

BRIEF COMMUNICATION

Peanut allergen in house dust of eating area and bed – a risk factor for peanut sensitization?

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Abstract

It has been hypothesized that high environmental exposure to peanut allergens may be a potent risk factor for cutaneous sensitization. Therefore, we wanted to investigate whether peanut proteins are detectable in house dust of different household areas. Peanut levels of dust samples were measured with ELISA. Overall, peanut was detectable in 19 of 21 households in the eating area and/or in bed. The frequency of peanut consumption correlated with peanut levels. Forty-eight hours after intentional peanut consumption, peanut levels were highly increased. Nevertheless, further research is required to prove whether peanut allergen in house dust can cause sensitization via skin.

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A high consumption of peanut in the household during infancy was found to be a possible risk factor for the development of peanut allergy (1). As sensitization to food allergens is often found in infants with moderate to severe atopic dermatitis, cutaneous exposure to food allergens seems to be an important way of sensitization (2, 3). Thus, high environmental exposure to peanut allergens in the household especially in the infant's bed may be a potent risk factor for cutaneous sensitization especially in young children with atopic dermatitis. In addition, it has been hypothesized that a permanent exposure of small amounts of food protein to the Langerhans cells can lead to an immune response (3). However, whether and to what extent peanut allergens are present in the household is not yet known. As house dust is easily distributed in all domestic areas, we wanted to investigate whether peanut proteins are detectable in house dust of different household areas and whether peanut levels correlate with the peanut consumption in the household.

Methods

We report on the results of 21 voluntary households that were included in the study. Overall, four single-person households

and 17 more-person households located in the town of Berlin were recruited. All participants completed a questionnaire regarding food frequency of peanut and peanut-containing products (listed as examples) over the last 4 weeks (never, at least one time/month, 1–2 times/week, and >3 times/week), habitual eating area of these products, and cleaning habits. House dust samples were collected with a vacuum cleaner using a special device (MITEST dust collector; Indoor Biotechnologies LTD, Cardiff, UK). Dust samples were taken in the habitual eating area (kitchen or dining room) and from bed sheets. All dust samples were taken in December 2012. Families were asked not to change their regular peanut consumption before dust samples were taken for the first time. Peanut protein was extracted from dust samples, and peanut levels were measured with a commercially available ELISA (RIDASCREEN®FAST peanut; R-Biopharm, Darmstadt, Germany) coated with polyclonal antibodies against peanut extract. The range of quantification was 2.5–20 µg/g dust (detection limit 1.5 µg/g dust). Samples with peanut levels above the range of the standard curve were further diluted. In households with low peanut levels at baseline, participants were asked to consume roasted peanut snacks in their habitual eating area. Dust samples were collected 48 h after peanut

consumption in the eating area and from bed sheets. Spearman's rank correlation coefficient was calculated to evaluate the relation between peanut levels and peanut consumption. Wilcoxon rank test was used to compare peanut levels at baseline and 48 h after intentional peanut consumption.

Results

Overall, peanut was detectable in 19 of 21 households (91%) in a wide range, and only in two households (household numbers 20 and 21), peanut levels were below the detection limit (Fig. 1). In 16 of 21 households (76%), peanut was detectable in the eating area (range 0–2178.5 µg/g dust). On bed sheets, we found peanut in 19 of 21 households (91%) (range 0.23–1154.4 µg/g dust). The value of one dust sample (number 8) was extrapolated due to insufficient sample volume for further dilution. Within the households, amounts of peanut in dust samples correlated moderately between eating area and bed sheets ($r = 0.474$, $P < 0.05$) (data not shown). When we compared the frequency of household peanut consumption to peanut levels in the house dust, we found a moderate positive correlation between peanut consumption and peanut levels in the eating area ($r = 0.559$, $P < 0.01$) (data not shown). There was no significant correlation between peanut consumption and peanut levels on bed sheets. Overall, eight participants declared not to consume any peanut or peanut products in their household over the last 4 weeks. Nevertheless, in these 'peanut-free' households, peanut was detectable in house dust samples of five of eight eating areas and six of eight bed sheets.

In 10 households with low peanut levels at baseline (range 0–14.4 µg/g), a second collection of dust samples was performed 48 h after roasted peanut snacks were consumed

in the habitual eating area. One dust sample of one eating area was not available for the analysis. In nine of 10 households, we could measure highly increased peanut levels after peanut consumption in the eating area (median at baseline 3.1 µg/g, median 48 h after peanut consumption 247.7 µg/g (range 0.1–2200 µg/g), $P < 0.05$) as well as on bed sheets (median at baseline 3.2 µg/g, median 48 h after peanut consumption 18.1 µg/g (range 1.3–986.2 µg/g), $P < 0.05$), whereas in one household, peanut levels remained stable (Fig. 2). Values of two dust samples of eating areas after peanut consumption were extrapolated, and no further dilution was possible due to little sample volume.

