

Dietary Exposure to Harmful Chemicals and Health Outcomes in Children: A Systematic Review

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ABSTRACT

Children may be exposed to hazardous chemicals through contaminated foods or chemicals introduced during food production, processing, storage, and packaging. However, evidence on dietary chemical exposure and child health remains fragmented. This systematic review synthesized evidence on food-related chemical exposure and health outcomes in children. PubMed and Scopus were searched for primary studies published between 2016 and 2026. Eligible studies included children or adolescents younger than 18 years, assessed dietary or food-related chemical exposure, and reported health outcomes or biological responses. Studies were excluded if they lacked relevant outcomes, focused on non-dietary exposure, included adults only, or investigated microbial contaminants. Of 838 records identified, 23 studies met the eligibility criteria. These examined endocrine-disrupting chemicals and plasticizers, mycotoxins, metals and metalloids, fluoride, melamine, processing-related contaminants, natural plant toxins, and chemical mixtures. Seventeen studies reported direct health outcomes, and six reported intermediate biological responses. Associations involved growth, neurodevelopment, cognitive and pubertal development, cardiometabolic risk, dental outcomes, renal and oxidative stress biomarkers, immune and microbiome responses, respiratory and allergic outcomes, and clinical toxicity. The strength and consistency of associations varied across chemical-outcome relationships. Overall, substantial heterogeneity, methodological limitations, and limited evidence for several chemical-outcome pairs constrain causal interpretation.

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INTRODUCTION

Changes in the food environment have influenced dietary patterns worldwide, including the increasing availability and consumption of commercially prepared and ultra-processed foods (Baker et al., 2020). Dietary patterns are also closely related to the nutritional quality of foods consumed. In Indonesia, the Indonesian Food Barometer has documented differences in the contribution of breakfast foods to daily energy and nutrient intake, including relatively high contributions of fat and saturated fat and low contributions of dietary fiber and several micronutrients (Khusun et al., 2023). Beyond their nutritional characteristics, foods and beverages can also be a source of exposure to hazardous chemicals through contamination or the introduction of chemicals during food production, processing, storage, and packaging. In school-aged children in Southern Italy, for example, reducing the use of plastic food packaging in school meals was associated with lower urinary bisphenol A concentrations, providing evidence that food-related practices can contribute to children's chemical exposure (Sessa et al., 2021).

Dietary exposure to hazardous chemicals can arise from multiple sources throughout the food system. Some

substances are intentionally added to foods, including preservatives, colorants, flavoring agents, and sweeteners, whereas other contaminants may be introduced or formed during food processing (Pakdel et al., 2023; Soni et al., 2024). In addition, chemicals associated with food-contact materials may migrate into food and subsequently contribute to human dietary exposure (Muncke et al., 2020). Food-contact materials encompass a wide range of materials used in food packaging and processing, including plastics, paper and paperboard, coatings, and other polymers, which may contain substances of potential health concern (Trasande et al., 2018). Thus, dietary exposure to hazardous chemicals can occur through multiple pathways and across a broad range of foods and beverages.

Children may be particularly vulnerable to chemical exposure because of their ongoing growth and physiological development. Infants and young children consume greater amounts of food and fluids relative to their body weight than adults, which may result in greater exposure per kilogram of body weight (Hauptman & Woolf, 2017). Furthermore, metabolic, renal, and other physiological systems continue to develop throughout childhood, potentially affecting the absorption, distribution, metabolism, and elimination of

chemical substances. Exposure during sensitive developmental periods may therefore be of particular concern, especially for chemicals with endocrine-disrupting or other toxicological properties (Rauh & Margolis, 2016).

Previous studies have reported associations between exposure to chemicals commonly encountered through food and food-related sources and a range of health outcomes in children. Bisphenols and phthalates, for example, have been investigated in relation to metabolic and endocrine-related outcomes, while other chemical exposures have been examined in relation to immune, behavioral, and neurodevelopmental outcomes (Astrada & Mulyono, 2021; Mihalache & Dall’Asta, 2023; Russ & Howard, 2016; Savin et al., 2022). However, the available evidence is heterogeneous with respect to chemical classes, exposure sources, exposure assessment methods, developmental stages, and health outcomes. Importantly, evidence of chemical presence in food, estimated exposure, potential toxicological risk, and observed health outcomes represent different levels of evidence and should not be interpreted interchangeably.

Despite increasing research on individual chemical classes and specific exposure sources, evidence concerning chemical exposure associated with processed foods and its relationship with children's health remains fragmented. Previous reviews have often focused on particular chemical groups, exposure sources, or specific health outcomes, limiting an integrated understanding across these dimensions. Therefore, this systematic review aimed to synthesize the available evidence on harmful chemical exposures associated with processed foods and food-related exposure pathways among children, with particular emphasis on the types of chemicals, their sources and exposure pathways, the developmental groups studied, and the reported health outcomes. By distinguishing evidence concerning chemical occurrence, exposure, potential risk, and reported health outcomes, this review seeks to provide a clearer characterization of the current evidence base, clarify its limitations, and identify important gaps for future research.

METHODS

Study Design and Reporting Framework

This study was conducted as a systematic review of the available evidence on chemical exposure associated with processed foods and food-related exposure pathways among children. The review was reported in accordance with the

Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. The review question and eligibility criteria were structured using the PECO framework. The review included relevant studies published between 2016 and 2026, with the final database search conducted in September 2026. The initial review protocol was registered in the International Prospective Register of Systematic Reviews (PROSPERO) under registration number CRD420251029052 (version 1.0). During peer review, the review question, eligibility framework, publication period, and evidence base were substantially revised in response to reviewer comments, including restructuring the framework using PECO. To reflect these revisions, the protocol was re-registered as a new PROSPERO record under registration number CRD420261517722 (version 1.0) on 25 September 2026. The present review follows the methodology and eligibility criteria specified in the latter registration.

