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Chronic Obstructive Pulmonary Disease: Role of cytokines and treatment optimization

Abstract:

Chronic obstructive pulmonary disease (COPD) is a slowly developing, progressive condition, characterized by basically irreversible changes resulting in long-term breathing difficulty, including shortness of breath, cough, and excessive sputum production. Airway obstruction results in persistent airflow limitation. Clinical phenotypes include chronic bronchitis and emphysema. The relative contribution of each differs between patients. Known causes include cigarette smoking, air pollutants, occupational exposure, and alpha-1 antitrypsin deficiency. A persistent inflammatory process involving activation of multiple types of immune cells and release of different pro-inflammatory cytokines, including interleukin (IL)-1b, tumor necrosis factor (TNF)-a, interferon (IFN)-g, IL-8, IL-17, IL-18, and IL-32, contribute to the development and progression of the disease. Resulting imbalances between proteases and antiproteases and between oxidants and antioxidants play important roles. Functional overlaps of different cytokines are due to high levels of pleiotropy and redundancy. In addition to commonly used treatments, such as smoking cessation, bronchodilators, steroids, oxygen, mucolytics, antibiotics, vaccinations, a-1 antitrypsin, pulmonary rehabilitation, and lung volume reduction surgery, new therapeutic approaches are needed to optimize the treatment based on specific pathophysiological disease phenotypes targeting specific pro-inflammatory mediators and associated inflammatory pathways.

Biography

John Klir is an MD/PhD medical scholar with broad experience of more than 25 years in academic medicine, biomedical research, clinical medicine, and administration. His research areas include immunophysiology, specifically the roles of pro-inflammatory cytokines in the pathophysiology of diseases. Dr Klir's work resulted in a significant contribution to the area of cytokine research, as evidenced by multiple publications.