**INTRODUCTION**

Manganese was first discovered by Sheele in Sweden in 1774, and named ‘manganese’ by Guyton de Marveau in 1785. South Africa, with more than 50 million tons of alumino-silicates, contains around 40% of the world’s known reserves of these minerals and accounts for nearly half of the Western world’s production. With manganese reserve in excess of 4000Mt, 80% of the world’s known manganese ore deposits are located in the Northern Cape and North West Province. The country’s annual output in 1999 was 3122Kt, of which 1569Kt was exported.

Couper first described industrial manganese poisoning in 1837 in five workers in France, who made bleaching powders using manganese dioxide. He found that long-term exposure to manganese dust caused a peculiar extra-pyramidal syndrome. However, his observations were forgotten for a long time, and it was only in 1919 that a definite relationship between the epidemiologic, clinical, and pathological effects that manganese poisoning has on the central nervous system was established by Edsall, Wilbur and Drinker in the USA. With the onset of World War II the accelerated growth of the steel industry led to a high incidence of manganese poisoning, crippling as many as 25% of their working population, as reported by Schualer et al. (1957), who described in great detail the extra-pyramidal syndrome.

Important uses for manganese include the production of steel, non-ferrous alloys, dry cell batteries, electrode coating in welding rods, glass industry, textile bleaching and in fertilizers. It is also used in the chemical industry. Considerable exposure takes place from manganese-containing ores, especially iron ores. Organic manganese has been used as a fungicide and is still used as an anti-knock agent in gasoline. Most hazardous manganese exposure occurs in the mining and the smelting of ore.

In this decade, manganese poisoning has become rare, even in developing countries. The recent cases of manganese poisoning in South Africa remind us of the need to keep a high index of suspicion to the possibility of excess exposure to hazardous chemicals in South Africa.

**EXPOSURE LEVELS**

Exposure levels differ worldwide; TLV – ACGIH: 0.2 mg/m³, OSHA 5.0 mg/m³ respectively from the US and SA. SA has a level 25 times higher than the US.

**ABSORPTION, DISTRIBUTION AND EXCRETION**

Manganese is an essential element for humans and is absorbed via food and water intake. It is part of the enzyme mitochondrial super oxide dismutase (SOD) in rats and is essential for many animal species for the formation of bone and connective tissue, as well as the metabolism of carbohydrates and lipids. The daily requirements for humans is about 2–3 mg (WHO 1981).

Manganese in the workplace is mainly absorbed through inhalation. After inhalation (or parenteral and oral exposure) the absorbed manganese is rapidly eliminated from the blood and distributed mainly to the liver. Manganese preferentially accumulates in tissue rich in mitochondria. It also penetrates the blood-brain barrier. The biological half-life for manganese is between 36 and 41 days, but for manganese in the brain it is considerably longer. Manganese in the blood binds to the erythrocyte porphyrin complex. Excretion of manganese is mostly through the faeces, thus the bile route is the main route of excretion for manganese. Only 0.1 – 1.3% of the daily intake of manganese is excreted through the urine.

**SYMPTOMS AND SIGNS**

The symptoms and signs of manganese poisoning or manganism can occur after variable periods of heavy exposure ranging from 6 months to 3 years at average air levels of 5 mg/m³. The disease begins insidiously with headache, irritability and occasionally psychotic behaviour. The latter, manganese psychosis, occurs most frequently in miners rather than in industrial workers, and consists of transitory psychological disturbances such as hallucinations, compulsive behaviour and emotional instability.
Severe somnolence, followed by insomnia, is often found early in the disease. As manganese exposure continues, symptoms include generalized muscle weakness, speech impairment, inco-ordination and impotence; tremors, paresthesia and muscle cramps have been noted. In advanced stages the subject exhibits excessive salivation, inappropriate emotional reaction and Parkinson-like symptoms, such as mask-like faces, severe muscle rigidity and gait disorders. The dystonic posture of the limb is often accompanied by painful cramps, and is characteristic of findings in manganese poisoning. Manganism is reversible if it is limited to psychological disturbances and the worker is removed from exposure early.

