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Failures in brain energy metabolism unveil therapeutic targets for Huntington's disease

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The brain makes up 2% of a person's weight. Despite this, even at rest, the brain consumes 25% of the body's energy. Most of the energy consumed in the brain is attributable to restoration of the membrane gradient following neuronal depolarization. Neurotransmitter recycling, intracellular signaling and dendritic and axonal transport also require energy. Even though neurons are responsible for massive energy consumption, the brain is made up of many cells, including neurons, glial and ependymal cells. Every brain cell has a specific function and thus every brain cell has different metabolic needs. Many of these specific functions are concerned with maintenance of neuronal transmission. Astrocytes play a central role in supporting neurons metabolically by producing lactate, through glycolysis and activation of glycogen catabolism. There have been several reports of metabolic impairment in a variety of neurodegenerative disorders such as Alzheimer's disease, Huntington's disease, amyotrophic lateral sclerosis and Parkinson's disease, among others. Moreover, deregulation of energy metabolism could be implicated in an increased production of oxidative species. During the last 10 years we have been making steady progress in the mechanisms of communication between neurons and glial cells, the way they regulate their metabolism and the use of ascorbic acid as inter cellular messenger. Here, we will describe the regulation of neuronal glucose, lactate and ascorbic acid transporters under synaptic activity in mice models of Huntington's disease. Experiments demonstrating a failure in astrocytic ascorbic acid recycling and ascorbic acid-dependent modulation on neuronal metabolism in Huntington's disease will be discuss. Brain is an expensive organ in energetic terms so disruptions in energy production may affect neuronal transmission and thus, neuronal survival.

Biography

Maite A Castro is a Professor in the Department of Biochemistry at the Universidad Austral de Chile since 2005. She has obtained her PhD in Biological Sciences at Universidad Austral de Chile in 2005. In 2009, she did a Postdoctoral training in Dr. Michael Levine's Laboratory at the University of California, Los Angeles, USA. During the last 15 years she has been making steady progress in the mechanisms of communication between neurons and glial cells and the way they regulate their metabolism. Presently, her interest is to study the correlation between failures in brain energy metabolism and the progression of Huntington disease.

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