ALLERGY, PHILOSOPHY

WORMY WORLD

23.04.2007

Allergy (or at least an associated trait) may have its roots somewhere in Africa – where helminth infections are frequent. A new Nature Immunol Review has an overview but I am quite disappointed. From the abstract

> There is no immunological mechanism to adequately explain the sudden epidemic in allergies noted in the last 30 years in developed countries.

I can recommend to read a recent PLoS paper with references to several immunological mechanisms.

> The reduction in the development of allergic disorders observed in individuals infected with parasitic helminths, however, supports a possible role for worms in suppressing allergies.

Has anyone ever infected a larger number of individuals with parasitic helminths and found a clear suppression of allergy?

> Helminths regulate the immunity of the host to ensure a mutually beneficial environment for the survival of both the parasite and the host.

I do not believe that helminths take over and regulate the immune system of the host.

> This interplay between helminths and allergic responses raises fundamental questions in immunobiology. Harnessing current mechanistic studies for translational research into helminth infections and atopy might have potential for the identification of novel biomarkers, and even therapeutics, in allergic diseases.

that may be true, there are fundamental questions... Check out the rest of the paper, I have stopped at the famous “genetically similar East and West Germans” hoax on the first page. At least do not miss ref. 36, a nice meta-analysis of earlier studies.

Addendum

The chitinase story goes on. Published first in Science 2004

> Chitin is a surface component of parasites and insects, and chitinases are induced in lower life forms during infections with these agents. Although chitin itself does not exist in humans, chitinases are present in the human genome. We show here that acidic mammalian chitinase (AMCase) is induced via a T helper-2 (Th2)â€“specific, interleukin-13 (IL-13)â€“mediated pathway in epithelial cells and macrophages in an aeroallergen asthma model and expressed in exaggerated quantities in human asthma.

there is an update in Nature yesterday

> chitin induces the accumulation in tissue of IL-4-expressing innate immune cells, including eosinophils and basophils, when given to mice. Tissue infiltration […] did not occur if the injected chitin was pre-treated with the IL-4- and IL-13-inducible mammalian chitinase, AMCase4, or if the chitin was injected into mice that overexpressed AMCase.

There is already a cross-link to human gene variation in chitinase (not cited by the authors)

> the newly identified amino acid exchange lysine to arginine on position 17 showed association with asthma. It is located only seven amino acids in front of the catalytic center of AMCase. It is therefore plausible that this amino acid exchange may modulate the catalytic activity of AMCase. An arginine-to-lysine substitution may be considered conservative, but there are examples of substantial modulation of activity by this substitution in enzyme systems.

on the other hand chitin is nothing specialof chitin. As the authors of the new Nature paper acknowledge chitin is the second-most abundant bioplymer in nature with “estimates of billions of metric tons produced annually in oceans alone”.