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Abstract Topic: - Molecular effects of genetic variation

Abstract Title: - Galectin-3 as an emerging molecule in pathophysiology of chronic obstructive pulmonary disease.

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Aims: - To assess the role of Galectin-3 in pathogenesis of Chronic Obstructive Pulmonary Disease.

Methods: - The relevant literature was retrieved from the NCBI (https://pubmed.ncbi.nlm.nih.gov) by using appropriate keywords.

Results: - Chronic Obstructive Pulmonary Disease (COPD) is a set of pulmonary diseases, characterized by persistent airflow limitation resulting from chronic inflammation caused by long-term exposure to harmful gases and particles. Globally, prevalence rate of COPD is 12.2%. In India's adult population, a meta-analysis of eight studies, involving 8,569 participants, found a combined prevalence rate of 7.4% for COPD. In addition to environmental factors, genetic variations play a substantial role in influencing the progression of COPD. Exposure of irritants to the respiratory system results in the activation of immune cells, particularly macrophages and neutrophils. Galectin-3 is expressed in various myeloid cell types and is involved in further recruitment and activation of neutrophils, which release harmful substances like oxygen radicals and elastase in response to irritants and noxious particles which are responsible for the destruction of the lung tissues that occur as a result of smoking cigarettes. Galectin-3, encoded by LGALS3, is a pro-inflammatory molecule that significantly contributes to inflammation, further exacerbating the progression of COPD. Galectin-3 boosts macrophage phagocytosis and promotes natural killer cell activity and production of IL-1. An increase in Galectin-3 expression in small airways has been reported among severe COPD patients. Neutrophils were stated to be specifically localized near the epithelium of small airways, and this presence was associated with increased epithelial proliferation and airflow obstruction. Serum Galectin-3 levels were shown to be elevated during the acute exacerbation phase in contrast to the convalescence phase of COPD. Additionally, Galectin-3 levels were higher in current smokers than in never-smokers, suggesting a potential link between circulating Galectin-3 levels and smoking history. Recent research has revealed a significant correlation between elevated Galectin-3 levels in blood and lung tissues and the occurrence of increased epithelial injuries and airflow obstruction in severe COPD patients. Genetic variation in LGALS3 can modulate expression and functional attributes of Galectin-3.

Conclusions: - These findings suggest that Galectin-3 might be a valuable biomarker and therapeutic target for COPD. Understanding its role in immune responses and inflammation may lead to development of novel treatment strategies.

Keywords: - Chronic Obstructive Pulmonary Disease (COPD) is a set of pulmonary diseases, characterized by persistent airflow limitation resulting from chronic inflammation caused by long-term exposure to harmful gases and particles. Globally, prevalence rate of COPD is 12.2%. In India's adult population, a meta-analysis of eight studies, involving 8,569 participants, found a combined prevalence rate of 7.4% for COPD. In addition to environmental factors, genetic variations play a substantial role in influencing the progression of COPD. Exposure of irritants to the respiratory system results in the activation of immune cells, particularly macrophages and neutrophils. Galectin-3 is expressed in various myeloid cell types and is involved in further recruitment and activation of neutrophils, which release harmful substances like oxygen radicals and elastase in response to irritants and noxious particles which are responsible for the destruction of the lung tissues that occur as a result of smoking cigarettes. Galectin-3, encoded by LGALS3, is a pro-inflammatory molecule that significantly contributes to inflammation, further exacerbating the progression of COPD. Galectin-3 boosts macrophage phagocytosis and promotes natural killer cell activity and production of IL-1. An increase in Galectin-3 expression in small airways has been reported among severe COPD patients. Neutrophils were stated to be specifically localized near the epithelium of small airways, and this presence was associated with increased epithelial proliferation and airflow obstruction. Serum Galectin-3 levels were shown to be elevated during the acute exacerbation phase in contrast to the convalescence phase of COPD. Additionally, Galectin-3 levels were higher in current smokers than in never-smokers, suggesting a potential link between circulating Galectin-3 levels and smoking history. Recent research has revealed a significant correlation between elevated Galectin-3 levels in blood and lung tissues and the occurrence of increased epithelial injuries and airflow obstruction in severe COPD patients. Genetic variation in LGALS3 can modulate expression and functional attributes of Galectin-3.