

Nutritional Surplus and the Allergenic Source Term: A Hypothesis Linking Nitrogen Input in Peanut Cultivation to Rising Peanut Allergy Prevalence

Dr PD Prideaux

Independent Researcher, Stockton-on-Tees, United Kingdom

Abstract

The prevalence of peanut (*Arachis hypogaea*) allergy has risen markedly over the late twentieth and early twenty-first centuries, particularly in industrialized nations. Dominant explanatory models have focused on changes in human immune development, such as the hygiene hypothesis, or on post-harvest processing effects, particularly thermal modification during roasting. This paper proposes an additional, upstream variable: a change in the biochemical composition of the peanut itself driven by modern agricultural practices. We advance a *Source Term Hypothesis*, suggesting that the transition from low-input cultivation in nitrogen-limited soils to intensive NPK fertilization regimes has increased the absolute concentration of nitrogen-rich seed storage proteins, including the major peanut allergens Ara h 1, Ara h 2, and Ara h 3. Even in the absence of altered human immune sensitivity, an increased allergenic protein density per unit mass of peanut would raise exposure flux, potentially lowering thresholds for sensitization and clinical reactivity at the population level. We outline the biochemical rationale for this hypothesis, address potential objections, and propose experimental approaches for its verification.

1 Introduction

Peanut allergy is among the most severe and persistent food allergies, affecting approximately 1–2% of children in Western nations—a prevalence that has increased substantially since the 1990s [1]. The rise has been particularly dramatic in countries such as the United States, United Kingdom, Canada, and Australia. This trend represents a significant public health concern given the severity of reactions, the persistence of the condition into adulthood, and the substantial impact on quality of life for affected individuals and their families.

Explanatory frameworks have largely centred on host-side factors, including reduced microbial exposure during early childhood (the hygiene hypothesis) [2], delayed dietary introduction of peanuts to infants [3], vitamin D deficiency, and immunological skewing during development. In parallel, food science research has identified post-harvest processing—particularly roasting—as a modifier of peanut allergenicity through structural and chemical alteration of allergenic proteins, with dry-roasting potentially enhancing allergenicity through Maillard reaction products [4].

These approaches implicitly treat the peanut as a biochemically static object whose intrinsic allergen content is invariant across historical and agricultural contexts. This assumption may be unwarranted. Over the same period that peanut allergy prevalence has increased, global peanut cultivation has undergone profound changes, transitioning from traditional, low-input systems—often on marginal, nitrogen-depleted soils—to high-yield, fertilizer-supported monocultures optimized for maximum productivity. The potential consequences of this transition for the seed proteome, and specifically for allergenic protein abundance, have received little attention in the allergy literature.

This paper proposes that changes in agricultural nitrogen availability may have altered the *source term* of allergenic exposure, independently of changes in human immune sensitivity or food processing practices.

2 Agricultural Nitrogen Input and Seed Protein Expression

Arachis hypogaea is a leguminous crop capable of biological nitrogen fixation via symbiosis with *Rhizobium* species. In nitrogen-limited soils, this symbiosis supplies nitrogen sufficient for vegetative growth and seed development. Under conditions of elevated soil nitrogen availability, however, legumes may partially or fully down-regulate fixation while still experiencing increased net nitrogen availability during seed filling, depending on cultivar, timing, and soil conditions.

Plants are known to exhibit “luxury consumption” of nutrients when availability exceeds immediate growth requirements [5]. In such conditions, excess nitrogen is commonly partitioned into storage compounds, particularly seed storage proteins (SSPs), which serve as nitrogen reservoirs for germination. This phenomenon is well-documented in agricultural research, where protein content of legume seeds typically increases with nitrogen fertilization.

The principal peanut allergens are themselves SSPs [6]:

- **Ara h 1 and Ara h 3**, members of the cupin superfamily (vicilin- and legumin-type globulins), accounting for substantial proportions of total seed protein, and
- **Ara h 2**, a 2S albumin, notable for its proteolytic and thermal stability and high allergenic potency despite representing a smaller fraction of total protein [7].

It is therefore plausible that increased nitrogen availability during seed development leads to an increase in the absolute abundance of these proteins within the mature seed. This hypothesis does not require preferential up-regulation of allergenic proteins relative to other SSPs; an increase in total SSP content alone would increase the absolute allergen dose per gram of peanut consumed.

