Distribution of peanut allergen in the environment

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Background: Patients with peanut allergy can have serious reactions to very small quantities of peanut allergen and often go to extreme measures to avoid potential contact with this allergen.
Objective: The purpose of this study was to detect peanut allergen under various environmental conditons and examine the effectiveness of cleaning agents for allergen removal.
Methods: A monoclonal-based ELISA for *Arachis hypogaea* allergen 1 (Ara h 1; range of detection, 30-2000 ng/mL) was used to assess peanut contamination on cafeteria tables and other surfaces in schools, the presence of residual peanut protein after using various cleaning products on hands and tabletops, and airborne peanut allergen during the consumption of several forms of peanut.
Results: After hand washing with liquid soap, bar soap, or commercial wipes, Ara h 1 was undetectable. Plain water and antibacterial hand sanitizer left detectable Ara h 1 on 3 of 12 and 6 of 12 hands, respectively. Common household cleaning agents removed peanut allergen from tabletops, except dishwashing liquid, which left Ara h 1 on 4 of 12 tables. Of the 6 area preschools and schools evaluated, Ara h 1 was found on 1 of 13 water fountains, 0 of 22 desks, and 0 of 36 cafeteria tables. Airborne Ara h 1 was undetectable in simulated real-life situations when participants consumed peanut butter, shelled peanuts, and unshelled peanuts.
Conclusion: The major peanut allergen, Ara h 1, is relatively easily cleaned from hands and tabletops with common cleaning agents and does not appear to be widely distributed in preschools and schools. We were not able to detect airborne allergen in many simulated environments. (J Allergy Clin Immunol 2004;113:973-6.)

Key words: *Food allergy, peanut allergen*

Peanut allergy is an enormous clinical problem. It is the third most common food allergy in young children[1] and the most common food allergy in older children, adolescents, and adults.[2] In addition to its substantial prevalence, it is the food allergen most capable of causing severe, life-threatening, and even fatal allergic reactions.[3,4] The diagnosis of peanut allergy therefore carries tremendous medical and emotional significance.

Abbreviation used
Ara h 1: *Arachis hypogaea* allergen 1

Because avoidance is the only available treatment for food allergy at this time, patients with peanut allergy must take extraordinary care to eliminate all peanut-containing foods from the diet. This is far more difficult than it sounds, especially because of the cross-contamination of foods that may occur in the manufacturing process. In addition to the obvious goal of avoiding peanuts in the diet, another key issue facing patients with peanut allergy and their families involves other potential sources of accidental exposure. Inadvertent exposure has been reported to occur in environmental settings such as restaurants,[5] schools,[6,7] and other public places—for instance, sporting events and commercial airline flights.[8] Although these reactions are presumed to occur by exposure through skin contact or inhalation of airborne allergen, in most of these reports, accidental ingestion of peanut could not be entirely ruled out. A recent study by Simonte et al[9] reported that casual contact or inhalation of peanut butter does not pose a significant risk for severe reactions, suggesting that many of the reports of casual contact or inhalation reactions may in fact be caused by inadvertent ingestion.

The purpose of the current study was to determine the prevalence and levels of exposure that may be encountered in home and school settings and under several simulated environmental conditions. We included environments such as those present in homes and public eating areas as well as those that may be present at sporting events or during commercial airline flights.

METHODS

Subjects

Participants included 19 adult volunteers without peanut allergy. Participants consumed various forms of peanut while wearing personal air monitors and cleaned peanut butter off their hands by using a variety of common cleaning agents. The study was approved by the Johns Hopkins Hospital Institutional Review Board.

