

Diet and allergy: You are what you eat?

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Key words: Diet, nutrition, allergy

Nutrition defines the process by which an organism assimilates food for normal growth and maintenance of the balance between health and disease. In the past few decades, the prevalence of atopic diseases, such as hay fever, asthma, eczema, and food allergy, has increased.^{1,2} It has been speculated that dietary changes and modifications in consumer habits influence the host early in life and thereby contribute to this trend. Therefore increased importance has been attached to the philosophy “you are what you eat” (Fig 1).

This is especially true for the state of malnutrition, which initiates a vicious circle leading to an impaired host defense combined with increased susceptibility to infections and further aggravation of malnutrition caused by enhanced nutritional requirements during infection.³ The major mechanism underlying this process is that inflammation induces a high number of oxygen free radicals. Therefore inflammation can be controlled by the supplementation of antioxidant enzymes.⁴ The mechanisms by which different types of malnutrition, such as malnutrition caused by genetic disorders, protein-calorie-associated malnutrition, or secondary malnutrition syndromes of childhood, cause immune deficiencies and the clinical pictures resulting from an imbalance of nutrients are nicely summarized in the review article by Cunningham-Rundles⁵ in the current issue of the Journal. In addition, the author describes the manner in which the deficiency of micronutrients, such as antioxidant enzymes (eg, copper and zinc), or combined deficiencies of proteins, vitamins and minerals modulate cytokine production, leading to impaired phagocytic activity and T-cell dysfunction. The author reports that the effects of primary malnutrition can be restored by supplementation of adequate nutrients,

whereas the cause of secondary malnutrition has to be abolished first before nutritional imbalance can be cured.

Relevant to atopic diseases, the effect of nutrition on the manifestation and severity of atopic disorders has been carefully reviewed in several studies.⁶ In this regard 2 different approaches, an elimination diet aimed at omitting causative nutrients on the one hand and a supplementary diet adding protective nutrients on the other hand, have been studied as preventive measures for various atopic diseases.

A central issue that has been raised by all of these studies is the optimal time period of life in which the different strategies might be most effective. However, the correct answer to this question would require a better understanding of the natural history of the atopic march. Furthermore, identification of the critical points of vulnerability for atopic sensitization in combination with identification of the specific subgroups of atopic patients that would have the greatest benefit from the different dietary strategies would be of enormous importance.

The most important and initial question frequently addressed to physicians during the first phase of the atopic career of an individual at risk is whether breast-feeding prevents or promotes the development of atopic diseases. In the current issue, Friedman and Zeiger⁷ provide an overview about the multitude of answers frequently given to this question. In their review entitled “The role of breast-feeding in the development of allergies and asthma,” they consider studies showing the beneficial effects of breast-feeding, studies showing that breast-feeding promotes the development of atopic diseases, and studies that conclude that breast-feeding does not have any influence on the development of atopic disorders. Taken together, the contradictory data reported by studies examining the role of breast-feeding might be the result of methodologic differences involving sample size, lack of blinding, duration of breast-feeding, and the presence of maternal avoidance diets during lactation. In addition, the complexity of atopic diseases themselves contribute to the heterogeneity of the results mirrored by different genetic backgrounds and familial risk of each individual, the effect of protective antibodies against bacterial and viral infections transferred by breast milk, or other variables, such as the type of atopic disorders studied.⁸ In summary, they conclude that the question of whether breast-feeding should be recommended should

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Disclosure of potential conflict of interest: all authors—none disclosed.

Received for publication March 24, 2005; accepted for publication April 4, 2005.

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J Allergy Clin Immunol 2005;115:1235-7.
0091-6749/\$30.00

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doi:10.1016/j.jaci.2005.04.004

