

Prevalence and diagnosis of sesame allergy in children with IgE-mediated food allergy

To the Editor

Sesame has emerged as an important food allergen around the world, including the United States (US) where it is the ninth most common childhood food allergy with a population prevalence of approximately 0.1%–0.2%.¹ Sesame is the most common seed to cause hypersensitivity reactions, likely due to its widespread use in the food, pharmaceutical, and cosmetic industries. The US Food and Drug Administration (FDA) is currently considering a petition to include sesame in the list of allergens that must be disclosed on food labels as is currently mandated in Europe, Australia, and Canada. Only an estimated 20%–30% of patients outgrow their sesame allergy, and severe anaphylactic reactions are common, underscoring the need to optimize recognition and diagnosis of this allergy.²

The diagnosis of sesame allergy is challenging as there are no identified sesame-specific IgE (sIgE) or skin prick test (SPT) thresholds that predict clinical reactivity. Recently, specific component testing against a recombinant Sesi 1 allergen has been reported, with serum reactivity to Sesi 1 yielding a specificity and sensitivity of 86.1% and 85.7, respectively.³ However, component testing for sesame is not currently available clinically. Many prior studies evaluating the utility of SPT and sesame sIgE testing as a diagnostic tool included children with a suspicion for sesame allergy.^{4,5} The goal of this study was to examine the prevalence and manifestations of sesame allergy in a population of US children with IgE-mediated food allergy and to determine the utility of sIgE testing in predicting sesame allergy in this cohort.

One hundred nineteen consecutive subjects (median age: 9.0 years; 61.3% male) with IgE-mediated food allergy enrolled on a Natural History of Food Allergy protocol at the National Institutes of Health (NIH) were assessed for sensitization and clinical reactivity to sesame (Table 1). Subjects with a history of an immediate hypersensitivity reaction to at least one food along with positive IgE testing to that food were eligible for enrollment. No eligible subjects declined participation. All subjects were offered an oral food challenge (OFC) to sesame unless they had a recent convincing history of a hypersensitivity reaction (within 3 years) to sesame or if they were known to tolerate concentrated sesame (tahini, hummus) in their diet. Sesame sensitization was defined as a sesame sIgE >0.35 kU_A/L, sesame allergy as a recent immediate hypersensitivity reaction following exposure to sesame at home or via clinic food challenge (tahini), and sesame tolerance as the ability to ingest concentrated sesame (tahini, hummus) with no symptoms. For clinic OFCs (n = 11), increasing doses of tahini, starting with 0.045 g

(1%) of sesame protein, were administered every 15–30 minutes up to a cumulative dose of at least 4.5 g. An OFC was considered positive if the subject experienced objective symptoms (based on PRACTALL guidelines)⁶ and negative if symptoms were absent after ingesting all doses. Food-specific and total IgE were measured by ImmunoCAP (Thermo Fisher). The study was approved by the NIH IRB, and informed consent/assent was obtained for all subjects. Comparisons between groups were made with Wilcoxon rank-sum tests, chi-squared tests, or Fisher's exact tests. Logistic regression was used to estimate predictors of allergy. *P*-values <.05 were considered statistically significant.

Of the 119 patients, 108 (91%) were sensitized to sesame, 15 (12.6%) were confirmed sesame allergic, and 73 (61.3%) were sesame tolerant (Table 1). Although all subjects were offered an OFC to sesame regardless of their sesame sIgE levels, sesame allergy status could not be determined for 31 (26.1%) subjects, most of whom reported no known exposure to sesame and were avoiding it as a result of previous positive sIgE testing. There were no statistically significant differences in age, gender, ethnicity, allergic comorbidities, total reported food allergies (excluding sesame), or total IgE levels between those allergic vs. tolerant to sesame (Table 1). Among the 88 subjects with confirmed allergic status, the prevalence of sesame allergy was 17.0%. Compared to those who were tolerant, sesame allergic subjects had higher sesame sIgE levels (median 31.2 vs. 2.2 kU_A/L) and sesame sIgE/total IgE ratio (median 0.029 vs. 0.002) (Table 1; Figure 1A,B). An ROC analysis (Figure 1C,D) and a univariate logistic model (Figure 1E,F) were performed on the 88 subjects with confirmed sesame allergic status. The sesame-specific IgE threshold that maximized the sum of estimated sensitivity and specificity was 11.6 kU_A/L (4.9, 19.7) with a sensitivity of 0.93 and a specificity of 0.85 (Figure 1C). Using logistic regression, another threshold was estimated as the value associated with 50% probability of tolerating sesame: 29.4 kU_A/L (17.4, 61.2) (Figure 1E). Dividing sesame IgE by total serum IgE did not appear to improve the ability of sesame sIgE to predict allergy in this cohort (Figure 1D,F). However, logistic regression analysis found that a model with the two variables, total IgE and sesame sIgE, provided statistically significant better prediction of sesame allergy than a model with sesame sIgE alone.

