

# Abstract

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## **Preliminary evidence for involvement of the folate gene polymorphism 19bp deletion-DHFR in occurrence of autism.**

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**BACKGROUND:** Folate has long been implicated in both the metabolism of neurotransmitter molecules, and as an agonist with a direct effect upon neuronal tissue. **Folates mediate transfer of one-carbon units into major biosynthetic pathways.**

**DISCUSSION:** From a developmental perspective, the most important reactions are de novo methionine and thymine synthesis, critical for DNA expression and elaboration, respectively. Dihydrofolate reductase (DHFR) is the sole enzyme responsible for maintaining the reduced state of the vitamin needed for these two pathways.

**CONCLUSION:** Here, **we report that the 19bp-deletion polymorphism of DHFR acts independently (OR 2.69, 95% CI; 1.00-7.28,  $p < 0.05$ ) and in concert with related folate polymorphisms as a significant risk factor for autism.** Possible consequences of this are discussed in the context of the interaction between folate and the glutamatergic nervous system, an area of promising candidate genes for contributing to autism.

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