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# *From to Intervention: Immune Mechanisms and Therapeutic Challenges in Peanut Allergy*

**Yating Yang**

*Faculty of Science, The University of Melbourne, Parkville, Australia*
*yating_yang_0102@163.com*

***Abstract.*** Although therapeutic immunotherapy, particularly peanut oral immunotherapy, demonstrate that clinical allergic reactivity can be modified by increasing reaction thresholds under ongoing treatment, seldom is long-term immune toleration or disease recovery achieved. This review will give a summary of the existing knowledge on the immunological pathways that cause peanut allergy and primarily dwell on the known genetic and environmental risk factors, the immunological pathway of the Th2-IgE, and immune regulation. It further examines the immunology and clinical results of oral immunotherapy (OIT) using peanuts, and has found that it has partial immunological effects instead of induction of immunological tolerance. Recurrent debates on the usefulness, lastingness and security of OIT are deliberated as well as the new substitute and adjunctive treatment. Finally, the review emphasizes the critical need for reliable biomarkers that identify those patients who are most likely to benefit from therapy and those at increased risk of adverse outcomes. Addressing these challenges will be essential to advance more personalized and safer therapeutic strategies for peanut allergy.

***Keywords:*** Peanut allergy, IgE-mediated hypersensitivity, Oral immunotherapy, Food allergy management.

## 1. Introduction

Peanut allergy is a world's common and urgent health issues, especially in industrialized countries [1]. The latest studies set the number of children with this type of allergy in Western countries at 1-3%, and they demonstrate a substantial growth in incidence over the past decade [2]. In contrast, available evidence suggests that prevalence remains lower in many Asian and developing regions, though comprehensive data continue to be inadequate [1]. Peanut allergy typically begins early in life and persists in 75-80% of affected children, so it presents as one of the least likely childhood food allergies to be outgrown [3]. Overall, these patterns indicate the growing burden of peanut allergy on both a national and global scale [4].

By all accounts, peanut allergy is a much more serious clinical manifestation compared to other types of food allergies [5]. Peanuts are a leading cause of anaphylaxis and are associated with a disproportionate number of life-threatening reactions [6]. In Western populations, studies and surveys show that peanut-allergic individuals report more severe past reactions than those with other food allergies. An estimated 33-50% patients may experience anaphylaxis at some period of time in

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their life [5]. Despite limited surveys reporting the extent of fatalities, peanuts are identified as the primary cause of fatal food-induced anaphylaxis [6]. Immunologically, peanut allergy is mediated by robust IgE- and Th2-skewed immune responses, with around 15-25% being able to recover from the allergy on the account of natural tolerance [7]. This strong IgE/Th2 skew helps explain both the high reactivity and the persistence of the condition.

Management options for peanut allergy remain limited and primarily focus on avoidance. However, complete avoidance is difficult due to peanut's widespread presence in food items, which causes accidental exposures in roughly 7-14% of patients per year. Families often experience dietary restriction, constant vigilance and substantial anxiety as a result. Recently, the FDA approved the first OIT product designed to raise reaction thresholds in children [5]. Nevertheless, OIT carries an important drawback, it concurrently raises the reaction thresholds while raising the rate of adverse events. One analysis reported a three times higher risk of anaphylaxis and increased epinephrine injection compared to avoidance alone [4]. In clinical practice, up-dosing can result in 10% of the patients who experience systemic reactions, and during maintenance, the figure goes up to 19%. Moreover, 15–30% of patients fail to attain meaningful desensitization [5]. While OIT represents an important advance in peanut allergy management, its clinical benefits are entangled with a number of difficulties, including the high number of adverse events, the interference with treatment, the variation in the responses of the treatment, and the doubting long-term outcomes.

The escalating rate, severity, and limitations of conventional treatments in the realm of peanut allergies compel the introduction of novel treatments for better management. Further understanding of the mechanisms of immune system in all responses to peanut allergy and treatment could be beneficial in forms of therapies with more effective and safe methodologies [8]. This study examines immunological markers associated with peanut allergy and responses to therapy, ultimately supporting the development of better long-term management strategies.

