially as a fume, is readily absorbed through the lungs. It is eliminated primarily through feces with a half-life average ranging from 4 to 39 days.

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Non-occupational exposed individuals have been reported to have a usual range of manganese blood level of 0.8-1.6 micrograms/dl and 0.11-2.67 micrograms/liter urine level. Water supplies in certain areas can be a considerable contributor to manganese levels amounting to an additional 3-5 mg per day.

Airborne manganese dust experienced during occupational exposure can consist of particles ranging from sub-micron levels to more than 10 microns in diameter. The current ACGIH threshold limit value (TLV) for manganese is a time weighted average of 0.2 mg/m³; however, prior to 1996, the recommended levels were much higher, typically 5 mg/m³.

Considerable research has not yielded a biomarker for manganese exposure applicable to all exposure situations. Recent work has, however, identified serum prolactin levels as a possible biomarker. (Smargiassi and Mutti, 1999). Air levels have not traditionally correlated well with blood or urine levels.

Welding

Welding is defined by the American Welding Society as “a metal-join process wherein coalescence is produced by heating to suitable temperatures with or without the use of filler metal.” It has been estimated that up to two percent of the working population in industrial countries perform some aspect of welding.

Manganese exposure is highest for those who work with manganese every day, including welders. Over the last several years, a large number of lawsuits have been filed against welding companies alleging injuries arising from the inhalation of manganese contained in welding fumes.

Exposure to welding fumes can cause a variety of diseases, including metal fume fever, which has symptoms similar to common influenza: fever chills, nausea, cough, fatigue and general weakness and aching of the head and body. Excessive manganese exposure causes Manganism or manganese induced Parkinson's Disease.

Other chemicals that may be present in the fluxes used or that may be produced by the welding operation are carbon dioxide, carbon monoxide, fluoride, nitrogen dioxide, nitric oxide, ozone and particulates.

Parkinson's Disease

Parkinson's Disease is a neurological illness named after Dr. James Parkinson, a London physician who was the first to describe it in 1817. Parkinson's disease (or PD) is a disorder that results from a degeneration of the dopamine – producing neurons in a portion of the brain referred to as the substantia nigra pars compacta (SNpc). Basal ganglia are critically dependent on the SNpc for normal movement and function. The loss (death) of these cells produces a reduction in a vital chemical called "dopamine," which causes symptoms including resting

tremor, a generalized slowing and loss of movement, rigidity and stooped posture. Other symptoms may include loss of facial expression, reduction in speech volume and clarity, difficulty swallowing, change in size of handwriting, dry skin, constipation, urinary difficulties, and depression. Because Parkinson's disease is a progressive disorder, these symptoms worsen with time.

Parkinson's disease occurs in approximately 150 people per 100,000 population and has an annual incident rate of 10 to 20 per 100,000. Although there is a sharp increase in incidents after age 50, the etiology of Parkinson's remains unknown. There is, however, a decrease in incidents among smokers, those exposed to pesticides and herbicides, and rural populations.

Parkinson's patients generally respond to treatment with the medication Levedopa, used to replenish the dopamine no longer effectively produced by the patient.

Scientists recently reported that welding might trigger the early onset of Parkinson's Disease. A research team led by neurologist Brad A. Racette, M.D., found that professional welders developed typical clinical and neurological signs of the disease an average of 15 years earlier than the general population. The study was featured in the professional journal Neurology with an accompanying editorial. Dr. Racette has however admitted on cross examination during litigation, that his article entitled "Welding-related Parkinsonism" does not prove that exposure to welding fumes causes earlier onset Parkinson's disease.

Manganism

Severe chronic exposure to manganese produces a condition known as manganism or manganese induced Parkinsonism. The first observation of manganism was made in was made in 1837 by John Couper in men grinding the black oxide of manganese. By 1940, 353 cases had been reported in the literature.

The clinical presentation of Manganism begins months to years after the first exposure. A 1955 study of Roier observed a latency period ranging from one month to 10 years. Latency of several years is typical depending upon the extent of exposure. Others have reported clinical onset years after exposure cessation. (Cook et al. 1974).

