Neuron-Glia crosstalk in the autonomic nervous system and its possible role in the progression of metabolic syndrome: A new hypothesis

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Metabolic syndrome (MS) is characterized by the following physiological alterations: increase in abdominal fat, insulin resistance, high concentration of triglycerides, low levels of HDL, high blood pressure, and a generalized inflammatory state. One of the pathophysiological hallmarks of this syndrome is the presence of neurohumoral activation, which involve autonomic imbalance associated to hyperactivation of the sympathetic nervous system. Indeed, enhanced sympathetic drive has been linked to the development of endothelial dysfunction, hypertension, stroke, myocardial infarct, and obstructive sleep apnea. Glial cells, the most abundant cells in the central nervous system, control synaptic transmission, and regulate neuronal function by releasing bioactive molecules called gliotransmitters. Recently, a new family of plasma membrane channels called hemichannels has been described to allow the release of gliotransmitters and modulate neuronal firing rate. Moreover, a growing amount of evidence indicates that uncontrolled hemichannel opening could impair glial cell functions, affecting synaptic transmission and neuronal survival. Given that glial cell functions are disturbed in various metabolic diseases, we hypothesize that progression of MS may relies on hemichannel-dependent impairment of glial-to-neuron communication by a mechanism related to dysfunction of inflammatory response and mitochondrial metabolism of glial cells. In this manuscript, we discuss how glial cells may contribute to the enhanced sympathetic drive observed in MS, and shed light about the possible role of hemichannels in this process. © 2015 Del Rio, Quintanilla, Orellana and Retamal.

Connexins

Glia

Hemichannels

Metabolic syndrome

Mitochondria

Tripartite synapse

adenosine triphosphatase (potassium sodium)

excitatory amino acid transporter

glial fibrillary acidic protein

interleukin 1beta

inwardly rectifying potassium channel

nestin

tumor necrosis factor alpha

vimentin

Article

astrocyte

autonomic nervous system

cell activation

cell communication

cell function

cell interaction

encephalitis

glia cell

gliosis

hemichannel

human

hypothesis

membrane channel

metabolic syndrome X

microglia

nerve cell

nonhuman

oxidative stress

paracrine signaling

sympathetic nerve cell

sympathetic tone

synaptic transmission