Carotid body type-I cells under chronic sustained hypoxia: Focus on metabolism and membrane excitability

and membrane excitability
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Chronic sustained hypoxia (CSH) evokes ventilatory acclimatization characterized by a progressive
hyperventilation due to a potentiation of the carotid body (CB) chemosensory response to hypoxia.
The transduction of the hypoxic stimulus in the CB begins with the inhibition of K+ currents in the
chemosensory (type-I) cells, which in turn leads to membrane depolarization, Ca2+ entry and the
subsequent release of one- or more-excitatory neurotransmitters. Several studies have shown that
CSH modifies both the level of transmitters and chemoreceptor cell metabolism within the CB. Most
of these studies have been focused on the role played by such putative transmitters and modulators
of CB chemoreception, but less is known about the effect of CSH on metabolism and membrane
excitability of type-I cells. In this mini-review, we will examine the effects of CSH on the ion channels
activity and excitability of type-I cell, with a particular focus on the effects of CSH on the TASK-like
background K+ channel. We propose that changes on TASK-like channel activity induced by CSH
may contribute to explain the potentiation of CB chemosensory activity. Copyright © 2018

Carotid body

Chronic hypoxia

Ion channels

Membrane depolarization

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TASK-like channel

adenosine triphosphate

calcium ion
chloride channel
cholinergic receptor
dopamine
endothelin 1
heme oxygenase 2
hypoxia inducible factor
hypoxia inducible factor 1beta
hypoxia inducible factor 2alpha
neurotransmitter
nitric oxide
potassium ion
sodium ion
voltage gated calcium channel
carotid body chemoreceptor
carotid body type I cell
cell activation
cell function
cell hypoxia
cell metabolism
cell proliferation
chemoreceptor cell
chronic sustained hypoxia
conductance
human
intracellular membrane

membrane depolarization
membrane potential
nerve cell excitability
neurotransmission
nonhuman
osmolarity
oxidative phosphorylation
protein expression
protein function
protein secretion
Review
sensitization