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Commentary

Mechanisms of Allergic Rhinitis and Advantages in Probiotic Medicine

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DESCRIPTION

Several mechanisms are probably involved in both tolerance and sensitization because allergy is not a single illness. This explains why associations between genes and diseases are so elusive. However, the results of research that have discovered links between gene alleles and allergy disease have limits. This may be because of inadequate definitions of sickness and health, restrictions in the selection of single-nucleotide polymorphisms, or improper segregation and interpretation.

Studies on mice have shown that oral probiotics can reduce allergic reactions in the lower respiratory tract and that antibiotics can boost allergic airway responses by changing the intestinal microbiome. However, probiotics have mostly been employed for prophylaxis through neonatal supplementation as mucosal immune modulators targeting asthma outcomes or characteristics. Certain probiotic strains may alleviate asthma symptoms such airway eosinophilia, local cytokine responses, and bronchial hyperresponsiveness when given to adult mice. In light of this, some researchers have looked at the potential of probiotic therapy in treating human asthma.

Pediatric rhinitis quality of life has significantly decreased to the use of fermented milk fortified with *L. paracasei* LP33 in the treatment of Perennial Allergic Rhinitis (PAR) in children. A difference in the cumulative incidence of rhinitis episodes was discovered in a trial of preschoolers given milk fermented with 1010 *cfu L. casei* or a placebo for 12 months. In a further research, a small group of children aged 6 to 12 with asthma and allergic rhinitis received either *L. gasseri* A5 supplements or a placebo. The treated group experienced a reduction in the rhinitis symptom score. Ultimately, a specific form of PAR was tested in amateur marathon runners, however supplementing with *L. rhamnosus* GG had no beneficial effects.

In vitro IgE tests and *in vivo* testing, most frequently skin prick tests, can be used to demonstrate atopic sensitization, or the generation of allergen-specific IgE antibodies. Unfortunately, the sensitivity and specificity of various commercial brands of *in vitro* tests for IgE detection vary. The sensitivity of commercial tests has also improved over time. In other instances, both the anti-IgE antibodies for IgE detection and the allergen source materials attached to the solid phase have been altered. However, the outcome of a skin prick test can vary based on the method employed as well as the strength and makeup of the allergen extracts. Thus, it is necessary to define these techniques in their context.

The only accurate way for comparing the rate of sensitization within and between laboratories over time is to evaluate all samples using *in vitro* IgE testing with the same brand and batch of antigen. When the same extracts from the same manufacturer are used, skin test results can be compared more accurately, however the clinician's technique is more difficult to standardise. It is impossible to compare studies that estimate allergen-specific sensitivity in different ways.

The same idea that underlies the terminology for allergies also holds true for allergic illnesses. As a result, "allergic asthma" can be split into "IgE-mediated asthma" and "non-IgE-mediated asthma" (i.e., asthma with a plausible or proven immunological mechanism). Contrast the latter, which involves cells, with "non-allergic asthma," which describes asthmatic diseases without immune system involvement (if they exist). Similarly, "IgE-mediated food allergy" and "non-IgE-mediated food allergy" should be distinguished.

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