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Manganese induces cholinergic transmission disruption in SN56 cholinergic neurons from basal forebrain

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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 or cholinergic transmission disruption in this region has been related with cognitive disorders. Besides, it has been reported that manganese can affect cholinergic transmission, which may explain its effects on learning and memory processes. According to these data, we hypothesized that Mn could induce basal forebrain cholinergic transmission alteration. To prove this hypothesis, we evaluated in SN56 cell culture from basal forebrain, the Mn toxic effects after 24 h and 14 consecutive days of exposure on cholinergic transmission. This study shows that Mn decreased acetylcholine levels after 24 h and 14 days of exposure. Mn did not have any effect on vesicular acetylcholine transporter (VAChT) expression, although it induced acetylcholinesterase (AChE) activity and decreased choline acetyltransferase (ChAT) activity and high-affinity choline transporter (CHT) gene expression after 24 h and 14 days exposure, which suggests that these alteration mediated acetylcholine levels disruption. Our present results provide a new view of the mechanisms contributing to Mn neurotoxicity and may explain cognitive dysfunctions observed after Mn exposure.

Biography

Paula Moyano received her 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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