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1: [Pediatr Res.](#) 2002 Oct;52(4):576-9.



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Increased monoamine concentration in the brain and blood of fetal thalidomide- and valproic acid-exposed rat: putative animal models for autism.

[Narita N](#), [Kato M](#), [Tazoe M](#), [Miyazaki K](#), [Narita M](#), [Okado N](#).

Neurobiology Laboratory, Institute of Basic Medical Sciences, University of Tsukuba, Ibaraki, Japan.

Autism is defined as a congenital neurodevelopmental disorder in which serotonergic dysfunction may be involved in its pathogenesis. One of the characteristic laboratory findings in autistic patients is hyperserotonemia, although its mechanism has not been elucidated to date because of difficulties in studying human patients. Recent reports have demonstrated that thalidomide or valproic acid exposure during early embryonic days (first trimester) in humans causes higher incidence of autism. Morphologic abnormalities found in autism (e.g. cerebellar anomalies, reduced motor neuron numbers) have been reported in animals administered with these teratogens prenatally, suggesting the possibility of the use of these animals as an experimental autistic model. In this study, we evaluated monoamine levels in the brain and blood of rats exposed to teratogens prenatally. Of the groups exposed to thalidomide on embryonic day (E)2, E4, E7, E9, and E11, a significant increase of hippocampal serotonin was only observed in the group exposed on E9. Furthermore, E9 thalidomide and valproic acid exposure both resulted in an increase of hippocampal serotonin, frontal cortex dopamine, and hyperserotonemia. These results thus indicate that two potentially autism-inducing teratogens, thalidomide and valproic acid, have the same effect on early monoamine system development in the brain and the blood, which may explain the pathogenesis of autism.

PMID: 12357053 [PubMed - indexed for MEDLINE]

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