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The Cause of Autoimmune and Allergic Diseases

by

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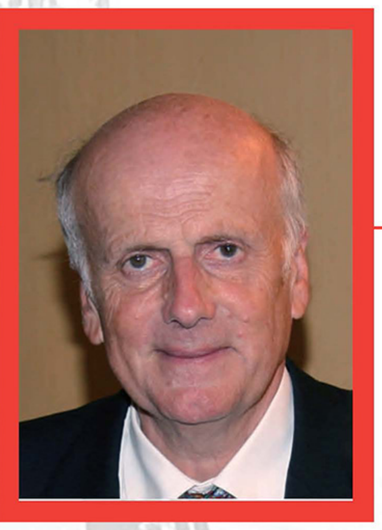

Abstract:

Autoimmune and allergic diseases show an increased incidence worldwide, especially in developed countries. There is no single cause for these diseases but a combination of genetic and acquired predisposing factors. Genetic determinism is complex involving many genes. It is still very difficult, with few exceptions, to assess the real importance of individual genes in the pathophysiology and the etiology of the disease. Environmental factors are very diverse. Some are clearly identified: a specific virus or bacterium in a small number of autoimmune diseases and, of course, allergens in allergic diseases, when they are identified. In fact, in most cases, the triggering environmental factor remains unknown. The study of experimental models, including insulin-dependent diabetes and atopic dermatitis in mice, shows that the occurrence of these dysimmune diseases does not necessarily depend on exposure to a given antigen. The problem is complicated by the fact that the environment often has a protective rather than a triggering role. A correlation has been observed, over the last decades, between the decline of certain infectious diseases and the increase in autoimmune and allergic diseases. The causal relationship between these two observations has been directly demonstrated in animal models and indirectly in man. The mechanisms underlying the protective effect of infections on autoimmune and allergic diseases are becoming better known with in particular the involvement of antigenic competition regulatory T cells and Toll-like receptors.

The situation has recently evolved with the demonstration that autoimmune and allergic diseases are often associated with abnormal composition of the intestinal microbiota. Then, hygiene conditions taken in the wide sense affect the gut microbiota as previously shown for occurrence of infections. The problem is posed of the respective role of commensal and pathogenic bacteria in dysimmune diseases. A possible common mechanism could be TLR stimulation by bacterial ligands contained in both types of bacteria.

It is hoped that a better understanding of these genetic and environmental phenomena will better explain the nosology and pathophysiology of these diseases. They should also allow the discovery of new treatments that will be more then just symptomatic and could, at best, generate a new avenue for the prevention of these diseases which now represent a major health burden.

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Time: 4:30 pm

Venue: Peter Ho Lecture Theatre (LT10)
4/F, Academic 1
City University of Hong Kong

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