MANGANESE and INORGANIC COMPOUNDS

CAS number: 7439-96-5 (Manganese)

Molecular formula: Mn

TLV–TWA, 0.2 mg/m^3, as Mn

Summary

A TLV–TWA of 0.2 mg/m^3, as Mn, is recommended for occupational exposure to elemental manganese and its inorganic compounds. This value is intended to minimize the potential for pre-clinical adverse effects in the lungs and CNS and adverse effects on the fertility of male workers exposed to manganese. The lowest exposure concentration of manganese at which early effects on the CNS and the lungs may occur is still unknown. However, once neurological signs are present, they tend to continue and worsen after exposure ends. Additional data are needed to more accurately determine the exposure doses necessary to protect nearly all workers. Sufficient data were not available to recommend Skin, SEN, or carcinogenicity notations or a TLV–STEL.

Chemical and Physical Properties

Elemental manganese (Mn), atomic number 25 in group VIIB of the periodic table, is a gray-white metal resembling iron, but it is harder and more brittle. It is a highly reactive metal and exists in seven oxidation states. Its most important ore is black dioxide (MnO_2), known as pyrolusite. Chemical and physical properties of elemental manganese include:[1]

Molecular weight: 54.9380
Specific gravity: 7.21 to 7.4, depending on allotropic form
Melting point: 1244°C
Boiling point: 1962°C
Solubility: soluble in dilute acids
Conversion factor at 25°C and 760 torr:
1 mg/m^3 = 0.45 ppm

Ferromanganese fume, generated in the pouring and casting of molten ferromanganese, was found to be largely manganese tetroxide (Mn_3O_4) by X-ray diffraction analysis. The fume[1] is formed whenever manganese oxide is heated strongly in air. Chemical and physical properties of the tetroxide include:[1]

Molecular weight: 228.79
Specific gravity: 4.876
Melting point: 1564°C
Solubility: insoluble in water; soluble in hydrogen chloride with evolution of chlorine
Conversion factor at 25°C and 760 torr:
1 mg/m^3 = 0.11 ppm

The U.S. Agency for Toxic Substances and Disease Registry [1] published an extensive review of the literature characterizing the toxicological and adverse health effects of manganese exposure.

Major Uses

Manganese has been especially noted for alloying with metals and imparting hardness, such as in the manufacture of steel. Inorganic compounds of manganese have a variety of uses. Manganese chloride ($MnCl_2$) has been used as a catalyst and as an animal feed supplement. Both manganese dioxide (MnO_2) and $MnCl_2$ have been used in the production of dry-cell batteries. The dioxide has also been used in the manufacture of fireworks, matches, porcelain, and glass-bonding materials. Manganese sulfate has been used in fertilizers, ceramics, glazes, and varnishes; as a nutritional supplement; and as a fungicide.

Oberdoerster and Cherian[2] reported that the size of airborne manganese dust particles can span a wide range, from submicron to more than 10 µm in diameter, whereas manganese fumes (melting and steel making) are mostly in the submicron range. Deposition of the inhaled manganese-containing particles on the respiratory tract of workers can vary considerably as a result of the manufacturing process and depending on the physical workload (tidal volume, breathing rate, nose or mouth breathing).

Animal Studies

Acute

Subcutaneous doses of 50 mg Mn/kg body weight, as $MnCl_2$, were fatal to mice, guinea pigs, and rabbits, whereas 18 mg/kg of the same preparation were lethal to rabbits by the intravenous route and 56 mg/kg were required for dogs.[3] Rats received separate intratracheal injections of MnO_2 and $MnCl_2$ in an effort to simulate manganese pneumonitis seen in humans. Characteristic histological changes in the lungs were produced. The $MnCl_2$ caused intense congestion and pulmonary edema that was often fatal.[4]

Suspensions of manganous oxide (MnO), MnO_2, and manganese tetroxide (Mn_3O_4) (particle sizes less than 3 µm) were injected intratracheally in young rats in studies by Levina and Robachevsky.[5] Pneumonitis and similar pulmonary effects were reported. Important additional findings were that the higher oxides were more toxic, and freshly prepared oxides exhibited greater toxicity than those stored 6 and 12 months.

