

X-Message-Number: 32236
Date: Fri, 18 Dec 2009 23:29:00 -0800 (PST)
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Subject: evidence based OTC treatment of Alzheimer's disease I

[Clinical trials with over-the-counter supplements have concentrated either on items which suppress inflammation, or on antioxidants which scavenge oxygen derived free radicals. Most of these items have proved to be worthless in the treatment of Alzheimer's disease. Similarly most drugs used to treat Alzheimer's disease do little to slow the deterioration, but instead offer a mild temporary symptom relief. However, evidence has been accumulating that the primary driver of Alzheimer's disease is a nitrogen derived free radical called peroxynitrite, which may mediate both amyloid and tau accumulation as well as their toxicity. Excellent results have been obtained with peroxynitrite scavengers, with reversals of Alzheimer's disease in human clinical trials being repeatedly demonstrated. IMHO, the only thing which may be preventing the abolition of Alzheimer's disease is the mental inertia of scientists, as well as the bureaucrats who fund them. Unfortunately, most bureaucrats keep throwing money into repeatedly testing discredited interventions, while ignoring successful ones. Common sense is anything but...]

[Abolition of amyloid induced memory deficits by rosmarinic acid is due to peroxynitrite scavenging.]

Behav Brain Res. 2007 Jun 18;180(2):139-45. Epub 2007 Mar 12.

A natural scavenger of peroxynitrites, rosmarinic acid, protects against impairment of memory induced by Abeta(25-35).

Alkam T, Nitta A, Mizoguchi H, Itoh A, Nabeshima T. Department of Neuropsychopharmacology & Hospital Pharmacy, Nagoya University Graduate School of Medicine, Nagoya 466-8560, Japan.

Peroxynitrite (ONOO(-))-mediated damage is regarded to be responsible for the cognitive dysfunction induced by amyloid beta protein (Abeta) in Alzheimer's disease (AD). In the present study, we examined the protective effects of rosmarinic acid (RA), a natural scavenger of ONOO(-), on the memory impairment in a mouse model induced by acute i.c.v. injection of Abeta(25-35). Mice daily received i.p. several doses of RA after the injection of Abeta(25-35). RA prevented the memory impairments induced by Abeta(25-35) in the Y maze test and novel object recognition task. RA, at the effective lowest dose (0.25mg/kg), prevented Abeta(25-35)-induced nitration of proteins, an indirect indicator of ONOO(-) damage, in the hippocampus. At this dose, RA also prevented nitration of proteins and impairment of recognition memory induced by ONOO(-)-i.c.v.-injection. Co-injection of the non-memory-impairing dose of ONOO(-) with Abeta(25-35) blocked the protective effects of RA (0.25mg/kg). These results demonstrated that the memory protective effects of RA in the neurotoxicity of Abeta(25-35) is due to its scavenging of ONOO(-), and that daily consumption of RA may protect against memory impairments observed in AD.

PMID: 17420060

[Peroxynitrite is linked to the accumulation of tau filaments.]

Am J Pathol. 2003 Sep;163(3):1021-31.
Nitration of tau protein is linked to neurodegeneration in tauopathies.

Horiguchi T, Uryu K, Giasson BI, Ischiropoulos H, LightFoot R, Bellmann C,