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

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Cadmium is an environmental pollutant, which is a cause of concern because it can be greatly concentrated in the organism causing severe damage to a variety of organs including the nervous system, which is one of the most affected. Cadmium is a neurotoxic compound which induces cognitive alterations similar to those produced by Alzheimer's disease (AD). However, the mechanism through which cadmium induces this effect remains unknown. In this regard, cholinergic system in central nervous system (CNS) is implicated on learning and memory regulation, and it has been reported that cadmium can affect cholinergic transmission and it also induces a more pronounced cell death on cholinergic neurons from basal forebrain, which may explain cadmium effects on learning and memory processes. According to all above, an alteration of cholinergic transmission in basal forebrain cholinergic neurons, may result in the cognitive disorders observed after cadmium exposures. The present study is aimed at researching the selective neurotoxicity induced by cadmium on cholinergic system in CNS. For this purpose we evaluated, in basal forebrain region, the cadmium toxic effects on cholinergic transmission in NS56 cholinergic murine septal cell line. This study proves that cadmium induces an alteration of Ach levels and acetylcholinesterase (AChE) and choline acetyltransferase (ChAT) activities in SN56 cells. The alteration of Ach levels was independent of alteration of AChE activity, but dependent of ChAT activity reduction. Our present results provide new understanding of the mechanisms contributing to the harmful effects of cadmium on cholinergic neurons and suggest that cadmium could mediate the cognitive disorders through alteration of cholinergic transmission.

Biography

Javier Del Pino received his PharmD degree at the University Complutense University of Madrid in 2004. He has two Master's in Sciences, 2009 and 2010. He specialized in Neurotoxicology and Neurodevelopmental Toxicology and received his PhD in Toxicology in 2009. In 2010, he worked in Institute of Health Carlos III in the National Center of Environmental Health. From 2010 to 2012 he was Associated 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 got a position as Associated Professor of Toxicology at the Complutense University of Madrid.

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