Sesame oil and intact seeds are sometimes considered less allergenic than sesame paste since highly processed oils may contain less intact protein and seeds can be passed without significant digestion; however, anaphylaxis to these forms of sesame has been reported.^{7,8}

TABLE 1 Characteristics of study participants (number, %)

	Overall (n = 119)	Sesame allergic (n = 15)	Sesame tolerant (n = 73)	P-value ^a
Age (years) ^b	9.0 (5.0, 14.0)	9.0 (4.0, 14.0)	9.0 (5.0, 14.0)	0.96
Ethnicity				
Asian	16 (13.4%)	2 (13.3%)	10 (13.7%)	0.37
Black	15 (12.6%)	4 (26.7%)	7 (9.6%)	
Hispanic	4 (3.4%)	0	3 (4.1%)	
Mixed	10 (8.4%)	2 (13.3%)	7 (9.6%)	
White	74 (62.2%)	7 (46.7%)	46 (63.0%)	
Gender				
Female	46 (38.7%)	4 (26.7%)	33 (45.2%)	0.30
Male	73 (61.3%)	11 (73.3%)	40 (54.8%)	
Asthma	71 (59.7%)	7 (46.7%)	44 (60.3%)	0.49
Atopic dermatitis	97 (81.5%)	13 (86.7%)	55 (75.3%)	0.54
Allergic rhinitis	101 (84.9%)	12 (80.0%)	60 (82.2%)	1.00
EGID	17 (14.3%)	3 (20.0%)	7 (9.6%)	0.48
Total number of reported non-sesame food allergies	4.0 (2.0, 7.0)	5.0 (3.5, 7.0)	3.0 (2.0, 5.0)	0.07
Total IgE (kU/L) ^b	1067.0 (350.0, 2306.0)	975.0 (463.0, 1754.0)	542.0 (306.0, 1854.0)	0.28
Sesame IgE (kU _A /L) ^b	4.5 (1.0, 20.5)	31.2 (20.0, 46.3)	2.2 (0.6, 6.0)	<0.0001
Sesame sensitized ^c	108 (90.8%)	15 (100%)	62 (84.9%)	0.24
Sesame sIgE/Total IgE ratio ^b	0.004 (0.002, 0.011)	0.029 (0.005, 0.054)	0.002 (0.001, 0.007)	<0.0001
Peanut sensitized ^c	106 (89.1%)	14 (93.3%)	62 (84.9%)	0.65
Peanut allergic ^d	48 (40.3%)	6 (40.0%)	32 (43.8%)	1.00
Peanut IgE (kU _A /L) ^b	29 (3.0, 100)	53.5 (8.2, 78.1)	13.5 (1.5, 100)	0.27
Any TN sensitized ^c	111 (93.3%)	15 (100%)	66 (90.4%)	0.47
Any TN allergic	24 (20.2%)	6 (40.0%)	14 (19.2%)	0.16
Almond sensitized ^c	26 (23%)	15 (100%)	24 (32.8%)	<0.0001
Almond allergic ^d	7 (5.9%)	2 (13.3%)	2 (2.7%)	0.27
Almond IgE (kU _A /L) ^b	2.1 (0.5, 7.8)	4.4 (2.0, 7.2)	1.1 (0.2, 4.5)	0.02
Brazil nut sensitized ^c	72 (60.5%)	12 (80.0%)	37 (50.7%)	0.07
Brazil nut allergic ^d	0	0	0	1.00 ^e
Brazil nut IgE (kU _A /L) ^b	1.3 (0.2, 7.8)	1.7 (0.7, 4.0)	0.7 (0.2, 3.8)	0.36
Cashew sensitized ^c	94 (79.0%)	14 (93.3%)	52 (71.2%)	0.14
Cashew allergic ^d	13 (10.9%)	2 (13.3%)	9 (12.3%)	1.00
Cashew IgE (kU _A /L) ^b	5.0 (0.7, 24.0)	5.0 (0.9, 19.9)	2.8 (0.2, 16.4)	0.57
Pistachio sensitized ^c	92 (77.3%)	15 (100%)	52 (71.2%)	0.04
Pistachio allergic ^d	9 (7.6%)	2 (13.3%)	6 (8.2%)	0.89
Pistachio IgE (kU _A /L) ^b	11 (1.1, 40.6)	10.2 (2.1, 28.9)	7.6 (1.0, 30.3)	0.76
Hazelnut sensitized ^c	97 (81.5%)	15 (100%)	56 (76.7%)	0.09
Hazelnut allergic ^d	5 (4.2%)	2 (13.3%)	3 (4.1%)	0.43
Hazelnut IgE (kU _A /L) ^b	10.6 (2.7, 27.6)	14.4 (4.3, 22.2)	9.3 (2.3, 19.5)	0.35
Pecan sensitized ^c	69 (58.0%)	11 (73.3%)	37 (50.7%)	0.19
Pecan allergic ^d	6 (5.0%)	1 (6.7%)	4 (5.5%)	1.00
Pecan IgE (kU _A /L) ^b	1.7 (0.2, 9.5)	3.6 (0.3, 14.4)	1.0 (0.2, 6.2)	0.37

