Peanut allergy: routes of pre-natal and post-natal exposure

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Background

In June 1998 the Department of Health (DH) published recommendations aimed at halting the rising incidence of peanut allergy. This guidance was based on the conclusion that peanut sensitization occurring as a result or exposure in utero or via lactation was mechanistically possible.

Most presentations of peanut allergy occur on the first known contact the child has had with peanut. The route by which sensitisation occurs is unclear. The possibilities are that sensitization is occurring in utero, via breast milk or via indirect low dose environmental exposure. Much work has focused on maternal consumption of allergen (during pregnancy or lactation) yet interventional studies have failed to demonstrate any benefit of dietary elimination. Recent data is supportive of the possibility of sensitization through low dose cutaneous exposure as a result of the application of arachis oil containing creams to inflamed skin.

This study aims to quantify the exposure to environmental allergen during the allergic child's infancy. Environmental peanut exposure can occur through a variety of ways as well as the application of peanut-containing creams. Other important environmental components include the peanut consumption of all household members and the cutaneous contact and vapour inhalation that can result from this.

If sensitisation is occurring through environmental exposure, this has important implications for current DH guidance on peanut avoidance and future allergy prevention studies.

Approach and results

The researchers adopted a retrospective, case-controlled, observational cohort approach to investigate low dose environmental exposure to peanut as a possible route of allergic sensitisation and the development of peanut allergy in young children. A validated questionnaire was developed and used to retrospectively quantify the individual child’s dietary consumption of peanut during their first year of life, as well as the total household weekly consumption of peanut by all family members (environmental exposure). Other relevant information, such as maternal consumption of peanut during pregnancy, and whether peanut containing creams were used, was also collected. The questionnaire was applied to families before their child’s peanut allergy had been diagnosed to avoid recall bias.

Results were collected on 133 children with peanut allergy, 150 children with no food allergies (controls) and 160 children with egg allergy (but not sensitive to peanut). Egg allergic children were included in the study as they are considered at high risk (30-50%) of developing peanut allergy, therefore studying those egg allergic children who did not develop peanut allergy may
provide clues as to what factors prevented them from becoming peanut allergic.

Median household peanut consumption during the first year of the child’s life was found to be significantly higher in peanut allergic children (77.2g/week) compared with the non-allergic controls (29.1g/week) and the egg-allergic (but not peanut allergic) children (8.1g/week). This relationship was found to be independent of the child’s own level of consumption of peanut. These results suggest that higher environmental exposure to peanuts during early life in the families of those children who went on to develop peanut allergy, may have promoted the development of peanut allergy.

Further, the even greater differences seen between the level of household consumption in peanut allergics compared with egg-allergics, suggests that the very low levels of household peanut consumption in the egg-allergic families, protected these high-risk children from becoming allergic to peanut. The results have also suggested that the form that the peanut was consumed in by household members is important in terms of risk, with the consumption of peanut butter associated with a greater risk of peanut allergy than consumption of other types of peanut containing food.

The researchers hypothesise that environmental exposure to peanuts through the skin or via inhalation, might be a significant factor in the development of sensitisation, as distinct from maternal or infant consumption of peanut. Their findings also raise the question that early oral exposure may play an important role in the development of tolerance to peanut.

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