Review Question and PECO Framework

The review question was formulated according to the PECO framework: Population (P): children and adolescents younger than 18 years; Exposure (E): dietary exposure to hazardous or potentially harmful chemicals through food or food-related sources, including chemicals associated with food processing, packaging, storage, and food-contact materials; Comparator (C): lower, non-detectable, or reference exposure levels, where applicable; and Outcome (O): health-related outcomes associated with dietary chemical exposure in children. The PECO framework was used to maintain consistency among the review question, eligibility criteria, search strategy, study selection, and evidence synthesis.

Literature Search Strategy

A systematic literature search was conducted using PubMed and Scopus to identify relevant studies published between 2016 and 2026. The search strategy combined terms related to children, chemical hazards or chemical exposure, food or dietary sources, and relevant food-related chemical exposures using Boolean operators (AND/OR). Search terms were adapted to the indexing systems and search functions of each database. The final database search was conducted in September 2026. The complete search strategies used to identify potentially relevant studies are presented in Supplementary Material 1. The article search and selection process is presented in Figure 1.

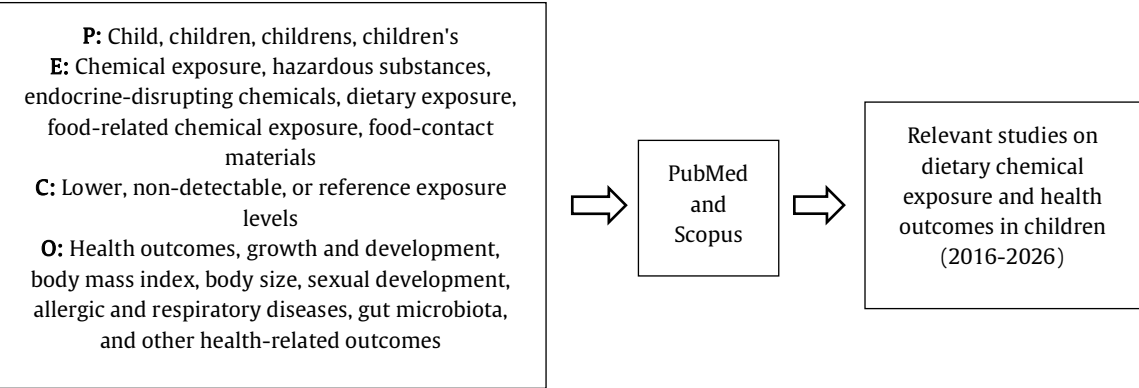


Figure 1. Article Search Flowchart

Eligibility Criteria

Studies were included if they (1) involved children, including infants, preschool children, and school-aged children, or reported pediatric data separately; (2) investigated exposure to hazardous or potentially harmful chemicals through food or dietary intake, including chemicals associated with food processing, packaging, storage, or food-contact materials; (3) assessed chemical exposure using dietary assessment, measurements of chemicals in food, or exposure biomarkers that were specifically linked to dietary exposure; (4) evaluated at least one health-related outcome or biological response in children, including growth, nutritional status, metabolic, endocrine, neurodevelopmental, immune, respiratory, allergic, reproductive, or other clinically relevant health outcomes, as well as biomarkers indicative of physiological or pathological changes relevant to health or disease processes; (5) were primary research articles published in English between 2016 and 2026; and (6) had accessible full text. Studies were excluded if they (1) involved adolescents or adults without separately reported data for children; (2) assessed exclusively non-dietary environmental, occupational, dermal, inhalational, or other non-food-related chemical exposure; (3) measured chemical concentrations, exposure biomarkers, internal-dose biomarkers, or estimated dietary exposure without evaluating a health-related outcome or a health-

relevant biological response; (4) focused on microbial foodborne diseases without a chemical exposure component; (5) were reviews, systematic reviews, meta-analyses, commentaries, policy statements, technical reports, protocols, or other non-primary research publications; (6) were not published in English; or (7) did not have accessible full text.

Study Selection

The study selection process followed the PRISMA 2020 framework. All records identified from PubMed and Scopus were imported into Rayyan, where duplicate records were removed before screening. Titles and abstracts were independently screened by two reviewers (DLT and AWMM) using Rayyan's Blind Mode, which concealed each reviewer's screening decisions from the other reviewer. Potentially eligible studies then underwent independent full-text assessment using the predefined eligibility criteria. After completion of each screening stage, disagreements between reviewers were identified and resolved through discussion and consensus. Studies that met all predefined eligibility criteria were included in the final synthesis. The overall study selection process is presented in the PRISMA flow diagram (Figure 2).

