Peanut allergen exposure through saliva: Assessment and interventions to reduce exposure

Jennifer M. Maloney, MD,[a] Martin D. Chapman, PhD,[b] and Scott H. Sicherer, MD[a]

New York, NY, and Charlottesville, Va

Background: Exposure to food allergens through saliva (kissing, utensils) can cause local and systemic allergic reactions.

Objective: To determine the time course of peanut allergen (Ara h 1) persistence in saliva after ingestion of peanut butter and to evaluate mouth cleansing interventions to reduce salivary peanut allergen.

Methods: Thirty-eight individuals ingested 2 tablespoons of peanut butter, and saliva was collected at various time points. At another time, samples were collected after 5 interventions (brushing teeth, brushing and rinsing, rinsing, waiting then brushing, waiting then chewing gum). Detection of Ara h 1 was performed by a monoclonal-based ELISA (detection limit, 15-20 ng/mL).

Results: Salivary Ara h 1 varied considerably immediately after ingestion, but included levels expected to invoke reactions (as much as 40 μg/mL). Most (87%) subjects with detectable peanut after a meal had undetectable levels by 1 hour with no interventions. None had detectable levels several hours later after a peanut-free lunch. This result indicates (95% confidence) that 90% would have undetectable Ara h 1 in saliva under these circumstances. All of the interventions reduced salivary Ara h 1, in some cases by >95%, but Ara h 1 remained detectable in ~40% of samples (though typically below thresholds reported to induce reactions).

Conclusion: Patients with peanut allergy require counseling regarding risks of kissing or sharing utensils, even if partners have brushed teeth or chewed gum. Advice to reduce risks, though not as ideal as total avoidance, includes waiting a few hours plus eating a peanut-free meal.

Clinical implications: Waiting several hours and ingesting a peanut-free meal were more effective at reducing salivary peanut protein concentration than simple, immediate interventions. (J Allergy Clin Immunol 2006;118:719-24.)

Key words: *Peanut allergy, kissing, anaphylaxis, saliva, Ara h 1*

Abbreviation used
PB: Peanut butter

Peanut allergy affects approximately 1.7 million Americans,[1] and adherence to dietary avoidance mandates constant vigilance on the part of the patient and, for children, the caregiver because of risks of exposure in a various environments.[2] Casual contact with peanut through inhalation or contact with the skin is a source of concern, although generally these exposures do not result in severe reactions.[3,4] Conversely, ingestion of peanut, even minute quantities, can result in reactions.[5-8] An inadvertent route of oral exposure is through passionate kissing of an allergic person with a partner who has consumed the allergen, or by sharing utensils, cups, and so forth. Case series and case reports reviewing allergic reactions through kissing have revealed a wide range of reaction severities, including anaphylaxis.[9-13] Severity of reactions via this mode of exposure likely depends on the amount of allergen present in saliva.

For practical patient care advice, it is essential to know how much peanut protein is residual in saliva after ingestion of peanut and the time course over which it diminishes in saliva. Moreover, a recommendation specifying an efficient way to remove peanut from the oral cavity would be helpful for guiding safe practices. We undertook the current study to determine the quantity of peanut in saliva, the duration of allergen persistence in saliva, and the effect of various practical interventions on the concentration of peanut allergen in saliva after ingestion of a significant quantity of peanut butter (PB). We measured the concentration of Ara h 1 as a marker for peanut protein. We conservatively sought an intervention in which the concentration of Ara h 1 in saliva would be below assay detection limits (<15-20 ng/mL) after intervention for 30 consecutive subjects. This goal was sought because it would indicate, with 95% confidence, that 90% of the time Ara h 1 would be undetectable.[14]

METHODS

Subjects and procedures

Participants included 42 adolescent and adult volunteers. Exclusion criteria were peanut allergy, wheat allergy, false teeth,

From [a]the Elliot and Roslyn Jaffe Food Allergy Institute, Division of Allergy and Immunology, Department of Pediatrics, Mount Sinai School of Medicine, New York; and [b]INDOOR Biotechnologies, Inc, Charlottesville.

