Journal of Asthma and Allergy

Dovepress
Taylor & Francis Group

Open Access Full Text Article

CLINICAL TRIAL REPORT

High- and Low-Dose Oral Sesame Immunotherapy: Interim Analysis of a Randomized Single-Center Open-Label Trial

Joanna Zielińska*, Anna Krupa-Łaska*, Marek Kulus, Katarzyna Grzela

Department of Paediatric Pulmonology and Allergology, Medical University of Warsaw, Warsaw, Poland

*These authors contributed equally to this work

Correspondence: Katarzyna Grzela, Department of Paediatric Pulmonology and Allergology, Medical University of Warsaw, 63A Zwirki i Wigury Street, Warsaw, 02-091, Poland, Email katarzyna.grzela@wum.edu.pl

Journal of Asthma and Allergy downloaded from https://www.dovepress.com/
For personal use only.

Purpose: To compare the efficacy and safety of two sesame protein maintenance doses (300 mg vs 1200 mg) in pediatric oral immunotherapy (OIT) for sesame allergy.

Patients and Methods: In this randomized, single-center, open-label trial (NCT05158413), 26 children aged 4–17 years with confirmed sesame allergy were allocated 1:1 to receive either 300 mg or 1200 mg sesame protein as maintenance therapy following dose escalation. Inclusion criteria included positive skin prick test (SPT) and/or elevated sesame-specific IgE (sIgE), as well as clinical reactivity during oral food challenge (OFC). The primary endpoint was the proportion of patients tolerating 4000 mg of sesame protein at the end-of-treatment OFC. Secondary outcomes included changes in immunologic markers (SPT wheal size, sIgE, IgG4) and safety.

Results: The rate of negative OFCs in our study (81.8% in the high-dose group and 69.2% in the low-dose group; $p = 0.649$) was comparable to outcomes reported in previous sesame OIT trials (19–22), indicating similar efficacy across different maintenance dose regimens. Both groups showed significant reductions in SPT wheal size and increases in sesame-specific IgG4 ($p < 0.005$), with no significant changes in sIgE levels. The rate of World Allergy Organization (WAO) grade I adverse events per dose was comparable between groups (4.74% vs 3.88%, $p = 0.706$), with three WAO grade II events (2 high-dose, 1 low dose) and no grade III or adrenaline-requiring reactions reported.

Conclusion: This interim analysis, based on a limited sample size, suggests that both low- and high-dose sesame OIT regimens are consistent with being effective and well tolerated in children, with similar immunologic responses and a favorable safety profile. Given the pilot nature of the study, the results should be interpreted as preliminary and hypothesis-generating, warranting confirmation in larger, long-term trials to optimize sesame OIT dosing strategies.

Keywords: sesame allergy, oral immunotherapy, food allergy, oral food challenge

Introduction

Sesame allergy is a relatively rare food allergy, with an estimated global prevalence of between 0.1% and 0.9%.[1–6] Despite its low prevalence, sesame avoidance is challenging because of its growing use in foods such as bread, cereals, and confectionery.[7,8]

Clinical manifestations of sesame allergy vary widely, ranging from mild symptoms, such as oral pruritus and urticaria, to severe, potentially life-threatening anaphylactic reactions.[9] In the vast majority of patients, sesame allergy is persistent, with spontaneous resolution in only 18–32% of cases.[10–13] Diagnostic methods include the skin prick test (SPT) using sesame extract or native products, sesame-specific IgE (sIgE) measurement to both sesame extract and its major allergenic component Ses i 1, and the basophil activation test (BAT).[14,15] The oral food challenge (OFC) remains the gold standard for diagnosis.[16,17]

https://doi.org/10.2147/JAA.S563421
Received: 26 August 2025
Accepted: 9 January 2026
Published: 5 February 2026

© 2026 Zielińska et al. This work is published and licensed by Dove Medical Press Limited. The full terms of this license are available at https://www.dovepress.com/terms.php and incorporate the Creative Commons Attribution – Non Commercial (unported, v4.0) License (http://creativecommons.org/licenses/by-nc/4.0/). By accessing the work you hereby accept the Terms. Non-commercial uses of the work are permitted without any further permission from Dove Medical Press Limited, provided the work is properly attributed. For permission for commercial use of this work, please see paragraphs 4.2 and 5 of our Terms (https://www.dovepress.com/terms.php).

Management is primarily based on a strict elimination diet and the provision of emergency adrenaline auto-injectors in the event of accidental exposure and severe reactions.[18] Recently, oral immunotherapy (OIT) has emerged as a promising therapeutic strategy for food allergies, including sesame allergy. Nachshon et al reported full desensitization in 88.4% of patients (53/60) following an initial dose escalation up to 4 g of sesame protein, followed by a maintenance dose of 1.2 g for at least six months, with reactions occurring in 4.7% of hospital and 1.9% of home doses; epinephrine was required in 16.7% and 8.3% of patients, respectively.[19] Chua et al demonstrated 85.7% efficacy (18/21) using a lower maintenance dose of 200 mg over one year in preschool-aged children, where 67.9% experienced only mild (grade 1–2) reactions and one case (3.6%) required epinephrine.[20] Salari et al achieved 100% efficacy (11/11) and favorable safety outcomes using omalizumab-assisted OIT with a 5 g maintenance dose.[21] Shah et al described a cohort of 84 patients, 65 of whom reached a 1000 mg maintenance dose.[22] Among the 31 participants who underwent a final OFC, 30 (96.8%) had a negative result, and 29 achieved sustained unresponsiveness. Overall, these studies demonstrate high efficacy and suggest that sesame OIT has a favorable safety profile, with most reactions being mild to moderate and severe events, including anaphylaxis, occurring rarely.