Table and surface wipe samples

One teaspoon (5 mL) peanut butter was applied to a 12-in × 12-in area of a clean table. By using a 37-mm glass fiber filter moistened with extract solution (PBS and 1% Tween-20 [Sigma-Aldrich, St Louis, Mo]), wipe samples were obtained before and after cleaning with various cleaning agents or plain water. The area was allowed to air dry before the postcleaning sample was taken. Cafeteria tabletops

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TABLE I. Results of table wipe samples*

Cleaner	No. detectable	Detectable range (ng/mL)
None (n = 5)	5/5	720-6.3 × 10^4
409 Cleaner (n = 12)	0/12	BD
Target cleaner with bleach (n = 12)	0/12	BD
Lysol wipe (n = 12)	0/12	BD
Dish soap (n = 12)	4/12	40-140
Water (n = 12)	0/12	BD

BD, Below detection.
*Areas 12 × 12 in were coated with 1 teaspoon of peanut butter, then cleaned using various cleaning solutions or plain water.

and other surfaces including desktops, water faucets, and food preparation areas were sampled at local schools and preschools. Cafeteria table samples were taken just after lunch and before cleaning from tables that included persons who consumed peanut products and from peanut-free tables.

Hand wipe samples

Approximately 5 mL peanut butter was applied to the hands of volunteers, and some samples were taken before hand washing. Persons were then asked to wash their hands by using their normal hand washing techniques with various cleaning agents or plain water. Participants were also asked to clean their hands with a nonsoap antibacterial hand sanitizer after the application of 1 mL peanut butter. Participants were not instructed on specific hand washing techniques but were told to wipe or wash their hands as they normally would to remove the peanut butter. After hand washing, a 37-mm glass fiber filter was moistened with extract solution, and wipe samples were taken from the hands of participants.

Collection of airborne peanut allergen

Participants consumed peanuts in various forms to simulate situations that might create airborne peanut allergen. While peanut products were consumed and open peanut butter jars were present, participants wore personal air monitors (Buck-Genie VSS-12 series, Orlando, Fla, or Gillian High Flow Sampler, West Caldwell, NJ) running at a mean rate of 4 L/min for 60 minutes. Area samples at rates of 4 to 30 L/min (mean, 9.55 L/min) were also collected during some of the eating sessions. To avoid cross-contamination, each simulated setting was performed on different days. To simulate a school cafeteria setting, participants consumed peanut butter sandwiches. For environments similar to sporting events, participants shelled and consumed roasted peanuts. Participants were encouraged to discard peanut shells on the floor and were also allowed to walk on the shells during many of the sessions. To simulate the environment on commercial airliners, each participant opened 15 bags of unshelled peanuts in small, ½-oz packages and consumed the nuts. During each simulated environment, air samples were collected from participants eating the peanut product, from participants sitting next to the peanut eater, and from distances 5 to 10 ft away from the eater. Area samples were also taken 2 in above open peanut butter jars at 4 L/min. Some sessions were performed with the room ventilation turned off to decrease the air exchange rate. All personal and area samples were collected on 37-mm glass fiber filters for 60 minutes.

Sample extraction

All samples were stored at −30°C until extraction. Extractions were performed as previously described for air filter samples[10,11] and wipe samples.[12] Briefly, filters for airborne samples were left in cassettes until extraction. While leaving the filter in place, the support pads were carefully removed from the cassettes. Then, 1.5 mL extract solution was added to the cassette. Cassettes were rotated overnight at 4°C. Visible fluid was suctioned from the cassette. The filter was then folded and compressed in a 3-mL syringe to express any residual fluid. Surface and hand wipe samples were carefully folded and placed in a 3-mL syringe, and 1.5 mL extract solution was added. Samples were rotated overnight at 4°C, and filters were compressed to remove the fluid. Because heat extraction has been reported to yield higher concentrations of *Arachis hypogaea* allergen 1 (Ara h 1) in food products, some samples were extracted at 60°C for 15 minutes, as previously described.[13]

Peanut ELISA

After extraction, peanut allergen was measured by using a monoclonal-based Ara h 1 ELISA (INDOOR Biotechnologies, Charlottesville, Va), as previously published.[13,14] The range of detection of the assay was 30 to 2000 ng/mL.

