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Abstract Topic: - Molecular effects of genetic variation

Abstract Title: - DC-SIGN in the pathophysiology of chronic obstructive pulmonary disease.

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Aims: - To assess the role of DC-SIGN in pathogenesis of Chronic Obstructive Pulmonary Disease.

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

Results: - Chronic Obstructive Pulmonary Disease (COPD) is a chronic inflammatory condition characterized by airflow limitation. Various environmental factors and genetic variations significantly contribute to COPD progression. COPD encompasses a group of conditions primarily chronic bronchitis and emphysema, often coexist and share similar symptoms. Innate inflammation and remodeling of small airways which start in the early stages of the disease are hallmarks of COPD. Dendritic Cells (DCs) play a key role in regulating each stage of these processes. They sense environment for microbial compounds and danger signals using pathogen recognition receptors and damage-associated pattern recognition respectively. Cigarette smoke is also linked to the movement of DCs from mucosa to nearby lymph nodes or a decrease in their recruitment and buildup within the bronchial mucosa or may trigger DC apoptosis. On surface of Dendritic Cells is a type II transmembrane protein receptor called Dendritic Cell-Specific Intercellular adhesion molecule-3-Grabbing Non-integrin or DC-SIGN, encoded by CD209. DC-SIGN recognizes and binds various pathogens (viruses and bacteria). It plays a role in capturing pathogens and facilitating their uptake by dendritic cells, leading to subsequent immune responses. Expression of DC-SIGN and number of DCs have been observed to be reduced in COPD patients who are current smokers whereas in ex-smokers these parameters seem to be similar to control samples. Thus, quitting smoking would appear to return the DC numbers in smokers with COPD to levels similar to those seen in non-smoking healthy individuals. Genotypic variants of CD209 have been studied in relation to many respiratory diseases including pulmonary tuberculosis, SARS and invasive pulmonary Aspergillosis infection, it is under scope that similar analysis can be done in relation to COPD. Genetic variation in CD209 can modulate expression and functional attributes of DC-SIGN.DC-SIGN has been seen to interact with bacterium Pseudomonas aeruginosa, infection of which further acts as a major cause of exacerbations and morbidity in COPD.

Conclusions: - A perusal of these studies indicates important role of DC-SIGN in pathophysiology of COPD and its exacerbations. Considering this, DC-SIGN has been suggested as a crucial molecule in shaping immune responses and outcomes in COPD.

Keywords: - Chronic Obstructive Pulmonary Disease (COPD) is a chronic inflammatory condition characterized by airflow limitation. Various environmental factors and genetic variations significantly contribute to COPD progression. COPD encompasses a group of conditions primarily chronic bronchitis and emphysema, often coexist and share similar symptoms. Innate inflammation and remodeling of small airways which start in the early stages of the disease are hallmarks of COPD. Dendritic Cells (DCs) play a key role in regulating each stage of these processes. They sense environment for microbial compounds and danger signals using pathogen recognition receptors and damage-associated pattern recognition respectively. Cigarette smoke is also linked to the movement of DCs from mucosa to nearby lymph nodes or a decrease in their recruitment and buildup within the bronchial mucosa or may trigger DC apoptosis. On surface of Dendritic Cells is a type II transmembrane protein receptor called Dendritic Cell-Specific Intercellular adhesion molecule-3-Grabbing Non-integrin or DC-SIGN, encoded by CD209. DC-SIGN recognizes and binds various pathogens (viruses and bacteria). It plays a role in capturing pathogens and facilitating their uptake by dendritic cells, leading to subsequent immune responses. Expression of DC-SIGN and number of DCs have been observed to be reduced in COPD patients who are current smokers whereas in ex-smokers these parameters seem to be similar to control samples. Thus, quitting smoking would appear to return the DC numbers in smokers with COPD to levels similar to those seen in non-smoking healthy individuals. Genotypic variants of CD209 have been studied in relation to many respiratory diseases including pulmonary tuberculosis, SARS and invasive pulmonary Aspergillosis infection, it is under scope that similar analysis can be done in relation to COPD. Genetic variation in CD209 can modulate expression and functional attributes of DC-SIGN.DC-SIGN has been seen to interact with bacterium Pseudomonas aeruginosa, infection of which further acts as a major cause of exacerbations and morbidity in COPD.