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editorial-office@sciformat.ca

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IMPACT OF FOOD ADDITIVES AND ULTRA-PROCESSED DIETARY PATTERNS ON THE PATHOGENESIS OF PEDIATRIC ASTHMA AND ALLERGIC DISEASES: A COMPREHENSIVE REVIEW

Michał Petkow *(Corresponding Author, Email: michal.petkow@gmail.com)*
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0008-3398-9069

Katarzyna Lipska
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0003-5670-7726

Aleksandra Oszczypała
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0006-5809-3934

Mateusz Żyła
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0003-9005-620X

Monika Szyszkowska
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0006-5367-0026

Aleksandra Potempa
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0004-2698-4957

Liwia Prorok
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0008-3762-514X

Aleksandra Ustaszewska
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0001-5651-5906

Zofia Parol
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0007-0885-2637

Zuzanna Strojek
Medical University of Warsaw, Warsaw, Poland
ORCID ID: 0009-0005-8857-5670

ABSTRACT

The global escalation of pediatric asthma and allergic diseases amounts to a significant public health challenge, frequently described as the second wave of the allergy epidemic. While genetic factors provide a baseline of susceptibility, the rapid increase in prevalence points to environmental factors, particularly the considerable shift in eating habits. The modern diet is characterized by a high intake of ultra-processed foods (UPF), which are industrial formulations containing multiple additives, including emulsifiers, synthetic colorants, and non-nutritive sweeteners. This review analyzes the epidemiological evidence and biological mechanisms underlying UPF consumption and specific food additives and their effects on respiratory and allergic outcomes in children, based on an extensive review of PubMed-indexed literature. Current evidence suggests that high UPF intake is associated with an increased risk of current asthma, wheezing, and allergic rhinitis. Longitudinal cohort data indicate that maternal consumption of artificially sweetened beverages during pregnancy substantially increases the risk of asthma in offspring. Key biological mechanisms identified include the disruption of the intestinal epithelial barrier, gut microbial dysbiosis—indicated by a reduction in short-chain fatty acid-producing taxa—and the induction of systemic inflammation through oxidative stress and the activation of the NF-κB pathway. Furthermore, additives such as tartrazine and sodium benzoate have been shown to exacerbate airway inflammation by upregulating leukotriene B4 and promoting Th2-skewed immune responses. These findings show the significant need for dietary interventions and stricter regulation of food additives to reduce the chronic inflammatory burden in the pediatric population.

KEYWORDS

Ultra-Processed Foods, Food Additives, Allergic Diseases, Gut-Lung Axis, Gut Microbiota

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Introduction

In the contemporary medical environment, the rising incidence of non-communicable diseases (NCDs) among children has turned into a focal point of pediatric research. Among these, asthma and allergic diseases—including atopic dermatitis, food allergies, and allergic rhinitis—have seen a precipitous global increase over the last four decades.1 This phenomenon is usually characterized as the "allergy epidemic," where a first wave of respiratory allergies has been followed by a second wave of food-induced anaphylaxis and complex multi-organ allergic manifestations.3 While the "hygiene hypothesis" initially sought to explain these trends through reduced microbial exposure in early life, modern research has progressively turned toward the "epithelial barrier theory" and the "nutritional transition" as main drivers of immunological dysregulation.5

The nutritional transition is defined by the displacement of traditional, minimally processed diets by ultra-processed foods (UPF). According to the NOVA classification system, UPFs are industrial formulations of food-derived substances that typically contain little to no whole foods and are characterized by the inclusion of additives created to increase palatability, shelf life, and sensory appeal.6 These additives include emulsifiers, preservatives, synthetic dyes, flavor enhancers, and artificial sweeteners. In many developed and developing nations, UPF consumption now accounts for almost half of daily energy intake in preschool and school-aged children.7

Ultra-processed foods are usually characterized by a poor nutritional profile, being high in saturated fats, trans-fatty acids, free sugars, and sodium, while lacking essential micronutrients, dietary fiber, and bioactive compounds.6 However, the health impact of UPFs goes past their basic nutrient composition. The industrial processing itself and the presence of exogenous chemicals have been implicated in disrupting physiological homeostasis. Specifically, the consumption of UPFs has been linked to obesity, metabolic syndrome, and cardiovascular diseases, but emerging evidence now strongly suggests a role in respiratory and allergic outcomes.1