Many attempts to induce characteristic manganese brain damage by feeding manganese compounds were only partially successful; however, these showed that manganese administered by mouth in the inorganic form was slowly and incompletely absorbed in the bloodstream.[6]

Repeated intraperitoneal injections of $MnCl_2$ into monkeys for 18 months resulted in characteristic lesions of basal nuclei. The animals exhibited choreiform movements and later displayed rigidity of the muscles, disturbances in motility, tremors, and contraction of the hands.[7]

Subchronic

Inhalation exposures of rabbits to MnO_2 dust 4 hours/day for 3 to 6 months at concentrations of 10 to 20 mg/m^3 resulted in decreased hemoglobin and number of erythrocytes.[8] Manganese pneumonitis did not occur, but fibrotic changes in the lung resembling those in silicosis were observed.

Pharmacokinetic/Metabolism Studies

Manganese has been shown to be an essential element in the nutrition of humans and for many animal species for the formation of connective tissue and bone, for carbohydrate and lipid metabolism, and as a catalyst in many metabolic pathways.[1,3] However, there are no well-defined symptoms of manganese deficiency for humans.[3] Manganese is absorbed through the epithelium of the gastrointestinal and respiratory tracts. For occupationally exposed persons, the lung constitutes the predominate route of manganese uptake. The available data show that manganese deposited in the lungs is readily absorbed.[3] In contrast, manganese taken orally is absorbed to a very low degree, usually about 3%.[2] Morrow et al.[9] found a mean retention half-time of 66 days in humans after inhaling submicron-sized $^{54}MnO_2$ particles.

Human Studies

The usual form of chronic manganese poisoning primarily involved the CNS. Early symptoms included languor, sleepiness, and weakness in the legs.[10] A stolid, mask-like appearance of the face, emotional disturbances such as uncontrollable laughter, and spastic gait with a tendency to fall when walking were found in more advanced cases.[11,12] In addition, a high incidence of pneumonia has been found in workers exposed to the dust or fume of some manganese compounds.[13] Fairhall and Neal[11] recorded reports from 12 countries of some 353 cases of manganese poisoning between 1837 and 1940. According to Hunter,[14] at least 118 more cases had been reported up to 1968.

The concentrations of manganese dust found in mines and workrooms where cases developed were usually excessively high. Rodier[15] noted manganese concentrations from 100 to 900 mg/m^3 and felt that values below 100 mg/m^3 would be reasonably satisfactory.

Flinn et al.[12] reported manganese concentrations up to 170 mg/m^3, averaging 47 mg/m^3, in a mill where 11 of 34 employees were found to suffer from manganese poisoning. No cases occurred among workers exposed at less than 30 mg/m^3. However, studies in another ore mill with dusty operations, where workers performed similar tasks with more modern equipment and local exhaust ventilation, revealed manganese concentrations averaging 2.3 mg/m^3 (from two air samples), with 6 mg/m^3 at the dustiest operation.

Lloyd Davies[13] found manganese concentrations averaging 210 mg/m^3 associated with pneumonia. Kesiæ and Hausler[16] reported cases of manganese poisoning in a plant in Yugoslavia where concentrations of MnO dusts were between 7 and 63 mg/m^3; however, the incidence of intoxication was low when concentrations ranged from 3 to 9 mg/m^3. In contrast, Schuler et al.[17] found chronic manganese poisoning in miners when only one-third of the air samples showed values above 5 mg/m^3.