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Reprint requests: Jennifer M. Maloney, MD, Mount Sinai School of Medicine, Division of Pediatric Allergy and Immunology, One Gustave L. Levy Place, Box 1198, New York, NY 10029. E-mail: jennifer.maloney@mssm.edu.

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braces, and/or bleeding gums. Participation required consumption of 2 tablespoons of commercially prepared PB on a sandwich and collection of a series of 1-mL salivary samples. Before each salivary collection, participants were asked to rub their tongue over their teeth and along their gums and palate to aid in the removal and dislodgement of peanut protein, which may have been fixed to the oral cavity. To simulate typical experiences and avoid biases, we did not inspect the oral cavity after the ingestion of peanut butter, or after interventions. Subjects recorded diet diaries and were instructed to avoid peanut, soy, and all other legumes during their involvement in the study. Dietary avoidance sheets and education were supplied to participants. If brushing teeth was a part of the assigned protocol, a new toothbrush and toothpaste was supplied to the participant. This study was approved by the Mount Sinai Institutional Review Board, and signed informed consents were obtained.

Time course studies

One milliliter of saliva was collected from each study participant before the PB sandwich to confirm that at baseline, allergen could not be found. Further saliva samples were collected at various time points throughout the day after PB ingestion: 5 minutes post-PB, 60 minutes post-PB, before lunch, after lunch, and at the end of the work day. There were an additional 7 study subjects with detectable Ara h 1 after the sandwich who were asked to collect saliva the following morning. Participants were instructed not to eat or drink between the 5-minute post-PB and 60-minute post-PB saliva collections.

Interventions

Our goal was to identify an intervention with which 30 consecutive subjects had no detectable Ara h 1 (95% confidence that 90% of such samples would be below detection). Persons with undetectable Ara h 1 at 5 minutes after ingesting the PB were excluded from analysis to avoid bias. One milliliter of saliva was collected after the sandwich to confirm the presence of peanut protein after ingestion (if there was none, they were excluded from analysis for interventions or time course). An intervention was assigned to each individual (participants may have performed different interventions on separate days). One of 3 different interventions was completed 5 minutes after sandwich ingestion as follows: brushing teeth for 2 minutes, brushing teeth for 2 minutes and rinsing the mouth with water twice (eg, a mouthful of water swished around for 10-30 seconds then spit), and rinsing the mouth twice without brushing teeth. Two interventions included a period of waiting longer: 1 hour after eating the sandwich and brushing teeth, or chewing gum for 30 minutes after a 30-minute period after the sandwich. Interventions were piloted in 5 to 10 individuals with an aim to include as many as 30 consecutive individuals if an intervention resulted in Ara h 1 below assay detection, as described.

Peanut ELISA and statistics

After salivary collections, samples were frozen at –4°C. Samples were shipped on dry ice to INDOOR Biotechnologies, Inc (Charlottesville, Va), where Ara h 1, 1 of the major peanut allergens,[15] was measured as a marker for peanut protein using a monoclonal-based technique as previously published.[16-18] The detection limit for the assay is 15 ng/mL or 20 ng/mL based on the specific assay run. Values under the detection limit were considered 14 ng/mL or 19 ng/mL (depending on the detection limit for the particular assay) for statistical calculation. In spiking experiments comparing saliva with buffered saline with albumin, peanut extract with known standard Ara h 1 content revealed identical results for Ara h 1 concentrations tested below 300 ng/mL (data not shown).