The relationship between diet and the immune system is mainly mediated through the gut-lung axis. The gut microbiota, shaped by dietary intake from birth, plays a key role in the development of oral tolerance and in the regulation of systemic inflammation.3 Ultra-processed foods and their additives have been shown to induce microbial dysbiosis, increase intestinal permeability (frequently known as "leaky gut"), and activate inflammatory cascades that affect distant mucosal sites, including the airways.11

This review aims to deliver a comprehensive synthesis of the current scientific literature regarding the impact of UPFs and common food additives on the development and exacerbation of asthma and allergies in children. By studying epidemiological data from major cohorts and exploring the molecular and cellular mechanisms of action, this report aims to provide an expert-level understanding of the dietary factors contributing to the pediatric allergy epidemic and to highlight the importance of nutritional strategies for disease prevention and management.

Materials and Methods

The evidence synthesized in this review is based on a systematic search and analysis of scientific literature available in the PubMed database. The search was conducted to identify relevant studies published primarily between 2006 and 2025, focusing on the intersection of dietary processing, food additives, and pediatric immunological outcomes.13

The search strategy utilized various combinations of MeSH terms and keywords, including "ultra-processed foods," "NOVA classification," "food additives," "synthetic colorants," "artificial sweeteners," "emulsifiers," "pediatric asthma," "childhood allergy," "atopic dermatitis," "gut-lung axis," and "intestinal permeability".1 Inclusion criteria were restricted to peer-reviewed original research articles, systematic reviews, meta-analyses, and longitudinal cohort studies that provided empirical data on the associations between diet and allergic or respiratory conditions in children and adolescents.

Particular emphasis was placed on high-quality longitudinal studies, such as the Danish National Birth Cohort (DNBC), and on population-based surveys, such as the National Health and Nutrition Examination Survey (NHANES), which provide reliable statistical evidence.7 To explain biological mechanisms, this review also incorporated relevant experimental studies using murine systems and in vitro systems, particularly where they addressed the effects of specific additives on immune cell differentiation, cytokine production, and barrier function.16

Studies focusing exclusively on adult populations were excluded unless they supplied key mechanistic insights appropriate to pediatrics. Furthermore, consistent with the objective of this review, we avoided relying on large, potentially unreliable aggregate numbers and instead focused on effect sizes (such as odds ratios and hazard ratios), confidence intervals, and specific p-values to indicate statistical significance. Data extraction focused on the frequency of UPF consumption, the specific types of additives involved, the timing of exposure (e.g., prenatal, infancy, childhood), and the clinical outcomes related to asthma, wheeze, and allergic sensitization.

Results

Consumption Trends and the NOVA Framework in Pediatrics

The classification of food products by degree of processing has become a standardized approach in nutritional epidemiology. The NOVA system classifies foods into four distinct groups: unprocessed or minimally processed (Group 1), processed culinary ingredients (Group 2), processed foods (Group 3), and ultra-processed foods (Group 4).6 Ultra-processed foods are defined by the inclusion of industrial substances—such as hydrogenated oils, modified starches, and protein isolates—and additives like stabilizers, emulsifiers, and colors that are not typically used in domestic kitchens.7

Among pediatric populations, the prevalence of UPF intake is remarkably high. In Canada, it has been reported that UPFs account for approximately 48% of total daily energy intake among preschool-aged children.8 Similarly, in a cohort of primary school children in Chile, the mean UPF intake was found to be 29% of total calories.18 In Brazilian adolescents, UPF consumption commonly accounts for more than 26% of daily energy intake, with individuals in the highest tertiles consuming significantly more simple carbohydrates, saturated fats, and sodium while receiving less protein and fiber.9

Epidemiological Evidence Linking UPF to Asthma and Wheezing

The association between the consumption of UPFs and respiratory symptoms in children has been demonstrated in multiple cross-sectional and longitudinal studies. Analysis of the NHANES data for children aged 2 to 19 years revealed that a higher contribution of UPF to total energy intake is positively associated with the risk of current asthma.7 Children in the highest quartile of UPF intake showed an increased risk of 76% (OR = 1,76; 95% CI; 1.10-2.82; p for trend = 0,0393) compared to those in the lowest quartile.7

Studies involving adolescents have found that frequent consumption of specific UPF categories, such as biscuits, sweets, processed meats, and packaged snacks, is correlated with a higher risk of wheezing and asthma diagnosis.6 However, the evidence is not entirely uniform across all cohorts. For instance, data from the Pelotas birth cohort in Brazil did not find a significant association between UPF consumption and asthma symptoms during certain periods of childhood, suggesting that the timing of exposure and the specific composition of the local UPF supply may be critical variables.7

