BIOLOGICAL THRESHOLD LIMIT VALUES FOR MANGANESE DUST EXPOSURE

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SYNOPSIS

Workers from two manganese ore milling plants and three dry-cell battery manufacturing plants in Singapore were studied to determine the extent of absorption and exposure of workers to manganese dioxide. An attempt was also made to determine the usefulness of blood and urine manganese estimations as biological indicators of exposure.

The highest manganese exposures were in the manganese mills. Fifteen of the twenty-eight samples collected exceeded the Threshold Limit Value (TLV). The battery factories had significantly lower exposure levels.

No case of chronic manganese poisoning or manganism was detected. However, four workers from the manganese mills had urine manganese concentrations exceeding the biological TLV of 50μg/litre. The blood manganese concentration of all the workers were below the biological TLV of 300μg/litre with an average ($\bar{x}_g$) of 22.59μg/litre.

Manganese concentrations in air correlated significantly with manganese concentrations in urine ($r = 0.77$) and blood ($r = 0.69$). At the TLV of 5mg/m^3, the corresponding blood manganese concentration was about 30μg/litre and that of urine also about 30μg/litre.

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INTRODUCTION AND OBJECTIVES

Manganese (Mn) is widely used in industry. There are three main uses:

(1) In the production of steel, it counteracts the harmful effects of sulphur and improves the quality of steel;

(2) In the manufacture of dry cell batteries, it acts as a depolariser in the battery and

(3) In the chemical industry eg. production of potassium permanganate, electrode coating in welding rods, dyeing and tanning of leather, drier in paints, anti-knock fuel additive, etc.

Mn enters the body through inhalation and intestinal absorption. Negligible amounts are absorbed through the skin. It leaves the blood quickly and is stored in parenchymatous tissues eg. liver, pancreas, pituitary gland, kidneys and long bones.

The excretion of Mn consists of a fast phase of four days followed by a slow phase of 39 days. 92% is excreted in the faeces and two to ten per cent in the urine. Hair is also an important route of elimination.

Acute and chronic Mn poisoning may occur following occupational exposure. So far, no cases of poisoning have been reported in Singapore.

The objective of this study was to determine the extent of Mn exposure and absorption in two industries in Singapore: battery manufacturing and Mn ore milling. An attempt was also made to determine the usefulness of blood and urine Mn as biological indicators of exposure.

MATERIALS AND METHODS

This study covered three dry-cell battery factories and two Mn ore milling plants. As far as could be ascertained, these five factories form the local universe population of dry-cell battery manufacturing and Mn ore milling in Singapore. Of the three dry-cell battery plants, two started operation in 1980 and one in 1947. The two Mn ore milling plants had started operation in 1973 and 1976.

Preliminary inspections were carried out on all the five factories (Plants A-E) to delineate the areas of Mn dust exposure.

During the visit, the operations, material inventories, work schedules, job characteristics, control measures and relevant records were examined.

There were two main operations in the manufacture of dry-cell batteries — mixing of the ingredients and compressing the mixture into a "cake". This "cake" was then placed in zinc cans and carbon rods were inserted centrally. These filled cells were then capped and sealed with hot pitch. After dressing, they were packed as the finished product.

It was observed that those doing mixing would be more exposed to Mn dust than those working in the compressing section. This was because the mixers handled dry manganese dioxide ($Mn-_2$) powder whilst those in the compressing section handled moist MnO_2 mixture.

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There were also two main operations in the Mn ore milling plants: (a) Feeding of the Mn ore into the ball mill and (b) Bagging of the milled Mn ore. The latter process was very dusty.

In this study the feeding and bagging sections were considered together as the same workers were involved in both processes.

Workers from all the five factories were divided into three groups, i.e. high, moderate and low exposure groups. The Mn ore mill workers were considered as belonging to the high exposure group. All the 22 workers were studied. The mixers in the battery factory were classified under moderate exposure. Only workers who had been in the area for at least three months were included in the study. Workers doing compressing were considered to be of low exposure. For this section, only workers who had worked for more than three years were included in the study. Hence, only 46 out of the 64 battery workers were investigated.

All control subjects were matched by age, sex and ethnic group. Workers from the same factory or factories nearby were chosen. A total of 58 workers were obtained.

Workers were all interviewed including taking a smoking history (1,2). They were also examined for early neurological changes. Venous blood and 24-hour urine were collected for determination of blood and urine Mn concentrations respectively.

Environmental Assessment

The environmental assessment consisted of particle size analysis of the crushed MnO_2 and the electrolytic MnO_2, and personal dust sampling.

