

## Developmental neurotoxicity of monocrotophos and lead is linked to thyroid disruption

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### Abstract

**Aim:** A role of thyroid disruption in developmental neurotoxicity of monocrotophos (MCP) and lead is studied.

**Materials and Methods:** A total of 24 female rats after conception were randomized into four groups of six each and treated as follows: Group I - Sham was administered distilled water orally, Group II - A positive control was administered methyl methimazole at 0.02% orally in drinking water, Group III - MCP orally at 0.3 mg/kg and Group IV - Lead acetate at 0.2% orally in drinking water. The drug was administered from gestation day 3 through post-natal day 21 in all the groups. Acetylcholinesterase (AChE) inhibition, thyroid profile (thyroid stimulating hormone, T<sub>3</sub> and T<sub>4</sub>), neurodevelopment (brain wet weights, DNA, RNA and protein), and neurobehavioral (elevated plus maze, photoacrometry, and Morris water maze) parameters were assessed in pups. A histopathology of thyroid of dams and brain of progeny was conducted.

**Results:** Inhibition of AChE was <20%. Thyroid profile decreased in the treatment groups. Neurodevelopmental and neurobehavioral parameters did not reveal any significant changes. Thyroid architecture was affected significantly with MCP and lead. Cortical layers too were affected. The three layers of cerebellum either had abnormal arrangement or decreased cellularity in all treated groups relating to thyroid disruption.

**Conclusion:** MCP and lead might have affected the development of cerebrum and cerebellum via thyroid disruption leading to developmental neurotoxicity.

**Keywords:** behavioral alterations, developmental neurotoxicity, lead, monocrotophos, thyroid disruption.

### Introduction

The potential harmful chemicals or substances, such as heavy metals, pesticides, and hydrocarbons, are dumped either or released into the water bodies [1]. When these pollutants flow into water bodies in a higher concentration than permissible limits then these result in the form of heavy mortalities of all life form residing in those aquatic systems such as fish and shellfish, etc., while in a lower concentration these lead to bioaccumulation of these pollutants and ultimately go through the food web to human beings. These pollutants can also alter other hormonal processes of fish like the development of bones and proper thyroid functioning. Dimethoate and lambda-cyhalothrin showed the lethal effect on thyroid hormone of *Labeo rohita* [2]. Fetal exposure to environmental chemicals could affect the development of nervous

system. In this chemical age, certain of the developmental defects do not have a definite etiology and the only pointer could be exposure during development that too at a critical time. With the rise in the use of pesticides, surfacing of behavioral disorders became common. A majority of children suffer from neurodevelopmental disorders and exposure to xenobiotics has been identified as one of the risk factors. About 8 million children suffer from one or other mental disorders, and 1.1 million are exposed to organophosphate (OP) insecticides above the safety levels. One of the facets of OP toxicity is chronic OP-induced neuropsychiatry disorders. While researching on the developmental neurotoxicity of OPs, especially chlorpyrifos (CPS), their cholinesterase-independent actions came into the fore and have surpassed the receptor level and are lingering at the cell signaling mechanisms. One aspect that has been attempted albeit on a lesser scale is the interference of endocrine mechanisms by OPs that could contribute to the existing neurotoxicity on in-utero exposure.

Lead (Pb) has been implicated in a variety of behavioral disorders since its use in 1900 as leaded gasoline and other forms. Although a unified mechanism of action has been elusive, it is believed to be

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# Developmental Neurotoxicity Of Lead

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