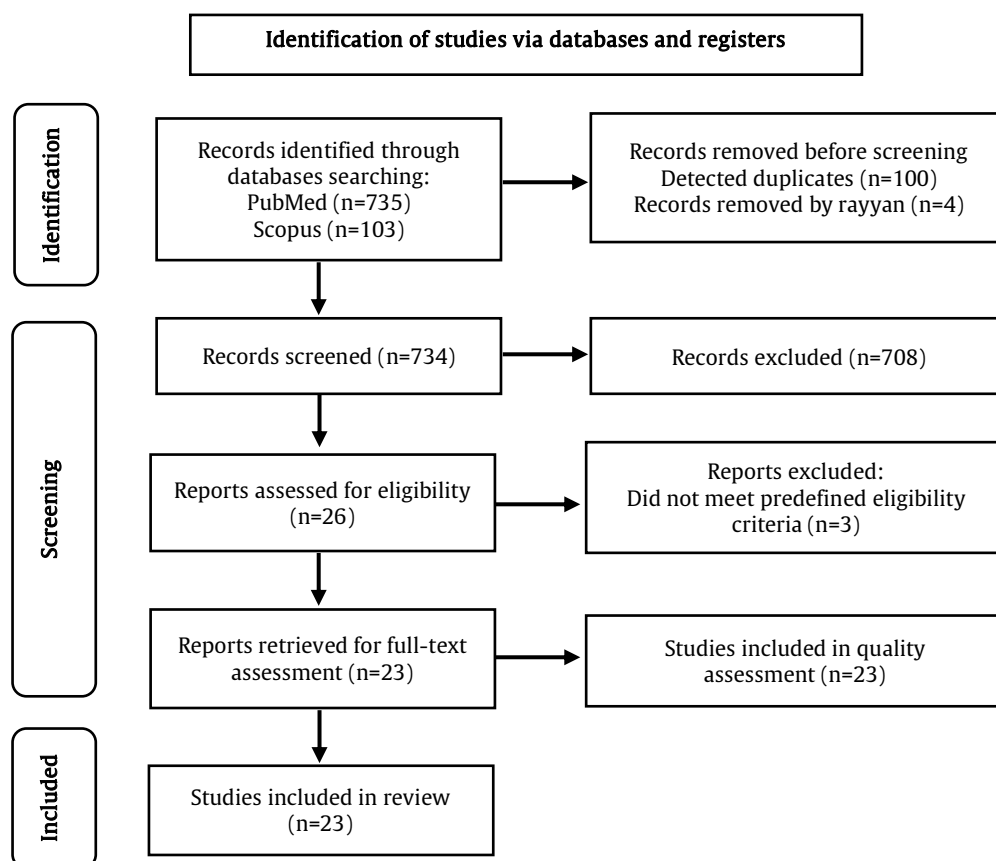


Figure 2. PRISMA Flowchart

Quality Assessment and Risk of Bias

The methodological quality and risk of bias of the included studies were assessed based on study design, participant selection, exposure measurement, outcome measurement,

confounding control, and other potential sources of bias. Studies were judged as having low risk of bias when no major methodological concerns were identified, moderate risk of bias when important but non-critical limitations were present, and high risk of bias when major limitations were

likely to substantially affect the validity or interpretation of the findings. The overall judgment was based on the balance of these methodological considerations. The assessment results were considered when interpreting and synthesizing the findings.

Data Extraction and Synthesis

Data extraction was performed using a standardized Microsoft Excel form. The extracted information included author and publication year, study title, study location, study design, population characteristics and developmental stage, chemical or chemical class investigated, food or food-related exposure source, exposure pathway, exposure assessment method, exposure levels or biomarkers where reported, comparator characteristics where applicable, and reported health outcomes. Because of heterogeneity in the chemical substances investigated, exposure assessment methods, study populations, and health outcomes, the findings were synthesized narratively rather than statistically pooled. The synthesis distinguished between chemical occurrence in food, measured or estimated exposure, potential health risks, and reported health outcomes, recognizing that these represent different levels of evidence and should not be interpreted interchangeably.

RESULTS OF STUDY

Study Selection

The study selection process is presented in the PRISMA flow diagram (Figure 2). The searches of PubMed and Scopus identified 838 records, comprising 735 records from PubMed and 103 from Scopus. Following duplicate detection and removal, 734 records remained for title and abstract screening. Of these, 708 records were excluded during title and abstract screening, and 26 records proceeded to full-text assessment. At the full-text stage, three studies were excluded because they did not meet the predefined eligibility criteria, including the requirements for the study population, dietary or food-related chemical exposure, empirical health-related outcomes, and publication or study type. Ultimately, 23 studies met the eligibility criteria and were included in the final qualitative synthesis.

Characteristics of the Included Studies

The characteristics of the included studies are summarized in Supplementary Material 2. The 23 included studies were conducted across 14 countries, including Spain, the United States of America, Tanzania, Portugal, Mexico, Taiwan, the Democratic Republic of the Congo, Vietnam, Kenya, Nigeria, Norway, Zambia, Lebanon, and France. The studies employed diverse designs, including cross-sectional, prospective or longitudinal cohort, case-control, randomized controlled, outbreak investigation, case investigation, and case report designs.

The included studies involved children across different developmental stages, ranging from neonates and infants to school-aged children and adolescents. Sample sizes varied considerably, ranging from 3 children in a case report to 5,279 children and adolescents in a population-based cohort. The chemicals investigated included metals and metalloids, mycotoxins, endocrine-disrupting chemicals and plasticizers, melamine, processing-related contaminants, natural plant

toxins, and complex mixtures of persistent environmental contaminants

Chemical exposure was assessed using several approaches, including dietary questionnaires and recalls, food diaries, estimated dietary intake, chemical analysis of food samples, and biological measurements in urine, blood, serum, hair, breast milk, or stool. Some studies combined dietary assessment with biomonitoring or direct chemical analysis of contaminated food products.

The reported outcomes covered growth and nutritional status, neurodevelopment and cognition, dental health, pubertal development, cardiometabolic risk, endocrine and reproductive responses, renal and oxidative-stress biomarkers, immune responses, gut microbiome composition, respiratory and allergic outcomes, and clinical toxicity. Given the heterogeneity across study designs, chemical exposures, exposure assessment methods, populations, and outcome measures, the evidence was synthesized narratively rather than quantitatively pooled.

Evidence Across the Exposure-Health Outcome Continuum

The included studies reported findings across different levels of the exposure-health outcome continuum. To distinguish the types of evidence provided, the findings were categorized into two groups: studies reporting direct health-related outcomes, and studies reporting intermediate biological responses. Direct health-related outcomes included clinically or functionally relevant outcomes such as growth, nutritional status, neurodevelopment, dental health, pubertal development, cardiometabolic outcomes, respiratory or allergic conditions, and clinical toxicity. Intermediate biological responses included biomarkers of renal injury, oxidative stress, reproductive or endocrine responses, immune responses, and changes in the gut microbiome.

