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MANGANESE-INDUCED NEUROTOXICITY: LESSONS FROM WORMS TO HUMAN NEONATES

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Manganese (Mn) is a trace metal required for normal physiological processes in humans. Mn levels are tightly regulated, as high levels of Mn results in accumulation in the brain and cause a neurological disease known as manganism. Manganism shares many similarities with Parkinson's disease (PD), both at the physiological level and the cellular level. Exposure to high Mn-containing environments increases the risk of developing manganism. Homozygous mutations in *SLC30A10* cause familial Parkinsonism associated with manganese (Mn) retention. We recently identified SLC30A10 to be a cell surface-localized Mn efflux transporter and demonstrated that Parkinsonism causing mutations block its intracellular trafficking and efflux function. In C. elegans, SLC30A10 over-expression protected against Mn-induced lethality and dopaminergic neurotoxicity, consistent with results in mammalian systems. SLC30A10 expression did not protect worms against $ZnSO_4$ toxicity, suggesting that SLC30A10 does not mediate Zn export in *C. elegans*.

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