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## **Taurine prevents the neurotoxicity of beta-amyloid and glutamate receptor agonists: activation of GABA receptors and possible implications for Alzheimer's disease and other neurological disorders.**

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### **Source**

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### **Abstract**

Alzheimer's disease (AD) and several other neurological disorders have been linked to the overactivation of glutamatergic transmission and excitotoxicity as a common pathway of neuronal injury. The beta-amyloid peptide (Abeta) is centrally related to the pathogenesis of AD, and previous reports have demonstrated that the blockade of glutamate receptors prevents Abeta-induced neuronal death. We show that taurine, a beta-amino acid found at high concentrations in the brain, protects chick retinal neurons in culture against the neurotoxicity of Abeta and glutamate receptor agonists. The protective effect of taurine is not mediated by interaction with glutamate receptors, as demonstrated by binding studies using radiolabeled glutamate receptor ligands. The neuroprotective action of taurine is blocked by picrotoxin, an antagonist of GABA(A) receptors. GABA and the GABA(A) receptor agonists phenobarbital and melatonin also protect neurons against Abeta-induced neurotoxicity. These results suggest that activation of GABA receptors decreases neuronal vulnerability to excitotoxic damage and that pharmacological manipulation of the excitatory and inhibitory neurotransmitter tonus may protect neurons against a variety of insults. GABAergic transmission may represent a promising target for the treatment of AD and other neurological disorders in which excitotoxicity plays a relevant role.

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