Tanaka and Lieben[18] recorded 7 cases and 15 borderline cases of manganism in 75 Pennsylvania plants where 144 workers were exposed to manganese dust or fume concentrations exceeding 5 mg/m^3. Of the seven cases, four resulted from exposure to manganese dust and three from manganese fumes. No cases were reported in 48 workers exposed at air concentrations of fume or dust of less than 5 mg/m^3. Because the only results reported and the criterion used was whether the exposure of the affected workers exceeded 5 mg/m^3, the study is of little value in pinpointing the relative degree of hazard between manganese fume and dust.

Whitlock et al.[19] reported two cases of manganese fume poisoning in a manganese steel plant. Air samples showed concentrations between 2.7 and 4.7 mg/m^3. Whitman and Brandt[20] considered the air analysis data reported by Whitlock et al.[19] to be inadequate and reported no evidence of manganese poisoning at a concentration in the vicinity of 5 mg/m^3, as a time-weighted average (TWA), in the exposed employees.

During a study period of 1957 to 1965 in a Pennsylvania steel plant, Smyth et al.[21] reported

five cases of chronic manganism in 71 workers. Three of the five cases resulted from exposure to ferromanganese fumes and two of the cases were from exposure to ferromanganese dust. Two of the three workers exposed to manganese fumes had worked in the pig-casting operation as pourers where their average concentration was estimated to be 13.3 mg/m^3 for a period of 5 years. The third individual worked in an area where exposure was to fumes from a pouring operation in which the manganese air concentrations were less than 1 mg/m^3; the development of manganism in this worker suggested the possibility of hypersusceptibility of certain individuals, inasmuch as others in similar positions and exposed at concentrations in the range of 1 to 3.6 mg/m^3 showed no signs of any adverse effects.

The other two employees adversely affected worked in a ferromanganese crushing and screening operation where air concentrations were approximately 30 to 50 mg/m^3 during most of the period of the study. Although others working in the same operations were probably exposed less than the two workers adversely affected, concentrations of ferromanganese were most likely considerably above the level of 5 mg/m^3 for an extended period of time.[21]

Neurologic/Psychomotor Study

Roels et al.[22] conducted a cross-sectional epidemiological study among 141 male workers exposed to inorganic manganese in a plant producing manganese oxides and salts from manganese ore. They reported on the effects of manganese on the lung and CNS and on some biological indices. The mean age of the workers was 34.3 years and the mean duration of exposure was 7.1 years with a range of 1 to 10 years. The results were compared with a matched control group of 104 workers. The TWA concentration of total airborne manganese dust (personal samples) during the survey ranged from 0.07 to 8.61 mg/m^3, with the overall mean and median of 1.33 and 0.97 mg/m^3, respectively.

In reporting the results of respiratory effects, the authors[22] found a significantly higher prevalence of cough in cold season, dyspnea during exercise, and recent episodes of acute bronchitis in the manganese-exposed group. With respect to lung ventilatory parameters, forced vital capacity (FVC), forced expiratory volume (FEV_2), and peak expiratory flow rate (PEFR) were only mildly altered in a manganese-exposed group of workers who smoked. The magnitude and prevalence of these changes were not related to manganese in blood and urine or the duration of exposure. The authors[22] did not observe any synergistic effect between manganese exposure and smoking in any of the spirometric parameters.

In order to detect early effects of manganese on the CNS, the authors[22] used a questionnaire, performed a standardized neurological examination, and conducted several psychomotor tests (hand tremor, short-term memory, and simple reaction time). The prevalence of subjective symptoms did not differ significantly between the controls and the manganese-exposed group except for 4 of 25 symptoms: fatigue, tinnitus, trembling of the fingers, and irritability. The standardized neurological examination did not reveal any specific difference between control and manganese-exposed workers except for rigidity of trunk. On the other hand, psychomotor tests showed that manganese-exposed workers exhibited a significantly longer mean reaction time than the control workers, performed significantly less well in the audio-verbal short-term memory test than the control group, and differed significantly in the prevalences of abnormal score values for eye–hand coordination and hand steadiness parameters than those found in the control group. This study indicated that a TWA exposure of total manganese dust of about 1 mg/m^3 might lead to the occurrence of clinical adverse effects in the lungs and the CNS in some workers exposed for less than 20 years.