Study Population	Exposure Measure	Outcome	Statistical Finding
US Children (NHANES)	UPF energy contribution (%)	Current Asthma	OR = 1,76 for highest quartile.7
Canadian Preschoolers	UPF intake at age 3	Internalizing/ Externalizing Symptoms	Higher UPF associated with behavioral issues at age 5.8
US Youth (NHANES)	Sugar-Sweetened Beverages (SSB)	Current Asthma	Heavy consumption: aOR = 2.01.20
Danish Birth Cohort	Artificially Sweetened Beverages (Prenatal)	Offspring Asthma (7 yrs)	OR = 1.30 (Carbonated ASB).14
Chilean Primary School	UPF intake tertiles	Systemic Inflammation	Suggestive trend for elevated IL-1B18

Allergic Sensitization and the IgE Paradox

The relationship between ultra-processed diets and allergic sensitization is complex in the NHANES cohort; higher UPF intake was associated with symptoms of eczema in girls.7 Interestingly, however, several studies have noted a "paradoxical" negative association between UPF consumption and total serum IgE levels.7 Specifically, children in the highest quartiles of UPF consumption had a reduced risk of elevated IgE by 34% (OR = 0.66; 95% CI: 0.49-0.89; p = 0.006).7

This evidence suggests that the respiratory and allergic symptoms associated with UPF may not always be mediated by traditional IgE-dependent pathways. Instead, they may reflect non-atopic inflammation, or perhaps a disruption of the regulatory mechanisms that normally balance IgE production. Furthermore, UPFs are more complex mixtures that tend to contain more allergens and are more prone to cross-contamination than minimally processed foods, which may increase the risk of adverse reactions in already sensitized individuals, regardless of total IgE levels.21

The Impact of Specific Food Additives

A significant portion of the risk associated with UPFs is attributed to the presence of industrial additives. These chemicals are added to improve texture, appearance, and shelf stability, but they often interact with the child's biological systems in ways that promote inflammation.

Synthetic Colorants and Preservatives

Synthetic food dyes, such as tartrazine (E102), Sunset Yellow (E110), and Carmoisine (E122), have long been suspected of triggering hypersensitivity reactions.22 Tartrazine, specifically, has been linked to asthma exacerbations, urticaria, and angioedema in sensitive children.23 Experimental data indicate that tartrazine can promote the oxidation of arachidonic acid and upregulate the production of leukotriene B4 (LTB4) in human neutrophils, which is a potent mediator of airway constriction and inflammation. 17

Preservatives like sodium benzoate and sulfites also have a part. Sulfites are well-known triggers for bronchial hyper-responsiveness and can cause severe asthma exacerbations and even anaphylaxis.10 Sodium benzoate has been associated with both behavioral changes and allergic manifestations, and its presence in children's medications (such as cough syrups) is a particular concern for sensitive populations.22

Artificial Sweeteners and Non-Nutritive Sweeteners (NNS)

The impact of artificial sweeteners on pediatric respiratory health is increasingly evident, beginning in the prenatal period. Data from the Danish National Birth Cohort showed that mothers who consumed artificially sweetened non-carbonated soft drinks during pregnancy were 1.23 times more likely to report a child's asthma diagnosis at 18 months.14 By age 7, the consumption of carbonated artificially sweetened beverages during pregnancy was linked to a higher risk of both asthma and allergic rhinitis (OR = 1.31).14

Mechanistic studies in murine models suggest that artificial sweeteners such as saccharin and neotame can inhibit the acquisition of oral tolerance.16 Mice treated with a mixture of food additives during the induction of oral tolerance showed higher levels of OVA-specific IgE and symptoms of food allergy compared to controls.16 These additives were found to decrease the proportion of CD25+ regulatory T cells (Tregs) in the mesenteric lymph nodes, consequently promoting a pro-inflammatory environment. 16

Emulsifiers and Thickening Agents

Emulsifiers such as Polysorbate 80 (P80) and Carboxymethylcellulose (CMC) are ubiquitous in UPFs. These substances directly affect the intestinal epithelial barrier. As detergents, they can disrupt the protective mucus layer of the gut, leading to increased intestinal permeability.11 This "leaky gut" allows for the translocation of bacterial components and food antigens, triggering systemic inflammation. In animal models, P80 and CMC have been shown to elicit Th1-prone and Th2-driven immune responses, respectively, promoting the development of inflammatory diseases and allergic sensitization.11