Typically Manganism begins as behavioral changes including apathy, asthenia, anorexia, lassitude, insomnia or somnolence and slowed or clumsy movements. Other reported symptoms may include myalgia, cramps, headaches, speech and gait problems, sweating, nightmares, and paresthesias. A psychiatric presentation known as manganese psychosis consisting of aggressive and mental excitement may also be observed.

Manganism is often compared to Parkinson's disease because it produces similar symptoms, although there are differences between the two. Manganism is believed to affect the basal ganglia of the brain specifically the globus pallidus while the main affect in Parkinson's disease is the substantia nigra.

Clinically, Manganism may be differentiated from Parkinson's disease. There are some similarities in neurological symptoms including changes to the gait and posture, but with less tremor and more dystonia (a distorted twisting or movement of a part of the body). Nonetheless, the clinical picture is markedly different in at least one respect: Manganism is not known to affect the SNpc. PET scans (positron emission tomography) have been used to establish whether or not there is damage to the SNpc. Generally, damage to the SNpc has resulted in the patient being diagnosed with idiopathic (the cause is unknown) Parkinson's disease rather than Manganism. MRI (magnetic resonance imaging) studies have suggested that the high intensity signals in the mid brain resolve following exposure cessation. Levedopa (L-Dopa) is useful in treating Parkinson's disease, but studies show it does not have the same effect in treating Manganism.

The tremor in Parkinson's disease has been classically identified as a resting tremor while that of Manganism has been described as an intentional tremor.

The average intake of manganese in adults eating a western type diet ranges from 0.7 to 10.0 mg of manganese per day. It is estimated that less than 3 percent of ingested manganese is absorbed and less than 1 percent is retained after biliary excretion. Manganese homeostasis is controlled via the liver with the majority of manganese being excreted as bile.

Manganese Medical Causation

Welding of mild steel is a process that produces fumes containing trace elements of manganese. These tiny particles that comprise this component of welding fumes are spherical in shape and coated with a glassy layer of silicon caused by the extremely high temperatures inherent in the welding process. Defendants argue that the characteristics of these particles, along with a very low concentration of these particles in air surrounding the welder, make it very difficult to hypothesize an effective mechanism for significant biological uptake of manganese in this form.

Dependants also argue that because manganese is an essential element the human body has evolved sophisticated defenses against this toxic substance when it enters the body. The body quickly identifies any excess and rapidly excretes it through the bile duct and liver. Finally, manganese is limited in its ability to cross the blood brain barrier.

Epidemiologists have concluded based on the current literature involving welders and neurological illness, that there is currently no scientific reliable epidemiological cause and effect relationship between exposure to welding fumes and Parkinson's disease, Manganism, Parkinsonism or other neuropsychological injury. Antidotal case reports and clinical observations, although of interest, cannot establish cause and effect association.

The conclusion of the epidemiologists is based in large part on the fact that although there have been hundreds of thousands of people employed as full time welders over many years, there are only a few case reports to support the hypothesis of an association between the disease and welding. The case reports, however, do not exclude other alternative causes for the identified disease in the welder-patient's studied, and cannot provide any support for conclusions of cause

and effect associations. For example, the liver is the organ that clears the body of excess dietary manganese. There was liver disease in some of the patients in the reported cases that cannot be excluded as a cause of the reported illness. More importantly, there are no reports of manganese from welders serving in the ship building industry during World War II when the number of American welders had increased 200%.

There are studies using animals that have hypothesized a direct olfactory mechanism for the passage of manganese into brain tissue. But all of these studies have used animals with much different olfactory systems than humans. No study has demonstrated that the human olfactory system is a source of direct manganese uptake to the brain. And as the U.S. Supreme Court affirmed in General Electric Company, et al. v Joiner, 522 US 136 (1997), animal studies are no substitute for good science in establishing chemical substances as the cause of diseases in humans.

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