Roels et al.[23] conducted another cross-sectional epidemiological study among 102 male workers exposed to MnO_2 in a dry alkaline battery factory to assess the validity of the conclusion drawn in their first study[22] and to better define an exposure limit to inorganic manganese dust. The mean age of the workers was 29.3 years, and the duration of exposure was 5.3 years with a range of 0.2 to 17.7 years. The results were compared with a matched control group of 104 workers. The authors[23] reported the current exposures to airborne manganese dust, determined using personal samplers, amounted on average (geometric mean) to 215 µg Mn/m^3 with respective geometric means of 792 and 3505 ìg Mn/m^3 $\times$ year. Although on a group basis, the concentrations of manganese in blood (MnB) and in urine (MnU) were significantly higher in the manganese-exposed group compared to the controls, on an individual basis, no statistically significant correlation was found between MnB or MnU and various external parameters, such as duration of exposure or current or integrated exposure to manganese.

A self-administered questionnaire of respiratory symptoms was analyzed separately for smokers and nonsmokers and for both groups combined. The analysis did not show any statistical difference in the prevalence of reported symptoms between the control and manganese group. The analysis of the questionnaire on neuro-vegetative complaints did not show any significant difference in the prevalence of reported symptoms between the control and manganese exposure. However, the manganese

workers performed several neurofunctional tests and measurements less satisfactorily than did the control workers. The manganese workers had a significantly longer reaction time over the whole testing period than the control group. The results of five eye–hand coordination parameters were more erratic in the manganese-exposed workers than in the control subjects, and the results of the hole tremometer test (hand steadiness) showed a systematic tendency towards higher mean tremor scores in the manganese group as compared with control group. Although the manganese-exposed workers performed less well in the audioverbal short-term memory test than the control group, the mean scores of correctly recalled or recognized words were not significantly different between the groups.

The authors[23] used a logistic regression model to analyze the data and found that hand tremor (hand steadiness) was the most appropriate parameter to define a threshold effect level. The study indicated that a lifetime integrated exposure to inhalable manganese dust above 3575 µg Mn/m^3 x year or lifetime integrated exposure to respirable manganese dust above 730 µg $Mn/m^3 \times$ year might lead to an increased risk of tremor.

At a symposium on manganese toxicity,[24] Roels and Lauwerys[25] recommended for a working lifetime of 40 years that the 8-hour TWA airborne concentration of manganese in inhalable dust should not exceed 90 µg Mn/m^3 and 18 µg Mn/m^3 for respirable dust, to protect the majority (95%) of the exposed workers from the neurotoxicity of manganese.

Mergler[26] reported on the results of a study performed on 74 manganese-exposed workers and their matched controls during the production of silicomanganese. Exposure concentrations measured with personal samplers amounted on average (geometric mean) to approximately 0.23 mg Mn/m^3 as inhalable dust. The results of neurophysiological and neuropsychological tests showed that manganese alloy workers differed from their controls on symptom reporting, emotional state, specific motor functions, hand steadiness, olfactory perception, and cognitive flexibility.

Lucchini[27] reported on the preliminary results of a study involving a group of 58 workers in a ferroalloy plant. The exposure levels had been reduced from about 1.6 to approximately 0.3 mg Mn/m^3 inhalable dust in the last 2 years. The workers were examined during a temporary lay-off period ranging from 1 to 42 days after cessation of exposure. The author[27] found that blood manganese levels were related to performance on tests of memory, motor speed, and other intellectual functions. However, no correlation was found for the reaction time test.