Additive Type	Examples	Biological Mechanism	Associated Clinical Outcome
Synthetic Colorants	Tartrazine (E102), Sunset Yellow (E110)	Oxidative stress (ROS), LTB4 upregulation	Asthma exacerbation, Urticaria.17
Preservatives	Sodium Nitrite, Sulfites, Sodium Benzoate	NF-κB activation, Bronchial hyper-responsiveness	Asthma attacks, Anaphylaxis.10
Emulsifiers	Polysorbate 80, CMC, Lecithin	Intestinal barrier disruption, Microbiota dysbiosis	Food allergy, Systemic inflammation.11
Sweeteners	Saccharin, Aspartame, Sucralose	Impaired oral tolerance, Treg depletion	Offspring asthma, Atopy.14

Systemic Inflammation and Cytokine Profiles

The consumption of UPFs is consistently linked to markers of systemic inflammation in children. In a study of primary school children in Chile, higher UPF intake was associated with a suggestive trend toward elevated interleukin-1β levels. In older children, higher UPF consumption was specifically associated with increased IL-6 levels.18

Adolescents in Brazil with high UPF consumption (≥30% of energy) exhibited a 79% increase in IL-8 levels compared to those in the lowest tertile.9 These individuals also had higher serum leptin and C-reactive protein (CRP) levels, indicating that the pro-inflammatory effects of UPF are established early in life and correlate with both metabolic and immunological disturbances.9

Discussion

The Gut-Lung Axis and Microbial Dysbiosis

The primary conduit through which a high-UPF diet influences respiratory health is the gut-lung axis. The gut microbiota plays a critical role as an interface between the environment and the host immune system, particularly during the "first thousand days" of life.2 A healthy gut microbiota, defined by high diversity and an abundance of beneficial taxa such as *Bifidobacterium*, *Faecalibacterium*, and *Clostridia*, is essential for the induction of regulatory T cells and the maintenance of oral tolerance.3

Ultra-processed foods and their additives significantly change this microbial ecosystem. Allergic children consistently show reduced microbial diversity and lower levels of these beneficial taxa.3 Instead, they commonly harbor higher proportions of *Enterobacteriaceae* and *Ruminococcus gnavus*, the latter of which has been associated with pro-inflammatory polysaccharides and reduced fiber degradation.3

A critical consequence of this dysbiosis is a reduction in short-chain fatty acids (SCFAs) such as butyrate and propionate.3 SCFAs are produced through the fermentation of dietary fiber, which is typically deficient in UPF-rich diets. SCFAs play a vital role in protecting the integrity of the intestinal epithelial barrier and suppressing Type 2 (T2) cytokines such as IL-4, IL-5, and IL-13.3 Without adequate SCFAs, the immune system is predisposed to Th2-skewed responses, leading to allergic sensitization and airway hyper-responsiveness.3

The Epithelial Barrier Theory and Modern Exposure

The "Epithelial Barrier Theory" provides an extensive framework for understanding how modern environmental exposures contribute to the rise in chronic non-communicable diseases.5 According to this theory, the ingestion of industrial chemicals—including emulsifiers, detergents, and microplastics—damages the protective mucosal surfaces of the gut and lungs.5

Common food additives such as Polysorbate 80 and CMC directly affect the intestinal epithelium, increasing its permeability.11 This allows for the translocation of microbes and allergens into the subepithelial space, where they encounter immune cells. The resulting local inflammatory response involves the release of "alarmins" and the activation of innate lymphoid cells (ILC2s), which propagate systemic inflammation.5 This systemic inflammation can manifest in distant organs, including the skin (eczema) and the respiratory tract (asthma), explaining the frequent co-occurrence of these conditions in what is known as the "atopic march".5

Oxidative Stress and Metabolic Mediators

Beyond their effects on the microbiota and barrier function, food additives contribute to the development of asthma by inducing oxidative stress (OS).23 Synthetic colorants, such as tartrazine, have been shown to increase the production of reactive oxygen species (ROS) and decrease the activity of endogenous antioxidant enzymes, such as superoxide dismutase (SOD) and catalase (CAT).23 This disproportion leads to lipid peroxidation, as evidenced by elevated malondialdehyde (MDA) levels, which contribute to tissue damage and the chronic inflammatory state of the airways.23