RESULTS

Time course

Thirty-eight subjects enrolled in the time course study. Results from 2 of the participants were subsequently excluded for technical reasons (inadequate sample volume, protocol deviation). Results from 36 participants were evaluated, but 6 subjects did not have detectable levels of Ara h 1 at 5 minutes after PB ingestion and were therefore excluded from further time course studies. The results from the 30 subjects with detectable Ara h 1 at 5 minutes after the sandwich are shown in Fig 1. The median concentration of Ara h 1 at the various time points for these 30 participants are as follows: 5 minutes: 1653 ng/mL (mean, 4040 ng/mL; range, 70-34,926 ng/mL); 60 minutes: 14 ng/mL (mean, 19 ng/mL; range, 14-70 ng/mL); before lunch: 14 ng/mL (mean, 15.4 ng/mL; range, 14-57 ng/mL); end of lunch: all values below detection.

Of the subjects with detectable Ara h 1 at 5 minutes after PB, 26 (87%) had undetectable levels of Ara h 1 at 60 minutes after ingestion. Of the 4 subjects (13%) who had detectable salivary Ara h 1 at 60 minutes after PB, all 4 had undetectable levels before lunch. Two subjects had undetectable concentrations of Ara h 1 at the 60-minute time point, but detectable levels were measured at 1 additional time. For the first participant, Ara h 1 peaked at 34,926 ng/mL at 5 minutes after PB ingestion, became undetectable at 60 minutes post-PB, and was detected at the before lunch time point (142 minutes post-PB) with a quantity of 57 ng/mL. Further samples from this subject (eg, after lunch) had undetectable levels. For the second participant, the Ara h 1 concentration at 5 minutes after PB was 6965 ng/mL, became undetectable at 60 minutes post-PB, and was also undetectable before and after lunch, but at the end of the day, 58 ng/mL Ara h 1 was measured. This participant had eaten other foods after lunch, though they were presumed to be peanut-free by diet records. Therefore, none of the participants had detectable Ara h 1 after lunch. Also, none of 7 subjects who collected a sample of saliva the morning after eating the PB had detectable levels of Ara h 1.

The intervals between collections varied after the 60-minute specified point because participants ate lunch at different times. The median amount of time that elapsed after sandwich ingestion and before lunch was 168 minutes (mean, 181; range, 99-312), after sandwich ingestion and after lunch was 227 minutes (mean, 232; range, 150-357), and after sandwich ingestion and the end of the workday was 433 minutes (mean, 417; range, 300-500). For the 4 subjects who had detectable Ara h 1 at 60 minutes after PB but clearing at the before lunch time point, lunch was eaten at a median time of 181 minutes after the sandwich (mean, 197; range, 155-270).

Interventions

Our aim was to identify a strategy that resulted in no detectable (<20 ng/mL) salivary Ara h 1 for 30 consecutive subjects, and we discontinued testing more subjects if preliminary assays revealed salivary Ara h 1.

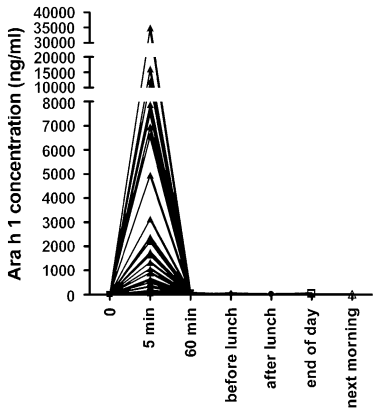

FIG 1. Time course study showing the concentrations of Ara h 1 before peanut butter ingestion and at various time points post-PB.

Twenty-three participants completed 5 interventions designed to remove peanut from the mouth, and 5 to 10 different subjects participated in each intervention (Table I). The results show that each of the 5 intervention procedures (including brushing teeth, rinsing, and chewing gum) led to significant reductions in Ara h 1 levels in saliva, compared with baseline levels that were determined immediately after consumption of the PB sandwich (Table I). In 20 interventions (57%), Ara h 1 was undetectable (<15 ng/mL) postintervention, and 20 interventions showed a ≥95% reduction in allergen levels. All of the interventions led to reductions in salivary Ara h 1 levels in subjects enrolled in the various groups, and in 90% of subjects, the reduction was >80%. We did not seek to evaluate large numbers of subjects to determine relative efficacy of procedures because we were piloting interventions to identify one with a high chance to essentially eliminate Ara h 1 from saliva. However, the intervention that included waiting for 60 minutes and then brushing teeth (wait then brush) appeared to be particularly effective in reducing allergen levels: 9/10 subjects showed a >95% reduction in Ara h 1 levels postintervention, and this group included several subjects with very high baseline salivary Ara h 1 (>50,000 ng/mL; Table I). Although Ara h 1 levels were not consistently reduced to undetectable levels after these interventions, marked reductions were noted, particularly for the 2 interventions with a waiting period.

