Journal of Clinical Respiratory Open Access

Extended Abstract

Vol. 1, Iss. 1

2019

COPD 2016: COPD and its association with gene polymorphism and cigarette smoke_Balagopalan Unni_CSIR-North East Institute of Science & Technology, India

Balagopalan Unni

Chronic obstructive pulmonary disease (COPD) is considered one of the most common preventable diseases. A large number of population studies have cited the polymorphism of the alpha-1 antitrypsin gene as one of the main genetic reasons for its pathogenesis. Among the environmental factors, along with the C-F compounds, cigarette smoke is considered to be the main factor in the progression of the disease. But the question is, how is cigarette smoke related to genetic polymorphism? The question is indeed complex. We are trying to understand the question, in terms of cigarette smoke compounds, of how they regulate antioxidant enzymes. We are getting some interesting results on how cigarette smoke extracts affect the machinery of the glutathione S-transferase enzyme. We are looking for the mechanism concerning the inflammatory effect of the extracts. From a genetic point of view, studies of gene expression of the determinants of cellular levels of GSH are also in progress. We have carried out experiments on genetic polymorphism studies of patients with COPD and have shown that the polymorphism of the GSTM1 gene is the main factor of the disease in coal miners.

Particulate toxic matter and toxic gases in the atmosphere are likely to be inhaled or often self-administered by cigarette smoke, causing lung damage. However, contamination of the atmosphere by anthropogenic sources such as coal mines, industrial sources as well as local conditions generated at home or in the workplace contributes significantly to the development of COPD. The relative prevalence and severity of occupational lung diseases linked to mining depend on the products used, levels of exposure to airborne risks, coexisting diseases or environmental conditions and lifestyle. Chronic obstructive pulmonary disease (COPD) is thought to be the result of the triggered environment in genetically sensitive individuals. Alpha 1 antitrypsin is the only known genetic cause of COPD. Bhattacharjee et al. previously studied the polymorphism of the α 1-antitrypsin gene in the population of the same area where we resumed the study. COPD is the consequence of an abnormal inflammatory response due to the inhalation of harmful agents such as smoking, occupational or environmental exposure. In fact, only a portion (10-20%) of heavy smokers develop a clinically detectable disease. Antioxidants and other less well understood protective mechanisms may also be important in preserving normal lung function in the face of lifetime exposure to potentially harmful environmental factors. Oxidative damage can also play an important role in the pathogenesis of COPD. Such an injury, resulting from an imbalance between free radicals and protective mechanisms, can alter the conformation of protease inhibitors and repairing enzymes, damage cell membranes and lead to mutagenesis. Free radicals appear in the lungs by inhalation of the environment or by their release of inflammatory cells inside the body. Genetically controlled antioxidant defense systems can also play an important role in determining sensitivity, both to free radicals released by inflammatory cells and to inhaled oxidants from the environment. The lung has several enzymatic trappers including glutathione which are under genetic control. The observation that the enzyme antioxidants are under genetic control and the allelic variations of these antioxidants modify their capacities to reduce free radicals, suggests that the genetic factors can expose certain individuals to a greater risk of oxidative injury. The glutathione system is the main antioxidant mechanism in the airways. Increased oxidative stress in the airways of patients with COPD may play an important pathophysiological role in the development of the disease by enhancing the inflammatory response in COPD.

Bottom Line: This abstract is partly presented at 3rd International Conference on Chronic Obstructive Pulmonary Disease on July 11-12, 2016 held at Brisbane, Australia

bgunni@yahoo.com