(Continues)

TABLE 1 (Continued)

	Overall (n = 119)	Sesame allergic (n = 15)	Sesame tolerant (n = 73)	P-value ^a
Walnut sensitized	91 (76.5%)	14 (93.3%)	51 (69.9%)	0.11
Walnut allergic ^d	8 (6.7%)	2 (13.3%)	4 (5.5%)	0.59
Walnut IgE (kU _A /L) ^b	5.8 (0.6, 27.0)	8.7 (2.6, 43.1)	1.9 (0.2, 21.5)	0.03

Abbreviations: EGID, eosinophilic gastrointestinal disease; TN, tree nut.

^aDifferences in distribution of categorical variables tested via chi-squared test; Differences in distribution of continuous variables tested via Wilcoxon rank-sum test.

^bMedian, (25th-75th percentile).

^cSensitized = sIgE ≥ 0.35 kU/L.

^dAllergic = convincing history of symptoms consistent with a hypersensitivity reaction immediately after eating the food.

^eFisher's exact test used due to cell counts incompatible with chi-squared test.

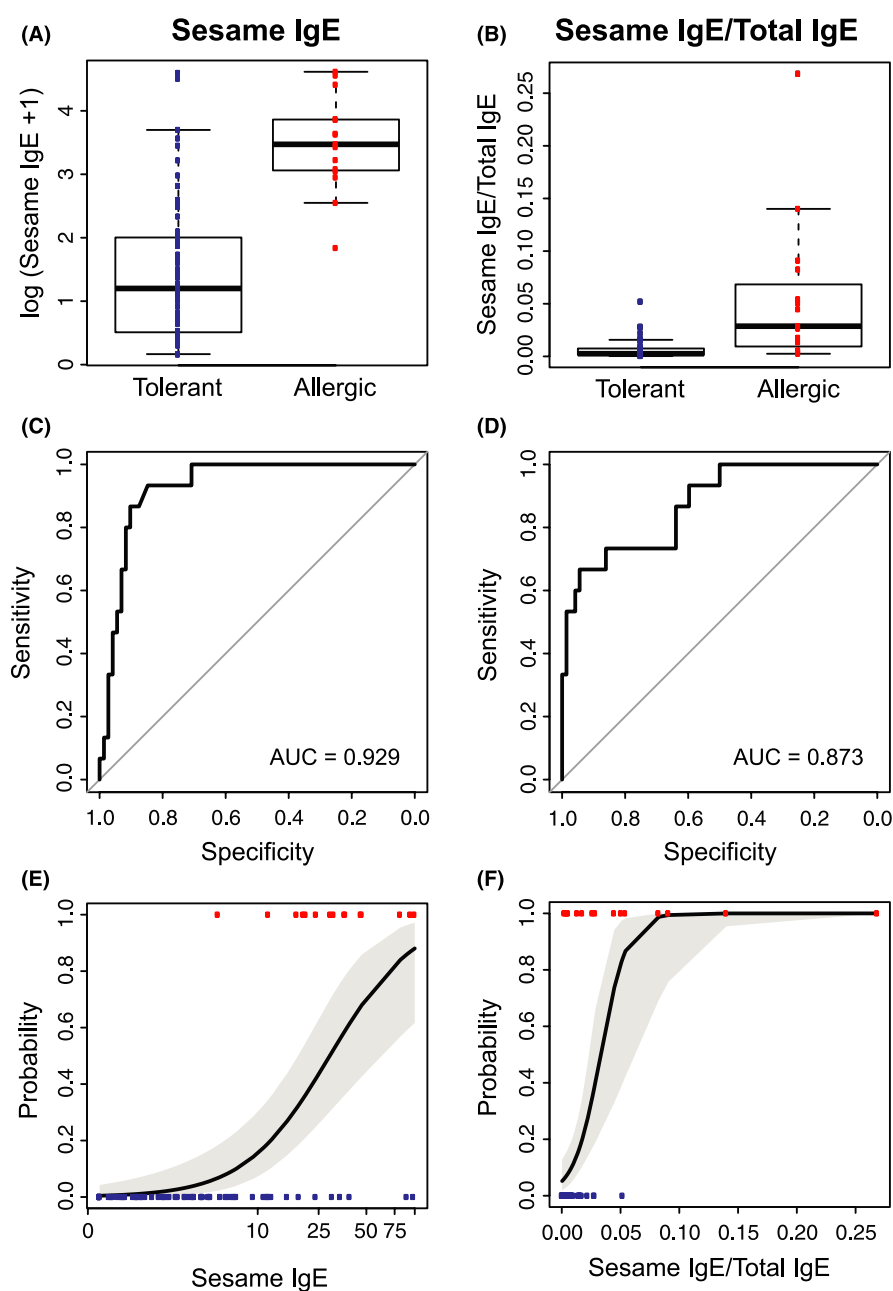


FIGURE 1 Sesame-specific IgE and ratio of sesame-specific/total IgE in subjects allergic or tolerant to sesame. Box plots (A and B) and ROC curves (C, D) depicting log (sesame sIgE + 1) (A, C) and ratio of sesame IgE/total IgE (B, D) in subjects allergic (red dots) or tolerant (blue dots) to sesame; logistic regression analysis (E, F) for sesame IgE and sesame IgE/total

Of the 15 sesame allergic subjects in this study, 4 could tolerate sesame oil and 2 could tolerate sesame seeds without any symptoms (Table S1). On the other hand, 3 subjects reported reactions to sesame oil and 7 to sesame