Discussion

There are few studies investigating the detection of environmental exposure to food allergens (4–7). Two studies detected peanut on tabletops and hands after the consumption of peanut products (4, 6). However, regarding the persistence of peanut allergens after using common cleaning agents, they showed different results. In our study, peanut was found in the house dust, not only in household areas where peanuts are habitually eaten but also in other areas such as in bed where peanut is usually not consumed, indicating a spreading of food allergens within the household. As infants and young children spend most of the time in bed, peanut containing house dust on bed sheets could be considered as an important risk factor for environmental sensitization. We found that peanut consumption is positively correlated with peanut levels in the eating area but not on bed sheets, and this could be due to several factors such as distance from the eating area to the bed room or washing hands after peanut consumption.

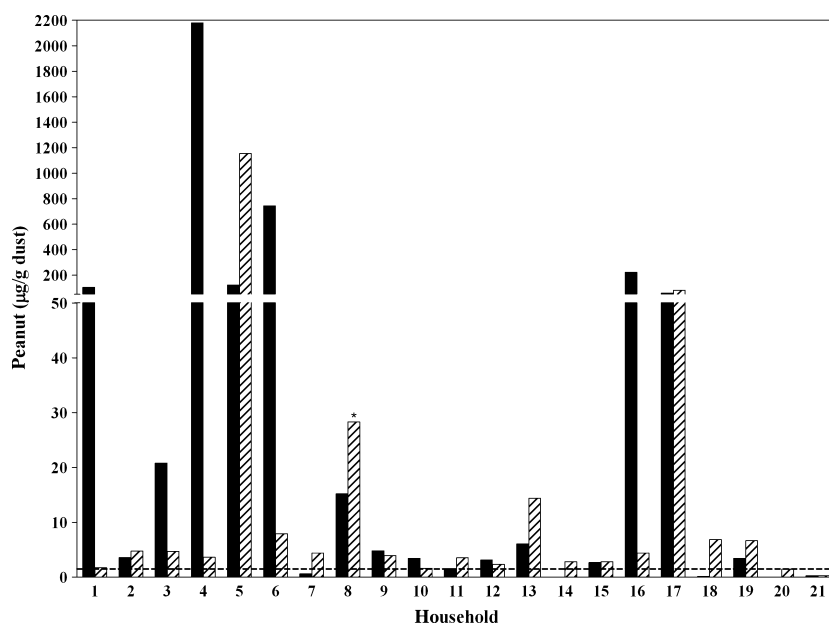


Figure 1 Peanut levels in dust samples of 21 households (black bars = eating areas; striped bars = bed sheets). 1.5 µg/g = detection limit. *Extrapolated value.

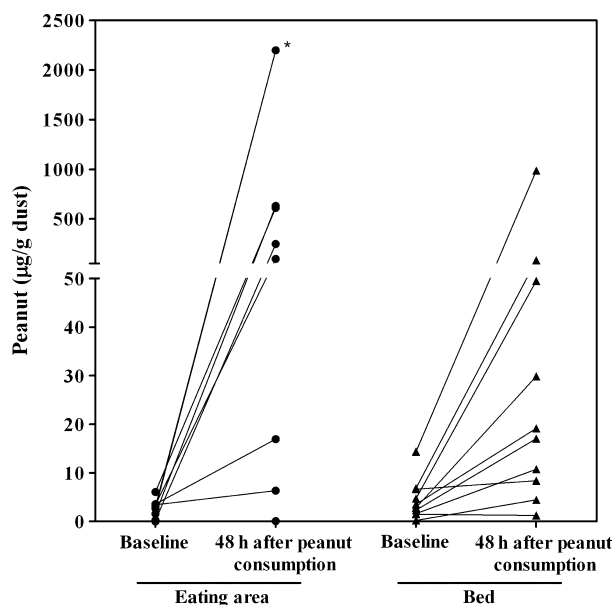


Figure 2 Peanut levels in dust samples at baseline and 48 h after intentional peanut consumption in the eating area ($n = 9$) and on bed sheets ($n = 10$). *Two identical peanut levels of eating areas of two households, extrapolated values.

Most recently, two studies showed similar results regarding the distribution of peanut within the household and the correlation of peanut consumption with peanut levels (6, 7). Furthermore, they demonstrated that peanut protein in house dust is biologically active and seems to be transferred by saliva and hands into the environment. Nevertheless, further

research is required to prove whether peanut allergen in the house dust can cause sensitization via cutaneous contact or inhalation (8).

Furthermore, our analysis reveals that even when patients are not aware of eating peanuts or peanut products at home, peanut is still found in their households. This could be due to the fact that people are often not aware of consuming peanut-containing products, especially regarding hidden allergens. In the regarding households, cereals and pastries could be identified as potential sources of hidden allergens. This should be considered when interpreting results of food frequency questionnaires regarding the consumption of hidden allergens.

In summary, peanut allergens are detectable in house dust of eating area and bed. The frequency of household peanut consumption seems to correlate with peanut levels. This supports the hypothesis that environmental peanut exposure could be a risk factor for sensitization via skin or trough airways.

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Conflict of interest

The authors have no conflict of interest to declare.

Declaration of funding

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