## 2. Immunological mechanisms of peanut allergy

### 2.1. Risk factors for peanut allergy

This section will discuss the genetic predisposition of peanut sensitization undergoes from sensitization to the development of a peanut allergy. Peanut allergy develops through a complex interaction between genetic susceptibility and environmental exposures. Findings have demonstrated that genes affecting skin barrier function and immunity are implicated. A good example is the loss-of-function mutations in filaggrin (FLG), an original skin system protein, which totally increase the chance of peanut sensitization [7]. Likewise, several HLA class II alleles and other immune-related genes, such as MALT1 have been found to contribute to peanut allergy [9]. The LEAP study established that the risk alleles category of three genes-FLG, MALT1, and HLA-DQA1, had a significant impact in enhancing peanut allergy incidents in infants who were peanut avoidant rather than peanut consume. This highlights the interaction of genetic susceptibility interacts with environmental factors [9]. Most recent genome-wide association studies have also indicated some new susceptibility loci related to peanut allergy such as C11orf30/EMSY, SKAP1, CTNNA3, ITGA6 and ANGPT4 thus confirming heterogeneous disease architecture genetically [3,10]. Family members with atopy and other food confirms are also a risk factor, the occurrence of which, together with the existence or presence of one first-degree relative has been shown to significantly predispose food allergy among adolescents [11]. Overall, these findings provide evidence of a model in which inherited defects in epithelial barrier function and immune regulation confer susceptibility to peanut

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allergy, but clinical disease expression depends on interaction with environmental exposures rather than genetic factors alone [9,11].

Moreover, the detrimental effects of environmental exposures on the risk of peanut allergy are significant. According to the dual-allergen-exposure hypothesis, exposure to peanut protein through skin barrier disruption (especially in eczema cases) often leads to sensitization, while periodic early oral exposure mostly results in tolerance [11]. Delayed peanut introduction in infancy markedly increases the risk of allergy: one study found that British infants (late introduction) had almost ten times higher peanut allergy than Israeli infants who consumed peanuts from the age of seven months [6]. However, while this observational difference is striking, it may lead to possible precautions regarding residual confounding factors, and thus the purpose of this provides only evidence rather than proof of a causal relationship. Other factors also influence immune priming, as early-life microbial exposures, such as household dogs or older siblings, which may lead to the reduced risk of food allergy. The correlation between vitamin D deficiency and Western lifestyle can be linked to increase in allergic diseases cases [11]. Migration and ancestry studies reveal complex gene–environment interactions, as children of Asian descent born in Australia have much higher rates of peanut allergy compared to those who were born in Asia, despite their shared ancestry. Such patterns underscore that diet, microbial environment, and mucosal health (e.g., skin integrity, vitamin D) can tip the balance toward or away from peanut sensitization [10,11].

## 2.2. Immune response mechanisms

Peanut sensitization occurs when antigen-presenting cells capture peanut proteins and prime CD4+ T cells to differentiate a Th2 phenotype [7]. In the gut or skin, peanut proteins presented on MHC class II molecules by dendritic cells touch naïve T cells. The T cells are then influenced by the early secretion of IL-4 from the basophils and innate immune cells and undergo a switch to the Th2 phenotype towards the end of the maturation process [7]. Th2 cells produce IL-4 and IL-13, thus, promoting B cell class switching and production of peanut-specific IgE antibodies. Knockout scientists who lack IL-4, IL-13, or STAT6 pathways reveal that these animals do not mount IgE responses. These observations demonstrate the pivotal function of the IL-4/IL-13/STAT6 signaling pathways in favoring the Th2 bias and switching immunoglobulins [12]. At the same time, regulatory T cells and innate cytokines can modulate the sensitization process. Nevertheless, the dominance of Th2 response fosters allergic symptoms [3]. This means that Th2-polarized effector T cell responses and induced regulatory T cells mediate critical balancing functions that determine whether peanuts provoke allergic sensitization or immune tolerance.

Upon re-exposure to peanuts, IgE can then bind to the mast cells and basophils through high-affinity FcεRI receptors, resulting in hypersensitivity of the individual due to the immediate reactions triggered [13]. IgE molecules bound to FcεRI on mast cells and basophils are cross-linked by the allergen, initiating an intracellular signaling cascade by ITAM motifs that involve kinases such as Lyn and Syk. This process is referred to the degradation of mediators such as histamine, leukotrienes, platelet-activating factor, and proteases [7]. These mediators produce the clinical manifestation of peanut allergy. To illustrate this, histamines cause smooth muscle contraction in addition to endothelial markers such as hypersensitivity hives, bronchospasm, and gastrointestinal symptoms, while cytokines like IL-4, IL-5, and IL-13 invoke consequences like eosinophilia and Th2 inflammation [14]. In more severe reactions, this process can progress to anaphylaxis and systemic shock. Hence, peanut allergy reflects a Th2-IgE axis both in inherited and environmental factors act together to bias the immune system towards sensitization mediated IgE.

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Subsequently, IgE-dependent interactions between allergens and effector cells, particularly mast cells, drive the clinical manifestations of allergic reactions.

## 3. Peanut oral immunotherapies

### 3.1. Immunological mechanisms of peanut oral immunotherapy

Peanut OIT is designed to desensitize an allergic person to peanut protein and allow the person to eat more of the food without a clinical reaction, as opposed to curing the allergy [5]. The desensitization is the most important immunological outcome of OIT that is based on the constant allergen exposure and is therefore reversible in case of treatment is discontinued [5]. The smaller percentage of patients attains the persistence of unresponsiveness, meaning that tolerance persists after a period of avoidance, but this outcome is not common and remains difficult to predict [15].

