New Neurotoxic Research Reveals Impact of Trehalose Sugar

Smart Sugars Lesson # 38

by JC Spencer

Physicians are learning how to help rid our bodies of neurotoxins. And, it is with a sugar. They call it alternative medicine. I call it food. More medical and healthcare professions are recommending their patients change the sugar in their sugar bowl.

A 2011 published paper verifies previous research that indicates the sugar trehalose is a powerful modifier of abnormal protein accumulation in neurodegenerative diseases.

I have reported on many university studies that verify it is the accumulation of neurotoxic proteins that cause neurodegenerative diseases like Parkinson's, Alzheimer's, and Huntington's. These and other diseases are due to accumulation of abnormal proteins which fold improperly and impair neuronal function.

Neurotoxins increase the rate of cell death. Researchers at the Departamento de Neurobiología-Investigación, CIBERned, Hospital Ramón y Cajal in Madrid, Spain investigated an alternative mechanism for elimination of abnormal proteins, by treatment with the sugar trehalose.

The Abstract states that the sugar trehalose is known to enhance autophagy, protects cells against various environmental stresses.

The medical dictionary's definition of autophagy is: a catabolic process involving the degradation of a cell's own components through the lysosomal machinery. It is a tightly regulated process that plays a normal part in cell growth, development, and homeostasis, helping to maintain a balance between the synthesis, degradation, and subsequent recycling of cellular products. It is a major mechanism by which a starving cell reallocates nutrients from unnecessary processes to more-essential processes.

The researchers in Madrid used trehalose as treatment which produced a dose and time-dependent increase in the number of autophagosomes and markers of autophagy in NB69 cells.

Trehalose did not change the number of total neither the number of dividing cells in the culture but it completely prevented the necrosis of NB69 induced by epoxomicin. In addition, the treatment with trehalose reverted the accumulation, induced by epoxomicin, of polyubiquitinated proteins, total and phosphorylated tau, p-GSK-3, and α-synuclein, as well as the α-synuclein intracellular aggregates.

The effects of trehalose were not mediated through activation of free radical scavenging compounds, like GSH, or mitochondrial proteins, like DJ1, but trehalose reduced the activation of ERK and chaperone HSP-70 induced by epoxomicin. Inhibition of ERK phosphorylation prevented the epoxomicin-induced cell death. Inhibition of autophagy reverted the neuroprotective effects of trehalose in epoxomicin-induced cell death. These results suggest that trehalose is a powerful modifier of abnormal protein accumulation in neurodegenerative diseases.

Source:
http://www.ncbi.nlm.nih.gov/pubmed/21232572

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