MANGANESE EXPOSURE

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1.0 OCCURRENCE AND USES OF MANGANESE

Manganese (Mn) is one of the most abundant elements in the earth's crust. Manganese does not have a special taste or smell. It is found in soils, sediments, rocks, water and biological materials. Pure manganese is a silver-coloured metal; however, it does not occur in the environment as a pure metal. Rather, it occurs in combination with other substances such as oxygen, sulphur, and chlorine. These forms (called compounds) are solids that do not evaporate. However, small dust particles of the solid material can become suspended in air.

Manganese are used:

- As a metal.
- In fertilizers and as driers for linseed oil.
- For glass and textile bleaching and for leather tanning.
- As a fuel-oil additive, a smoke inhibitor, and as an antiknock gasoline additive.
- In the production of batteries, in dietary supplements, and as ingredients in some ceramics and pesticides.
- In the production of steel as a reagent
- As an alloying agent for special steels, aluminium and copper.
- For electrode coating in welding rods and for rock crushers, railway points and crossings.
- In the ceramics, match, glass and dyestuff industries.

Manganese is an essential nutrient, and eating a small amount of it each day is important to stay healthy. Manganese is present in many foods, including grains and cereals, and is found in high concentrations in many foods, such as tea. The amount of manganese in typical western diets (about 1–10 mg per day) appears to be enough to meet daily needs.

Manganese is an essential trace element in order for the body to function properly. The body normally controls the amount of absorbed manganese, e.g. if

large amounts of manganese are absorbed through the diet, the body excretes large amounts in the faeces. Therefore, the total amount of manganese in the body tends to stay about the same, even when exposure rates are higher or lower than usual. However, if too much manganese is being absorbed, the body may not be able to adjust and excrete the excessive amounts. Manganese is and is necessary for good health. The human body typically contains small quantities of manganese, and under normal circumstances, the body controls these amounts so that neither too little nor too much is present.

2.0 ABSORPTION, DISTRIBUTION AND EXCRETION OF MANGANESE

Humans are exposed to manganese in the food and water they eat and drink and in the air they breathe. Infants absorb manganese that is present in breast milk, soy-based infant formulas, or cow's milk. The amount of manganese in these sources is generally not a problem, and they provide the manganese that is necessary for normal functioning of the body.

The contribution of toxicity via food, water, diet, etc. is uncertain. In general, adverse effects in people exposed through these routes have only been reported when environmental manganese levels were quite high. If you breathe air containing manganese dust, many of the smaller dust particles will be trapped in the lungs.

In occupational situations manganese is primarily absorbed by inhalation. Manganese dioxide and other manganese compounds, which occur as volatile by-products of metal refining are practically insoluble in water. Thus, only particles small enough to reach the alveoli are eventually absorbed into the blood. Large inhaled particles may be cleared from the respiratory tract and swallowed. Manganese may also enter the gastrointestinal tract with contaminated food and water. The dietary level of manganese and iron, the type of manganese compound, iron deficiency and age can influenced the rate of

absorption. However, the risk of intoxication by this route is not great. Absorption of manganese through the skin is negligible.

The absorbed manganese is rapidly eliminated from the blood and distributed mainly to the liver. The kinetic patterns for blood clearance and liver uptake of manganese are similar, indicating that these two manganese pools rapidly enter equilibrium. Excess metal may be distributed to other tissues such as kidneys, small intestine, endocrine glands and bones. Manganese preferentially accumulates in tissues rich in mitochondria. It also penetrates the blood-brain barrier and the placenta. Higher concentrations of manganese are also associated with pigmented portions of the body, including the retina, pigmented conjunctiva and dark skin. Dark hair also accumulates manganese. It is estimated that the total body burden for manganese is between 10 and 20 mg for a 70 kg male. **The biological half-life for manganese is between 36 and 41 days, but for manganese sequestered in the brain, the half-life is considerably longer.**

Bile flow is the main route of excretion of manganese. Consequently, it is eliminated almost entirely with faeces, and only 0.1 to 1.3% of daily intake with urine.

Some of the manganese in these small particles may then dissolve in the lungs and enter the blood. The exact amount that may enter the blood is not known. Larger particles and those that do not dissolve will be coughed up, in a sticky layer of mucus, out of the lungs and into the throat, where they will be swallowed and will enter the stomach.

