

### **L-Glutamine (Glutamine)**

L-Glutamine (also known as Glutamine) is the most abundant amino acid in the body. L-Glutamine is classified as a non-essential amino acid (meaning that it can be produced in the body) and plays an important role in maintaining the health of numerous body functions. Glutamine is utilized as a source of energy by all rapidly dividing cells and is an important muscle-building amino acid. It also increases production of new cells in the intestines, where it may help heal the damage caused by NSAIDs (non-steroidal anti-inflammatory drugs). In the brain, Glutamine is necessary for the production of neurotransmitters like GABA (gamma-aminobutyric acid).

Glutamine is plentiful in both animal and plant protein. The typical diet provides between 3.5 - 7 grams of glutamine, though heavy stress, such as strenuous exercise, infectious disease, surgery or other acute trauma may lead to Glutamine depletion with consequent immune dysfunction, intestinal problems and muscle wasting. Glutamine supplements cause a rapid rise in cellular Glutamine levels and Glutamine stores in muscle, and seem to be well-tolerated.

When the brain produces low energy, excitotoxins, such as glutamate, become even more toxic. It has been shown that the reason for glutamine toxicity under these conditions is because it is converted to the excitotoxin – glutamate

Of particular concern is the finding that people with multiple sclerosis have increased levels of the enzyme glutaminase (the enzyme that converts glutamine into glutamate) in areas of nerve fiber damage. High levels of glutamine in the diet would increase glutamate levels near these injured areas magnifying the damage. It has been shown that excitotoxicity plays a major role in multiple sclerosis by destroying the cells (oligodendrocytes) that produce myelin. Russell L. Blaylock, M.D.

Glutamate toxicity may conceivably be operative in neuropathological conditions that disrupt neuronal/oligodendrocyte interactions in axons, e.g. multiple sclerosis. Rosin et al 2004

Excitotoxic damage is a common pathologic event in a number of neurologic diseases occurring after accumulation of excess extracellular glutamate in the CNS and subsequent overstimulation of glutamate receptors

Oligodendrocytes appear to be predominant cells for glutamate clearance in human white matter. Glutamate receptor expression and glutamate removal were defective in MS white matter, possibly mediated by TNFalpha, changes that might underlie high extracellular glutamate and an increased risk for glutamate excitotoxicity.

Recent evidence suggests an altered glutamate homeostasis in the brain of patients with multiple sclerosis (MS), as seen in experimental models of MS. Cerebrospinal fluid glutamate levels were significantly higher in patients assessed during relapse compared with those of the patients with relapsing-remitting MS examined during the stable clinical phase and the controls ( $P < .001$ ). The levels of glutamate detected in patients with relapsing-remitting MS during the stable phase who had active lesions were significantly higher than in those without neuroradiological evidence of disease activity ( $P < .001$ ). Significantly higher levels of glutamate were found in patients with secondary progressive MS with an increase of 1 or more points on the Expanded Disability Status Scale score compared with stable patients with secondary progressive MS and control subjects ( $P < .001$ ). CONCLUSIONS: Neurotoxic events occur in MS patients, and they can be responsible for oligodendrocyte and neuronal cell death in patients with this demyelinating disease. The manipulation of glutamate-altered homeostasis or antagonizing glutamate receptor-mediated excitotoxicity may have therapeutic implications in MS patients.

Glu excitotoxicity has been proposed as one of the mechanisms of the demyelination