Direct health-related outcome studies

Of the 23 included studies, 17 reported direct health-related outcomes in children in relation to dietary chemical exposure, as summarized in Table 1. The direct health-related outcomes covered six broad domains: growth and nutritional status; neurodevelopment and cognition; endocrine, pubertal, and metabolic outcomes; dental health; acute toxicity; and respiratory/allergic outcomes. While most studies reported statistically significant associations, one study by Kvestad et al. (2018), which assessed methylmercury exposure from fatty fish consumption in a randomized controlled trial, found no association with cognitive outcomes, providing contrasting evidence for this particular exposure-outcome relationship. The individual findings are summarized below according to the main health outcomes identified across the included studies.

Chen et al. (2018) reported that fumonisin B1 exposure was associated with lower WAZ and HAZ among children. Huong et al. (2019) similarly found that combined exposure to aflatoxin B1, ochratoxin A, and fumonisins was associated with lower HAZ and WHZ. Alamu et al. (2020) reported higher odds of stunting and underweight among children with higher AFB1-lysine levels. In contrast, Leroy et al., (2018) found that higher serum AFB1-lysine was associated with greater linear growth at four months, suggesting that the observed associations between aflatoxin exposure and growth were not consistent across studies.

Kashala-Abotnes et al. (2018) found that higher cyanogen exposure from cassava flour was associated with poorer developmental and motor scores. Signes-Pastor et al. (2019)

reported that inorganic arsenic exposure was associated with lower global, gross, and fine motor function scores. Ramírez et al. (2024) found that interactions between bisphenol exposure and genetic polymorphisms were associated with cognitive scores. In contrast, Kvestad et al. (2018) found no clear association between total hair mercury and WPPSI-III scores despite increased methylmercury exposure.

Costa et al. (2026) reported that acrylamide was negatively associated with pubertal development in girls, whereas BPA was positively associated with pubertal development in boys. Gálvez-Ontiveros et al. (2024) found that BPA exposure from meat and eggs was associated with higher odds of overweight/obesity among females. Magalhães et al. (2024) reported that higher BPA exposure was associated with higher odds of belonging to a higher-risk cardiometabolic pattern among adolescents.

Cantoral et al. (2021) found that dietary fluoride intake was inversely associated with dental caries indices. In

contrast, Castiblanco-rubio et al. (2025) reported that dietary fluoride intake at age one was associated with higher odds of dental fluorosis during adolescence.

Viveiros et al. (2021) reported vomiting and diarrhea among children exposed to cake decorations containing copper and lead, with a blood lead level of 12 µg/dL. Troeschel et al. (2025) reported lead exposure following consumption of contaminated cinnamon-containing applesauce, with a median maximum venous blood lead level of 7.2 µg/dL and gastrointestinal and developmental/behavioral symptoms. Choudhury et al. (2025) reported gastrointestinal and neurological symptoms among children exposed to thallium-contaminated kale chips, which improved after exposure ceased. Ghosal et al. (2025) reported associations between specific chemical mixture clusters and asthma, allergy, and allergic/respiratory multimorbidity. The mixtures included furans, dioxins, PAHs, PCBs, BFRs, and pesticides, representing exposure to multiple contaminants through the infant diet.

Table 1. Direct Health Outcome Studies

No	Study	Chemical exposure	Age	Health outcome	Main finding
1	Alamu et al. (2020)	AFB1	6-24 months	Underweight, stunting	AFB1-lysine associated with higher OR for stunting and underweight
2	Cantoral et al. (2021)	Fluoride	12-18 years	Dental caries	Dietary fluoride intake inversely associated with caries indices
3	Castiblanco-rubio et al. (2025)	Fluoride	1-5 years	Dental fluorosis	Fluoride intake at age 1 year associated with higher OR for fluorosis
4	Chen et al. (2018)	AFB1, FB1	0-36 months	Growth (HAZ/WAZ/WHZ)	FB1 associated with lower WAZ and HAZ
5	Choudhury et al. (2025)	Thallium	2-5 years	GI and neurological symptoms	Symptoms improved after exposure ceased
6	Costa et al. (2026)	Acrylamide, BPA	4-13 years	Pubertal development	AA negatively associated with puberty in girls; BPA positively associated in boys
7	Gálvez-Ontiveros et al. (2024)	BPA, BPS	3-12 years	Overweight/obesity	BPA (meat & eggs) associated with higher OR for overweight in females
8	Ghosal et al. (2025)	Mixtures (furans, dioxins, PAHs, PCBs, BFRs, pesticides)	8-12 months	Asthma, allergy, multimorbidity	Specific chemical mixture clusters associated with higher OR for asthma/allergy
9	Huong et al. (2019)	AFB1, OTA, fumonisins	<5 years	Stunting/wasting	Mycotoxin mixture associated with lower HAZ/WHZ
10	Kashala-Abotnes et al. (2018)	Cyanogen/cyanide	12-48 months	Neurodevelopment & motor development	Higher cyanogen associated with poorer developmental and motor scores
11	Kvestad et al. (2018)	Methylmercury (MeHg)	4-6 years	Cognitive function	No association despite increased THHg
12	Leroy et al. (2018)	AFB1	6-12 months	Linear growth	Higher serum AFB1-lysine associated with greater growth at 4 months
13	Magalhães et al. (2024)	BPA	13 years	Cardiometabolic risk pattern	BPA associated with higher OR for high-risk cardiometabolic cluster
14	Ramírez et al. (2024)	BPA, BPS	6-12 years	Cognitive function	Gene-bisphenol interactions modified cognitive scores
15	Signes-pastor et al. (2019)	Inorganic arsenic (iAs)	4-5 years	Neuropsychological development	iAs associated with lower motor function scores
16	Troeschel et al. (2025)	Lead chromate (PbCrO4)	0-6 years	Lead poisoning	Median BLL 7.2 µg/dL; GI and developmental/behavioral symptoms
17	Viveiros et al. (2021)	Cu, Pb (luster dust)	1-11 years	Metal poisoning	Vomiting/diarrhea; blood lead 12 µg/dL