In the pilot study described here, we implemented an OIT protocol using two different maintenance doses (300 and 1200 mg) of sesame protein to compare the efficacy and safety of both regimens. We hypothesized that the lower dose would be associated with fewer adverse events, lower dropout rates, and improved treatment adherence, while maintaining comparable efficacy. This paper presents an interim analysis of our findings. An interim analysis was conducted after approximately 50% of the planned sample had been enrolled and completed the treatment phase, with the primary objective of assessing whether the predefined pilot sample size was adequate to detect meaningful trends in efficacy and safety outcomes. This paper presents the results of that interim analysis.

Materials and Methods

Patients

This prospective randomized, single-center, two-armed, open-label interventional trial study with an allocation ratio of 1:1 was performed on 26 patients with allergy to sesame (NCT05158413). These patients were recruited among patients of the Pediatric Hospital of the Medical University of Warsaw, Poland, from January 2022 to April 2024. Inclusion criteria included age 4–17 years, confirmed sesame allergy based on a positive SPT (wheal ≥3 mm) and/or sIgE >0.35 kUA/L, clinical reaction to sesame during an open OFC, signed informed consent (by parent/guardian and patients ≥16 years), and willingness of both patient and caregivers to comply with study procedures. Exclusion criteria included common comorbidities that could affect safety or efficacy, including severe or uncontrolled asthma, ongoing immunotherapy, chronic diseases requiring continuous treatment, and recent steroid or biologic therapy, as well as pregnancy and lack of consent or cooperation.

The study protocol was approved by the Ethics Committee of the Medical University of Warsaw (approval number: KB/147/2021). Informed consent was obtained from all participants or their legal representatives. The study complies with the Declaration of Helsinki.

Randomization

Participants were randomized 1:1 to high-dose (1200 mg) or low-dose (300 mg) groups using permuted block randomization with random block sizes, generated independently in R (randomizeR v.2.0.0). Allocation codes were sealed in numbered envelopes and revealed sequentially upon patient enrollment.

Study Design

In the first step of the study, a complete medical history was obtained from each patient. At the baseline and the end of the study, an SPT, blood analysis, and OFC were performed.

An SPT with native sesame (raw sesame-paste tahini, a commercial product containing 200 mg protein/g; Quality Food Tahini Premium) was performed on the volar surface of the forearm. Histamine (1 mg/mL; Diater, Spain) and saline (0.9% NaCl) were used as positive and negative controls, respectively. The blood sample was analyzed for sIgE and IgG4

levels (ImmunoCAP). Each patient underwent an open OFC with sesame protein using the PRACTALL stopping criteria.[23] On day 1, sesame protein was administered in incremental doses at 30-minute intervals (1.7 mg, 3 mg, 30 mg, 300 mg, 1000 mg, and 3000 mg); on day 2, a single 4000 mg dose was given. Patients who tolerated the full 4000 mg dose on day 2 were considered to have a negative OFC result and were not eligible for inclusion in the oral immunotherapy (OIT) protocol. These patients were advised to freely include sesame in their diet. The source of sesame protein was tahini, as a recent study showed that tahini can be more allergenic than sesame.[17] The tahini dose at which predefined stopping criteria were met was defined as the eliciting dose.

OIT consisted of two phases: a build-up phase and a maintenance phase. The selection of the initial oral food challenge dose was based on the individual's eliciting dose of sesame protein. For eliciting doses of 1.7 mg, 3 mg, 30 mg, 100 mg, 300 mg, 1000 mg, and 3000 mg, the corresponding initial OIT doses were 0.5 mg, 1.7 mg, 3 mg, 12 mg, 40 mg, 120 mg, and 300 mg, respectively. During the build-up phase, sesame protein doses were increased sequentially, typically following the progression: 0.5 mg, 1.7 mg, 3 mg, 6 mg, 12 mg, 20 mg, 40 mg, 80 mg, 120 mg, 160 mg, 200 mg, 240 mg, 300 mg, 600 mg, 900 mg and 1200 mg. Each dose was maintained for two weeks prior to escalation. Some patients completed the build-up phase at a target dose of 300 mg, while others continued dose escalation to 1200 mg, depending on protocol assignment. All patients who remained in the study successfully reached their planned maintenance dose.

The first and escalating doses were administered under medical supervision. After 2 hours of observation, if the patient tolerated the dose, they were instructed to consume this dose daily at home. During each build-up phase visit, assessments of coexisting chronic condition control and a review of daily symptoms were performed. After achieving tolerance of the maximal dose (depending on the assigned group), immunotherapy was continued daily for 3 months at a maintenance dose of 300 mg or 1200 mg of sesame protein, according to randomization. At the end of OIT, the final OFC and evaluation of the desensitization to sesame protein were performed. Confirmation of the total desensitization to sesame was the tolerance of a single dose of 4000 mg sesame protein.