seeds. The remaining subjects had never tried sesame oil or seeds since their initial allergic reaction was after ingestion of concentrated sesame, and they were advised to subsequently avoid all sesame products (Table S1). Many of the sesame allergic patients were able to tolerate other seeds, and in fact, none reported an allergic reaction to other seeds although nearly half of the subjects had not ingested other seeds (Table S1).

Both serologic cross-reactivity and clinical cross-reactivity have been observed between sesame and peanut and tree nuts.² In this cohort, 93.3% of subjects with confirmed sesame allergy were sensitized to peanut and 100% to at least one tree nut, compared with 84.9% and 90.4%, respectively, in those with known tolerance to sesame. Of the 15 patients allergic to sesame, 6 (40.0%) had a history of an acute hypersensitivity reaction to peanut and 6 (40.0%) to at least one tree nut, compared with 32 (43.8%) and 14 (19.2%) in the tolerant group. In general, subjects with sesame allergy had higher rates of allergy to individual tree nuts, although this did not always reach statistical significance (Table 1). Of note, a large group of subjects with confirmed sesame allergy and tolerance had never ingested peanut ($n = 7$; 46.7%; $n = 26$; 35.6%, respectively) or any tree nuts ($n = 8$; 53.3%; $n = 45$; 61.6%, respectively). Peanut and tree nut sIgE levels were generally higher in allergic than tolerant subjects (Table 1).