Metabolomics profiling has identified specific serum metabolites that mediate the relationship between food additives and childhood asthma. These include various glycerophospholipids, sphingosine-1-phosphate, and amino acids involved in glutathione and histidine metabolism.17 Mediation analysis has shown that additives such as benzoic acid can affect these metabolic pathways, leading to metabolic disturbance of helper T cells and antigen-presenting cells.17 These data suggest that the impact of additives is not simply a superficial hypersensitivity but a central metabolic reprogramming of the immune system.17

The Synergistic Effect of Diet Quality and Processing

The risk associated with UPFs is likely synergistic. Not only do these foods contain harmful additives and processing-induced contaminants (such as Advanced Glycation End-products), but they also displace protective nutrients.6 A "Westernized" diet high in UPF is typically low in Vitamin D, omega-3 polyunsaturated fatty acids, and dietary fiber, all of which have been shown to have anti-inflammatory and allergy-preventative effects.28

Furthermore, the high free sugar content in UPF-rich diets, particularly in the form of sugar-sweetened beverages, can promote systemic inflammation and worsen pulmonary health.20 The relationship between sugar intake and asthma has been found to be dose-dependent, with heavy consumers of fruit drinks and non-diet soft drinks exhibiting twice the odds of current asthma compared to non-consumers.20 This effect appears to be independent of obesity status, suggesting that the metabolic and inflammatory impact of a high-sugar, high-UPF diet is a direct pathway to respiratory disease.20

Second and Third-Order Insights

When evaluating the impact of UPFs, it is essential to consider the broader socio-ecological and behavioral implications. High UPF consumption in early childhood is not only a marker of poor physical health but also correlates with adverse behavioral and emotional functioning.8 The shared inflammatory pathways between the gut, brain, and lungs suggest that the "allergy epidemic" may be a component of a broader "inflammation epidemic" affecting multiple systems in children.5

The displacement of minimally processed foods by UPFs also leads to a loss of nutritional diversity. Early-life diet diversity is a key factor in the development of a resilient immune system.28 For every additional food group introduced during the first year of life, there is a measurable reduction in the odds of food allergy

by age 10.28 By homogenizing the diet with industrial formulations, UPFs may prevent the necessary "training" of the immune system, thereby raising susceptibility to both allergies and chronic inflammatory conditions. 28

Mechanism	Primary Impact	Downstream Effect on Asthma/Allergy
Microbial Dysbiosis	Loss of *Bifidobacterium* and *Clostridia*; reduction in SCFAs	Th2 skewing; loss of oral tolerance; systemic T2 inflammation.3
Barrier Injury	Increased intestinal permeability ("Leaky Gut")	Antigen translocation; systemic "alarmin" release; atopic march.5
Oxidative Stress	ROS production; depletion of SOD and Catalase	Lipid peroxidation; chronic airway inflammation; DNA damage.23
Metabolic Shift	Alteration in sphingolipid and amino acid pathways	Dysregulation of Treg and Th17 cell balance.17

Conclusions

This extensive review spotlights the key role of ultra-processed foods and common food additives in the pathogenesis of pediatric asthma and allergic diseases. The contemporary dietary transition toward high-UPF consumption signifies a fundamental shift in the developing child's environment, promoting immunological dysregulation through multiple linked pathways.

The epidemiological evidence consistently links high UPF intake—and particularly the prenatal and early-life exposure to synthetic sweeteners and synthetic dyes—with an increased risk of asthma, wheezing, and eczema. These outcomes are driven by a combination of gut microbial dysbiosis, disruption of the intestinal epithelial barrier, and induction of chronic systemic inflammation. The "IgE paradox" observed in some cohorts suggests that UPF may promote a relatively complex, non-atopic form of inflammation that is not always reflected in traditional allergy markers but that nonetheless increases the burden of respiratory disease.

From a clinical and public health perspective, these findings stress the necessity of viewing diet as a primary modifiable risk factor in the allergy epidemic. Promoting minimally processed, high-fiber, and nutrient-dense dietary patterns is important for maintaining the integrity of the gut-lung axis and establishing immunological homeostasis. Furthermore, there is a strong urgency for stricter regulatory monitoring regarding the use of synthetic additives in foods marketed to children and pregnant women. Addressing the proliferation of ultra-processed foods in the pediatric diet is not only a matter of metabolic health but is a vital strategy for curbing the global rise of chronic respiratory and allergic conditions.

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