Statistical Analysis

All statistical analyses were conducted using R software (R Core Team, 2024) and related packages, including dplyr, tidyr, purrr, stringr, car, epitools, and ggplot2.[24–30] Continuous variables are summarized as either the mean with standard deviation (SD) for normally distributed data or as the median and interquartile range (IQR) for variables not following a normal distribution. Distribution normality was assessed using the Shapiro–Wilk test and supported by examination of skewness and kurtosis values. Levene's test was used to assess the equality of variances where applicable. To compare differences between independent groups, Student's *t*-test or the Mann–Whitney *U*-test were employed, depending on data distribution. Categorical variables were analyzed using Pearson's chi-square test or Fisher's exact test, as appropriate. For paired comparisons, either a paired *t*-test or a Wilcoxon test was applied, based on the distribution of differences. All statistical tests were two-tailed, with results considered significant at *p*-values below 0.05.

Results

Study Group Characteristics

Patient characteristics are shown in Table 1. Study participants were mainly male (80.0%). The median age of the total group was 5.63 years. Groups treated with high and low doses did not differ in terms of sex structure or age ($p > 0.999$ and $p = 0.880$, respectively). Atopic dermatitis and allergic rhinitis were reported for more than half of the participants (57.7% and 80.8%, respectively), and 30.8% of the participants had asthma. Food allergies other than sesame were confirmed for 88.5% of the study group, and sesame-related reactions were reported by 50.0% of the study participants. The median initial protein dose was 3 mg in both groups ($p = 0.417$). No significant differences were found between the study groups in terms of allergy-related characteristics.

Treatment Outcomes Between Study Groups

Table 2 shows the clinical and immunological outcomes at the end of the treatment. The proportion of negative OFCs at the end of the treatment was 81.8% (9/11) in the high-dose group and 69.2% (9/13) in the low-dose group, which was not

Table 1 Study Group Baseline Characteristics

Variable	Total Group (n = 26)	High-Dose Group (n = 13)	Low-Dose Group (n = 13)	MD or RR (95% CI)	*p*-value
Female, n (%)	5 (19.2)	2 (15.4)	3 (23.1)	–	>0.999
Male, n (%)	21 (80.8)	11 (84.6)	10 (76.9)		
Age, years, median (IQR)	5.63 (4.76–8.41)	5.56 (4.75–9.17)	5.70 (4.76–8.33)	−0.14 (−1.54–2.26)	0.880
Atopic dermatitis, n (%)	15 (57.7)	10 (76.9)	5 (38.5)	2.00 (0.95–4.23)	0.112
Allergic rhinitis, n (%)	21 (80.8)	11 (84.6)	10 (76.9)	1.10 (0.75–1.60)	>0.999
Asthma, n (%)	8 (30.8)	4 (30.8)	4 (30.8)	–	>0.999
Food allergies (other than sesame), n (%)	23 (88.5)	10 (76.9)	13 (100.0)	0.77 (0.57–1.04)	0.220
Reported sesame-related reactions, n (%)	13 (50.0)	8 (61.5)	5 (38.5)	1.60 (0.71–3.60)	0.433
Initial protein dose (mg), median (IQR)	3.00 (1.70–12.00)	3.00 (1.70–12.00)	3.00 (3.00–12.00)	0.00 (−10.30–1.30)	0.417
Baseline tahini SPT wheal size (mm), median (IQR)	10.00 (8.25–15.00)	9.00 (8.00–15.00)	11.00 (10.00–15.00)	−2.00 (−5.00–5.00)	0.409
Baseline sIgE (kUA/L), median (IQR)	7.37 (3.02–30.32)	5.85 (2.53–33.20)	8.07 (4.06–26.20)	−2.22 (−9.18–15.00)	0.511
Baseline IgG4 (mgA/L), median (IQR)	1.78 (0.49–2.73)	1.34 (0.49–2.61)	2.00 (0.49–3.10)	−0.66 (−1.97–1.23)	0.682

Notes: MD was reported as effect size for continuous variables, RR was reported as effect size of categorical variables. RR not calculated for sex as both sex were reported and not reported for asthma due to identical proportion in both groups. Groups were compared with the Mann–Whitney *U*-test (all continuous variables), Pearson's chi-square test (atopic dermatitis, reported sesame-related reactions), or Fisher's exact test (other variables).
Abbreviations: IQR, interquartile range; MD, median difference (high dose vs low dose); RR, relative risk (high dose vs low dose); CI, confidence interval.

statistically significantly different between groups (p = 0.649). There were two dropouts in the high-dose group, one due to uncontrolled asthma and the other due to chronic otitis media with effusion, which worsened during OIT. All patients with positive OFCs at the end of treatment demonstrated an increase in the eliciting dose threshold.

Tahini SPT wheal size, sIgE, and IgG4 at the end of treatment were not significantly different between groups. The percentage of World Allergy Organization (WAO) grade I adverse effects per dose was 4.74% in the high-dose group and 3.88% in the low-dose group, with no difference between study groups. There were three WAO grade II adverse events: two occurred in a single patient from the high-dose group, and one occurred in a patient from the low-dose group. No adverse effects of WAO grade III or more were observed, and no adrenaline administration was needed. The most frequently reported adverse events were oral itching and mild rash (Table 3). Patients recorded all events in diaries, marking predefined symptoms or describing others in their own words, ensuring complete data capture.

The study groups differed regarding OIT duration, with longer OIT observed in the high-dose group (MD = 60.74; 95% confidence interval (CI): 6.07–115.41; $p = 0.031$). A significant difference was also observed in terms of the number of visits: five more visits were reported in the high-dose group compared to the low-dose group (MD = 4.77; 95% CI: 1.59–7.95; $p = 0.005$).