In therapy, the peanut-specific IgE antibodies levels usually increase initially during the course of the initial up-dosing phase and then decrease slowly throughout the long-term therapy [16]. In contrast, the level of peanut-specific IgG4 antibodies continue to rise steadily and are thought to be to be a significant predictor of successful desensitization [16]. Competition with the peanut allergens by IgG4 antibodies causes dichain complexes to form between the mast cell or basophil activation and the target cell, and lengthen the period of desensitization [15]. Notably, recent mechanistic studies indicate that neutralization, as opposed to excessive levels of IgG4, of certain IgG4 antibodies is associated with enduring and significant benefit to patients following OIT [15].

OIT takes effect on cell immune response as well. Several studies have shown that improved generation of Th2-type cytokine does so, such as IL-4 and much like IL-5 is linked with effective OIT [13,17]. Meanwhile, there is an increase in the pathway of T cell activities, that is regulatory T cells. This therefore assists in the inhibition of allergic inflammation [8]. Also, these findings give evidence of OIT not only reshaping antibody profiles but also changing T cell polarization and commanding immune regulation. Multi-omics analysis further demonstrates indicates a high level of heterogeneity in the treatment response with complete responders exhibiting the presence of transcriptional and epigenetic signatures related to immune regulation, and incomplete responders the signature of innate immune activation and metabolic stress presence. Multi-omics studies also show that patients that respond to OIT have transcriptional and epigenetic profiles of immune regulation, and incomplete responders have patterns that are consistent with innate immune activation and metabolic stress [8]. Accordingly, effectiveness of OIT is by biological variation and can not only be correlated to the dose or period of treatment.

The suppression of effector cell reactivity is another major immunological mechanism. Basophil activation markers including CD63 expression and SYK phosphorylation, decrease with OIT and therefore can be measured early in the treatment plan within the first three months [16]. The resulting decrease of basophil responses suggests a downstream inhibition of IgE signaling pathways, progressively filling out a greater clinical threshold during food challenges [13]. However, basophil hypo responsiveness cannot predict which patients will sustain unresponsiveness because thus it cannot be an independent immunological marker [16].

### 3.2. Clinical application of peanut oral immunotherapy

Large randomized trials and meta-analyses consistently show that OIT increases reaction thresholds in most children with anaphylaxis, reducing the risk of severe reactions involuntarily exposed and supervised eating environment, despite the treatment sometimes can be accompanied by allergic

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reactions [4,5]. Patients report a significant improvement in their quality of life, especially in terms of reduced anxiety due to accidental ingestion [18]. However, OIT is also linked with many adverse effects, covering gastrointestinal symptoms and systemic allergic reactions, mainly in the course of dose escalation [4].

The safety of long-term therapy still remains a huge problem to be solved. Editorial analyses of extended OIT trials indicate that the patients still experience immune-mediated responses, which usually lead to systemic reactions during preserve maintenance phase and even are higher than the reactions observed under strict avoidance alone [19]. Adherence is another challenge, since daily long-term dosing is commonly required to maintain desensitization. This may cause some of the patients to suspend their therapy [19]. Adult OIT studies conducted with real peanut products show a significant increase in dosages tolerated. However, withdrawal due to adverse events remains frequent, and evidence for older age groups, such as adults, still remains limited compared with pediatric populations [18].

Overall, peanut OIT represents a meaningful but imperfect therapeutic option. Given that, biomarkers that identify patients with a better chance of responding and whose treatment reaction leads to meaningful outcomes, can be realized, such as sustained unresponsiveness or higher reaction thresholds. At the same time, it can prevent those at increased risk of severe adverse events or poor treatment adherence, should be discovered. Its benefits are strongest for reducing treatment-related risks linked to unintentional exposure, in comparison to inducing permanent tolerance, but outcome varies a lot between patients [8]. Current evidence supports careful patient selection and shared decision-making, with ongoing research needed to identify biomarkers that can predict long-term treatment success and improve safety [15].

## 4. Controversies and future directions in peanut allergy treatment

A major controversy in the treatment of peanut allergy shows how therapeutic success should be defined and interpreted in real-world contexts, particularly whether it refers to desensitization achieved under ongoing treatment, sustained unresponsiveness after treatment cessation, or a clinically meaningful reduction in the risk of accidental reactions [20].

