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1: [Reprod Toxicol.](#) 1997 Mar-Jun;11(2-3):417-22.

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#### Linking etiologies in humans and animal models: studies of autism.

[Rodier PM](#), [Ingram JL](#), [Tisdale B](#), [Croog VJ](#).

Department of Obstetrics and Gynecology, University of Rochester, New York, USA.

Thalidomide has been shown to lead to a high rate of autism when exposure occurs during the 20th to 24th d of gestation. Both the critical period and the neurological deficits of the autistic cases indicate that they have sustained injuries to the cranial nerve motor nuclei. To determine whether such lesions characterize other cases of autism, the brain stem of an autistic case was compared to that of a control. The autopsy case showed abnormalities predicted by the thalidomide cases and evidence of shortening of the brain stem, a defect that could have occurred only during neural tube closure. To test whether animals can be similarly injured but remain viable, rats were treated with 350 mg/kg of valproic acid on day 11.5, 12, or 12.5 of gestation. Neuron counts showed reductions of cell numbers in the cranial nerve motor nuclei. Rats with motor neuron deficits also had cerebellar anomalies like those reported in studies of autistic cases, supporting the idea that these animals may be a useful model of the developmental injury that initiates autism.

PMID: 9100317 [PubMed - indexed for MEDLINE]

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