Intermediate biological response studies

Six studies did not assess a clinical endpoint directly but instead measured biological responses or biomarkers that may be relevant to disease processes, as summarized in Table

2. These included biomarkers of early renal injury and oxidative stress (urinary ACR, NAG, MDA, and 8-OHdG) in relation to DEHP and melamine exposure from contaminated foods, reproductive hormone alterations (FSH, LH, and SHBG) in relation to phthalate metabolites, immune biomarkers,

including reduced hepatitis B vaccine-induced antibody titers, in relation to aflatoxin exposure; and changes in gut microbiome composition, including altered bacterial community structure and increased *Clostridioides difficile* abundance, in relation to mixed mycotoxin exposure in infants.

Tsai et al. (2022) reported significant associations between DEHP and melamine exposure and urinary ACR, while melamine exposure was also associated with MDA and 8-OHdG. Wu et al. (2018) similarly found correlations between melamine exposure and urinary ACR and NAG. Wen et al. (2017) reported associations between phthalate exposure and reproductive hormone measures, including FSH and SHBG. Githang'a et al. (2019) found that AFB1 exposure was associated with lower anti-HBs levels following hepatitis B vaccination. Ayeni et al. (2024) reported an association between mycotoxin detection and altered gut microbiome composition, while Daou et al. (2025) found a weak correlation between OT α and urinary NAG. These findings reflect biological responses or biomarker changes rather than diagnosed clinical conditions. They were therefore considered separately from the 17 studies reporting direct health-related outcomes.

Findings by Chemical Group and Food-Related Exposure Pathways

Across the 23 studies, eight broad chemical groups were represented, with their distribution and food-related exposure pathways summarized in Table 3. Metals and metalloids (lead, copper, mercury, arsenic, and thallium) were represented in five studies, reflecting both acute contamination incidents (lead chromate in cinnamon-containing products, copper/lead in cake decorations, and thallium in kale chips) and chronic dietary exposure (methylmercury and inorganic arsenic). Evidence included direct outcomes such as poisoning and neurodevelopmental effects. Fluoride was represented in two studies, both examining dietary fluoride exposure in relation to dental outcomes, including dental caries and dental fluorosis.

Endocrine-disrupting compounds and plasticizers (BPA, BPS, and phthalates) were equally prominent (7 studies), with dietary exposure occurring primarily through food-contact packaging and contaminated processed foods, and evidence spanning both direct outcomes (obesity, pubertal development, cognition, and cardiometabolic risk) and intermediate biomarkers (reproductive hormone alterations and early renal injury). A related but mechanistically distinct group, melamine and food-adulteration contaminants, was identified in two studies (Tsai et al., 2022; Wu et al., 2018), both involving melamine exposure and reporting renal injury and oxidative-stress biomarkers. Mycotoxins (aflatoxins, fumonisins, ochratoxins, and related compounds) were similarly well represented (7 studies), primarily linked to maize- and cereal-based foods, breast milk, and complementary foods, with evidence mainly involving growth outcomes, together with immune and gut microbiome responses.

The remaining three chemical groups were each represented in a single study: processing-related contaminants (acrylamide), natural plant toxins (cyanogenic glycosides), and complex environmental contaminant mixtures. These studies reported evidence related to pubertal development, neurodevelopment, and allergic or respiratory outcomes, respectively. Overall, the chemical-group distribution showed that food-related chemical exposures in children involved both individual contaminants and complex

mixtures, with exposure pathways spanning contaminated staple foods, food-contact materials, food adulteration, processed foods, and acute contamination events.

DISCUSSION

This systematic review synthesized evidence from 23 studies examining food-related chemical exposure and health outcomes in children across 14 countries. The evidence spanned three levels of the exposure-health outcome continuum: direct health-related outcomes ($n = 17$ studies) and intermediate biological responses ($n = 6$ studies). Eight chemical groups were represented, with mycotoxins and endocrine-disrupting chemicals/plasticizers each examined in 7 studies and metals/metalloids examined in 5 studies. Across these groups, the review identified associations between food-related chemical exposure and a range of health-related outcomes, including impaired growth, neurodevelopmental and cognitive outcomes, altered pubertal development, cardiometabolic risk, dental outcomes, renal and oxidative-stress biomarkers, immune and gut microbiome responses, and acute clinical toxicity. However, the consistency of these associations varied across chemical-outcome relationships, with some supported by several studies and others based on single-study evidence or showing inconsistent findings. These findings highlight the importance of food as a route of chemical exposure during childhood, while also indicating substantial variation in the strength and consistency of the available evidence.

The included studies involved children and adolescents across different developmental stages, ranging from infancy to adolescence, allowing the review to capture health effects of food-related chemical exposure across a broad pediatric age range. This broad age range is relevant because dietary patterns, exposure levels, biological responses, and developmental susceptibility may differ across childhood. However, the variation in age groups also limits direct comparison of findings across studies.

The strength and consistency of the evidence varied across chemical-outcome associations. Some relationships were examined in several studies, particularly mycotoxin exposure in relation to child growth and endocrine-disrupting chemicals/plasticizers in relation to developmental and metabolic outcomes. In contrast, several associations were supported by single studies, including cyanogenic toxin exposure and neurodevelopment, acrylamide exposure and pubertal development, and complex chemical mixtures and asthma or allergy outcomes. Other relationships showed inconsistent findings across studies, such as aflatoxin exposure and child growth and methylmercury exposure and cognitive function. Thus, the number of studies supporting an association, the consistency of their findings, and differences in study design and exposure assessment should be considered when interpreting the overall evidence.