The diagnosis of sesame allergy remains a clinically challenging problem. Our results suggest that sesame IgE levels may have diagnostic utility. A recent study also found sesame IgE levels to be significantly higher in children allergic versus tolerant to sesame, although Sesi 1-IgE levels were most effective at predicting the result of an OFC for sesame while sesame SPT was ineffective, in contrast to other studies^{9,10} that have found SPT to be predictive. Sesame component IgE and skin prick testing were not performed in this study, and therefore, we cannot comment on the relative utility of these different diagnostic tools.

One major limitation of this study is that allergic status to sesame could not be determined for 31 (26.1%) patients, as they had never consumed concentrated sesame. These subjects either declined a sesame OFC ($n = 24$; reasons included anxiety, distance from our institution, or no interest in eating sesame-containing products), or had medical conditions such as active eosinophilic esophagitis that contraindicated an OFC ($n = 7$). Over half of these 31 subjects who had never ingested concentrated sesame were tolerating sesame seeds and/or oil without a subsequent hypersensitivity reaction. The sesame IgE level of those with unknown sesame allergic status was intermediate (8.92 kU_A/L) between those with confirmed sesame allergy and tolerance, and thus, the true prevalence of sesame allergy in our cohort is likely to be higher than 12.6% (Figure S1, Table S1). Other clinical and serologic features of those with unknown status are listed in Table S1. Additionally, subjects were required to have IgE-mediated food allergy to enroll in our study, and therefore, our findings may not be applicable to the general population. Our cohort

may also represent a more severely affected group of children with food allergy as the majority were allergic to more than one food. Sesame component testing was not performed, and therefore, we are not able to distinguish whether the high rate of peanut and tree nut sensitization and allergy in this cohort represents sensitization to cross-reactive epitopes or co-reactivity. For example, the major peanut allergens 7S globulin (Ara h1) and 11S globulin (Ara h3) are highly homologous (including IgE-binding epitopes) to the corresponding proteins in sesame.¹¹ A major strength of our study is that consecutive patients with IgE-mediated food allergy were enrolled as opposed to only those with suspected sesame allergy or known sensitization, which distinguishes our approach from many prior studies.

In summary, sesame allergy is common among children with IgE-mediated food allergy and often co-occurs with peanut and tree nut sensitization and allergy. A subset of sesame allergic children are able to tolerate sesame seeds and oil, although these products can cause severe reactions in some patients. Sesame-specific IgE levels were identified that may be helpful in deciding when to consider oral food challenges.

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CONFLICTS OF INTEREST

None.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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No association between prenatal antibiotic exposure and atopic dermatitis among Japanese infants

To the editor,

Prenatal antibiotic exposure has been suggested to be a risk factor for childhood allergic disease, possibly by affecting the development of a healthy and diverse microbiome, which is important for immune maturation.¹⁻³ Atopic dermatitis (AD) is often the first manifestation of the so-called "atopic march" and has a prevalence of over 10% among children in many developed countries.⁴ We aimed to investigate the association between prenatal antibiotic exposure and AD at age 1 in the Japan Environment and Children's Study (JECS).

The Japan Environment and Children's Study is a nationwide government-funded birth cohort study that aimed to evaluate the effects of

environmental factors on children's health and development. The study method is described in detail elsewhere.⁵ Briefly, women in early pregnancy were recruited at 15 regional centres in Japan between 2011 and 2014 and followed up by self-administered questionnaires, interviews and obstetric medical records. The JECS protocol was approved by the Institutional Review Board on Epidemiological Studies of the Ministry of the Environment and by the Ethics Committees of all participating institutions. A written informed consent was obtained from all participants.