Iregren[28] reviewed psychological testing for neurotoxic effects from manganese exposure in active workers and reported that there was a coherent picture of effects on response speed, motor functions, and memory. However, Iregren concluded this coherent picture was somewhat blurred by studies[26,27] where the previously sensitive tests, regarding response speed, motor function, and memory did not consistently prove their sensitivity.

Reproductive Study

Lauwerys et al.[29] conducted a fertility study of 85 male workers in a plant producing manganese salts (dioxide, carbonate, sulfate) from concentrated ore. The airborne concentration of manganese inhalable dust personal samples ranged from 0.07 to 8.61 mg/m^3 with a median value of 0.97 mg/m^3. The overall arithmetic and geometric mean values were 1.33 and 0.94 mg/m^3, respectively. The authors[29] applied the method of Levine et al.[30], consisting of a questionnaire for monitoring male workers exposed to environmental agents that may impair fertility. Questions covering duration of employment, residence, and smoking and drinking habits were also contained in the surveys. The control group of workers consisted of 81 male subjects never occupationally exposed to manganese.

The matching of the exposed and control group was considered satisfactory. There was no marked difference between exposed and control groups with regard to age of the examined person and his wife, age of wife at marriage, age difference between husband and wife, duration of employment in the plant, smoking habits, alcohol consumption, educational level, and professional activity of the spouse.

The results of the study showed that the number of children in the age groups 16 to 25 and 26 to 35 born to manganese workers during exposure to the metal was significantly lower than the expected number. There was no indication that other factors might account for the marked difference between the control and manganese-exposed groups. This study suggests that a TWA exposure to manganese dust of about 1 mg/m^3 may be sufficient to interfere in male reproductive capacity, resulting in a significant deficit in the number of children born to manganese-exposed workers.

TLV Recommendation

The first cross-sectional study by Roels et al.[22] demonstrated that TWA exposures of approximately 1 mg/m^3 of manganese in air might lead to pre-clinical adverse effects in the lungs and nervous system in some workers exposed for less than 20 years. In a second cross-sectional study, Roels et al.[23] found neurotoxic effects among workers whose average lifetime integrated exposure to manganese dust was 50% less than that of the workers exposed during the authors' previous study[22] and suggested an 8-hour TWA of 0.09 mg/m^3 to protect the majority of workers from the neurotoxicity of manganese.

However, in this study, there was no indication of lung impairment, deficit in birth rate, or short-term memory deficit, and there were no differences with respect to subjective symptoms. In addition, the authors[23] concluded that to better define an acceptable occupational exposure limit, further studies were needed to determine whether the pre-clinical signs observed in their 1987 study[22] would be reversible when exposure to manganese had decreased or ceased.

The male fertility study by Lauwerys et al.[29] suggested a TWA exposure to about 1 mg/m^3 of total manganese dust might interfere in male reproductive capacity.

Clinical signs of manganism have been seldom reported below the former TLV–TWA of 5 mg/m^3, as Mn, for manganese dust and compounds. The World Health Organization (WHO)[31] concluded that signs of adverse effects on the CNS may occur at manganese concentrations of 2 to 5 mg/m^3.

The U.S. Environmental Protection Agency (EPA)[32] stated that epidemiological studies in humans indicate effects on the respiratory system at levels below 1 mg/m^3, whereas studies of effects on the CNS below this level are equivocal or negative.

The lowest exposure concentration of manganese at which early effects on the CNS and the lungs may occur is still unknown. However, once neurological signs are present, they tend to continue and worsen after exposure ends. Therefore, it is very important to detect and prevent adverse manganese effects while the disease is in its pre-clinical and possibly reversible stage of development. Accordingly, a TLV–TWA of 0.2 mg/m^3, as Mn, for manganese and inorganic compounds is recommended to reduce the potential for pre-clinical adverse effects in the lungs and CNS and adverse effects on the fertility of male workers exposed to manganese.

