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LETTER TO THE EDITOR

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NEW INSIGHT INTO MODE OF ACTION OF COLOSTRININ™

Dear Editor,

The paper by Dariush Douraghi-Zadeh et al. (1) provides insight into a likely mechanism by which Colostrinin™ provides protection against cell death (apoptosis) in Alzheimer's disease pathogenesis. Alzheimer's is a progressive neurodegenerative illness which is on the increase in developing countries. Most current clinical approaches to neurodegenerative disease such as Alzheimer's involve symptomatic treatment, and do not deal with the causes of the disease (2). The nutraceutical Colostrinin™ is a proline-rich polypeptide that has been isolated from both ovine and bovine colostrum, and has considerable potential to treat neurodegenerative diseases, particularly the early stages of cognitive decline. Previous studies have shown that Colostrinin™ decreases intracellular levels of reactive oxygen species and modulates activities of antioxidant enzymes and mitochondrial function (3). Moreover, recent microarray data have demonstrated altered expression profiles of several genes that are directly or indirectly involved in the maintenance of the cellular redox state and may have anti-tau activity (4).

Degenerative diseases such as Alzheimer's disease, are characterised by considerable neuronal and synaptic loss in areas of the brain involved in cognition, especially the temporal lobes including the hippocampus. The neuronal loss in Alzheimer's is correlated with the presence of tangles composed of hyper-phosphorylated tau, and senile plaques comprised principally of amyloid β-protein (A β). Determining the role played by amyloid β - is thus of great importance in understanding Alzheimer's disease. The paper by Douraghi-Zadeh et al. examines how Colostrinin™ prevents apoptosis in SH-SY5Y human neuroblastoma cells induced by aggregated β-amyloid. Cytotoxicity assays (using MTT and LDH) demonstrated that pre-treatment of human neuronal SHSY-5Y cells with 5 μg/ml Colostrinin™, for 24 hours, conferred neuroprotection against Aβ-induced neurotoxicity. Moreover 24 hours of pre-treatment with 5 mg/ml Colostrinin™ was also shown to reduce Aβ1-40- induced apoptosis in human neuronal cells as determined via qualitative and quantitative apoptosis assays.

Intriguingly the authors demonstrated that the neuroprotection conferred by Colostrinin™ pre-treatment was reduced with the Fas ligand (FasL) binding antibody Nok1, suggesting that the effects of Colostrinin™ may involve a Fas:soluble FasL interaction.

These findings indicate that Colostrinin™ could possibly play a role in the prevention of AD pathogenesis, through the inhibition of Fas-mediated apoptosis and coming on top of the recent microarray studies described above, demonstrates very clearly the enormous potential of Colostrinin™ as a nutraceutical product for use in treating cognitive decline in humans.

References

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