TABLE I. Salivary Ara h 1 concentrations after interventions to remove peanut residues from the oral cavity*

Intervention	Baseline Ara h 1 (ng/mL)	Postintervention Ara h 1 (ng/mL)	Percent reduction in Ara h 1 levels
Brushing teeth alone	12,315	111	99
	64	<15	>78
	3138	357	89
	53,295	434	99
	1297	162	88
Brushing teeth + rinse	3075	163	95
	519	171	67
	2027	189	91
	1184	166	86
	479	<15	>97
Rinse alone	11,392	423	96
	485	69	86
	2676	510	81
	156	<15	>91
	66	43	35
Wait (60 min) then brush	25,000	<15	>99
	694,000	180	>99
	3350	<15	>99
	2250	<15	>99
	330	<15	96
	3400	<15	>99
	2220	270	88
	350,000	<15	>99
	1,110,000	<15	>99
	690	<15	>98
Chewing gum (30 min and wait 30 min)	4796	<15	>99
	720	<15	>98
	<15	<15	—
	70	<15	>80
	3327	<15	>99
	200	174	13
	193	<15	>93
	1664	<15	>99
	822	<15	>98
	2127	<15	>99

*Five interventions were piloted, and each result represents data from a single study participant. Baseline Ara h 1 levels were measured in saliva samples immediately after the study subject had consumed a PB sandwich. Ara h 1 levels were determined postintervention at 5 minutes (brushing teeth alone, brush + rinse, and rinse alone) and at 60 minutes (wait then brush, or chewing gum).

Concentration variability of Ara h 1

Extraordinary variability existed between Ara h 1 concentrations among participants in the study, ranging from undetectable levels to 1,110,000 ng/mL after PB ingestion. Several subjects were enrolled into different arms of the investigation; therefore, intrasubject comparisons were made among Ara h 1 concentrations in participant's saliva 5 minutes post-PB. Results are displayed in Table II.

DISCUSSION

This study was performed to investigate the duration of peanut allergen persistence in saliva after consumption of peanut and to pilot practical interventions designed to remove peanut allergen from the mouth after the peanut was consumed. The study used a monoclonal assay for Ara h 1 as a surrogate for total peanut measurements.[16-18] This assay had been shown to measure Ara h 1 effectively in saliva samples in preliminary studies (data not shown). The results show that Ara h 1 was readily detectable in saliva 5 minutes after consumption of 2 tablespoons peanut butter. In most (~90%) subjects, the level of Ara h 1 in saliva was undetectable 60 minutes postconsumption.

The amount of Ara h 1 detected in saliva after PB ingestion (as much as 10-40 ug Ara h 1/mL) may result in

TABLE II. Intrasubject variability of Ara h 1 after ingestion of a PB sandwich

Subject	Samples (n)	Median (ng/mL)	Range (ng/mL)
A	4	1,707	70-12,315
B	4	128.5	0-479
C	4	2,635	2,027-11,392
D	4	612.5	485-2,250
E	3	2,676	602-3,400
F	2	4,883	4,786-4,970
G	2	555,829	1,657-1,110,000
H	2	1,569	0-3,138
I	2	33	0-66
J	2	34,713	15,131-53,295
K	2	1,842	1,297-2,386
L	2	78	0-156
M	2	1,272	200-2,343
N	2	1,657	1,650-1,664
O	2	588	354-822

