

Childhood Asthma may Trigger by Common Food Additives

A recent study published utilized metabolomics to investigate the mechanisms underlying the association between food additives and [childhood asthma](#).



Study

The current study quantified correlations between childhood asthma and food additives using logistic regression analyses and chi-square tests. The researchers also performed a non-targeted metabolic profile of serum samples collected from children 15 years of age and younger. Asthma-related metabolites were identified, and models were devised to elucidate how the differentiation of CD4+ T-cells and dendritic cells (DCs) is affected by [food additives](#).

[Serum concentrations](#) of ten food additives were measured using ultra-high pressure liquid chromatography (UPLC)-mass spectroscopy (MS)/MS. These ten additives were neotame, aspartame, sodium saccharin, ponceau 4R, sucralose, benzoic acid, cyclamate, acesulfame, dehydroacetic acid, and sunset yellow.

The authors also noted that certain additives, such as aspartame, neotame, sucralose, and sunset yellow, were poorly detected in serum because they are minimally absorbed and rapidly broken down in the gut rather than being absent from [children's diets](#).

Asthma-associated metabolites were identified and examined for mediation effects and pathway enrichment. Other metabolites corresponding to airway inflammation, immune cell differentiation, [immunoglobulin E](#) (IgE), interleukin-4 (IL-4), IL-17A, and CD4+ T-cell metabolomics profiles were also measured.

Results

Dehydroacetic acid, benzoic acid, and cyclamate were detected at the highest rates of 99.58%, 99.17%, and 69.17%, respectively. Aspartame and neotame were not detected, with low detection rates observed for sodium saccharin, [sucralose](#), acesulfame, ponceau 4R, and sunset yellow.

In the asthmatic group, the mean concentrations of dehydroacetic and benzoic acids were significantly higher than those of the control group. Chi-square tests and logistic regression analyses revealed significant associations between childhood asthma and exposure to dehydroacetic acid, [benzoic acid](#), and acesulfame.

Multivariate analysis led to the identification of seventy-three statistically significant asthma-associated metabolites. Several of these metabolites mediated the association between

childhood asthma and exposure to benzoic acid and dehydroacetic acid, including PC (14:0/14:0), glycerophosphocholine, glutamine, [histidine](#), spermine, LysoPC(17:0), acetylcholine, and others.

In a murine model, a significant increase in inflammatory cells was observed in food additive-treated groups as compared to controls. The proportion of eosinophil granulocytes and IL-17A in [bronchoalveolar lavage fluid](#) (BALF) was significantly higher in the food-additive treated groups, as were IgE levels in the BALF and serum, as well as serum IL-4 levels.

Mice consuming food additives also exhibited higher proportions of allergic DCs, Th2 cells, and Th17 cells. Taken together, the in vivo results indicate that food additives may disrupt [immune cell](#) differentiation pathways and potentially induce abnormal differentiation of helper T cells, which could facilitate the development of asthma.

When CD4+ T-cells were isolated from the [mesenteric lymph node](#) (MLN) and metabolomics analysis was performed, differential metabolites were identified. For example, the acesulfame-treated group showed lower levels of Cer(d18:2/20:0) and higher levels of fatty acyls, glycerophospholipids, and amines as compared to the oral tolerance and control groups.

The sodium saccharin-treated group exhibited lower levels of L-tyrosine and greater levels of amines, glycerophospholipids, nucleotides, and specific lipid metabolites. Significant alterations were also reported in [phenylalanine](#), tryptophan, tyrosine biosynthesis, and glycerophospholipid metabolism pathways among mice consuming food additives.

The authors also proposed that these immune and metabolic changes may be influenced by interactions along the [gut-lung axis](#), in which food additives alter intestinal permeability and the microbiota, allowing inflammatory metabolites and immune cells to affect lung immune balance.

The primary limitation of the current study is the lack of generalizability, as the sample cohort was exclusively recruited from Nanjing, China. The regression analysis also failed to control for confounding factors like parental smoking and [body mass index](#) (BMI), which could influence the results.

The current study explored correlations between childhood asthma and exposure to food additives; however, it did not provide causal explanations for these correlations. The authors emphasize that future research should include broader populations, additional types of additives, and direct mechanistic validation of metabolic effects on [immune pathways](#).

Conclusion

Childhood asthma may be exacerbated by exposure to food additives, which can metabolically dysregulate homeostasis between antigen-presenting cells and [helper T-cells](#).

These findings suggest that metabolic and immune disturbances caused by certain preservatives and sweeteners could contribute to airway inflammation through disrupted immune tolerance and [gut-lung interactions](#), although further studies are needed to confirm causality.

Source:

<https://www.news-medical.net/news/20251006/Common-food-additives-may-trigger-childhood-asthma.aspx>