Treatment Outcomes Before and After Treatment

Table 4 and Figure 1 show the immunological changes observed after treatment. Tahini SPT wheal size decreased significantly after the treatment in the high-dose group (MD = −4.00; 95% CI: −14.00–−2.50); $p = 0.004$) and in the low-dose group (MD = −6.08; 95% CI: −8.66–−3.50; $p < 0.001$). IgG4 increased significantly after the treatment in both the high-dose group (MD = 8.06; 95% CI: 4.06–145.82; $p = 0.001$) and the low-dose group (MD = 1.61; 95% CI: 1.04–10.16; $p = 0.002$). Specific IgE levels decreased in the high-dose group and increased in the low-dose group, but these changes were not statistically significant ($p = 0.765$ and $p = 0.588$, respectively).

Table 2 Treatment Outcomes Between Study Groups

Variable	Total Group (n = 24)	High-Dose Group (n = 11)	Low-Dose Group (n = 13)	MD or RR (95% CI)	*p*-value
OIT duration (days), mean ± SD	285.92 ± 70.12	318.82 ± 56.51	258.08 ± 70.21	60.74 (6.07–115.41)	0.031
No of visits, mean ± SD	12.58 ± 4.58	15.27 ± 3.47	10.31 ± 4.23	4.97 (1.65–8.28)	0.005
Tahini SPT wheal size at the end of treatment (mm), mean ± SD	5.33 ± 2.88	4.55 ± 3.14	6.00 ± 2.58	−1.45 (−3.88–0.97)	0.226
Delta, median (IQR)	−5.00 (−10.00–−2.75)	−5.00 (−10.00–−2.00)	−5.00 (−8.00–−3.00)	0.00 (−5.00–4.00)	>0.999
sIgE at the end of treatment (kUA/L), median (IQR)	8.27 (3.49–14.83)	4.51 (2.37–14.80)	9.18 (5.12–13.30)	−4.67 (−8.79–3.90)	0.207
Delta, median (IQR)	−0.42 (−2.43–2.21)	−0.14 (−2.00–1.68)	−0.71 (−2.33–2.16)	0.57 (–4.23–6.10)	0.910
IgG4 at the end of treatment (mgA/L), median (IQR)	7.16 (2.60–18.92)	9.40 (4.37–24.25)	3.61 (2.26–14.80)	5.79 (−2.35–16.64)	0.331
Delta, median (IQR)	5.66 (1.39–13.29)	7.79 (2.91–20.33)	2.12 (0.43–9.45)	5.67 (−0.88–15.16)	0.134
Open OFC eliciting dose (mg), median (IQR)	30.00 (3.00–300.00)	30.00 (3.00–35.00)	30.00 (30.00–300.00)	0.00 (−270.00–1.30)	0.275
Adverse effects WAO I, median (IQR)	12.00 (2.75–18.50)	13.00 (3.50–24.00)	10.00 (3.00–17.00)	3.00 (−6.00–15.00)	0.706
Adverse effects WAO I (% of doses), median (IQR)	4.19 (1.22–5.99)	4.74 (0.99–6.91)	3.88 (1.30–5.39)	0.87 (−3.04–3.93)	>0.999
Adverse effects WAO II (% of doses), median (IQR)	0.00 (0.00–0.00)	0.00 (0.00–0.00)	0.00 (0.00–0.00)	0.00 (0.00–0.00)	0.904
Adverse effects WAO II n (%)	2 (8.3)	1 (9.1)	1 (7.7)	1.18 (0.08;16.78)	>0.999
Adverse effects WAO III (% of doses), median (IQR)	0.00 (0.00–0.00)	0.00 (0.00–0.00)	0.00 (0.00–0.00)	-	-
Adverse effects WAO III – positive outcome, n (%)	0 (0.0)	0 (0.0)	0 (0.0)	-	-
Negative final OFC, n (%)	18 (75.0)	9 (81.8)	9 (69.2)	1.18 (0.75–1.87)	0.649

Notes: MD was reported as effect size for continuous variables, RR was reported as effect size of categorical variables. MD and RR not calculated for adverse effects WAO III due to no positive cases observed. Delta was calculated as the difference between measurements at the end of the treatment and at baseline. Groups were compared with Student's *t*-test (OIT duration, no of visits, tahini SPT wheal size at the end of the treatment), Mann–Whitney *U*-test (all variables unless indicated otherwise), or Fisher's exact test (positive outcomes of WAO II).

Abbreviations: SD, standard deviation; IQR, interquartile range; MD, mean/median difference (high dose vs low dose); RR, relative risk (high dose vs low dose); CI, confidence interval.

Table 3 Frequency of Adverse Events During Hospital, Home Escalation, and Home Maintenance Doses (Only Adverse Events Actually Observed in Study Participants are Included)

Adverse Event	Hospital Doses – Build-Up Phase n=330	Home Doses – Build-Up Phase n = 5345	Home Doses – Maintenance n=2104
Oral itching	16	87	7
Skin itching	0	10	10
Mild rash (including perioral)	9	92	2
Mild urticaria (<3 wheals)	11	25	2
Urticaria (≥3 wheals)	5	5	0
Tearing/itching of eyes	0	3	0
Mild sneezing (<10 episodes)	2	37	0
Throat itching/soreness	1	73	14
Hoarseness/stridor	0	1	0
Mild nausea/abdominal pain	5	4	0
Vomiting/diarrhea (<2 episodes)	0	2	0
Significant change in mental status	0	1	0

