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Imbalance between Increased Oxidative Sources and Defective Anti-oxidant Mechanisms

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Description

Oxidative stress is defined as an imbalance between increased oxidative sources and reduced or defective antioxidant mechanisms. While this is conceptually adequate, it fails to capture how dynamic and highly complex the process actually is. This definition suggests that a certain balance does exist and that deviations from this point can affect homeostasis and potentially cause or worsen disease. Thus, many research projects have attempted to restore this "balance" by providing antioxidants, with mostly disappointing results. This lack of therapeutic efficacy is likely due to several factors, including our inability to distinguish when, in a given disease process, oxidative stress is the cause of the disease or merely an epiphenomenon; even worse, antioxidant treatment can prevent the occurrence of oxidative stress, which may play an important role in controlling inflammation and cellular adaptive responses. Nowhere are these complexities of oxidative stress more evident than in the airways, which are constantly exposed to oxidant compounds from the outside and from the inside the effects of recruited and activated inflammatory cells. Given the lack of proper standardization of many biomarkers used to determine airway oxidative stress, it is difficult to fully assess their overall contribution to the development or progression of airway diseases.

Metabolic dysregulation as a source of airway oxidative stress

There is increasing evidence that obesity is a major comorbidity that may exacerbate asthma severity and reduce control through mechanisms other than increased allergic airway inflammation. In fact, paradoxically, among subjects with asthma, increased Body Mass Index (BMI) was associated with fewer airway eosinophils and lower exhaled NO. Some studies have shown that with increasing BMI, there are increased levels of biomarkers of oxidative stress in the airways, which are also associated with reduced corticosteroid responsiveness in vitro. Airway oxidative stress in obese and asthmatic patients may be explained by an enhanced inflammatory response associated with leptin. Experimentally, leptin increases the number of inflammatory cells and cytokines in the airways. Obese people with asthma have the highest levels of leptin in the airways and have been shown to release pro-inflammatory cytokines from activated alveolar macrophages. Another source of oxidative stress may be the formation of hypochlorous acid from MPO in airway neutrophilia, which has been described to be associated with obesity and occurs after a high-fat diet. Another recently proposed source of airway oxidative stress in obesity is related to the generation of superoxide anion from airway Inducible Nitric Oxide Synthase (INos).

During NOS uncoupling, this enzyme produces superoxide anion instead of NO, with the dual effect of promoting oxidative stress while reducing NO bioavailability. Uncoupling has been shown to occur with less enzyme substrate (L-arginine) and/or with increased levels of endogenous NOS inhibitors. Asymmetric Dimethyl Arginine (ADMA) results from post-translational methylation of L-arginine and is one of three NOS inhibitors that can uncouple all NOS isoforms. Subjects with asthma, especially those with more severe disease, have been shown to have lower L-arginine levels. In addition, obesity and metabolic syndrome were associated with higher ADMA levels.

Antioxidants mechanism in the lung

The lungs are equipped with enzymatic (SOD, catalase, glutathione peroxidase) and non-enzymatic antioxidant systems (ceruloplasmin, ferritin, ascorbic acid, uric acid, thionine and carotene), which allow the lungs to function with constant buffering of a wide range of environmental oxidants. The presence of insufficient antioxidant levels or mechanisms is a hallmark of airway oxidative stress that is well known and the subject of excellent reviews. Therefore, the purpose of this section is not to review each pathway, but to discuss these antioxidants as a dynamic component of oxidative stress with clinical and redox signaling implications.

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