3.0 STANDARDS FOR MANGANESE

3.1 Occupational Exposure Limit for Manganese

The Occupational Exposure Limit (OEL or American TLV) for Manganese and

manganese compounds as Mn is 5 mg/m^3 and Manganese fume as Mn is 1 mg/m^3.

International exposure limits are often more stringent, as reflected below.

The American Congress of Governmental Industrial Hygienists (ACGIH) sets the TLV for Manganese and inorganic compounds as Mn are 0.2 mg/m^3, whereas the National Institute of Occupational Safety and Health (NIOSH) has acceptable level for Manganese compounds and fume as Mn as 1 mg/m^3.

3.2 Biological exposure indices for Manganese

A Biological exposure indices (BEI) for Manganese does not exist, however, it is general accepted that 12,6 µg// manganese is an acceptable guideline. This value is general used as a reference value for unexposed employees or thus for the general population. Germany has established that exposed employees have an average manganese level of 20 µg//.

4.0 RISKS OF EXPOSURE TO NEUROTOXINS

4.1 General

A variety of neurobehavioural effects occur due to excessive exposure to lead, welding, aluminium, manganese and pesticides.

Neurotoxicant syndromes, brought about by substances, which adversely affect nervous tissue constitute one of the ten leading occupational disorders in the United States. Neurotoxicant effects constitute the basis for establishing exposure limit criteria for approximately 40% of agents considered hazardous by the United States National Institute for Occupational Safety and Health (NIOSH).

A neurotoxin is any substance capable of interfering with the normal function of

nervous tissue, causing irreversible cellular damage and/or resulting in cellular death.

Table 1: Grouping neurotoxic effects to reflect their relative strength for establishing neurotoxicity

Level	Grouping	Explanation/Examples
6	Morphological changes	Morphological changes include cell death and axonopathy as well as subcellular morphological changes.
5	Neurological changes	Neurological change embraces abnormal findings in neurological examinations on single individuals.
4	Physiological/behavioural changes	Physiological/behavioural changes comprise experimental findings on groups of animals or humans such as changes in evoked potentials and EEG, or changes in psychological and behavioural tests.
3	Biochemical changes	Biochemical changes cover changes in relevant biochemical parameters (e.g., transmitter level, GFA-protein content (glial fibrillary acidic protein) or enzyme activities).
2*	Irreversible, subjective symptoms	Subjective symptoms. No evidence of abnormality on neurological, psychological or other medical examination.
1*	Reversible, subjective symptoms	Subjective symptoms. No evidence of abnormality on neurological, psychological, or other medical examination.

* Humans only

The nervous system generally reacts rather stereotypically to exposure to neurotoxic substances (Figure 1). Some typical symptoms are indicated below.

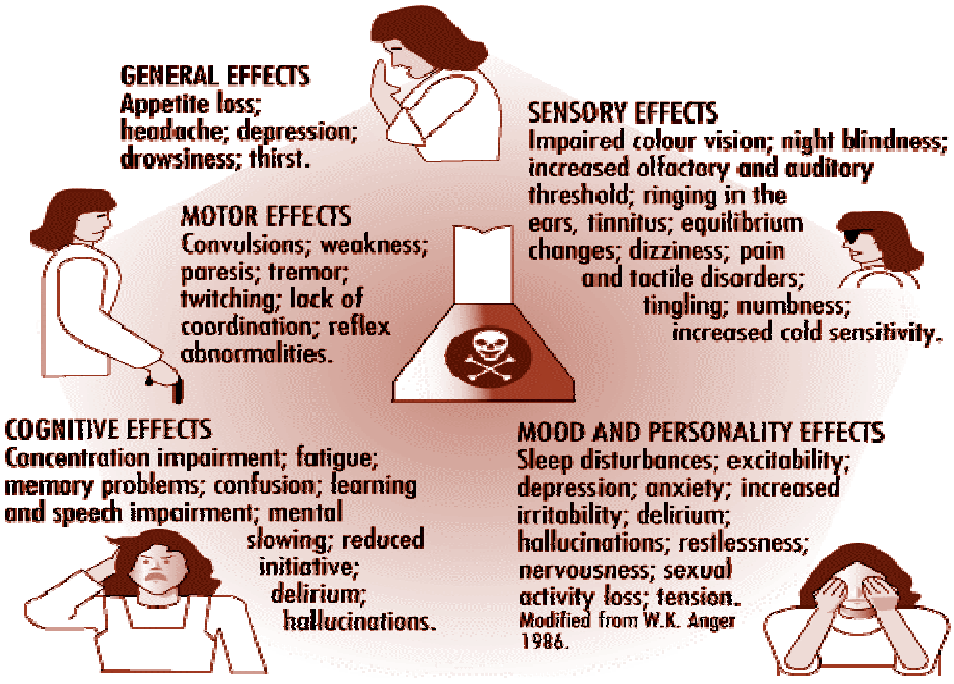

Figure 1: Neurological and behavioural effects of exposure to neurotoxic chemicals