The methodological quality varied across the included studies, with most studies (15/23) assessed as having a moderate risk of bias, while 6 had low risk and 2 had high risk of bias. The predominance of moderate-risk studies indicates that the findings provide useful evidence of potential relationships between food-related chemical exposure and child health, but the overall certainty of the evidence remains limited. This should be considered particularly when interpreting associations derived from observational studies, small samples, or studies with less comprehensive control of potential confounding factors.

Table 2. Studies Reporting Intermediate Biological Responses

Study	Chemical exposure	Biomarker	Main finding
Tsai et al. (2022)	DEHP, melamine	Oxidative stress & early renal injury	DEHP and melamine associated with ACR and MDA/8-OHdG
Wen et al. (2017)	Phthalates (DEHP, etc.)	Reproductive hormones	DEHP associated with higher FSH and SHBG
Githang'a et al. (2019)	AFB1	Hepatitis B vaccine-induced immunity	AFB1 associated with lower anti-HBs levels
Ayeni et al. (2024)	Mixed mycotoxins (AFM1, FB1/2, OTA, etc.)	Gut microbiome composition	Mycotoxin detection associated with altered microbiome composition
Wu et al. (2018)	Melamine, DEHP	Early renal damage	Melamine correlated with urinary ACR and NAG
Daou et al. (2025)	OTA, OTα	Renal biomarkers	OTα weakly correlated with urinary NAG

Table 3. Chemical Groups and Food-Related Exposure Pathways

Chemical group	Specific chemicals	No. of studies*	Main dietary sources	Evidence type
Endocrine-disrupting chemicals / plasticizers	BPA, BPS, phthalates (DEHP, DiNP, DBP, BBzP)	7	Food-contact packaging, contaminated processed foods	Direct outcomes (puberty, obesity, cognition, cardiometabolic risk) & intermediate responses (reproductive hormones, renal biomarkers)
Mycotoxins	AFB1, AFM1, FB1, FB2, OTA, OTα, CIT, AME	7	Maize-based foods, breast milk, complementary foods, contaminated cereals	Direct outcome (growth) & intermediate responses (immune, renal, microbiome)
Metals & metalloids	Cu, Pb, PbCrO4, Hg (MeHg), iAs, Tl	5	Cake decorations, fatty fish, cereals/rice, kale chips, cinnamon-containing applesauce	Direct outcomes (poisoning, neurodevelopment)
Fluoride	F ⁻	2	Drinking water and dietary sources	Direct outcomes (dental caries and fluorosis)
Melamine / food-contact adulteration contaminants	Melamine	2	Melamine-adulterated dairy/food products	Intermediate responses (renal injury and oxidative-stress biomarkers)
Natural plant toxins (cyanogenic glycosides)	Cyanogen/cyanide	1	Cassava/cassava flour	Direct outcome (neurodevelopment)
Complex environmental contaminant mixtures	Furans, dioxins, PAHs, PCBs, BFRs, pesticides, trace elements, acrylamide	1†	Infant diet (early solid foods; French Infant Total Diet Study)	Direct outcomes (asthma/allergy)
Processing-related contaminants	Acrylamide	1	Fried/baked starchy foods	Direct outcome (pubertal development)

*The number of studies sums to 26 across chemical groups because three studies contributed to two groups: (Costa et al., 2026) which assessed acrylamide and BPA, and (Tsai et al., 2022) and (Wu et al., 2018), which assessed DEHP and melamine. Thus, the table represents 23 unique studies overall.

†(Ghozal et al., 2025) is classified under Complex environmental contaminant mixtures only. Its trace-element, PAH, PCB, BFR, pesticide, and acrylamide components were analyzed jointly as latent mixture clusters (sparse NMF) rather than as separate single-chemical effect estimates.

Endocrine-active Chemicals and Metabolic/developmental Outcomes

Bisphenols (BPA and BPS) and phthalates were among the most frequently studied chemical groups, with seven included studies examining their associations with developmental, metabolic, cognitive, and biological outcomes. Exposure was assessed using dietary questionnaires, estimated intake, dietary records, and urinary biomarkers, with several studies examining exposure in relation to food-contact materials or contaminated foods. Across these studies, associations between endocrine-active chemicals and child health outcomes varied by chemical, outcome, and exposure assessment approach, indicating heterogeneity in the observed relationships.

Recent evidence further contextualizes phthalate exposure among children. Jung et al. (2025) reported

exposure to phthalates and nonphthalate plasticizers among children from Korea, Thailand, Indonesia, and Bangladesh, with DEHP representing a major contributor to exposure among Indonesian children. Complementing these findings, a recent systematic review documented the migration of phthalates, bisphenols, and per- and polyfluoroalkyl substances (PFAS) from food packaging into food (Tanzer et al., 2025), highlighting food-contact materials as a potential pathway of dietary exposure to these chemicals. Similar concerns are reflected in the health outcomes examined in the included studies, which covered young children to adolescents, including periods of rapid growth and puberty. BPA and BPS exposure was associated with overweight/obesity (Gálvez-Ontiveros et al., 2024), pubertal development (Costa et al., 2026), cardiometabolic risk (Magalhães et al., 2024), and cognitive function in interaction with genetic polymorphisms (Ramírez et al., 2024).

The findings suggested associations across several developmental and metabolic outcomes, but the direction and consistency of associations varied across chemicals and outcomes. The sex-specific association between BPA and pubertal development observed by Costa et al. (2026), is consistent with evidence from previous studies. Berger et al. (2018) reported associations of prenatal BPA and high-molecular-weight phthalates with later pubertal development in girls but earlier pubertal development in boys, while Freire et al. (2024) also identified sex- and chemical-specific associations between prenatal exposure to phthalates and synthetic phenols and pubertal development. These findings indicate that the observed associations may differ according to sex and the specific chemical examined.

