

Sharper Mind™ Product Science – Vinpocetine Abstracts

{Note: the underlined sections within the text of the abstracts are highlighted for emphasis by us, not the authors}

(1)

Brain-Res-Bull. 2000 Oct; 53(3): 245-54

Role of sodium channel inhibition in neuroprotection: effect of vinpocetine.

Bonoczka, P : Gulyas, B : Adam Vizi, V : Nemes, A : Karpati, E : Kiss, B : Kapas, M : Szantay, C : Koncz, I : Zelles, T

Vinpocetine (ethyl apovincamate) discovered during the late 1960s has successfully been used in the treatment of central nervous system disorders of cerebrovascular origin for decades. The increase in the regional cerebral blood flow in response to vinpocetine administration is well established and strengthened by new diagnostic techniques (transcranial Doppler, near infrared spectroscopy, positron emission tomography). The latest in vitro studies have revealed the effect of the compound on Ca(2+)/calmodulin dependent cyclic guanosine monophosphate-phosphodiesterase 1, voltage-operated Ca(2+) channels, glutamate receptors and voltage dependent Na(+)-channels; the latest being especially relevant to the neuroprotective action of vinpocetine. The good brain penetration profile and heterogeneous brain distribution pattern (mainly in the thalamus, basal ganglia and visual cortex) of labelled vinpocetin were demonstrated by positron emission tomography in primates and man. Multicentric, randomized, placebo-controlled clinical studies proved the efficacy of orally administered vinpocetin in patients with organic psychosyndrome. Recently positron emission tomography studies have proved that vinpocetine is able to redistribute regional cerebral blood flow and enhance glucose supply of brain tissue in ischemic post-stroke patients.

(2)

Free-Radic-Res. 2000 Jan; 32(1): 57-66

Synaptosomal response to oxidative stress: effect of vinpocetine.

Santos, M S : Duarte, A I : Moreira, P I : Oliveira, C R

It has been suggested that reactive oxygen species (ROS) play a role in the neuronal damage occurring in ischemic injury and neurodegenerative disorders and that their neutralization by antioxidant drugs may delay or minimize

neurodegeneration. In the present study we examine whether **vinpocetine** can act as an antioxidant and prevent the formation of ROS and lipid peroxidation in rat brain synaptosomes. After ascorbate/Fe²⁺ treatment a significant increase in oxygen consumption (about 5-fold) and thiobarbituric acid reactive substances (TBARS) formation (about 7-fold) occurred as compared to control conditions. **Vinpocetine** inhibited the ascorbate/Fe²⁺ stimulated consumption of oxygen and TBARS accumulation, an indicator of lipid peroxidation, in a concentration-dependent manner. The ROS formation was also prevented by **vinpocetine**. Oxidative stress increased significantly the fluorescence of the probes 2',7'-dichlorodihydrofluorescein (DCFH₂-DA) (about 6-fold) and dihydrorhodamine (DHR) 123 (about 10-fold), which is indicative of intrasynaptosomal ROS generation. **Vinpocetine** at 100 microM concentration decreased the fluorescence of DCFH₂-DA and DHR 123 by about 50% and 83%, respectively. We conclude that the antioxidant effect of **vinpocetine** might contribute to the protective role exerted by the drug in reducing neuronal damage in pathological situations.

(3)

J-Am-Geriatr-Soc. 1987 May; 35(5): 425-30

A double-blind placebo controlled evaluation of the safety and efficacy of vinpocetine in the treatment of patients with chronic vascular senile cerebral dysfunction.

Balestreri, R : Fontana, L : Astengo, F

In a double-blind clinical trial, **vinpocetine**, a synthetic ethyl ester of apovincamine, was shown to effect significant improvement in elderly patients with chronic cerebral dysfunction. Forty-two patients received 10 mg **vinpocetine** three times a day (tid) for 30 days, then 5 mg tid for 60 days. Matching placebo tablets were given to another 42 patients for the 90 day trial period. Patients on **vinpocetine** scored consistently better in all evaluations of the effectiveness of treatment including measurements on the Clinical Global Impression (CGI) scale, the Sandoz Clinical Assessment-Geriatric (SCAG) scale, and the Mini-Mental Status Questionnaire (MMSQ). There were no serious side effects related to the treatment drug.

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