MANGANESE EXPOSURE AND BIOLOGICAL INDICATORS

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To evaluate an occupational health hazard often requires it to be quantified in order to assess the degree of the hazard to workers. A toxic chemical vapour in a workroom, for example, can be measured as to its level or concentration in the air, and the result compared to the recommended threshold limit value or TLV which is considered as a relatively "safe" level for the majority of workers. Such levels, however, only serve as guidelines in the control of potential health hazards.

Because of personal factors and susceptibility of workers, however, knowledge of the air level of a toxic chemical alone may not be sufficient to accurately evaluate a hazard. Biological indicators are useful in monitoring the health of individual workers and these are used in medical surveillance programmes. Such biological TLVs or exposure indices (BEIs) "represent warning levels of biological response to the chemical, or warning levels of the chemical or its metabolic product(s) in tissues, fluids or exhaled air of exposed workers" (1). Lead is a classical example of a substance for which blood concentrations have long been considered of critical value in determing "safe" and "unsafe" exposures.

In the case of manganese, however, the situation is less certain. Although manganese is an essential trace element in man, being a metalloprotein component of some enzymes like pyruvate decarboxylase, in excessive amounts it is a toxic metal which is known to be able to cause parkinsonism. Milder manifestations of poisoning include headache, restlessness, irritability and dysarthria (2,3). Cases of manganese poisoning have been reported among miners, and in workers in the production of alloys and dry cell batteries. A TLV of 5 mg/m^3 air for an 8 hour-a-day exposure has been set (1). This was based on a few epidemiological studies which reported that poisoning cases had occurred at exposures much above this level and cases did not seem to occur below it (4). However, there have been other reports of adverse effects to the central nervous system in some workers exposed to air levels of only 2 to 5 mg/m^3 (2).

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There is poor correlation between mean manganese-in-air levels and the degree of disorders (2). Various authors found no correlation between the level of manganese in the blood and that in the air. Most authors have found no direct relationship between blood and urine manganese levels and the occurrence or severity of poisoning (5). Studies of individuals with well developed signs and symptoms have revealed blood manganese levels within the normal range. In contrast, healthy miners may have increased blood levels (6). Individual susceptibility is thought to be a decisive factor in manganese toxicity (7).

However, Tanaka and Lieben found, on a group basis, that the urine level of manganese had some correlation with the average air concentration (8). A level of 50 μg Mn/1 urine was proposed by the UK authority some years ago as a guideline to safe exposures (9).

Generally, the level of manganese in the blood, and especially in the urine, may be used to indicate the average level of exposure, on a group basis, but not on an individual basis (2,7). In individual workers, the blood and urine levels may be used to confirm exposure to manganese and possibly manganism. But no biological TLV or BEI can yet be proposed (2,5). The principle of biological monitoring for manganese exposure can only be recommended with reservation. Studies to date do not show a dose-response relationship for urine manganese and health disorders (7).

The paper on "Study of workers exposed to manganese dust in the dry cell battery manufacturing and manganese milling industries" in this journal reports that the air levels of manganese correlated significantly with those in the urine and blood of exposed workers, and that at the TLV of 5 mg/m^3, the corresponding blood manganese concentration was about 30 $\mu g/1$ and that in the urine was also about 30 $\mu g/1$. These are interesting findings and more such studies need to be done to confirm the relationship. If confirmed, such biological levels could be used, especially on a group basis, to complement the information derived from air monitoring. However, before a "biological TLV" can be proposed, especially for individual workers, it would be necessary to determine the levels of manganese in biological media (eg. blood, urine) at which adverse health effects are observed (2).

Further research also needs to be undertaken into the mechanisms of uptake and clearance of manganese from the respiratory system and gastrointestinal tract (2). It is thought that particle size plays a key role in this as it influences the deposition sites in the respiratory tract and the solubility rate (2,10). Only particles which are "respirable" (ie less than about 10 μ in size) can reach the alveoli. Larger particles are cleared from the respiratory tract and eventually swallowed. In the gastrointestinal tract, only about 3% of the ingested manganese is absorbed (2). It is interesting to note that the paper by S L Gan et al in this journal reported that the manganese dust was "non-respirable", the particle size ranging from 12.53 to 55.73 μ. This would suggest that absorption of the manganese by the workers was via the gastrointestinal tract.

Much as a biological TLV is desirable in the monitoring of workers, especially on an individual basis, more research needs to be done before one can be proposed. However it would still be useful to serially compare the group average blood and urine manganese levels as an indication of improving or deteriorating exposure levels. This would complement air monitoring results. The WHO recommends "repeated screening of subjective symptoms and thorough clinical examination" at regular intervals together with estimations of the level of manganese in blood and urine samples (6).

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