RESULTS

Table wipe samples

Five table samples were taken before cleaning, and the range of Ara h 1 on tables was 720 to 6.3 × 10^4 ng/mL. Table wipe samples were obtained after cleaning with each of the following: plain water, dishwashing liquid, Formula 409 cleaner (Clorox Company, Oakland, Calif), Lysol sanitizing wipes (Reckitt Benckiser, Wayne, NJ), and Target brand cleaner with bleach (Target Corporation, Minneapolis, Minn) (Table I). All cleaning techniques except dish soap removed residual Ara h 1. Dish soap left residual Ara h 1 on 4 of 12 samples with levels of 40, 62, 128, and 140 ng/mL.

School surface samples

Six preschools and schools participated in the study. Two schools had peanut-free tables or peanut-free food preparation areas, and 1 school was entirely peanut-free. Ara h 1 was found on 1 of 13 water fountains (level, 130 ng/mL). None of the 36 eating or food preparation areas sampled contained detectable Ara h 1, including 9 samples taken from peanut-free tables or food preparation areas. None of the 22 desks sampled had detectable Ara h 1.

Hand wipe samples

Nine hand samples were taken before cleaning, and the range of Ara h 1 was 480 to 5.6 × 10^4 ng/mL. Hand wipe samples were taken after each of the following cleaning methods: plain water, antibacterial hand sanitizer, Tidy Tykes wipes (Pampers, Procter and Gamble), Wet Ones antibacterial wipes (Playtex Products, Dover, Del), liquid soap, and bar soap (Table II). Water and hand sanitizer left residual Ara h 1 on 3 of 12 and 6 of 12 hands each (range, 164-8274 ng/mL and 132-1711 ng/mL, respectively). Ara h 1 was undetectable with all other hand cleaning techniques.

Airborne samples

Airborne Ara h 1 was undetectable under all simulated environmental settings, including sessions during which participants were allowed to walk on peanut shells (Table III) and sessions with decreased room ventilation.

TABLE II. Results of hand wipe samples*

Cleaner	No. detectable	Detectable range (ng/mL)
None (n = 9)	9/9	480-5.6 × 10^4
Water (n = 12)	3/12	164-8274
Antibacterial hand sanitizer (n = 12)	6/12	136-1711
Commercial wipes		
Wet Ones (n = 12)	0/12	BD
Tidy Tykes (n = 10)	0/10	BD
Liquid soap (n = 12)	0/12	BD
Bar soap (n = 10)	0/10	BD

BD, Below detection.
*Participants cleaned 5 mL peanut butter off hands using various cleaning agents or plain water. One milliliter was applied to hands before cleaning with hand sanitizer.

DISCUSSION

Reports of severe allergic reactions after skin contact or airborne peanut exposure have led to some of the more controversial issues in allergy in recent years, including the need for peanut-free tables in schools and preschools and the provision for peanut-free flights on commercial airlines. A great many decisions have been made on these and similar issues primarily on the basis of fear and anecdotal reports of allergic reactions. Often patients and families avoid situations that would otherwise seem harmless such as eating at school, dining out, or vacationing because of concern of possible inadvertent exposure to peanut allergen. Similarly, recommendations are made regarding the steps that are needed to clean peanut protein from cafeteria tables and other objects, as well as from children's hands, without any data to support them.

In the current study, we used a monoclonal ELISA to measure the presence of the major peanut allergen, Ara h 1, in various settings. We found that Ara h 1 was removed from tabletops with usual cleaning techniques with a variety of different cleaners. The amount of peanut butter used in this study (5 mL) was much greater than expected to be found on tabletops contaminated by someone eating a peanut butter sandwich or other peanut-containing food product. After contamination with this high concentration of peanut allergen, 4 dishwashing liquid samples left residual low concentrations of peanut allergen. No residual Ara h 1 was found after routine cleaning with all other cleaning products or plain water. These data suggest that there is relatively low risk of exposure to peanut allergen when tables contaminated with small quantities of peanut butter are carefully cleaned with most common household cleaning products or water. Similarly, relatively large amounts of peanut butter were removed from hands after using several different cleaning products, although it is important to note that plain water and antibacterial hand sanitizer both left residual Ara h 1 at relatively high concentrations. These cleaning methods therefore appear to be less than ideal for cleaning peanut-contaminated hands and may result in contamination of other objects.