4.2 Manganism

Manganese miners or steel workers exposed to high levels of manganese dust may experience mental and emotional disturbances, and their body movements may become slow and clumsy. The symptoms is a disease called 'manganism.' **Workers do not usually develop symptoms of manganism unless they have been exposed to manganese for many months or years.** Manganism occurs because too much manganese injures a part of the brain that helps to control body movements. Some of the symptoms of manganism may improve upon certain medical treatments, but the improvements are usually temporary as **the brain injury is permanent.** Manganism has been reported most often in miners. It has only been reported a few times in other workers exposed to the metal, such as steel workers. The symptoms most commonly observed in occupational workers (other than miners) include difficulty in motor skills, such as holding one's hand steady, performing fast hand movements, and maintaining balance when tested. These symptoms are not as severe as those related to manganism, indicating that the effects caused by manganese over-exposure are related to the level of exposure.

Inhaling too much manganese over a short or long time may cause irritation of the lungs, which makes breathing difficult and it also increases the chance of getting lung infection, such as pneumonia. However, this also can happen due to exposure of many kinds of dust particles, other than that contains manganese.

A common effect in men who are exposed to high levels of manganese dust in the air over a long time is impotence.

4.3 Metal Fume Fever

Metal fume fever is another self-limited, flu-like illness that develops after inhalation of metal fumes. The syndrome most commonly develops after zinc

oxide inhalation, as occurs in brass foundries, and in smelting or welding galvanized metal. Oxides of copper and iron also cause metal fume fever and vapours of aluminium, arsenic, cadmium, mercury, cobalt, chromium, silver, manganese, selenium and tin have been occasionally implicated. Workers develop tachyphalaxis, which are symptoms that appear only when the exposure occurs several days after exposure.

4.4 Diagnosis of manganism

Chronic manganese poisoning can take either a nervous or pulmonary form.

4.4.1 Nervous form

If the nervous system is attacked, three phases can be distinguished. During the initial period, diagnosis may be difficult. Early diagnosis, however, is critical because cessation of exposure appears to be effective in arresting the course of the disease.

4.4.2 Pulmonary form

Reports of “manganese pneumoconiosis” have been contested in view of the high silica content of the rock. Manganese pneumonia has also been described. There is also controversy over the correlation between pneumonia and manganese exposure unless manganese acts as an aggravating factor.

4.4.3 General

Manganese poisoning ultimately becomes chronic. However, if the disease is diagnosed while still at the early stages and the patient is removed from exposure, the course may be reversed. Once well established, it becomes progressive and irreversible, even when exposure is terminated. The nervous

disorders show no tendency to regress and may be followed by deformation of the joints. Although the severity of certain symptoms may be reduced, gait remains permanently affected. The patient's general condition remains good, and he or she may live a long time, eventually dying from an intercurrent ailment.

Diagnosis is based primarily on the patient's personal and occupational history (job, length of exposure and so on). However, the subjective nature of the initial symptoms makes early diagnosis difficult; consequently, at this stage, questioning must be supplemented by information supplied by friends, colleagues and relatives. During the intermediate and full-blown stages of the intoxication, occupational history and objective symptoms facilitate diagnosis; laboratory examinations can provide information for supplementing the diagnosis.

Differential diagnosis may be difficulty in distinguishing between manganese poisoning and some other diseases such as Parkinson's disease.

4.5 Conclusion

The differentiation of a neurotoxicant syndrome from a primary neurological disease poses a formidable challenge to physicians in the occupational setting. Obtaining a good history, maintaining a high degree of suspicion and adequate follow-up of an individual, as well as groups of individuals, is necessary and rewarding. Early recognition of illness related to toxicant agents in their environment or to a particular occupational exposure is critical, since proper diagnosis can lead to early removal of an individual from the hazards of ongoing exposure to a toxicant substance, preventing possible irreversible neurological damage. Furthermore, recognition of the earliest affected cases in a particular setting may result in changes that will protect others who have not yet become affected.