Enhanced carotid body chemosensory activity and the cardiovascular alterations induced by intermittent hypoxia

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The carotid body (CB) plays a main role in the maintenance of the oxygen homeostasis. The hypoxic stimulation of the CB increases the chemosensory discharge, which in turn elicits reflex sympathetic, cardiovascular and ventilatory adjustments. An exacerbate carotid chemosensory activity has been associated with human sympathetic-mediated diseases such as hypertension, insulin resistance, heart failure and obstructive sleep apnea (OSA). Indeed, the CB chemosensory discharge becomes tonically hypereactive in experimental models of OSA and heart failure. Chronic intermittent hypoxia (CIH), a main feature of OSA, enhances CB chemosensory baseline discharges in normoxia and in response to hypoxia, inducing sympathetic overactivity and hypertension.

Oxidative stress, increased levels of ET-1, Angiotensin II and pro-inflammatory cytokines, along with a reduced production of NO in the CB, have been associated with the enhanced carotid chemosensory activity. In this review, we will discuss new evidence supporting a main role for the CB chemoreceptor in the autonomic and cardiorespiratory alterations induced by intermittent hypoxia, as well as the molecular mechanisms involved in the CB chemosensory potentiation. ©

Autonomic dysfunction

Carotid body

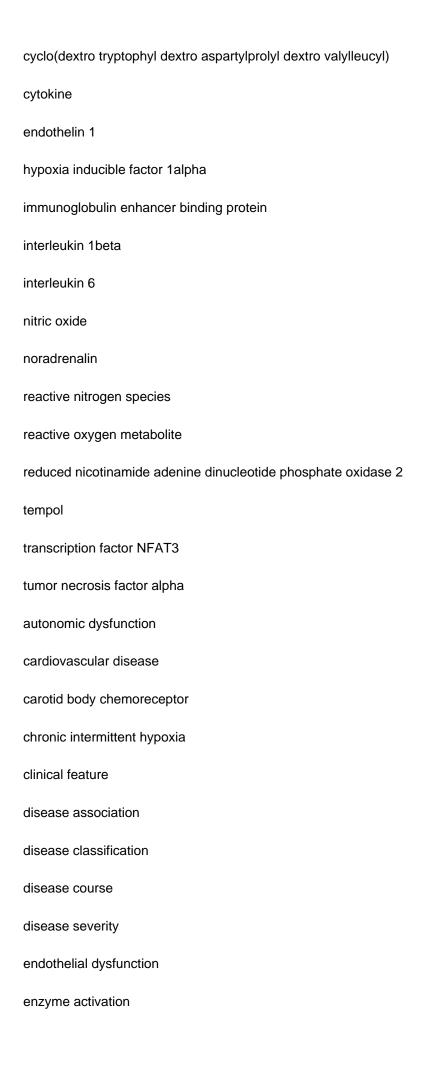
Hypertension

Intermittent hypoxia

Oxidative stress

angiotensin II

bosentan



human
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