TABLE III. Results of airborne peanut analysis during consumption of various forms of peanut at varying distances from the peanut source

Form of peanut Distance from peanut source	Airborne Ara h 1 level
Peanut butter	BD
Consumer (n = 23)	
Next to consumer (n = 18)	
Peanut butter jar	BD
2 in (n = 6)	
Shelled peanuts	BD
Consumer (n = 9)	
Next to consumer (n = 9)	
5 ft (n = 3)	
10 ft (n = 1)	
Unshelled peanuts	BD
Consumer (n = 19)	
Next to consumer (n = 10)	
5 ft (n = 5)	
10 ft (n = 2)	

BD, Below detection.

Samples taken from schools and preschools revealed no detectable Ara h 1, except 1 water faucet sample taken from a preschool. This finding was unexpected, because no residual Ara h 1 was detected for many wipe samples taken from tables that included persons who had eaten peanut butter-containing foods. Although these findings cannot be used to make firm conclusions about school settings, they suggest that there is relatively low risk of significant exposure for children with peanut allergy attending similar preshcools and schools.

We were not able to detect airborne peanut allergen under any conditions, including settings that far exceeded what would be encountered by a patient with peanut allergy under usual circumstances. These exagerrated settings included sessions with decreased room ventilation and multiple persons simultaneously walking on peanut shells. It is most likely that this finding is a result of an insufficient sensitivity of the assay, because it is extremely likely that there was some airborne peanut allergen in some of those conditions. The lower limit of detection of the assay is 30 ng/mL, which would correspond to an airborne allergen level of 187 ng/m^3 for samples taken at 4 L/min for 60 minutes. Another possiblity is that Ara h 1 is not the allergen responsible for previously reported airborne reactions and that other peanut allergens, such as *Arachis hypogaea* allergen 2, may become airborne more readily and illicit such reactions. Future studies that include challenging patients with peanut allergy in these settings will be needed to elucidate the risks of airborne exposures more fully.

Previous studies have investigated the threshold oral dose of peanut protein capable of illiciting subjective and objective symptoms in patients with peanut allergy. Hourihane et al[15] found that subjective symptoms were illicited at doses as low as 100 μg, whereas objective symptoms were illicited at doses of 2 to 50 mg. Similarly, Wensing et al[16] found subjective symptoms at oral doses

as low 100 μg. In a roundtable conference reported by Taylor et al,[17] the lowest provoking doses ranged from 0.25 to 125 mg peanut protein. When comparing these findings with results of residual Ara h 1 on wipe samples in the current study, we found that none of the positive surface or hand wipe samples approached the peanut protein concentrations reported to be capable of illiciting reactions. However, it is also important to note that these provocation studies report values for total peanut protein and not for specific allergens, such as Ara h 1, which makes up only a fraction of the total peanut protein. Therefore, although it is very unlikely that the levels we detected would be of clinical significance, it is not possible to make firm conclusions in that regard, because the threshold dose of Ara h 1 may be significantly lower than that reported for total peanut protein.

This study provides novel information about the distribution and removal of a major peanut allergen in the environment. Information gained from this study provides a better understanding of the extent and likelihood of potential exposure to peanut allergen in various settings. We conclude that there is relatively low risk of exposure to significant concentrations of Ara h 1 when table surfaces and hands are cleaned with common household cleaning agents and that school cafeteria tables and desks are not likely to be a source of significant exposure for most peanut allergic patients. Because we were not able to detect airborne Ara h 1 under any circumstances, we are unable to make specific conclusions regarding the risk of airborne exposures for persons with peanut allergy.

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