Exposure levels associated with advanced manganism typically have to be very high. 150 cases were found in three mines where air dust levels reached 450 mg/m³. Recent studies revealed seven cases with defined signs and symptoms out of 117 workers exposed to 5 mg/m³.

An association between manganese exposure and pulmonary effects including pneumonia, chronic bronchitis and airway impairment has been observed. Extrapolation from animal studies suggests that it is unlikely that manganese could be the sole etiologic agent responsible for serious pathological changes in the lungs. Instead it is possible that susceptibility to infection is increased.

Acute poisoning by manganese is rare, but may occur following large ingested or inhaled amounts of manganese.

Metal-fume fever, an influenza-like illness is characterized by chills, fever, sweating, nausea and coughing. The syndrome begins 4–12 hours after exposure to manganese oxide fumes and lasts 24 hours without causing permanent damage.

**DIAGNOSIS**

There is no specific diagnostic test for manganese poisoning, and the diagnosis depends chiefly upon a combination of characteristic neurological features and an occupational history of exposure (Cook et al., 1974).

Diagnosis has been based on the following points:
1. Symptoms and signs appear after exposure to dust or fumes of manganese for more than 3 months.
2. Symptoms and signs appear gradually and then become progressive, to some extent.
3. The initial symptoms are various and vague. They are asthenia, anorexia, apathy, insomnia and daytime-drowsiness, and a slowing down in performing motor acts.
4. Main manifestations are psychiatric and neurological.

Neither blood nor urinary manganese levels correlate with symptom and signs or the neurological and psychiatric manifestations. Therefore blood and urinary concentration are of little value in the diagnosis of manganese poisoning other than confirming exposure or assisting in differential diagnosis.

**BIOLOGICAL EFFECT MONITORING**

The organ systems affected can be monitored with chest X-rays, lung functions, liver functions, full blood counts, specialized neurological and psychiatric questionnaires and medical examinations that elicit specific symptoms and signs.

**BIOLOGICAL MONITORING**

Normal concentrations of manganese in blood range from 7–12 µg/l (Chang, W.). Analysis of manganese levels in biological samples for exposure is a very important component in determining individual and group trends of exposure in the work environment. Due to the short biological half-life in blood of less than 5 minutes, blood concentration will only be elevated very shortly after manganese exposure. The homeostatic mechanisms involved in trying to keep tissue concentrations on a constant level make it more difficult to detect increased blood levels. Blood manganese is mainly a reflection of body burden of the metal. Because of the very low percentage excretion in urine, this medium is also not a good indicator of exposure but if high may assist in proving exposure. On a group basis, manganese in urine, to some extent, seems to reflect recent exposure and will be higher than the non-exposed group. The main importance of biological monitoring is to determine trends to prevent increasing levels of manganese. Normal concentrations of manganese in urine range from 1–8 µg/l (Chang, W.).

**SOUTH AFRICAN MANGANESE MONITORING RESULTS**

Figures 1 and 2 reflect the exposure to manganese for South Africa during the period 2003 – 2005 from bio-monitoring results. Significant increases in urine levels (above 40 µg/l), is noticeable over the period 2004 and 2005. Blood manganese levels are showing some improvement in 2005, however, the sample size is small. No relationship between the airborne manganese concentration and the biological indicator of exposure could be established. Reliable air levels were available when compiling this report.
**CONCLUSION**

Even though there is little if any correlation between the level of manganese as measured by urinary and blood samples, biomonitoring may be of value in assessment of exposure or in differential diagnosis.

Body burden and exposure monitoring may assist in prevention or early disease diagnosis but in uncertain exposure scenarios as in developing countries or high exposure legislative levels as in South Africa it can be a good proxy for prioritisation and prevention measures.

There is a clear need to establish non-exposed population manganese levels as well as research on the correlation between exposure and blood and urine levels in SA working populations. Legislated exposure levels should be reviewed as a matter of urgency. A review of manganese medical surveillance protocols for early detection and removal from exposure should be established and standardised through tripartite consultations.

**REFERENCES**

3. NIOSH – Manganese and compounds. Medical Surveillance 2002.