The present study was based on the dataset of jecs-an-20180131, released in March 2018. Prenatal antibiotic exposure was defined as systemic antibiotic use (orally or by injection) reported (a) by the mother at the first interview (at recruitment) or second interview (during second/third trimester) or (b) in medical records

Figure E1

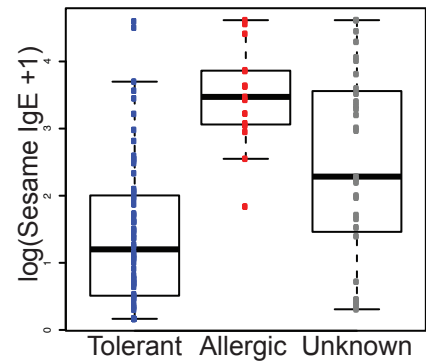


Table S1. Clinical and laboratory characteristics of sesame allergic subjects

Pt	Symptoms of hypersensitivity reaction to sesame	Form of sesame	Last reaction to sesame	Sesame IgE (kU _A /L)	Year sesame IgE measured	Total IgE (kU/L)	PN allergy	Any TN allergy	Tolerant to sesame seeds	Tolerant to sesame oil	Other seeds tolerated
1	Hives, throat tightening	Seeds	2017	37.0	2018	14,727	Y	Y	N	U	Mustard
2	Vomiting	Seeds, Oil (sauce)	2012	81.4	2015	581	N	N	N	N	Mustard, pumpkin
3	Hives, pruritis, flushing	Sesame paste	2014	20.7	2016	463	N	Y	U	U	Mustard
4	Throat pruritis, throat tightening	Seeds, hummus w/ tahini	2010	46.3	2012	1754	U	Y	N	Y	Mustard, poppy, pumpkin
5	Hives	Sesame paste, seeds	2017	18.1	2017	1351	Y	U	N	Y	Mustard
6	Hives, vomiting, wheezing, generalized pruritis, angioedema, nasal congestion	Tahini, seeds, oil	2009*	24.1	2015	265	Y	Y	N	N	Mustard, poppy, pumpkin
7	Mouth and throat pruritis	Seeds and hummus w/ tahini	2015	94.3	2018	1862	Y	U	N	U	U
8	Wheezing, angioedema, hives	Hummus w/ tahini	2011	>100	2014	22,672	U	U	U	U	U
9	Hives	Hummus w/ tahini	2017	20.0	2018	3715	Y	U	U	U	U
10	Dx'd by OFC: Nausea, throat clearing, hives	Tahini (4.5 g sesame protein)	2018	11.8	2017	669	U	U	Y	Y	Sunflower
11	Dx'd by OFC:n Vomiting, angioedema, hypotension	Tahini (300 mg sesame protein)	2018	31.2	2018	378	U	U	U	U	U

12	Vomiting, lip swelling	Hummus w/ tahini	2016	46.9	2018	1635	U	U	U	U	U
13	Vomiting, throat pruritus and tightness, generalized pruritus, face swelling	Hummus w/ tahini, ground sesame flour, sesame seeds, oil	2017	29.8	2018	111	Y	Y	N	N	U
14	Hives, difficulty breathing, angioedema	Tahini	2017	5.28	2018	975	U	Y	Y	Y	Pumpkin, sunflower
15	Dx'd by OFC: Vomiting, sneezing, generalized pruritis, hypotension	Tahini (300 mg sesame protein)	2019	36.6	2018	675	U	U	U	U	U

Pt=patient; PN=peanut; Y=Yes; N=No; U=unknown (because never eaten); Dx'd = diagnosed; OFC = oral food challenge

*Recent hypersensitivity was defined as a reaction to sesame within 3 years of the date when sesame IgE levels were measured; this subject was an exception and declined a sesame OFC to confirm the diagnosis

Table S2. Characteristics of participants with unknown allergic status to sesame (number, %)

