PHILOSOPHY

I LIKE DISCUSSIONS

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Just recently I cam across a long forgotten CIBA Foundation Symposium and they had some nice discussion printed there – conserved for eternity while I think that all these re-searchgate whatever forums are volatile, yea, yea.

most people do not produce IgG. It may be different if a person is heavily exposed to the allergen.

Turner: In this group in New Guinea the density of mites in the immediate environment (in the blankets, which often cover the head) close to the respiratory tract is very high. People are probably exposed to a higher dose of mite allergens locally than we would be from our bedding. That may be enough to induce significant IgG responses to mites. The lack of concomitant IgE may be related to parasitization, perhaps by antigen competition, in infants who are more heavily parasitized than adults. The problem then is why they produce IgG responses.

Ring: What is so unusual about the Western Caroline Islands, with 75% of children suffering from asthma?

Turner: Asthma there was well defined by respiratory function tests and case history, and the death rate in childhood from asthma is high. Nobody has looked further at that situation, or defined any allergen, so I can't answer you.

Finkelman: Are there technical problems in measuring the specific IgE response against, say, the house dust mite if there is also a large IgG antibody response to the same antigen and a very large total IgE concentration?

Turner: RAST and other radioimmunoassay systems have been criticized on the basis that IgG antibody may inhibit the read-out system for IgE antibody. We think not, because the adult asthmatics have incredibly high mite IgE antibody scores, yet their IgG antibody scores are just as high.

Finkelman: But the polyclonal IgE levels have come down?

Turner: Yes. That is interesting too, because the atopic adult in the tropics will produce a higher level of total IgE than other corresponding non-atopics also infected with parasites. Therefore, superimposed on the IgE polyclonal response, which all subjects with helminthic infections express, is a genetic control of IgE production. I would not have expected to be able to differentiate between these IgE responses in terms of atopic versus polyclonal stimulation, but you can.

Galli: Are there any experimental data in animals or humans to indicate, if one has to be exposed to a certain amount of antigen, whether it would be better to be exposed to small amounts of many different antigens or to large amounts of a few antigens? This issue may have some clinical relevance. For example, the distribution of species of flowering plants in the world is very uneven; the number of species in the tropics is vastly greater than in most parts of the developed world. To trigger effector cells one needs two IgE molecules of

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