

Protection via Methylcobalamine

Life Extension magazine republishes abstracts on health and longevity topics in each issue, drawn from research papers originally published in science and medical journals throughout the world.

Protective effects of a vitamin B12 analogue, methylcobalamin, against glutamate cytotoxicity in cultured cortical neurons Akaike A Tamura Y Sato Y Yokota T, Eur J Pharmacol (1993 Sep 7) 241(1):1-6 The effects of methylcobalamin, a vitamin B12 analogue, on glutamate-induced neurotoxicity were examined using cultured rat cortical neurons. Cell viability was markedly reduced by a brief exposure to glutamate followed by incubation with glutamate-free medium for 1 h. Glutamate cytotoxicity was prevented when the cultures were maintained in methylcobalamin-containing medium. Glutamate cytotoxicity was also prevented by chronic exposure to S-adenosylmethionine, which is formed in the metabolic pathway of methylcobalamin. Chronic exposure to methylcobalamin and S-adenosylmethionine also inhibited the cytotoxicity induced by methyl-D-aspartate or sodium nitroprusside that releases nitric oxide. In cultures maintained in a standard medium, glutamate cytotoxicity was not affected by adding methylcobalamin to the glutamate-containing medium. In contrast, acute exposure to MK-801, a NMDA receptor antagonist, prevented glutamate cytotoxicity. These results indicate that chronic exposure to methylcobalamin protects cortical neurons against NMDA receptor-mediated glutamate cytotoxicity.