Similarly, phthalate exposure was associated with alterations in reproductive hormones and early renal biomarkers (Wen et al., 2017; Tsai et al., 2022; Wu et al., 2018). Recent evidence further supports these findings, with the Hokkaido study reporting associations between urinary phthalate metabolites and reproductive, adrenal, and gonadotropic hormones in peripubertal children (Yasuda et al., 2025), while a longitudinal panel study in Chinese children found associations between urinary phthalate metabolites and early indicators of kidney injury, including cystatin C, β 2-microglobulin, and cystatin C-based estimated glomerular filtration rate (Zheng et al., 2024).

The BPA-overweight/obesity association requires careful interpretation because energy intake, physical activity, socioeconomic status, pubertal stage, and sex may influence both chemical exposure and obesity risk. Zhang et al. (2014) adjusted phthalate-obesity associations for pubertal onset, socioeconomic level, physical activity, and dietary nutrient intake, while Li et al. (2023) found that associations between urinary MMP and MnBP and childhood obesity were no longer significant after adjustment for physical activity and dietary intake.

Biologically, the observed associations with bisphenols are plausible because BPA can interfere with endocrine signaling through interactions with hormone receptors and can modulate transcription factors involved in metabolic regulation, including PPARs. BPA has also been associated with epigenetic alterations, including changes in DNA methylation, histone modifications, and non-coding RNA expression, which may affect gene regulation during development (Besaratnia, 2023). Recent mechanistic evidence further describes oxidative stress, inflammation, and DNA damage as potential pathways underlying BPA-related biological effects (Ahmad et al., 2024). However, most evidence was observational, and differences in exposure assessment, age, outcome definitions, and biomonitoring time points may contribute to heterogeneity and limit causal interpretation.

Mycotoxins and Child Growth

Mycotoxins were among the most frequently studied chemical groups, with seven studies examining aflatoxins, fumonisins, ochratoxins, and related compounds. Dietary exposure was primarily linked to maize- and cereal-based foods, complementary foods, and, in some studies, breast milk, assessed through dietary records, food analysis, and biomarkers. Consistent with our findings, Shirima et al. (2013) linked higher maize intake with aflatoxin biomarker levels in Tanzanian children and used plasma AF-alb and urinary FB1 to assess internal exposure. A recent systematic review also identified multiple mycotoxins in infant foods, particularly cereal-based products (Ullah et al., 2025).

Across the included studies, mycotoxin exposure was generally associated with poorer growth outcomes, including lower height-for-age, weight-for-age, or weight-for-height and greater odds of stunting or underweight (Alamu et al., 2020; Chen et al., 2018; Huong et al., 2019). This pattern is consistent with a systematic review and meta-analysis reporting negative associations between AFB1 exposure and HAZ and WAZ, as well as increased risks of underweight and stunting in prospective cohorts (Nejad et al., 2023). Similarly, Andrews-Trevino et al. (2021) found that higher AFB1-lysine exposure was associated with poorer growth and increased odds of stunting in a longitudinal cohort of children in Nepal. However, findings were not fully consistent, as Leroy et al. (2018) reported slightly greater linear growth with higher serum AFB1-lysine.

The observed associations are biologically plausible because mycotoxins can impair intestinal function through disruption of the gut microbiota, intestinal barrier, inflammation, and oxidative stress. Aflatoxin exposure has been proposed to contribute to environmental enteropathy, which may impair nutrient uptake and cause malabsorption, potentially contributing to childhood stunting (Urugo et al., 2024). Recent toxicological evidence further indicates that mycotoxins can disrupt intestinal barrier integrity, induce oxidative stress and inflammation, and alter the gut microbiota (Li et al., 2025). Nevertheless, causal interpretation remains limited because studies varied in exposure assessment and control of confounding. Seasonal and individual variation in aflatoxin exposure may complicate exposure characterization (Tessema et al., 2021), while residual and time-varying confounding and potential reverse causation cannot be excluded in longitudinal studies (Becerra-tom et al., 2024).

Metals/metalloids and Neurodevelopment or Clinical Toxicity

Metals and metalloids were examined in six studies, including lead (Pb), copper (Cu), methylmercury (MeHg), inorganic arsenic (iAs), and thallium (Tl). Dietary exposure occurred through contaminated foods and food ingredients and was assessed using dietary questionnaires, urinary biomarkers, blood lead levels, hair mercury concentrations, and chemical analysis of food samples. Recent studies in children identified cereals and potatoes as important dietary sources of iAs and fish as a major source of MeHg (Becerra-tom et al., 2024). A Swedish total diet study further demonstrated the relevance of food-based assessment for estimating metal exposure in children (Bjermo et al., 2025), while a study of Mexican adolescents linked dietary intake with blood Pb levels (Rodríguez-carmona et al., 2025).

The reported outcomes ranged from neurodevelopmental and cognitive effects to dental effects and acute clinical toxicity. Dietary iAs exposure was associated with poorer neuropsychological performance, particularly global motor development (Signes-pastor et al., 2019), consistent with a recent systematic review reporting an inverse association between arsenic exposure and cognitive performance in children (Tian et al., 2025). Evidence for mercury-related neurodevelopmental effects was less consistent, as Kvestad et al. (2018) found no notable association between hair mercury and cognitive performance, while Klus et al. (2023) similarly found no significant associations between postnatal MeHg exposure and 17 neurodevelopmental outcomes at age 7 years. For Pb, a recent systematic review found no clear association between maternal Pb exposure in the context of seafood consumption and child cognitive or behavioral

outcomes, although a weak negative association with motor development was reported (Balalian et al., 2025). In contrast, several included studies documented clinically important Pb exposure following contaminated food products, while thallium exposure was accompanied by clinical symptoms. Overall, the findings suggest that health effects vary across metals and exposure contexts.