Particle size analysis was carried out with a light microscope using a Porton graticule in a 10x eyepiece and a 44x objective. The graticule was calibrated with a stage micrometer. A total of eight specimens were analysed, including five crushed MnO_2 and three electrolytic MnO_2 samples. For each specimen, the number-size distribution of the particles was obtained by grouping particles into ranges defined by successive graticule circle sizes. The results were then plotted on a probability graph paper as cumulative percent for each group versus the size of that group. A total of more than 400 particles was measured for each specimen.

To maximise the effectiveness of sampling to assess exposure, individual plants were divided into different zones. These zones were the characteristic grouping of workers based on their job similarity and similarity of the environment in which they worked.

Total airborne Mn concentrations were determined by personal sampling in the breathing zones of workers randomly selected from the various exposure zones. A total of 83 personal samples was collected from 38 workers. Continuous sampling strategy was used. For each subject monitored, at least 2 consecutive samples were taken in order to cover as much of the entire workshift as possible. Usually one was taken in the morning and another in the afternoon.

Each sample was collected on a 37mm diameter membrane cellulose ester filter of 0.5μ pore size. This was mounted on a three piece cassette holder attached to the worker's collar. The cassette was sealed and connected by plastic tubing to an MSA sampling pump attached to the worker's belt. The flow rate was 1.5 litre/min and the average total sampling time per subject was about six hours. The flow rate was checked hourly using a calibrated rotameter.

Several eight-hour continuous samples were taken in the ambient air to determine the background Mn concentration.

Analysis

The total Mn content in the samples of air, blood and urine were determined by atomic absorption (3).

RESULTS

The majority of the workers in the battery factories were males. Females were only involved in compressing. In the Mn mills, there were no females. In the sample studied, the subjects were all males except for one female. Hence, for all subsequent discussion, they would be considered together.

83.6% of the workers studied were below 45 years of age. None of the workers in the mills exceeded 45 years. This was probably because the work required physical strength.

The Mn mills had a rather high turnover rate. 77.3% of the workforce had been employed for less than three years. In contrast, 68.9% of the workforce in the battery factories had worked for more than 3 years.

No case of manganism was found amongst the workers studied. Several of the workers complained of tiredness, irritability, drowsiness and muscular cramps. On examination, some demonstrated hand and tongue tremors, disdiadochokinesia, brisk Achilles tendon reflex and slurred speech. However, there were no significant differences in the prevalence of the above symptoms and signs between the exposed and control groups except for the prevalence of tiredness.

Blood and Urine Manganese Concentrations

The blood and urine Mn concentrations were spread over a wide range, thus log normal distributions were assumed when analysing the data.

The blood Mn concentration in both the exposed and control groups were below 300μg/l, the level thought to be suspicious by the International Labour Organisation (4). The average (x_g) blood Mn concentration was 22.59μg/l for the exposed and 13.04μg/l for controls (Table 1).

However, four workers from the Mn mills had urine Mn concentrations exceeding 50μg/l (5), the limit proposed by the Employment Medical Advisory Service (5). The average urine Mn concentration was 5.97μg/l for the exposed and 2.40μg/l for the controls (Table 1).

Consistent with the higher Mn-in-air levels, the blood and urine Mn concentrations of the workers in the milling plants were significantly higher than those in the battery plants ($p < 0.001$).

Both blood and urine Mn concentrations were not related to age, ethnic group, smoking status, duration of exposure and type of work.

TABLE 1

BLOOD AND URINE MANGANESE CONCENTRATIONS IN EXPOSED AND CONTROL WORKERS

	Exposed n = 67	Control n = 58	Level of Significance
Blood Manganese (μg/litre)	4.0–49.0 $x_g = 22.59$ (1.79)	1.0–46.0 $x_g = 13.0$ (2.07)	$p < 0.001$
Urine Manganese (μg/litre)	1.0 = 110.5 $x_g = 5.97$ (4.87)	1.0–15.0 $x_g = 1.73$ (2.40)	pμ0.001

SD is given in brackets

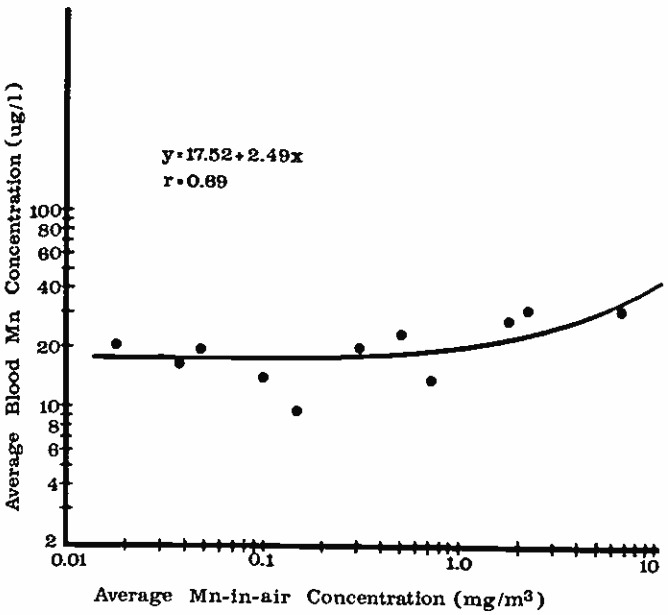

Fig. 1 Relationship between Mn-in-air and Blood Mn concentrations.

