

CAITLIN E. MURPHY and CHARLES B. ANTHONY. Department of Chemistry/Biochemistry, West Virginia Wesleyan College, Buckhannon, WV 26201. Expressional changes in amino acid glutamate transporters: 2 and 3, in the presence of plaque severities in Alzheimer's disease.

The primary goal of this experiment was to determine if there was a correlation between the expression of EAAT-2 and EAAT-3 in the glutamate reuptake pathway in astrocytes and the production of plaques associated with Alzheimer's Disease. Since both the EAAT-2 and EAAT-3 transporter proteins are involved in glutamate transport under normal pre-synaptic and post-synaptic neuron signaling, then altered expression of either EAAT-2 or EAAT-3 can have catastrophic effects on the functions of neurons in the brain. In Alzheimer's Disease it is believed that there is an under expression of both EAAT-2 and EAAT-3 which in turn effects the glutamate transport pathway by preventing glutamate uptake and causing synaptic suppression. Accumulation of extracellular glutamate can cause neurodegeneration and allow for the reduction of glutamine needed by the presynaptic neuron. We anticipated an inverse correlation between the expression of both EAAT-2 and EAAT-3 and the formation of plaques, revealing that the expression of EAAT-2 and EAAT-3 is decreased most in patients with severe Alzheimer's Disease.