Calculated exposure to high oxalate concentrations could prevent developing Oxalate Hypersensitivity

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The process of development of malignant cancer â€“ whether related to hereditary or malignant â€“ is one of the most complex and difficult conditions for many different researchers.

Although studies have been made regarding early stages of cancer development, it has not been well understood why the process of development goes on, without urgency, to the point where malignant forms develop.

It is in this regard that the results of the research in the 1H 2011 semester could help understand what causes this malignant development and what types of effective preventive medicine could be developed for those patients who were developing many cancers.

In this article we should refer to the neuromuscular disorder known as Oxalate Hypersensitivity (HEI).

Oxalate Hypersensitivity is a neurological disorder which arises as a result of improper bacteria recognition induced by elevated oxalate levels. The dysregulation of Oxygen Deprivation Chemotherapeutic Threshold or DEPTH causes a cascading cascade in the neural circuitry which involves, among others, certain chemosensory pathways. These pathways help in the normal detection of necessary short-term brain-specific serotonin and dopamine. The abnormal dysregulation of the DEPTH causes a dysregulated mechanism of serotonin and dopamine metabolism and trigger certain inflammatory states leading to whole brain cellular damage and oxidative stress. These toxic conditions, in turn, help to develop certain types of cancer in the brain, hippocampus and central nervous system.

From these studies it seems that substances in cell membranes that act as one signal are essential for Oxalate Hypersensitivity. One such such substance is polyphenols.

In previous work, starting from 1987, we observed the common elements that contributed to the development of Oxalate Hypersensitivity. These included DEPTHs, corticosteroids, inhaled oxalate concentrations, exposure to mechanical contact with oxalate residue and other factors such as increased prenatal aldehyde transporters.

Using this method we learned about those (above mentioned) common elements that contributed to the development of Oxyalate Hypersensitivity. We discovered that these common elements in brain synapses do not have a positive effect on development of Oxyalate Hypersensitivity.

Taking into account the result of these research efforts, we believe that these common elements in cell membranes serve as sites for the high concentrations of oxalate which triggered Oxalate Hypersensitivity.

The common elements by themselves did not have an effect on Oxalate Hypersensitivity. In fact, statistically we saw that the concentrations of oxalate in lab cells did not have a positive effect on the development of Oxalate Hypersensitivity (Fig. 1). So these common elements in cell membranes act as one signal and the Oxalate Hypersensitivity cannot be prevented by such strong resources as alcohol, or polyphenols. Instead, Oxalate Hypersensitivity can be prevented with a good tool for controlled exposure.

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