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Biography

John Klir has completed his PhD in Physiology from University of Illinois, MD from Saba University School of Medicine and postgraduate training from University of Michigan School of Medicine in areas of Immune-Pathophysiology, namely roles of cytokines as pro-inflammatory mediators. His work resulted in significant contribution to area of cytokine research, as evidenced by multiple publications.

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E-BABE ROLE OF PRO-INFLAMMATORY CYTOKINES IN DEVELOPMENT AND PROGRESSION OF CHRONIC OBSTRUCTIVE PULMONARY DISEASE

Chronic obstructive pulmonary disease (COPD) is a slowly progressive condition with high morbidity and mortality. Inhaled irritants are linked to COPD development, with cigarette smoke being the important factor. Manifestation of COPD includes two main components, emphysema and chronic obstructive bronchitis. An important feature is persistent inflammatory process, characterized by involvement of many immune cells, including neutrophils, macrophages, T lymphocytes, B lymphocytes, and eosinophil's. Activation of these cells is mediated by different immune mediators. Several pro-inflammatory cytokines play important roles during development and progression of COPD. Cytokines identified as pathophysiological mediators of COPD are interleukin (IL)-1β, IL-6, tumor necrosis factor TNF-α interferon (IFN)-γ, IL-8, IL-17, IL-18 and IL-32. Cigarette smoke can directly activate multiple cells, including cells such as pulmonary macrophages and bronchial epithelial cells, resulting in release of pro-inflammatory cytokines. Increased levels of these cytokines result in activation and recruitment of inflammatory cells, leading to inflammation and eventually to pulmonary tissue destruction. Numbers of immune cells reactive to IL-1β are increased in bronchial biopsies from patients with stable COPD along with elevated IL-1β levels in sputum, bronchoalveolar lavage and serum. Systemic and local levels of IL-6 are elevated in patients with stable COPD. IL-6 may also significantly contribute to progression of COPD by playing an important role in autoimmune response in patients with more severe stable COPD. TNF-α activates immune cells and bronchial epithelial and smooth muscle cells to release inflammatory mediators, such as oxidants, resulting in progressive airway remodeling. Sputum levels of these cytokines are elevated in patients during COPD exacerbation.