Table 4 Treatment Outcomes Before and After Treatment

Variable	Baseline	At the End of the Treatment	MD (95% CI)	*p*-value
High-dose group (n = 11)				
Tahini SPT wheal size (mm), median (IQR)	9.00 (7.50–17.50)	5.00 (3.00–5.50)	−4.00 (−14.00–−2.50)	0.004
sIgE, median (IQR)	5.57 (2.18–20.53)	4.51 (2.37–14.80)	−1.06 (−13.80–2.89)	0.765
IgG4, median (IQR)	1.34 (0.77–2.38)	9.40 (4.37–24.25)	8.06 (4.06–145.82)	0.001
Low-dose group (n = 13)				
Tahini SPT wheal size (mm), mean ± SD	12.08 ± 4.65	6.00 ± 2.58	−6.08 (−8.66–−3.50)	<0.001
sIgE, median (IQR)	8.07 (4.06–26.20)	9.18 (5.12–13.30)	1.11 (−8.15–2.16)	0.588
IgG4, median (IQR)	2.00 (0.49–3.10)	3.61 (2.26–14.80)	1.61 (1.04–10.16)	0.002

Notes: Comparisons made with paired *t*-test (tahini SPT wheal size in the low-dose group) or Wilcoxon test (all other comparisons).
Abbreviations: SD, standard deviation; IQR, interquartile range; MD, mean/median difference (at the end of the treatment vs baseline); CI, confidence interval.

Discussion

The aim of developing new treatment regimens is to improve efficacy and compliance, and to reduce the adverse effects of therapy. Trials using low doses of allergens appear to be a new trend in food-specific immunotherapy. The partial results of our study are consistent with the existing literature on this topic. The rate of negative OFCs in our study (81.8% in the high-dose group and 69.2% in the low-dose group; p = 0.649) was comparable to outcomes reported in previous sesame OIT trials,[19–22] indicating similar efficacy across different maintenance dose regimens. Both OIT groups showed

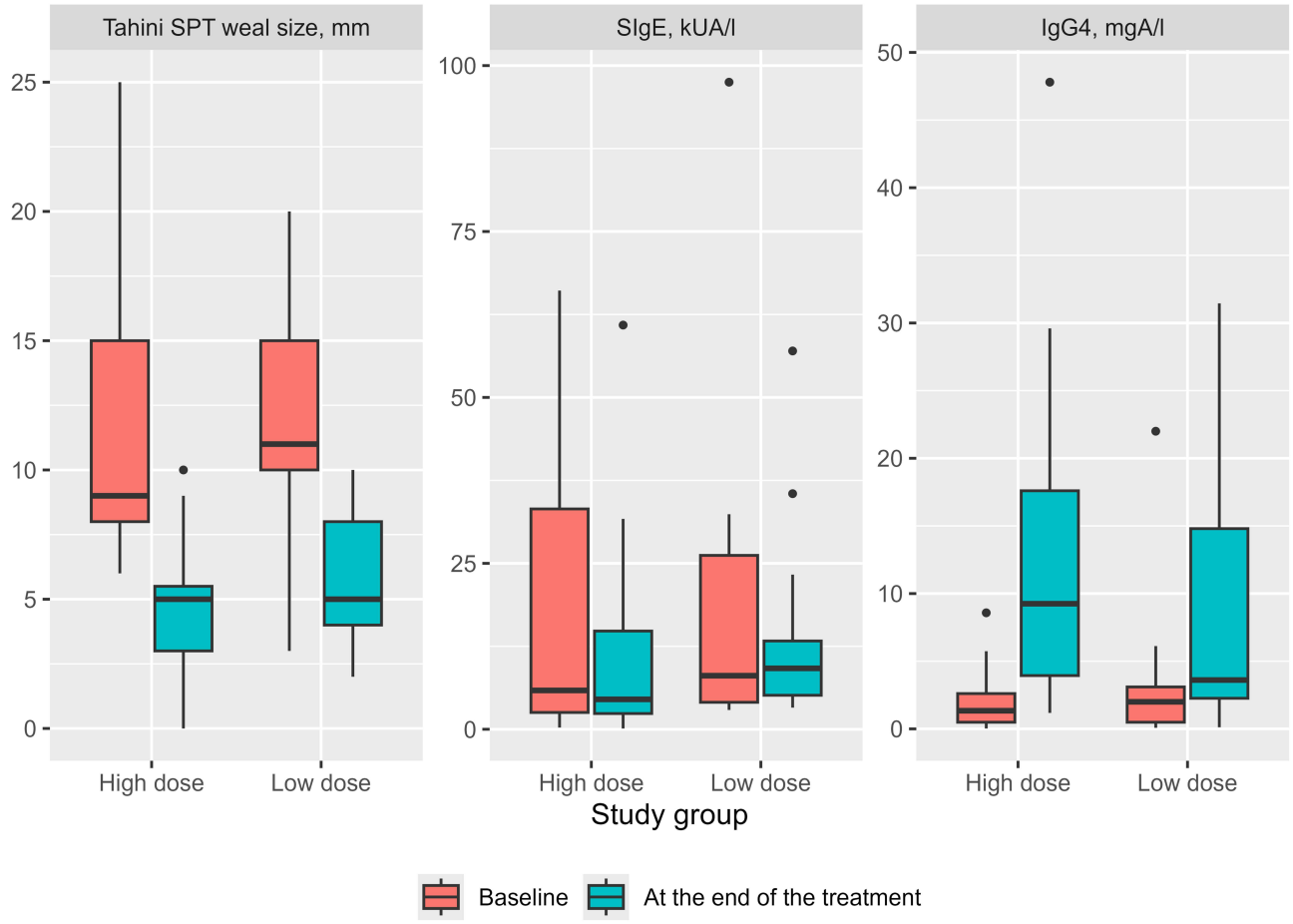

Figure 1 Change in tahini SPT wheal size and IgG4 between the baseline and the end of the treatment in high- and low-dose study groups *(excluding an outlier for IgG4 at the end of the treatment in the high-dose group, IgG4=292.00 mgA/l)*, p values were calculated as paired comparisons between baseline and the measurement at the end of the treatment.

