Cadmium induced β-amyloid proteins formation in SN56 basal forebrain cholinergic neurons mediated through muscarinic M1 receptor by alteration of AChE expression

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Abstract

Cadmium is a toxic compound reported to produce cognitive dysfunctions, though the mechanisms involved are unknown. Previously, it has been reported that cadmium induces a greater cell death in primary cholinergic neurons from the basal forebrain. It also induces cell death in SN56 cholinergic neurons from the basal forebrain partially through increase of amyloid-beta (Aβ) protein levels, among other mechanism. It was observed that the silencing or blockage of muscarinic 1 receptor (M1R) altered *acetylcholinesterase* (*AChE*) splice variants gene expression and Aβ protein formation. Furthermore, *AChE*-S variants were associated with the same actions modulated by M1R. Accordingly, we hypothesized that cadmium induced Aβ production in basal forebrain cholinergic neurons is mediated by M1R blockage, which triggers this effect through alteration of the expression of *AChE* variants. To prove this hypothesis, we evaluated that in SN56 cholinergic neurons from basal forebrain region, whether M1R silencing mediated Aβ production observed after cadmium exposure through alteration of the expression of *AChE* variants. Our results proved that cadmium induce Aβ production and this effect was mediated by M1R through *AChE* altered expression. Thus, our results help to explain the mechanism by which cadmium induces Aβ production in basal forebrain cholinergic neurons and may explain cognitive dysfunctions observed in cadmium toxicity.

Biography

Paula Moyano received her Juris Doctor degree from the Complutense University of Madrid in 2013. She has completed his/her Master's degree in Pedagogical Sciences. She specialized in Neurotoxicology and Legal Sciences and received his/her PhD in Toxicology and Legal Medicine in 2016.

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