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Effects from Occupational and Environmental Exposure

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ABSTRACT

Although much literature is available on the clinical manifestation of Mn poisoning, quite a few studies have been conducted on the early effects of prolonged occupational and environmental exposure to "low" doses. They are mainly of cross-sectional type, but some follow-up experience is now available. Important information can be derived from this research, useful for the identification of more precise health-based limit values.

Sensitive neurobehavioral and neurophysiological methods have been used to assess the central nervous system functions, together with biochemical and neuroendocrine indicators to evaluate the integrity of neurotransmitter systems. All these different methodologies coherently support the hypothesis of an impairment of extrapyramidal function and a tendency to aggressive behavior, which are possible consequences of an interference with the dopaminergic system. Since Mn accumulates in the brain and is released with an extremely slow elimination kinetics, the exposure limit values should be able to protect the integrity of neurobehavioral functions over a prolonged period of exposure, to prevent the onset of parkinsonian disturbances in the geriatric age. Therefore, the annual average airborne Mn concentrations must be low enough not to exceed a certain cumulative dose that can be reached in the long term.

Taken together all the indications provided by the occupational studies, a TLV-TWA of 100 $\mu\text{g}/\text{m}^3$ can be derived for the airborne concentration of Mn in total dust of industrial settings, as measured with personal sampling. Although a lack of accuracy of biological monitoring may occur due to high individual variability, the concentration of 7.5 $\mu\text{g}/\text{l}$ can be tentatively suggested for Mn in total blood as a possible biological exposure limit.

As for environmental exposure, derivation of the EPA's exposure limit for environmental exposure (Reference Concentration - RfC) for Mn (0.05 $\mu\text{g}/\text{m}^3$) was based on observations of neurobehavioral deficits in workers with subchronic exposure to mean airborne Mn at 150 $\mu\text{g}/\text{m}^3$. More recently, a population-based study observed subtle Parkinson-like alterations in adults with mean airborne-Mn exposure of 0.02 $\mu\text{g}/\text{m}^3$ (range=0.01-0.035) and a low mean drinking-water level of 4.4 $\mu\text{g}/\text{L}$. Residence in areas with higher airborne-Mn levels was associated with higher blood-Mn levels, which were associated with neurological deficits, particularly in men ≥ 50 years of age.

A study where Mn drinking-water levels ranged from 4-15, 82-253, and 1,800-2,300 $\mu\text{g}/\text{L}$ across three areas, also observed Parkinson-like signs in men ≥ 50 years of age. A composite Parkinson's score increased with Mn drinking-water levels, as did hair Mn levels. A further study observed learning and neurobehavioral deficits as well as neurotransmitter alterations in school children where Mn drinking-water levels ranged from 241-346 $\mu\text{g}/\text{L}$, relative to the control children's range of 30-40 $\mu\text{g}/\text{L}$. Hair Mn levels were elevated in the affected children, as were Mn levels in field-irrigation sewage water in the exposed town, but Mn levels in the air were not reported.

Based on this new evidence, exposure limit values should be also lowered to about $0.02 \mu\text{g}/\text{m}^3$. In addition, further research is needed to describe relationships between total Mn exposure, body burdens, and health outcomes in adults, women, children and susceptible populations such as people affected by hepatopatic diseases.