a significant increase in the threshold compared to baseline. In a study, Upton et al compared the efficacy of low (30 mg) and high (300 mg) doses of peanut immunotherapy.[31] In the study, adverse events were less frequent in the group receiving a low-maintenance dose. These results are consistent with the safety assessment of immunotherapy for treating egg and milk allergies.[32,33] Immunologic markers, including reductions in SPT wheal size and increases in sIgG4, followed similar trends in both arms, these findings align with other food OIT trials showing parallel immunological effects across dosing regimens.[34] Changes in concentrations of specific IgG4 have been assessed during OIT with many allergens, e.g. cow milk, peanut or eggs. High levels of allergen-specific IgG4 are observed in successful allergen immunotherapy and are responsible (as one of the immune factors) for achieving tolerance.[35–37]

Most AIT studies investigating sIgE have shown an increase in sIgE level in the beginning of therapy, followed by a decrease during prolonged therapy. Studies on peanuts immunotherapy suggest that the success of treatment depends not only on sIgE concentration, but also on the IgE peptide repertoire. This repertoire was broad at baseline and tended to diminish during therapy. Although sIgE levels decreased, some patients developed novel IgE specificities.[38] This may be one reason why there was no decrease in sIgE concentrations in our study. Another possible explanation is the mechanism whereby IgG4 competes with IgE for allergen binding. The presence of increased food-specific IgG4 alongside persistent sIgE has led to the concept that IgG4 acts as a "blocking antibody" to counteract the adverse functions of IgE.[39]

From a safety perspective, the absence of any WAO grade III (or higher) reactions and the occurrence of only three grade II events across both groups, none requiring adrenaline, reflect a favorable tolerability profile. These observations

https://doi.org/10.2147/JAA.S563421

are consistent with published literature indicating that most adverse events during OIT are mild to moderate when dose escalation protocols are carefully supervised.[40]

Nevertheless, several methodological limitations hinder the interpretation of our findings. First, the interim analysis was unplanned and not specified in the original study protocol. Such post hoc evaluations are exploratory in nature and may increase the risk of type I errors. Second, as a pilot study, a formal sample size calculation was not performed prior to initiation. At the time of study design, the data supporting the efficacy of sesame immunotherapy were limited; therefore, sample size calculations for adequate statistical power were not feasible. Consequently, our study may lack sufficient power to detect true differences between groups, consistent with concerns raised in the pilot and precision-driven experimental design literature. Third, this study was conducted without blinding because the distinctive taste and aroma of tahini make effective masking extremely difficult. Fourth, the study did not include a control group, which limits the ability to distinguish the effects of the intervention from possible spontaneous development of sesame tolerance, as reported in previous studies. Given the limited sample size of this pilot study, the possibility of missing rare but severe reactions cannot be excluded, underscoring the need for larger, adequately powered studies to confirm these findings.

Despite these limitations, our trial provides valuable preliminary insights. It represents one of the first randomized dual-dose pediatric sesame OIT studies, suggesting comparable trends in efficacy and safety between low- and high-dose regimens. These findings suggest that a lower maintenance dose may achieve similar clinical benefits while offering practical advantages in terms of tolerability and patient adherence. This aligns with emerging dose-optimization strategies seen in other food OIT studies.[41]

Further investigations are planned to evaluate long-term outcomes such as sustained unresponsiveness.

Conclusion

This pilot study provides preliminary evidence that both 300 mg and 1200 mg sesame protein maintenance regimens may be effective and well tolerated in pediatric sesame OIT. Both dosing strategies showed similar trends in immunologic responses, clinical efficacy, and safety, with the lower dose potentially offering practical advantages in tolerability and adherence. While these findings are consistent with emerging literature on low-dose approaches in food-specific immunotherapy, they should be interpreted with caution due to the interim, unblinded, and small-scale nature of the study, as well as the absence of a control group. Further adequately powered, blinded, and long-term trials are needed to confirm these observations, optimize dosing strategies, and evaluate sustained unresponsiveness.

Abbreviations

SPT, skin prick test; sIgE, specific immunoglobulin E; BAT, basophil activation test; OFC, oral food challenge; OIT, oral immunotherapy; WAO, World Allergy Organization.

Data Sharing Statement

Individual participant data that underlie the results reported in this article, after deidentification (text, tables, figure) will be available beginning 9 months and ending 36 months following article publication. The study protocol will also be available. Data access will be granted to qualified researchers who submit a reasonable request to the corresponding author.

Acknowledgments

The authors thank all the physicians and nurses involved in this study in our Allergy Clinic.

Disclosure

The authors report no conflicts of interest in this work.