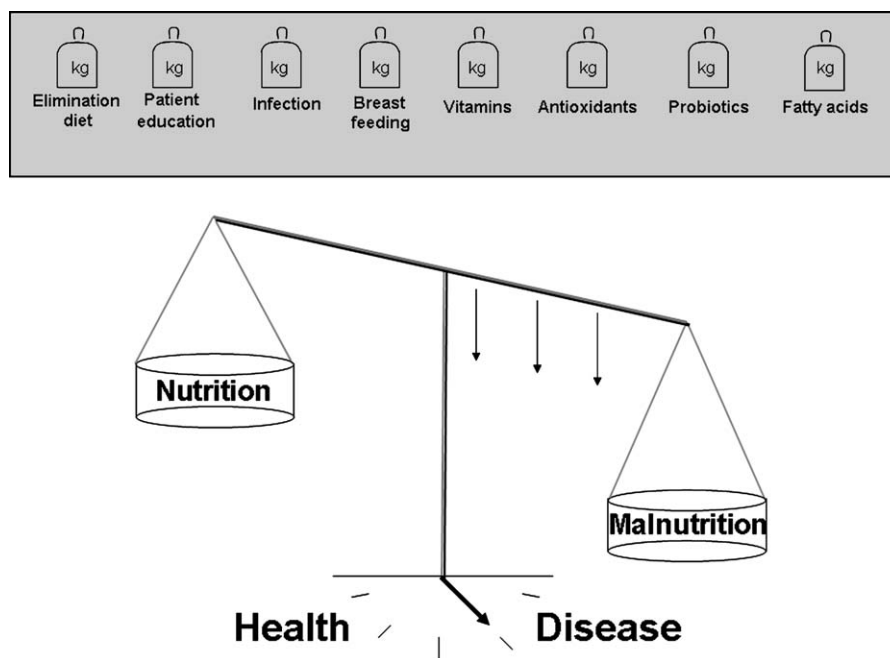


FIG 1. The balance between nutrition and malnutrition and the factors that preponderate on the outcome of health and disease in this interplay are shown.

be replaced by a reliable risk-benefit analysis, keeping in mind the critical consideration on what would be lost by not recommending breast-feeding.

Another issue that has been debated with similar intensity is the hygiene hypothesis, proposed more than 2 decades ago, which postulates a weaker influence of bacterial and viral components that promotes the development of T_H1 immune responses. As a result, tolerogenic and protective pathways might contribute to the retention of a T_H2 predominance as the basis for progression of the atopic career.⁹⁻¹¹ In this context dietary approaches have been conducted to pose the question of whether probiotics might serve as antiatopics. The main principle of the probiotic strategy is to alternate nutrition from passive elimination diets to an active stimulation, resulting in maturation of the immune system achieved by the supplementation of probiotic microbes.^{12,13} In the review entitled "New therapeutic strategies for combating the increasing burden of allergic diseases: probiotics," Isolauri et al¹⁴ comprehensively describe the complex interaction of different probiotic strains with the gut epithelium and the complex intestinal homeostasis resulting from the contacts of probiotics with respective pattern recognition receptors in the gastrointestinal epithelium. Furthermore, they call our attention to the variety of host-microbe cross-talks and the multitude of evolving immune responses related to different probiotic strains or mixtures of these strains. In addition, they focus on the diversity of effects achieved during particular periods of life and in different diseases, pointing to the need for age- and disease-specific probiotics adapted to the individual risk profile. They also suggest that genetic differences associated with atopic diseases might lead to modified responses

to different probiotics in atopic patients and should be considered to optimize these approaches. For example, polymorphisms in gene regions of pattern recognition receptors in our immune system, such as toll-like receptors 2 and 4 or the caspase recruitment domain binding protein, which have been found to be associated with atopic traits, might be of critical importance.^{15,16}

Another important issue that must be addressed is whether changes in our eating habits might contribute to the increasing incidence of atopic diseases and, if so, whether dietary intervention strategies would be able to modulate this process. In the review "Diet as a risk factor for atopy and asthma," Devereux and Seaton¹⁷ discuss studies on the effect of increasing dietary intake of n-6 polyunsaturated fatty acids on the one hand and the decreasing use of n-3 polyunsaturated fatty acids on the other hand, which might be one major nutrition-related factor contributing to the predominance of T_H2 immune responses and proinflammatory factors, such as prostaglandins, in atopic patients.

An inverse correlation of vitamin C, vitamin E, vitamin A, and selenium levels in the blood of atopic patients with parameters of atopic diseases such as serum IgE levels or FEV₁ in asthmatic subjects has also been observed. Although intervention strategies on the basis of dietary programs, such as the supplementation of antioxidative vitamins, show promising results, there is still a need for further randomized controlled trials because most of the past studies have reported equivocal results. The complexity of the issue of a nutritional effect on atopic disorders is mirrored in a recent research study that shows that simple modification of the gastric pH achieved by the intake of H₂-receptor blockers and proton pump inhibitors

increased significantly the risk for the development of sensitizations against digestion of labile food components.¹⁸ It is obvious that long-term follow-up studies have to consider a multitude of factors interacting with the development of sensitization in atopic individuals.¹⁹

Despite the need for intensive nutritional consulting of patients with food allergy in whom ingredients and certain types of food must be avoided and in whom preventive components should be supplemented in patients at risk, patient education strategies aimed at changing the behavior of patients become increasingly important and should be integrated into common therapeutic schemes. In the review "Challenges in asthma patient education," Cabana and Le²⁰ provide an overview about recent developments in the field of asthma education programs and strategies to increase the effectiveness and reduce the costs of such strategies. One example of such a model would be to perform group sessions for asthma education instead of individual training. In addition, they address the question of who should perform this education program, what type of training should be chosen, how it should be adapted to cultural differences, and in which way new technologies could be integrated.

Taken together in view of recent developments in the field of nutrition and diet, detailed nutritional counseling aimed at improving disease management and quality of life in atopic patients should be considered. However, the complex pathophysiologic background of atopic disorders must be kept in mind. As such, adequate diet and nutrition strategies might not completely prevent, but could hopefully attenuate, the atopic career of patients at risk for a lifetime of allergy.

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