symptoms for patients with peanut allergy who are orally exposed to saliva. This conclusion is based on several assumptions about exposure volume and Ara h 1 as a marker for peanut proteins. We conservatively assume, without documentation, that approximately 5 mL saliva is transferred during a passionate kiss, and that ~15% of peanut protein is Ara h 1.[17,19] Using these assumptions, the maximum level of Ara h 1 measured 5 minutes after PB ingestion was 1110 μg/mL. If 5 mL saliva is transferred during a kiss, then 5.6 mg of Ara h 1 could be exchanged. This amount of Ara h 1 represents approximately 37 mg of peanut protein present in 5 mL saliva (~1/10-1/5 of a peanut). Even if a more conservative assumption is used, such as 1 mL saliva, this would equate to a transfer of 8 mg peanut protein during a kiss. These amounts of peanut protein could elicit symptoms in individuals with peanut allergy, as illustrated by threshold investigations showing that subjective symptoms occur with 100 μg peanut protein, whereas objective symptoms have been triggered by doses as low as 250 μg.[5-8,20] Applying these assumptions (using 5 mL as an estimation for saliva exchange), 30% of the participants in the time course study attained levels after the sandwich that could trigger symptoms in those with more sensitive peanut allergy.[5-7]

With a conservative approach in regard to practical advice, we sought to determine time or interventions that could reduce Ara h 1 to levels undetectable by the assay. Of the time course participants, 87% (26/30) had undetectable levels of Ara h 1 one hour after PB. For the subjects who had detectable Ara h 1 at 60 minutes post-PB, 70 ng/mL was the maximum amount of Ara h 1 found, which, using the previous assumptions, is equivalent to 0.0023 mg of total peanut protein in a total of 5 mL saliva. Though representing an amount detectable by the assay, this is 100-fold below the lowest dose reported to cause objective symptoms.[7] However, for conservative safety reasons, we aimed for a point where all samples were undetectable for Ara h 1. This was only achieved after a peanut-free lunch eaten a mean of 3.8 hours after the peanut meal.

Two subjects in the time course experiments had undetectable levels of Ara h 1 at an early time point, with reappearance later. This may have been a result of dislodgement of PB remnants in the oral cavity, or the subject may have inadvertently eaten something with peanut that was not recorded on the diet diary. The possibility that an ingested allergen can later be resecreted into saliva has been proposed by Maleki et al.[21] Regardless of the mechanism in which the allergen reappeared, the levels of Ara h 1 detected at these points were minute, 57 ng/mL and 58 ng/mL. This amount is equivalent to 0.002 mg peanut protein (130-fold below amount shown to trigger objective symptoms).

The intervention part of the study was experimental and was designed to determine a method of cleansing the mouth that reduced salivary Ara h 1 to undetectable levels for 30 consecutive subjects. Although all of the procedures resulted in significant reduction of Ara h 1 levels in saliva, none were consistently reducing levels to undetectable during piloting, so the interventions were not performed in larger numbers of subjects. The maximum amount of Ara h 1 detected after an intervention was 510 ng/mL, which, on the basis of the previous assumptions, is equivalent to 17 μg peanut protein in 5 mL saliva (15-fold below the dose reported to elicit objective symptoms). It is arguable whether these levels would be a threat to subjects with peanut allergy. However, we aimed for the assay limits in this study (equivalent to about 0.00067 mg peanut in 5 mL saliva), which is the amount of peanut protein Bindslev-Jensen et al[22] suggested would theoretically cause a reaction in only 1 out of 1,000,000 subjects with peanut allergy. Though participant numbers are too small for final conclusions, the apparent effectiveness of the wait and brush procedure suggests that this would be a useful procedure to evaluate further in risk assessment studies. However, reactions from kissing despite brushing teeth have been reported.[11] In fact, Wuthrich et al[10] described a kiss-related reaction caused by peanut despite the partner's attempts at removing allergen (2-hour wait period, teeth brushing, rinsing the mouth, and chewing gum), indicating that future studies would focus on determining a degree of risk reduction from such procedures.