References

1. Bedolla-Barajas M, Bedolla-Pulido TR, Macriz-Romero N, Morales-Romero J, Robles-Figueroa M. Prevalence of peanut, tree nut, sesame, and seafood allergy in Mexican adults. *Rev Invest Clin*. 2015;67(6):379–386.
2. Ben-Shoshan M, Harrington DW, Soller L, et al. A population-based study on peanut, tree nut, fish, shellfish, and sesame allergy prevalence in Canada. *J Allergy Clin Immunol*. 2010;125(6):1327–1335. doi:10.1016/j.jaci.2010.03.015
3. Osborne NJ, Koplin JJ, Martin PE, et al. Prevalence of challenge-proven IgE-mediated food allergy using population-based sampling and predetermined challenge criteria in infants. *J Allergy Clin Immunol*. 2011;127(3):668–76.e1–2. doi:10.1016/j.jaci.2011.01.039
4. Peters RL, Koplin JJ, Gurrin LC, et al. The prevalence of food allergy and other allergic diseases in early childhood in a population-based study: healthNuts age 4-year follow-up. *J Allergy Clin Immunol*. 2017;140(1):145–153.e8. doi:10.1016/j.jaci.2017.02.019
5. Sokol K, Rasooly M, Dempsey C, et al. Prevalence and diagnosis of sesame allergy in children with IgE-mediated food allergy. *Pediatr Allergy Immunol*. 2020;31(2):214–218. doi:10.1111/pai.13143
6. Warren CM, Chadha AS, Sicherer SH, Jiang J, Gupta RS. Prevalence and severity of sesame allergy in the United States. *JAMA Network Open*. 2019;2(8):e199144. doi:10.1001/jamanetworkopen.2019.9144
7. Lemoine-Courcelles C, Abrams EM, Kim H, Povolo B, Gerdts J, Protudjer JLP. Sesame allergy a time burden for families of children with multiple food allergies. *J Allergy Clin Immunol Pract*. 2021;9(4):1753–1755.e1. doi:10.1016/j.jaip.2020.11.031
8. Nguyen K, Greenthal E, Sorscher S, Lurie P, Spergel JM, Kennedy K. Adverse events and labeling issues related to suspected sesame allergy reported in an online survey. *Ann Allergy Asthma Immunol*. 2022;128(3):279–282. doi:10.1016/j.anai.2021.12.005
9. Oriel RC, Elizur A, Sicherer SH. Comprehensive diagnosis, management, and treatment of sesame allergy. *J Allergy Clin Immunol Pract*. 2024;12(3):590–597. doi:10.1016/j.jaip.2023.11.008
10. Mahlab-Guri K, Guri A, Kadar L, et al. Characteristics of patients with spontaneous resolution of sesame allergy. *Ann Allergy Asthma Immunol*. 2022;128(2):206–212. doi:10.1016/j.anai.2021.10.031
11. Cohen A, Goldberg M, Levy B, Leshno M, Katz Y. Sesame food allergy and sensitization in children: the natural history and long-term follow-up. *Pediatr Allergy Immunol*. 2007;18(3):217–223. doi:10.1111/j.1399-3038.2006.00506.x
12. Aaronov D, Tasher D, Levine A, Somekh E, Serour F, Dalal I. Natural history of food allergy in infants and children in Israel. *Ann Allergy Asthma Immunol*. 2008;101(6):637–640. doi:10.1016/s1081-1206(10)60228-1
13. Kazancioglu A, Ocak M, Sahiner UM, Soyer O, Sekerel BE. Natural history of sesame allergy in pediatric patients: insight from a retrospective analysis. *Pediatr Allergy Immunol*. 2025;36(1):e70022. doi:10.1111/pai.70022
14. Riggioni C, Ricci C, Moya B, et al. Systematic review and meta-analyses on the accuracy of diagnostic tests for IgE-mediated food allergy. *Allergy*. 2024;79(2):324–352. doi:10.1111/all.15939
15. Goldberg MR, Appel MY, Nachshon L, et al. Combinatorial advantage of Ses i 1-specific IgE and basophil activation for diagnosis of sesame food allergy. *Pediatr Allergy Immunol*. 2021;32(7):1482–1489. doi:10.1111/pai.13533
16. Santos AF, Riggioni C, Agache I, et al. EAACI guidelines on the diagnosis of IgE-mediated food allergy. *Allergy*. 2023;78(12):3057–3076. doi:10.1111/all.15902
17. Ocak M, Sahiner UM, Soyer O, Sekerel BE. The role of diagnostic tests and oral food challenge results to predict sesame allergy. *Ann Allergy Asthma Immunol*. 2022;128(1):46–52.e1. doi:10.1016/j.anai.2021.09.005
18. Saf S, Borres MP, Södergren E. Sesame allergy in children: new insights into diagnosis and management. *Pediatr Allergy Immunol*. 2023;34(8):e14001. doi:10.1111/pai.14001
19. Nachshon L, Goldberg MR, Levy MB, et al. Efficacy and safety of sesame oral immunotherapy-A real-world, single-center study. *J Allergy Clin Immunol Pract*. 2019;7(8):2775–2781.e2. doi:10.1016/j.jaip.2019.05.031
20. Chua GT, Soller L, Kapur S, et al. Real-world safety and effectiveness analysis of low-dose preschool sesame oral immunotherapy. *J Allergy Clin Immunol Glob*. 2024;3(1):100171. doi:10.1016/j.jacig.2023.100171
21. Salari F, Bemanian MH, Fallahpour M, et al. The effectiveness of oral immunotherapy in patients with sesame anaphylaxis using omalizumab. *Clin Med Res*. 2022;20(3):125–132. doi:10.3121/cmr.2022.1730
22. Shah A, Cox AL, Groetch M, et al. Sesame oral immunotherapy outcomes in a pediatric cohort. *J Allergy Clin Immunol Pract*. 2025;13(4):938–940.e1. doi:10.1016/j.jaip.2025.01.036
23. Sampson HA, Gerth van Wijk R, Bindslev-Jensen C, et al. Standardizing double-blind, placebo-controlled oral food challenges: American academy of allergy, asthma & immunology-european academy of allergy and clinical immunology PRACTALL consensus report. *J Allergy Clin Immunol*. 2012;130(6):1260–1274. doi:10.1016/j.jaci.2012.10.017
24. R: a language and environment for statistical computing. Version 4.4.2. R Foundation for Statistical Computing; 2024. Available from: https://www.R-project.org/. Accessed January 28, 2026.
25. stringr: simple, consistent wrappers for common string operations. Version 1.5.1. *R package*; 2023. Available from: https://CRAN.R-project.org/package=stringr. Accessed January 28, 2026.
26. tidyr: tidy messy data. Version 1.3.1. *R package*; 2024. Available from: https://CRAN.R-project.org/package=tidyr. Accessed January 28, 2026.
27. purrr: functional programming tools. Version 1.0.2. *R package*; 2023. Available from: https://CRAN.R-project.org/package=purrr. Accessed January 28, 2026.
28. dplyr: a grammar of data manipulation. Version 1.1.4. *R package*; 2023. Available from: https://CRAN.R-project.org/package=dplyr. Accessed January 28, 2026.
29. Fox JWS. *An R Companion to Applied Regression*. Third edition ed. Sage; 2019.
30. epitools: epidemiology tools. Version 0.5-10. *R package*; 2024. Available from: https://CRAN.R-project.org/package=epitools. Accessed January 28, 2026.
31. Upton JEM, Rivero DT, Ke D, et al. Peanut oral immunotherapy using 30 mg and 300 mg maintenance doses. *J Allergy Clin Immunol Pract*. 2025;14(1):223–232.e7. doi:10.1016/j.jaip.2025.10.007
32. Yanagida N, Sato S, Asaumi T, Nagakura K, Ogura K, Ebisawa M. Safety and efficacy of low-dose oral immunotherapy for Hen's egg allergy in children. *Int Arch Allergy Immunol*. 2016;171(3–4):265–268. doi:10.1159/000454807