Sufficient data were not available to recommend Skin, SEN, or carcinogenicity notations or a TLV–STEL. The reader is expected to be familiar with the section on *Excursion Limits* in the "Introduction to the Chemical Substance TLVs" of the current edition of the *Documentation of the TLVs and BEIs* for the guidance and control of excursions above the TLV-TWA, even when the 8-hour TWA is within the recommended limit.

Historical TLVs

Manganese

1946–1947: MAC–TWA, 6 mg/m^3
1948–1959: TLV–TWA, 6 mg/m^3
1960–1962: TLV–TWA, 5 mg/m^3
1963–1969: TLV–CEILING, 5 mg/m^3

Manganese and Compounds

1970–1981: TLV–CEILING, 5 mg/m^3, as Mn

Manganese Fume

1977: *Proposed*: TLV–TWA, 1 mg/m^3, as Mn
1979–1994: TLV–TWA, 1 mg/m^3, as Mn; STEL, 3 mg/m^3, as Mn

Manganese Dust and Compounds

1982–1987: TLV–CEILING, 5 mg/m^3, as Mn
1986: *Proposed*: TLV–TWA, 5 mg/m^3, as Mn
1988–1994: TLV–TWA, 5 mg/m^3, as Mn

Manganese, Elemental, and Inorganic Compounds

1992: *Proposed*: TLV–TWA, 0.2 mg/m^3, as Mn
1995: TLV–TWA, 0.2 mg/m^3, as Mn

References

1. U.S. Agency for Toxic Substances and Disease Registry: Toxicological Profile for Manganese and Compounds. TP-91/19. ATSDR, U.S. Public Health Service, Atlanta, GA (July 1992).
2. Oberdoerster, G.; Cherin, G.: Proceedings, 17th Rochester Int'nl. Conf. Environ. Toxicol. Biological Monitoring of Toxic Metals. T.W. Clarkson, Ed. Plenum Press, New York (1988).
3. Beliles, R.P.: The Metals. In: Patty's Industrial Hygiene and Toxicology, 4th ed., Vol. II, Part C, Toxicology, pp. 2106–2124. G.D. Clayton and F.E. Clayton, Eds. John Wiley & Sons, New York (1994).
4. Lloyd Davies, T.A.; Harding, H.E.: Manganese Pneumonitis. Further Clinical and Experimental Observations. Br. J. Ind. Med. 6:82–90 (1949).
5. Levina, E.N.; Robachovsky, E.G.: Gigiena i Sanit. 1:25 (1955); abstract in Ind. Hyg. Digest 19(948) (1955).
6. Von Oettingen, W.F.: Manganese: Its Distribution, Pharmacology and Health Hazards. Physiol. Revs. 15(2):175–201 (1935).
7. Mella, H.: The Experimental Production of Basal Ganglion Symptomatology in *Macacus rhesus*. Arch. Neurol. Psychiat. 11:405–417 (1924).
8. Carratala, R.; Carboneschi, C.L.: The Toxicity and Fixation of Manganese. Chem. Abstr. 31:8697 (1937).
9. Morrow, P.: Retention Rate of Inhaled Submicron Manganese Dioxide. In: Inhaled Particles III, Vol. II. W.H. Walton, Ed. Unwin Bros., Ltd., Gresham Press. Old Woking, Surrey, U.K. (1970).
10. Fairhall, L.T.: Industrial Toxicology, p. 74. Williams & Wilkins, Baltimore (1957).
11. Fairhall, L.T.; Neal, P.A.: Industrial Manganese Poisoning. National Institutes of Health Bull. No. 182. NIH, Washington, DC (1943).
12. Flinn, R.H.; Neal, P.A.; Reinhart, W.H.; et al.: Chronic Manganese Poisoning in an Ore-Crushing Mill. U.S. Public Health Service Bull. No. 247. USPHS, Washington, DC (1940).
13. Lloyd Davies, T.A.: Manganese Pneumonitis. Br. J. Ind. Med. 3:111–135 (1946).
14. Hunter, D.: The Diseases of Occupation, 8th ed. P. Reffle, P. Adams, P. Baxter, and W. Lee, Eds. Little Brown & Co., Boston (1994).
15. Rodier, J.: Manganese Poisoning in Moroccan Mines. Br. J. Ind. Med. 12:21–35 (1955).
16. Kesiæ, B.; Hausler, V.: Hematological Investigation on Workers Exposed to Manganese Dust. Arch. Ind. Hyg. Occup. Med. 10:336–343 (1954).
17. Schuler, P.; Oyanguren, H.; Maturana, V.; et al.: Manganese Poisoning. Ind. Med. Surg. 26:167–173 (1957).
18. Tanaka, S.; Lieben, J.: Manganese Poisoning and Exposure in Pennsylvania. Arch. Environ. Health

