



Short Communication

The concept of oral tolerance induction to foods

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Introduction

In the UK, US, and Canada, peanut allergy (PA) in early childhood is estimated at between 1 and 2% and food allergy (FA) at between 4 and 8%. The pathogenesis of FA is unclear and a common assumption is that early introduction of food allergens in infancy or during pregnancy leads to the development of FA. Thus avoidance of foods in early infancy, delayed introduction of solids, prolonged breast feeding and even dietary manipulation of the maternal diet have been proposed to reduce the development of FA.

These avoidance policies have been utilized in interventional trials in early childhood but have failed to demonstrate significant effects [1] in reducing IgE mediated hypersensitivity to foods. One explanation is that allergen avoidance was not sufficiently stringent. An alternative explanation is that early allergen exposure may not lead to allergy but could lead to tolerance. It has recently been suggested that sensitization to peanut is a post-natal event and rather than being caused by peanut consumption in early childhood or during pregnancy, it may be linked to low levels of cutaneous exposure [2].

More recently it has been proposed that allergic sensitization to food occurs through low dose cutaneous sensitization and that early consumption of food protein induces oral tolerance [3]. Oral tolerance in murine models is recognized. Many studies have shown that early high-dose oral exposure leads to immunologic and clinical tolerance.

In a cross-sectional study [4] the route of peanut exposure in the development of allergy was assessed. Maternal peanut consumption during pregnancy, breast-feeding, and in the first year of life was captured using a questionnaire. Additionally, all household peanut consumption was quantified. The median weekly household peanut consumption in the patients with PA was significantly increased (18.8 g) compared with control subjects without allergy (6.9 g) and high-risk control subjects (1.9 g). These findings suggest that high levels of environmental exposure to peanut during infancy can promote sensitization. This supports the hypothesis that peanut sensitization occurs as a result of environmental exposure.

The rate of PA is higher in industrialized societies where peanuts are avoided in pregnancy and infancy [5]. In countries where peanut is

consumed in high amounts during infancy (Middle East, Southeast Asia, Africa), PA is reportedly rare [6,7].

In a cross-sectional study among Israeli ($n = 55,615$) and UK ($n = 55,171$) Jewish children, the prevalence of PA was 10 times higher in the UK (1.85%) than in Israel (0.17%, $P < .001$) [8]. This difference is not explained by differences in atopy, social class, genetic background, or peanut allergenicity and raises the question whether early introduction rather than avoidance of peanut during infancy prevents the development of PA. This study found that peanut is introduced earlier, eaten more frequently and in larger quantities in Israel than in the UK.

Studies are now underway to see whether oral tolerance to peanuts can be induced by early consumption in infancy [9] and whether complementary feeding at 3 months of life in infants who are breast feeding can prevent the development of FA [10].

References

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