

The Pathogenesis of Obstructive and Restrictive Lung Diseases

Elena Bargagli* and Paolo Cameli

Section of Respiratory Medicine, Department of Medical Sciences, Surgery and Neurosciences, University Hospital Siena, Italy

***Corresponding Author:** Elena Bargagli, Professor, Section of Respiratory Medicine, Department of Medical Sciences, Surgery and Neurosciences, University Hospital Siena, Siena, Italy.

Received: April 23, 2019; **Published:** August 30, 2019

Keywords: *Interstitial Lung Diseases; Obstructive Pulmonary Disease; Prognosis*

The pathogenesis of lung diseases is a relevant topic of the research. In the last years, a wide spectrum of lung diseases including obstructive and restrictive lung diseases, rare lung disorders such as cystic fibrosis, occupational diseases, acute lung injury and drug-induced lung diseases have been explored through different methodologies. Some studies, for example, have analysed changes in protein expression in patients with different pneumopathies, i.e. proteins that are up-or down regulated, modified or secreted under particular pathological conditions and therefore implicated in the pathogenetic mechanisms of the diseases [1,2]. Some research groups adopted this approach to study of serum, BAL using 'omic' approaches, some others preferred the analysis of trace elements to analyse distinct pathogenetic pathways [3,4]. Trace elements for example are essential for cell metabolism regulation, including the activation or inhibition of enzymatic reactions, and the regulation of gene and membrane functions [5]. During the last decades, knowledge of metal transport proteins and the transfer of their different chemical forms to different organs and tissues has significantly improved [6]. Unlike other organs, lungs are directly and continuously exposed to high oxygen concentrations, exogenous oxidants and pollutants; thus, they have the greatest susceptibility to oxidative stress and pollutant toxicity, from which they protect themselves through the action of own constitutive and inducible antioxidants and detoxification mechanisms [7]. It is useful to study the metabolism and impact of essential and toxic metals in the respiratory system rather than by traditional exposure biomarkers such as blood or urine. Among essential trace elements, Fe metabolism is the most studied [8]. Iron plays a fundamental role in the respiratory chain and it is essential for repairing damaged lung tissue [8]. However, if not appropriately chelated, Fe can promote the formation of harmful free radicals. Many other trace elements are involved in the regulation of Fe metabolism and contribute to the functioning and protection of the lung. Thus, a better knowledge of the occurrence and distribution of trace elements in the respiratory system and changes occurring during lung diseases could give new insights for diagnostic, therapeutic and preventive actions, especially for severe and complex diseases. The lung is particularly susceptible to oxidative stress being continuously exposed to oxygen radicals and other exogenous oxidants such as the ozone and sulphur dioxide [9]. The endogenous defence against oxidative stress induced by free radicals stress involves several preventive, repair, enzymatic, and non-enzymatic mechanisms [10]. As the oxidant/antioxidant imbalance is implicated in the pathogenesis of miscellaneous diseases affecting the lung [10], the research is highlighting its involvement in the chronic obstructive pulmonary diseases, lung cancer and interstitial pulmonary fibrosis providing an interesting overview of new therapeutic strategies.

Conflict of Interest

No conflict of interest related to this manuscript has to be declared from the author.

Bibliography

1. Rottoli P, *et al.* "Proteomic analysis of interstitial lung diseases: a review". *Current Opinion in Pulmonary Medicine* 15.5 (2009): 470-478.

2. Landi C., *et al.* "Towards a functional proteomics approach to the comprehension of idiopathic pulmonary fibrosis, sarcoidosis, systemic sclerosis and pulmonary Langerhans cell histiocytosis". *Journal of Proteomics* 83 (2013): 60-75.
3. Barnes PJ. "Cellular and molecular mechanisms of asthma and COPD". *Clinical Science* 131.13 (2017): 1541-1558.
4. Strzelak A., *et al.* "Tobacco smoke induces and alters immune responses in the lung triggering inflammation, allergy, asthma and other lung diseases: A mechanistic review". *International Journal of Environmental Research and Public Health* 15.5 (2018): E1033.
5. Bargagli E., *et al.* "Analysis of trace elements in bronchoalveolar lavage of patients with diffuse lung diseases". *Biological Trace Element Research* 124.3 (2008): 225-235.
6. Monaci F., *et al.* "Concentrations of major elements and mercury in unstimulated human saliva". *Biological Trace Element Research* 89.3 (2002): 193-203.
7. Van der Vliet A., *et al.* "Oxidative stress in chronic lung disease: From mitochondrial dysfunction to dysregulated redox signaling". *Molecular Aspects of Medicine* 63 (2018): 59-69.
8. Rubinsztein DC., *et al.* "Autophagy and aging". *Cell* 146.5 (2011): 682-695.
9. Chilosi M., *et al.* "Premature lung aging and cellular senescence in the pathogenesis of idiopathic pulmonary fibrosis and COPD/emphysema". *Translational Research* 162.3 (2013): 156-173.
10. Mariani TJ. "Respiratory disorders: Ironing out smoking-related airway disease". *Nature* 531.7596 (2016): 586-587.

Volume 8 Issue 9 September 2019

© All rights reserved by Elena Bargagli and Paolo Cameli.