

# Abstract Book

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## Manganese Induced Parkinsonism: New Evidence and Hypothesis for a Complex Neurodegenerative Illness

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Manganese (Mn) is a well known neurotoxic agent that can cause a neuro-psychiatric intoxication called "manganism". This clinical manifestation was clearly described in many reports and studies on miners and ferroalloy workers exposed to very high concentrations. According to an extensive review by the World Health Organization clinical manganism can occur starting from the level of 2-5 mg/m<sup>3</sup>, but considering the highly variable individual susceptibility, even at 1 mg/m<sup>3</sup> of Mn in total dust (WHO, 1981). The neurological and neuropsychological features of manganism have been identified and distinguished from those typically expressed by patients affected by Idiopathic Parkinson Disease (IPD). In addition, response to Levodopa treatment, imaging and histological evidences have been addressed for typical manganism (Calne et al., 1994).

More recently, epidemiological evidence of increased parkinsonian disturbances and impaired motor functions has been shown in groups of population with environmental exposure to manganese (Øygard et al., 1992, Santos-Burgoa et al., 2001, Lucchini et al., 2003). Prevalence of parkinsonian disturbances in the vicinities of ferroalloy plants has been observed in the range of 250-450/100,000 population. Similar findings have been reported for welders with prolonged exposure to manganese, with a prevalence of 977 to 1,336 cases/100,000 population (Racette et al., 2005). These prevalence rates are significantly higher compared to the average rates reported in community based studies, indicating around 130/100,000.

A new hypothesis is based on these studies, indicating that high exposure levels of manganese can cause typical manganism, whereas prolonged exposure to much lower levels may act as an "environmental trigger" able to accelerate the physiological ageing of the brain. In this second case, the distinction between typical manganism and IPD would be less evident, with the expression of a new phenotype overlapping the features of both clinical entities.

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