A large number of clinical trials and meta-analyses illustrate that OIT elevates reaction thresholds in the clinical environment, thereby reducing risk of significant clinical reactivity to accidental exposure [4]. However, these outcomes are often measured by double-blind, placebo-controlled food challenges and may not fully reflect the safety and improvements achieved in the everyday life conditions [20]. This is of clinical importance because immunotherapy may confer fluctuating tolerance parameters, as the presence of co-factors (viral illnesses, intra-exercise periods, non-steroidal anti-inflammatory medications, and sleep deprivation) may further lower reactivity threshold [3]. OIT illustrates a central conflict in peanut allergy research-a net improvement in desensitization efficacy is accompanied by higher rates of adverse clinical reactions, which may surpass those sustained by strict reactivity avoidance, even in desensitized patients [4]. Considered effective in desensitizing peanut-allergic patients, meta-analyses also evidence higher rates of allergic and anaphylactic reactions compared with avoidance [4]. This impacts the determination of relative benefit versus the risk of sequential routine accidental exposure [4]. Lack of durability represents a fundamental constraint on OIT as a disease-modifying therapy, as sustained unresponsiveness after treatment discontinuation appears rare, and reducing or stopping maintenance dosages is often associated with a return of clinical reactivity [3]. Finally, long-term daily dosing poses adherence challenges, which may limit feasibility for some patients and families [20].

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However, these limitations have prompted interest in treatment modalities that are more focused on the immune pathways rather than the effect of allergen exposure alone. Biologic therapy study, including the therapeutic effect of anti-IgE therapy, have been increasingly explored [20]. Recent studies and reports show that omalizumab can effectively increase reaction thresholds in both peanut and other allergen reactions, even at the lowest level of allergen molecule. This proves its current status as a risk-reduction agent, though not a treatment in itself, and its effect as an adjunct in different groups of patients may influence its potential to moderate risk of clinical reactivity [21]. Nevertheless, it is not yet clear that biologics have long-term significant effects on the natural history and may only have effects in the short-term [20]. In addition, clinical methods of biologics and OIT with combined therapy have not always managed to remove treatment-related side effects, underscoring that targeting a single immune pathway response can prove ineffective [22].

Future Directions Biomarker study is a significant emphasis of peanut allergy treatment strategy [20]. Currently, no biomarkers are accepted as primary efficacy endpoints for regulatory approval, and clinicians rely heavily on food challenges that are resource-intensive and carry inherent risks [20]. Clinical prognostic instruments are required in order to aid in clinical treatment discrimination [23]. In particular, biomarkers could help address two critical clinical questions: identifying patients most likely to achieve meaningful benefit, such as high potential for sustained unresponsiveness and clinically significant elevations in reaction thresholds, as well as those likely to sustain statistics of serious adverse reactions and poor adherence [3,20].

Addressing these challenges will enhance the safety and efficacy of beneficial peanut therapy treatment strategies [20].

## 5. Conclusion

Peanut allergy is a complex reaction between genetic susceptibilities and environmental reactive exposures, which causes the elaboration of Th2-dominated reactions and the activation of effector cells by IgE. Current evidence shows that allergic sensitization is characterized by a biased immune response and not a unitary pathway, whereas IgE-dependent mast cells and basophil trigger clinical responses. The combination of these processes constitutes the cause of peanut allergy persistence, as well as its possible response to immunomodulatory treatments.

Development of immunotherapy, especially OIT, proves that the disease reactivity may be modulated, particularly by potentially substantial increase of reactivity thresholds, sustained as longer treatment proceeds. These effects are significant risk reduction, which is observed under controlled conditions, and are likely to reduce the risk of adverse reactions after unintentional exposure. Nonetheless, durable disease remission or immune tolerance is scarcely ever seen. The phenomenon of desensitization is therefore a phenomenon of reduced short time reactivity but not permanent organic immunological remission. The observation that sustained unresponsiveness after treatment discontinuation is uncommon highlights a fundamental limitation of OIT as a disease-modifying therapy, rather than merely an unresolved technical challenge.

Moreover, OIT treatment benefits are further negated by risks associated with treatment, sustained adverse reactions, compliance issues as well as massive inter-individual response heterogeneity. These restrictions have provoked increasing interest in both alternative and adjunctive therapies, including alternative immunotherapies by different routes, and anti-IgE therapy as a biologic treatment. Although these strategies can be effective risk-reduction measures or more lenient treatment facilitators, there is no doubt that they cannot have any meaningful effect on peanut allergy natural history in the long run.

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The future of the management of peanut allergy will rely on the further publication of pertinent insights into the heterogeneity of the immunity and the creation of dependable biomarkers. Biomarkers that can foresee clinical benefit with any meaning, risk during treatment, or prognosis over time are required in assisting in personalized decisions. Combining mechanistic and clinical stratification will be of paramount importance in developing safer, more effective, and patient-centered approaches to the therapy.

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