33. Takaoka Y, Yajima Y, Ito YM, et al. Single-center noninferiority randomized trial on the efficacy and safety of low- and high-dose rush oral milk immunotherapy for severe milk allergy. *Int Arch Allergy Immunol*. 2020;181(9):699–705. doi:10.1159/000508627
34. Nurmatov U, Devereux G, Worth A, Healy L, Sheikh A. Effectiveness and safety of orally administered immunotherapy for food allergies: a systematic review and meta-analysis. *Br J Nutr*. 2014;111(1):12–22. doi:10.1017/s0007114513002353
35. Alves-Correia M, Gaspar Â, Borrego LM, Azevedo J, Martins C, Morais-Almeida M. Successful oral desensitization in children with cow's milk anaphylaxis: clinical and laboratory evaluation up to nine-years follow-up. *Allergol Immunopathol*. 2019;47(2):133–140. doi:10.1016/j.aller.2018.07.007
36. Chinthrajah RS, Purington N, Andorf S, et al. Sustained outcomes in oral immunotherapy for peanut allergy (POISED study): a large, randomised, double-blind, placebo-controlled, Phase 2 study. *Lancet*. 2019;394(10207):1437–1449. doi:10.1016/s0140-6736(19)31793-3
37. Giavi S, Vissers YM, Muraro A, et al. Oral immunotherapy with low allergenic hydrolysed egg in egg allergic children. *Allergy*. 2016;71(11):1575–1584. doi:10.1111/all.12905
38. Vickery BP, Lin J, Kulis M, et al. Peanut oral immunotherapy modifies IgE and IgG4 responses to major peanut allergens. *J Allergy Clin Immunol*. 2013;131(1):128–34.e1–3. doi:10.1016/j.jaci.2012.10.048
39. Santos AF, James LK, Bahnson HT, et al. IgG4 inhibits peanut-induced basophil and mast cell activation in peanut-tolerant children sensitized to peanut major allergens. *J Allergy Clin Immunol*. 2015;135(5):1249–1256. doi:10.1016/j.jaci.2015.01.012
40. Lodge CJ, Waidyatillake N, Peters RL, et al. Efficacy and safety of oral immunotherapy for peanut, cow's milk, and hen's egg allergy: a systematic review of randomized controlled trials. *Clin Transl Allergy*. 2023;13(7):e12268. doi:10.1002/clt2.12268
41. Sato S, Nagakura KI, Yanagida N, Ebisawa M. Recent advances in oral immunotherapy for food allergies. *Clin Rev Allergy Immunol*. 2025;68(1):89. doi:10.1007/s12016-025-09108-4

Journal of Asthma and Allergy

Dovepress
Taylor & Francis Group

Publish your work in this journal

The Journal of Asthma and Allergy is an international, peer-reviewed open-access journal publishing original research, reports, editorials and commentaries on the following topics: Asthma; Pulmonary physiology; Asthma related clinical health; Clinical immunology and the immunological basis of disease; Pharmacological interventions and new therapies. The manuscript management system is completely online and includes a very quick and fair peer-review system, which is all easy to use. Visit http://www.dovepress.com/testimonials.php to read real quotes from published authors.

Submit your manuscript here: https://www.dovepress.com/journal-of-asthma-and-allergy-journal