	Sesame Allergic (n=15)	Unknown Allergic Status (n=31)	p-value [†]
Age*	9.0 (4.0, 14.0)	9.0 (5.0, 12.0)	0.96
Ethnicity: Asian	2 (13.3%)	4 (12.9%)	0.42
Black	4 (26.7%)	4 (12.9%)	
Hispanic	0	1 (3.2%)	
Mixed	2 (13.3%)	1 (3.2%)	
White	7 (46.7%)	21 (67.7%)	
Gender: Female	4 (26.7%)	9 (29.0%)	1.00
Male	11 (73.3%)	22 (71.0%)	
Asthma	7 (46.7%)	20 (64.5%)	0.40
Atopic dermatitis	13 (86.7%)	29 (93.5%)	0.83
Allergic rhinitis	12 (80.0%)	29 (93.5%)	0.38
EGID	3 (20.0%)	7 (22.6%)	1.00
Total number of reported food allergies*	5.0 (3.5, 7.0)	6.0 (3.5,8.0)	0.78
Total IgE (kU/L)*	975.0 (463.0, 1754.0)	2891.0 (395.0, 8238.0)	0.23
Sesame IgE (kU _A /L)*	31.2 (20.0, 46.3)	8.92 (3.0, 32.8)	0.03
Sesame sensitized [§]	15 (100%)	31 (100%)	1.00 ^{&}
Sesame IgE/Total IgE ratio*	0.029 (0.005, 0.054)	0.004 (0.002, 0.011)	<0.0001
Peanut sensitized [§]	14 (93.3%)	30 (96.8%)	1.00
Peanut allergic [‡]	6 (40.0%)	10 (32.2%)	0.85
Peanut IgE (kU _A /L)*	53.5 (8.2, 78.1)	40.8 (9.4, 100.0)	0.95
Any TN sensitized [§]	15 (100%)	30 (96.8%)	1.00
Any TN allergic [‡]	6 (40.0%)	4 (12.9%)	0.09
Almond sensitized*	15 (100%)	29 (93.5%)	0.81
Almond allergic [‡]	2 (13.3%)	3 (9.6%)	1.00
Almond IgE (kU _A /L)*	4.4 (2.0, 7.2)	4.0 (1.4, 36.7)	0.62
Brazil nut sensitized [§]	12 (80.0%)	3 (74.2%)	<0.0001
Brazil nut allergic [‡]	0	0	1.00 ^{&}
Brazil nut IgE (kU _A /L)*	1.7 (0.7, 4.0)	4.8 (0.8, 23.6)	0.16
Cashew sensitized [§]	14 (93.3%)	28 (90.3%)	1.00
Cashew allergic [‡]	2 (13.3%)	2 (6.5%)	0.83
Cashew IgE (kU _A /L)*	5.0 (0.9, 19.9)	16.3 (1.1, 62.4)	0.14
Pistachio sensitized [§]	15 (100%)	25 (80.6%)	0.17
Pistachio allergic [‡]	2 (13.3%)	1 (3.2%)	0.51
Pistachio IgE (kU _A /L)*	10.2 (2.1, 28.9)	29.7 (2.2, 86.3)	0.14
Hazelnut sensitized [§]	15 (100%)	26 (83.9%)	0.09
Hazelnut allergic [‡]	2 (13.3%)	0	0.19
Hazelnut IgE (kU _A /L)*	14.4 (4.3, 22.2)	26.2 (3.3, 47.4)	0.35

Pecan sensitized [§]	11 (73.3%)	21 (67.7%)	0.96
Pecan allergic [‡]	1 (6.7%)	1 (3.2%)	1.00
Pecan IgE (kU _A /L)*	3.6 (0.3, 14.4)	3.0 (0.7, 11.9)	0.99
Walnut sensitized [§]	14 (93.3%)	26 (83.9%)	0.67
Walnut allergic [‡]	2 (13.3%)	2 (6.5%)	0.83
Walnut IgE (kU _A /L)*	8.7 (2.6, 43.1)	11.3 (2.2, 31.5)	0.58

[†] Differences in distribution of categorical variables tested via Chi-Squared Test;
Differences in distribution of continuous variables tested via Wilcoxon Rank Sum Test

*median, (25th-75th percentile)

[§]Sensitized = sIgE $\geq$ 0.35 kU/L

[‡]Allergic = convincing history of symptoms consistent with a hypersensitivity reaction immediately after eating the food

[&]Fisher's exact test used due to cell counts incompatible with Chi-Squared Test

EGID = eosinophilic gastrointestinal disease

TN = tree nut