19:674–684 (1969).
19. Whitlock, C.M.; Amuso, S.J.; Bittenbender, J.B.: Chronic Neurological Disease in Two Manganese Steel Workers. Am. Ind. Hyg. Assoc. J. 27:454–459 (1966).
20. Whitman, N.E.; Brandt, A.D.: Comments. Am. Ind. Hyg. Assoc. J. 27:459 (1966).
21. Smyth, L.T.; Ruhf, R.C.; Whitman, N.E.; Dugan, T.: Clinical Manganism and Exposure to Manganese in the Production and Processing of Ferromanganese Alloy. J. Occup. Med. 15:101–109 (1973).
22. Roels, H.; Lauwerys, R.; Buchet, J.P.; et al.: Epidemiological Survey Among Workers Exposed to Manganese: Effects on Lung, Central Nervous System, and Some Biological Indices. Am. J. Ind. Med. 11:307–327 (1987); Erratum. Am. J. Ind. Med. 12:119--120 (1987).
23. Roels, H.; Ghyselen, P.; Buchet, J.; et al.: Assessment of the Permissible Exposure Level to Manganese in Workers Exposed to Manganese Dioxide Dust. Br. J. Ind. Med. 49(1):25–34 (1992).
24. Symposium on Manganese Toxicity. Proceedings. International Manganese Institute, Paris (November 19–20, 1992).
25. Roels, H.; Lauwerys, R.: Health Risk Assessment of Chronic Exposure to MnO_2 Dust. An Epidemiological Study in a Battery Plant. Symposium on Manganese Toxicity. Proceedings. International Manganese Institute, Paris (November 19–20, 1992).
26. Mergler, D.: Early Nervous System Dysfunction Among Canadian Workers in a Ferro- and Silico-Manganese Alloy Plant. Symposium on Manganese Toxicity. Proceedings. International Manganese Institute, Paris (November 19–20, 1992).
27. Lucchini, R.: Neurobehavioral Effects in a Ferromanganese Group of Workers after Temporary Cessation of Exposure. Symposium on Manganese Toxicity. Proceedings. International Manganese Institute, Paris (November 19–20, 1992).
28. Iregren, A.: Psychological Testing for Neurotoxic Effects from Manganese in Active Workers. Symposium on Manganese Toxicity. Proceedings. International Manganese Institute, Paris (November 19–20, 1992.)
29. Lauwerys, R.; Roels, H.; Genet, P.; et al.: Fertility of Male Workers Exposed to Mercury Vapor or Manganese Dust: A Questionnaire Study. Am. J. Ind. Med. 7.(2):171–176 (1985).
30. Levine, R.J.; Symons, M.J.; Balogh, S.A.; et al.: A Method for Monitoring the Fertility of Workers. I. Method and Pilot Studies. J. Occup. Med. 22:781–791 (1980).
31. International Programme on Chemical Safety: Manganese Environmental Health Criteria 17. World Health Organization, Geneva (1981).
32. U.S. Environmental Protection Agency: Health Assessment Document for Manganese. Final Report. Environmental Criteria and Assessment Office, U.S. EPA, Cincinnati, OH (1984).