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Striatal alteration of monoaminergic neurotransmitters systems in rats, after prenatal and postnatal exposure to chlordimeform

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Formamidine pesticides induce permanent sex- and region-dependent effects on development of monoaminergic neurotransmitter systems. These effects could be related to monoamine oxidase (MAO) inhibition. However, chlordimeform is a very weak MAO inhibitor, which suggests that other mechanism should be involved. In this regard, chlordimeform, a formamidine pesticide may alter the enzymes' expression that mediates the synthesis and metabolism of monoaminergic neurotransmitters systems. Therefore, an alteration of these enzymes could also mediate the effects observed. In order to confirm that the formamidines produce permanent alterations of the monoamine neurotransmitter systems in the striatum, by alteration of the expression of the enzymes that synthesize and/or metabolize these neurotransmitters, we evaluated, in striatum of male and female rats, the effect on the expression of MAO, COMT, BDH, TH, TRH and AD enzymes at 60 days of age after maternal exposure to chlordimeform (5 mg/kg body weight). Chlordimeform induced a significant decrease in the expression of the enzymes COMT, BDH, TH and TRH in both males and females. We observed a bigger increase in the expression of BDH [44, 65% (P<0,001)] enzyme in females than in males. Chlordimeform treatment did not alter the expression of MAO and AD enzymes. The present findings indicate that after maternal exposure to formamidines, in general, and chlordimeform, in particular, a permanent alteration of monoaminergic neurotransmitters, through alteration of the enzymes that synthesize these neurotransmitters, in striatum, is induced.

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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