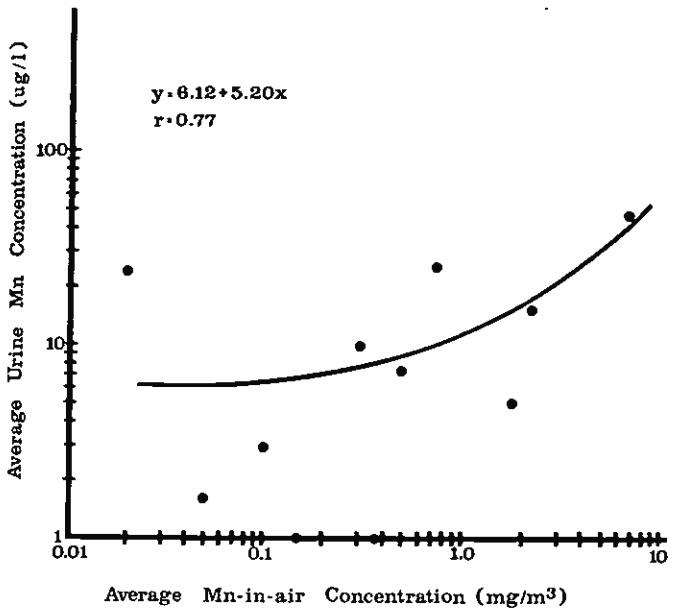

Fig. 2 Relationship between Mn-in-air and Urine Mn concentrations.

Particle size analysis

The number-size distribution of the crushed MnO_2 and electrolytic MnO_2 follows a log-normal distribution. The range of the count median diameter was 0.35μ-0.90μ. The geometric standard deviation ranged from 2.35–3.10μ. This implies a polydisperse dust sample. Using the density of 4.2gm/cm^3 for MnO_2, the mass median equivalent diameter (MMED) was found to range from 12.53μ-55.73μ. This means that the dust was non-respirable.

Environmental Assessments

Similarly, log normal distributions were assumed when calculating this data. The highest Mn exposures were in the Mn mills, with a mean of 4.16mg/m^3. Fifteen of the twenty-eight samples collected exceeded the Threshold Limit Value of 5mg/m^3(6). The battery factories had significantly lower exposure levels. None of the samples collected here exceeded the threshold limit value. In the mixing and compressing sections, the mean was 0.62mg/m^3 and 0.08mg/m^3 respectively. The background air Mn level was 0.0003mg/m^3.

Relationship between air, blood and urine manganese concentrations

A linear relationship (correlation coefficient, r=0.69) was obtained between blood Mn concentrations in individual exposure zones and corresponding mean air Mn concentrations (Fig 1):

$$\text{Blood Mn} = 17.52 + 2.49 \times \text{Air Mn}$$

The Mn content in blood was relatively constant around 18μg/l when the Mn-in-air level was below 0.5mg/m^3. At the Threshold Limit Value (TLV) of 5 mg/m^3, the corresponding blood Mn concentration was approximately 30μg/l.

Using the same exposure zone concept, a linear relationship was found between the mean urinary concentration and the Mean Mn concentration in air (Fig. 2).

$$\text{Urine Mn} = 6.12 + 5.20 \times \text{Air Mn}$$

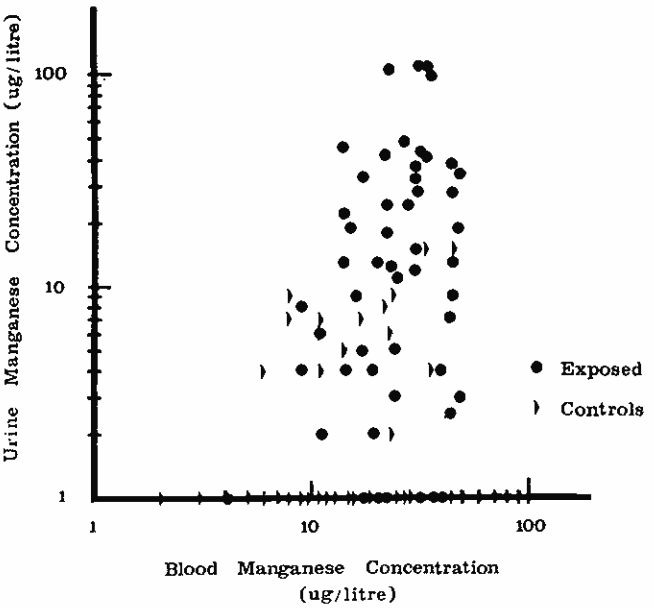

Fig. 3 Relationship between Blood and Urine Mn concentrations.

