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Exposure of Allergens in Different Environments and their Pathophysiology

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Commentary

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DESCRITPION

The development of sensitization and allergies is the result of a very complex gene-gene and gene environment interaction. Some of the recent studies have shown that there is a lower sensitization in children who live in farms possibly due to endotoxin, higher bacterial and viral infections and higher omega 3 fatty acids from cow's milk exclusively fed on fresh grass. Also some of the allergic sensitization related to CD14, TLR-2 and TLR-4 leads to higher levels of sensitization. A complex interplay occurs between the allergen exposure and environmental factors in the development of clinical allergy. In susceptible individuals, the effect of allergen exposure is enhanced when combined with anthropogenic factors such as diesel exhaust particles or environment tobacco smoke. The protein nutrition that follows many allergens can lead to development of neo-allergens. Many of these anthropogenic cofactors act as Th2 adjuvants and increase Th2 responses. Many types of pollen, themselves contribute to the development of sensitization in susceptible individuals by release of plant associated lipid mediators such as oxylipins and phytoprostanes. Oxylipins influence the chemo taxis of pro-inflammatory cells such as eosinophil's and neutrophils and result in increased eosinophil cationic proteins and myeloperoxidase. Phytoprostanes influence the dendritic cells and increase Th2 responsiveness. Pollens also contain NADPH oxidases with resultant increase in reactive species that can damage the airway epithelium leading to barrier dysfunction and increased risk of sensitization and clinical allergy.

Some of the recent developments on the key cells involved in the process of sensitization and clinical allergy are the dendritic cells and T regulatory cells. Dendritic cells are one of the most important cells in the pathophysiology of sensitization and allergy. The antigens are presented to the T cells *via* the antigen presenting cells which is the mucosal dendritic cell. Depending on the co-factors associated during the antigen presentation, different kinds of T effector cell subsets can be activated that either lead to protection or the development of sensitization and allergy. The control of further downstream pathways is by the T regulator cells both activate and suppress different effector cells. It also has regulatory effects on other T cell subsets such as Th9, Th17, Th1 and Th2 and in the normal person leads to the generation of immunoglobulin-G antibodies and thus protection against allergies. One of the most important pathways of T regulator cell activation depends on the co-factors associated with antigen presentation by the dendritic cells, whether it happens in a pathogen associated molecular patterns rich environment or without. In a pathogen associated molecular pattern rich environment the dendritic cells have a high density of pathogen recognition receptors on its surface. A PAMP rich environment is associated with the antigen presentation to the T cell in the presence of IL-2 which leads to the differentiation of T cell to T regulatory and Th1 cells thus conferring antigen recognition without allergy. In a PAMP deficient environment antigen presentation by the DCs occur without IL-12 leading to the T-cells differentiation into Th2, thus causing sensitization and clinical allergy with the production of IL-4, IL-13 and other inflammatory cytokines. This is the basis of the hygiene hypothesis that explains lower incidence of sensitization and allergy.