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Cadmium induced β-amyloid proteins formation in SN56 basal forebrain cholinergic neurons mediated by oxidative stress generation

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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 by increasing amyloid beta (Aβ) protein levels among other mechanisms. Moreover, this mechanism for inducing cell death on SN56 cells after cadmium exposure is partially mediated by muscarinic 1 receptor (M1R), through alteration of AChE expression, suggesting other mechanisms are involved. In this regard, cadmium is also able to induce reactive oxygen species (ROS) formation, related with the Aβ production and Tau abnormal phosphorylation that could also contribute to explain the effect observed. Accordingly, we hypothesized that cadmium induced Aβ production on basal forebrain cholinergic neurons is mediated by ROS generation. To prove this hypothesis, we evaluated, in SN56 cholinergic neurons from basal forebrain region, cadmium Aβ production and whether ROS mediated this effect observed after cadmium exposure. Our results prove that cadmium induces Aβ production and ROS generation mediates this effect. Thus, our results help explain the mechanism by which cadmium induces Aβ production in basal forebrain cholinergic neurons and may explain cognitive dysfunctions observed in cadmium toxicity.

Biography

Javier Del Pino has received his PharmD degree at the University Complutense University of Madrid in 2004. He has specialized in Neurotoxicology and Neurodevelopmental Toxicology and received his PhD in Toxicology in 2009. In 2010, he has worked in Institute of Health Carlos III in the National Center of Environmental Health. From 2010 to 2012, he was an Associate Researcher at University of Massachusetts (UMASS) working in Sandra Petersen´s Lab in a National Institute of Health (NIH) project on developmental effects of TCDD endocrine disruptor on sexual differentiation. In 2016, he became Associate Professor of Toxicology at the Complutense University of Madrid, Spain.

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