



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

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INTRODUCTION

Manganese (Mn) is an essential metal with industrial applications that has 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.

METHODS

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 cholinergic transmission.

RESULTS

This study shows that Mn decreased acetylcholine levels after 24 h and 14 days exposure. Mn 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, suggesting that these alteration mediated acetylcholine levels disruption.

CONCLUSIONS

Our present results provide new view of the mechanisms contributing to Mn neurotoxicity and may explain cognitive dysfunctions observed after Mn exposure.

