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OXIDATIVE AND NITROSATIVE STRESS AND BRONCHIAL ASTHMA IN CHILDREN

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Bronchial asthma (AB) is a common disease that affects children and adults of all ages. Although the pathogenesis of AB remains incompletely understood, it is associated with chronic airway inflammation and increased oxidant stress. Although oxygen is a prerequisite to life, at concentration beyond the physiological limits it may be hazardous to the cells. Since the lung are directly exposed to very high amounts of oxygen, it is imperative for the organ to possess defenses against possible oxidative challenge. Many studies conducted on the human asthmatic subjects or laboratory animal models of asthma revealed that numerous biologically active pro-inflammatory mediators lead to increased and uncontrolled production of reactive oxygen species (ROS) and the gaseous molecule nitric oxide (NO). Oxidant-antioxidant imbalance may play an important role in the pathogenesis of AB, especially during acute exacerbations. The ROS likely play a vital role because these have been shown to be associated with many pathophysiologic changes that are relevant in asthma, such as increased lipid peroxidation, increased airway reactivity and secretion, increased production of chemoattractants and increased vascular permeability. Persistently increased ROS and NO in asthma lead to reactive nitrogen species (RNS) formation and subsequent oxidation and nitration of proteins, which may cause alterations in protein function that are biologically relevant to airway injury and inflammation. Another important source of NO-derived oxidants is eosinophil peroxidase and myeloperoxidase and leucocyte-derived enzymes. Concomitant with increased oxidative stress in asthmatics, loss of protective antioxidant defense, especially superoxide dismutase, contributes to the overall toxic environment of the asthmatic airway. Oxidative metabolites may play a direct or indirect role in the modulation of airway inflammation. It was shown also that decreased intake of antioxidant is connected with increased risk of exacerbation of wheezing symptoms. Conversely, e.g. the consumption of fruit rich in vitamin C (important dietary antioxidant), even at a low level of intake may reduce wheezing symptoms already susceptible individuals. There are alterations in a wide array of oxidants and antioxidants, with imbalance shifting toward increased oxidative stress in AB. Increased oxidative stress is likely to contribute to perpetuation and amplification of the inflammatory response in asthmatics. Therapeutic augmentation of the antioxidant defenses might be beneficial. Further studies are necessary to clarify the exact position of oxidative stress in the pathogenesis of asthma: it plays important role in the initial phase of clinical expression of asthma, co-works in asthma exacerbation and contributes to the maintenance of chronic inflammation in asthmatics.