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Manganese induced cell death of SN56 basal forebrain cholinergic neurons mediated by oxidative stress generation

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Manganese (Mn) is an essential metal with industrial applications that have been shown to produce memory and learning deficits after acute and repeated exposure similar to those induced in Alzheimer's disease (AD). However, the complete mechanisms through which it induces these effects are unknown. In this regard, basal forebrain is one of the main regions involved in regulation of learning and memory processes and a degeneration of cholinergic neurons in this region has been related with cognitive disorders. Besides, it has been reported that manganese can induce cell death and oxidative stress on brain regions different from basal forebrain. Whether manganese-induced these effects in basal forebrain cholinergic neurons it would explain its effects on learning and memory processes. According to these data, we hypothesized that Mn could induce basal forebrain cholinergic cell death through oxidative stress generation. To prove this hypothesis, we evaluated in SN56 cell culture from basal forebrain, the Mn toxic effects after 24 h and 14 consecutive days exposure on cell viability and reactive oxygen species (ROS) formation and lipid peroxidation induction. This study shows that Mn increased the formation of ROS and induced lipid peroxidation after 24 h and 14 days of exposure. Mn also produced a decrease in cell viability of SN56 cells after 24 h and 14 days exposure, mediated partially by oxidative stress, although other mechanisms seem to be involved. Our present results provide the new view of the mechanisms contributing to Mn neurotoxicity and may explain cognitive dysfunctions observed after Mn exposure.

Biography

Paula Moyano received his JD degree at the University Complutense University of Madrid in 2013. She has a Masters in Pedagogical Sciences 2017. She specialized in neurotoxicology and legal sciences and received his PhD in Toxicology and legal medicine in 2016.

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