Hypoxia-related lipid peroxidation: Evidences, implications and approaches

Behn, Claus

Araneda, Oscar F.

Llanos, Aníbal J.

Celedón, Gloria

González, Gustavo

Hypoxia may be intensified by concurrent oxidative stress. Lack of oxygen in relation to aerobic ATP requirements, as hypoxia has been defined, goes along with an increased generation of reactive oxygen species (ROS). Polyunsaturated fatty acids (PUFAs) range among the molecules most susceptible to ROS. Oxidative breakdown of n-3 PUFAs may compromise not only membrane lipid matrix dynamics, and hence structure and function of membrane-associated proteins like enzymes, receptors, and transporters, but also gene expression. Eicosapentaenoic acid depletion, products of lipid peroxidation (LP), as well as, lack of oxygen may combine in exacerbating activity of nuclear factor kappa B (NF?B), an ubiquitous pro-inflammatory and anti-apoptotic transcription factor. Field studies at high altitude show malondialdehyde (MDA) content in exhaled breath condensate (EBC) of mountaineers to correlate with Lake Louis score of acute mountain sickness. A pathogenic role of LP in hypoxia can therefore be