Commentary: The defence of dirt

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As the Duchess of Windsor once remarked, one can never be too thin or too rich—wisely, she did not add too clean. The hygiene hypothesis aims to explain why some people have allergies and some do not, and why the prevalence of allergic disorders has been increasing over the past century. It suggests that the modern obsession with cleanliness may be counterproductive; in childhood, at least, it may encourage the development of allergic disorders. Benn and colleagues have attempted to untangle the link between infections in infancy and atopic disease.[1]

Formalised in the late 1980s, the hypothesis seemed to fly in the face of common sense. After all, hadn't the hygiene movement ushered in a dramatic reduction in infectious disease? This initial scepticism was compounded by the suspicion that infections might actually provoke allergy. Despite these doubts, the evidence kept piling up.[2] In epidemiological studies, factors such as large household size, a poor standard of living, not using antibiotics, and early exposure to farm animals all emerged as protective.

The original attempt to explain the immunological basis of the hygiene hypothesis invoked a loss of balance between two sets of the body's immune cells—the Th1 and Th2 lymphocytes.[3] A reduced exposure to childhood infection, it was argued, meant a low level of Th1 activity in the body, so bringing about a relative excess of Th2 activity—and a consequent tendency to allergy. This neat explanation began to fall apart when it was appreciated that Th1-mediated conditions, such as type 1 diabetes and inflammatory bowel disease, were also on the increase, and in the same regions of the world.

Clearly, any reinterpretation of the hygiene hypothesis had to take account of this parallel increase in conditions associated with Th1 and Th2. A recent attempt manages to do just this.[4] It suggests that the effect of hygiene is to diminish the body's production of a third group of players in the immune drama—the regulatory T cells. It is a shortage of these that results in the emergence of allergy. The driving force in this case, goes the argument, is our freedom not from pathogenic microbes but from a group of organisms including mycobacteria, lactobacilli, and helminth worms. These "old friends," as Rook describes them,[4] have lived with us for countless generations. In adapting to their more or less benign presence, the immune system has learned not to over-react. It exercises this self suppression by generating regulatory T cells.

In the absence of its "old friends" the system produces fewer of these cells. The consequence is a state of relatively uncontrolled effector T cell exuberance and, depending presumably on genetic factors of some kind, a predisposition to allergy or to more serious autoimmune conditions such as inflammatory bowel disease and diabetes. The findings reported by Benn and colleagues,[1] which refer only to "clinically apparent" infections, are entirely consistent with this view of the "old friends" being the organisms responsible for the protection against allergy.

Does this version of the hygiene hypothesis suggest a method of vaccination against allergy based on stimulating the body's production of regulatory T cells? It does, and preliminary experiments are already under way.[5] Immunology's love affair with dirt is blossoming and may yet bear fruit.

Competing interests: None declared.

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