The correlation coefficient, r was 0.77. At the TLV of 5mg/m^3, the corresponding urine Mn level was about 30μg/l.

A poor correlation (r=0.31) was found between urinary and blood Mn concentrations on an individual basis (Fig. 3).

DISCUSSION

The Mn dust was found to be non-respirable. The particle-size distribution was similar to that obtained by Smyth et al (7). A walk-through survey by NIOSH(8) in a dry-cell battery plant also demonstrated that most of the Mn dust was non-respirable. This, together with the systemic effects of Mn, suggests that total dust sampling should be used for Mn exposure evaluation.

The breathing zone dust levels may however be different from the actual occupational exposure as dust masks were provided in all the plants studied. However, these masks were for non-toxic dust and their use was not strictly enforced.

The dust monitoring results showed that the bagging process in the Mn mills had the highest dust exposure, followed by mixing and compressing in the dry-cell battery plants. In general, the dust exposure was related to the type of work and the engineering control measures used. In all cases, the Mn-in-air levels were significantly above the background Mn concentration of 0.0003mg/m^3. This level was comparable to other reports (9,10).

It should also be mentioned that besides exposure to Mn dust, workers were also exposed to other airborne contaminants. Workers in the mills may be exposed to trace copper, arsenic, sulphur, phosphorus, tin, titanium dioxide, alumina, silicon dioxide and iron, which may be present as impurities in the raw ore. Workers in the battery plants may be exposed to ammonium chloride dust, acetylene black and graphite particles which were used in the mix preparation.

A good linear correlation ($r = 0.69$) was noted between the blood Mn and Mn-in-air on a group basis. There was a significant difference between the exposed and control groups with regard to the blood Mn levels. A good linear relationship ($r = 0.77$) was also found between the urine Mn and Mn-in-air on a group basis. Tanaka and Lieben (1)) reported a linear correlation ($r = 0.67$) on a group basis. Smyth and co-workers (7) however obtained a poor correlation of $r = 0.12$.

A poor correlation ($r = 0.31$) between the Mn content in the whole blood and in urine was obtained on an individual basis. This result was similar to that obtained by Horiuchi and co-workers (12).

The results also imply that the proposed level (5) of 50μg/l for urine Mn is quite comparable to the TLV of 5mg/m^3. However, the blood Mn level of 300μg/l (4) appears to be too high. Fig. 1 suggests that a blood Mn level exceeding 30μg/l may be more appropriate in indicating excessive or harmful exposure. In view of the wide variation in the normal Mn content in blood of healthy men, the blood Mn criterion should be further looked into.

Pleban and Pearson (13) obtained an average of 9μg/l (range 3.9 to 15μg/l) in the blood of normal adults. Other studies (14,15) were also in general agreement with this data. In the present study, the average concentration of Mn in blood of the unexposed subjects was 13μg/l. This level is slightly higher than those sited above. Differences in dietary pattern eg. high tea consumption and problems with contamination may account for this.

In this study, the blood Mn was affected by the type of work and not by other factors. The average blood Mn correlated with the mean air Mn and not with the duration of exposure. This may be explained by its rapid clearance from the body. Several investigators (16,17) reported that even cases of chronic Mn poisoning had normal blood Mn levels. Thus, determination of Mn in blood may not be adequate for detection of Mn poisoning.

Urinary Mn concentrations in normal persons have been reported to range from less than one to ten microgrammes per litre (9,15,18,19). In this study, a range of one to fifteen microgrammes per litre with an average of 2.8μg/l was obtained. For the exposed group, the urine Mn concentration ranged from one to 110.5μg/l with four workers having levels exceeding 90μg/l. Of these four workers, one complained of tiredness alone, whilst the other had in addition hand tremors. The third worker had brisk Achilles reflex and the fourth was asymptomatic. These four were re-examined by a senior neurologist. No neurological deficits were detected.

Cholak and Hubbard (9) reported concentrations of Mn in urine of as high as 600μg/l in persons with Mn exposure but without clinical evidence of manganism. Nilubol et al (18) reported urine concentrations in asymptomatic workers ranging from 25 to 124μg/l. Tanaka and Lieben (11) showed that individuals with high urinary Mn concentrations do not necessarily have symptoms, and asymptomatic patients do not necessarily have low urinary Mn levels. This is probably due to the fact that the urinary Mn concentrations more or less reflect the levels of recent absorption.

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