The study limitations include the fact that the assay for peanut measured only Ara h 1. However, Ara h 1 is stable under a variety of manufacturing/food processing conditions[17] and in conditions that mimic human digestion.[23] We assume that the other peanut allergens are also found in saliva and would behave in a similar manner to Ara h 1. However, it is possible that retention of other allergens in saliva occurs over a different duration. Peanut may remain stuck to teeth or lips despite various interventions or time. Study participants were instructed to rub their tongue over their teeth and gums before collecting saliva samples; however, it is possible that remnants of the food may lodge between teeth, in dental work, or within areas of the mouth allowing longer persistence of the protein in some people.

Our intervention participants knew the reason for the study and may have been more detail-oriented about cleaning their mouths. Whether the result would have been different if casual brushing was used is not known. Importantly, our data applies only to PB, and for an additional margin of safety, a large serving of peanut butter was used. Although one would assume that many food allergens behave similarly, physical characteristics of foods are different and may influence their retention in the oral cavity. For example, eating actual peanuts may leave particles between teeth longer than PB, and these particles could later dislodge and enter the saliva. There is also a possibility that retained allergens in a toothbrush may reintroduce an allergen to the mouth at a later time. We used new toothbrushes in this study, and so this possibility was not fully explored and remains a theoretical concern. Importantly, no other food allergens (eg, milk, shrimp) were evaluated in this study, so our comments are limited to peanut in the form of peanut butter. We also make conservative recommendations because we found levels of Ara h 1 to be extremely variable among saliva samples (and even within the same individual) with a wide range of reduction percentage after interventions. Although explanations for this variability remain speculative, possibilities include differences in the manner in which individuals chew and swallow, or differences in salivary secretion rates.

Allergic reactions to food allergens via kissing are relatively common. In 2 case series of patients with food allergy, investigators illustrated that significant numbers of patients reported such reactions (5.3% and 12%).[11,13] In addition, there are several case reports describing kiss-induced allergic events.[9,10,12] A common theme for most of the described reactions is that the kiss of the allergic person occurred shortly after ingestion of the causative food by the partner. Furthermore, presumed passionate kissing (between romantic partners on the lips or mouth) resulted in more significant manifestations compared with a casual kiss on the cheek, which usually caused local symptoms (contact urticaria).

Our study confirms these previous patient reports and highlights the importance of educating patients with food allergy about the possibility of reactions via saliva exchange. Although our focus has been kissing, with larger saliva exchange, exposure can also occur when individuals share eating utensils, straws, cups, and other items. With respect to advice regarding avoidance of kiss related reactions, the safest approach we advocate is for the partner of the individual with allergy to avoid the allergenic food. For peanut butter specifically, we have shown that interventions that include a waiting period and brushing teeth/chewing gum appear to reduce the concentration to levels that are unlikely to induce reactions, but did not reduce Ara h 1 to undetectable in all cases. Waiting several hours after PB consumption and eating a meal within that time frame should reduce protein levels in saliva to clinically insignificant quantities. On the basis of our time course results from 30 participants, there is 95% confidence that levels of Ara h 1 in saliva will reach undetectable levels (<20 ng/mL) for 90% of people several hours after PB consumption and after a meal.

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Correction

With regard to the July 2006 articles entitled ''Diagnosis and treatment of atopic dermatitis in children and adults: European Academy of Allergology and Clinical Immunology/American Academy of Allergy, Asthma and Immunology/PRACTALL Consensus Report'' (2006;118:152-69) and ''Loss-of-function variations within the filaggrin gene predispose for atopic dermatitis with allergic sensitizations'' (2006;118:214-9): Disclosure of conflict of interest statements for Natalija Novak were incomplete. The following information should have appeared in the footnotes on pages 152 and 214:

Disclosure of potential conflict of interest: N. Novak serves on the Novartis Advisory Board.