3 The Allergenic Source Term and Exposure Flux

In physical detection systems, event probability is determined by the product of incident flux and detector cross-section. An analogous framing can be applied to food allergy. The probability of sensitization or elicitation depends both on host immune sensitivity (the “cross-section”, σ) and on the quantity and biochemical form of allergen encountered (the “flux”, Φ).

$$P_{\text{reaction}} \propto \Phi_{\text{source}} \times \sigma_{\text{host}} \quad (1)$$

Most contemporary allergy research has focused on the cross-section term: genetic predisposition, immune regulation, microbiome composition, and timing of exposure. The present hypothesis instead addresses the *flux*: the allergenic protein density of the food source. An increase in the absolute concentration of Ara h proteins per unit mass of peanut would have several potential consequences:

- **Lower effective thresholds for reaction**, increasing the likelihood that low-sensitivity individuals exhibit clinical symptoms.
- **Enhanced early-life sensitization**, if higher allergen doses overwhelm mechanisms of oral tolerance during immune development.
- **Amplification of processing effects**, whereby thermal or chemical modifications act upon a larger initial allergen pool, potentially producing more immunogenic products.

This framework is compatible with, rather than exclusive of, existing immunological and processing-based models. The Source Term Hypothesis suggests that multiple factors may act multiplicatively: changes in immune development, processing methods, and intrinsic allergen content could all contribute to the observed epidemiological trends.

4 Distinction from Stress-Induced Allergen Models

Previous plant-based explanations for variable allergenicity have emphasized abiotic stress, such as drought or pathogen pressure, leading to increased expression of defensive proteins (Pathogenesis-Related proteins). The present hypothesis differs fundamentally in mechanism and implication. Rather than stress-induced defence, it proposes that agricultural optimization itself—through nutrient surplus—has unintentionally increased allergenic load by enhancing storage protein accumulation.

Paradoxically, this suggests that highly “successful” agricultural systems, optimized for yield and protein content, may produce food that is immunologically more potent than their low-input predecessors.

5 Addressing Potential Objections

5.1 Geographic Variation in Allergy Prevalence

One potential objection to this hypothesis is that countries with intensive peanut agriculture (notably China and India) do not exhibit the high allergy prevalence observed in Western nations. Several factors may contribute to this discrepancy:

First, consumption patterns differ markedly: boiled peanuts are traditional in many Asian countries, whereas dry-roasted peanuts predominate in Western markets [8]. Second, early-life exposure patterns differ, with earlier and more consistent peanut introduction in some Asian populations potentially promoting tolerance even in the presence of higher allergen doses [3]. Third, Western markets increasingly source peanuts internationally, and export-oriented production may employ different fertilization regimes than domestic cultivation.

5.2 Other Legumes and Agricultural Crops

A related question is whether allergies to other legumes have risen comparably. Peanuts are botanically distinct and consumed in larger quantities in more allergenic forms (roasted) than many other legumes. Furthermore, the specific allergenic proteins in peanuts, particularly Ara h 2, are unusually stable and potent [7]. Nevertheless, the hypothesis would predict that if similar allergen-content increases have occurred in other crops, effects should be observable at the population level.

6 Proposed Experimental Tests

The Source Term Hypothesis is experimentally tractable. A direct test could involve:

- **Cultivation conditions:** Genetically identical cultivars grown under controlled nitrogen regimes, ranging from reliance on biological fixation to escalating NPK supplementation.
- **Proteomic analysis:** Quantification of Ara h 1, 2, and 3 using Liquid Chromatography–Mass Spectrometry (LC-MS) [6].
- **Functional immunological assays:** Testing of protein extracts from different conditions using sera from allergic patients to assess IgE binding and basophil activation.

7 Implications and Conclusion

If peanut allergenicity has increased due to agricultural inputs, it may reflect a shift in the biochemical composition of the peanut itself—an overlooked consequence of agricultural intensification. Mitigation strategies might therefore include agronomic interventions, such as optimized nitrogen management or cultivar selection for lower allergen:protein ratios.

The author welcomes collaboration with researchers interested in pursuing experimental validation of this hypothesis.

Correspondence: Dr PD Prideaux, pdp22@cantab.net

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