The biological plausibility of these findings is supported by the ability of metals and metalloids to disrupt neurological and cellular processes. Exposure to lead, mercury, and arsenic has been associated with impaired neurocognitive development in children, including deficits in IQ attention, memory, and behavior (Guo et al., 2025). These effects may involve oxidative stress, altered neurotransmission, neuroinflammation, and mitochondrial dysfunction. For thallium, toxicity involves disruption of potassium-dependent cellular processes and enzyme activity, together with reactive oxygen species generation and mitochondrial dysfunction (Fujihara & Nishimoto, 2024). These mechanisms are consistent with the neurological and systemic manifestations reported in the included thallium poisoning investigation.

However, the evidence should be interpreted cautiously because the included studies differed in design, exposure biomarkers, chemical species, dose, and outcome assessment. Co-exposure and exposure timing may also influence neurodevelopmental associations. Bauer et al. (2020) highlighted the importance of metal co-exposure and exposure windows, while Farías et al. (2022) found that associations between prenatal Pb exposure and infant neurodevelopment varied with concurrent Hg and Mn exposure. These differences may limit comparisons across studies.

Chemical Mixtures and Multiple Health Outcomes

Real-world exposure to food-related chemicals is unlikely to occur in isolation, as children may be simultaneously exposed to multiple contaminants through the same or different foods. However, among the 23 studies included in this review, only Ghozal et al. (2025) explicitly evaluated mixtures of food-related chemicals in relation to multiple health outcomes. Evidence from the French ELFE cohort similarly showed that prenatal dietary exposure to specific chemical mixtures was associated with eczema, food allergy, or wheezing in children (Ghozal et al., 2024). Beyond dietary exposure, Amine et al. (2025) found that early-life exposure to mixtures of parabens and phthalates was associated with poorer health across cardiometabolic, respiratory, and neurodevelopmental domains at age 3 years. Together, these findings indicate that mixture-based approaches can capture potential health effects of simultaneous chemical exposures across multiple outcomes.

These findings are biologically plausible because multiple food contaminants may act through common or distinct pathways, and the effects of one chemical may vary with co-exposure to others. Evidence from pediatric studies has reported interactions between metals, although these findings have not been consistent across exposure measures and analytical approaches (Claus Henn et al., 2014). From a food-contaminant perspective, Vejdovszky et al. (2021) found that combined exposure to several contaminants contributed to cumulative risks for pre- and neonatal development, with no single substance clearly dominating these risks. Thus, the associations observed by Ghozal et al. (2025) should be interpreted as associations with combined exposure patterns

rather than evidence that individual chemicals independently or interactively caused the observed outcomes.

Nevertheless, evidence on chemical mixtures remains limited in this review because it was derived from a single study and relied on estimated dietary exposure. Recent longitudinal studies demonstrate the potential value of repeated exposure measurements and mixture-based approaches. Zhang et al. (2025) used repeated urinary measurements in a panel of children to examine associations between EDC mixtures and metabolic outcomes, while Amine et al. (2025) assessed repeated early-life chemical measurements in relation to a multi-domain health score.

Strengths and Limitations

At the review level, between-study heterogeneity is a major limitation. The included studies differed in sources and types of exposure, measurement methods, study designs, age ranges, sample sizes, and definitions and measurements of outcomes. Exposure was assessed using food frequency questionnaires, dietary recall, food diaries, intake estimates, analysis of food contaminants, and biomarkers in urine, blood, hair, or tissue. These differences limit direct comparisons between studies and make it difficult to draw quantitative conclusions regarding the magnitude of the association between exposure and health outcomes. A wide age range can also influence dietary patterns, exposure levels, metabolism, and developmental vulnerability. Furthermore, most of the evidence comes from observational studies, so confounding and reverse causality remain important considerations. Socioeconomic factors, dietary patterns, nutritional status, physical activity, family characteristics, and developmental factors may be associated with both exposure and health outcomes. Differences in the selection and control of confounders across studies may also contribute to the heterogeneity of results. In addition, the review was restricted to studies published in English, which may have introduced language bias and resulted in the omission of relevant evidence published in other languages.

In terms of the level of evidence, support for chemical-outcome associations varies across different groups of chemicals and outcomes. Some associations are supported by more than one study, particularly the association between mycotoxin exposure and child growth, as well as associations between phthalate or bisphenol exposure and developmental, metabolic, or biological biomarker outcomes. However, the direction and strength of these associations are not always consistent, so the evidence for these associations cannot yet be considered uniform. Some associations are supported by only one study, including the association between exposure to cyanogenic toxins and neurodevelopment, exposure to acrylamide and pubertal development, and exposure to mixtures of various contaminants and asthma/allergy multimorbidity. The relationship between methylmercury exposure and cognitive function also remains inconclusive in this review, as the available findings did not indicate a significant association. Furthermore, some studies on exposure to metals and contaminants in food are case-report studies or clinical case investigations, thus providing evidence that differs in nature from epidemiological studies on chronic exposure in populations.

Overall, limitations in the level of evidence are primarily related to the still-limited number of studies for most chemical-outcome pairs, variations in methodological quality, and inconsistencies in the measurement of exposure and outcomes. Therefore, findings supported by multiple studies may indicate a more consistent association, whereas

associations reported by only one study should be viewed as preliminary evidence. Associations with inconsistent results or limited evidence still require confirmation through studies with longitudinal designs, repeated exposure measurements, more comprehensive control for confounding factors, and adequate sample sizes.

A limitation of this review concerns the timing and iterative nature of the protocol revision. The revised protocol (CRD420261517722), reflecting the PECO framework, updated publication period, and expanded evidence base, was registered on 25 September 2026, after re-screening in response to peer review had already begun; the amended methodology therefore does not meet the strict definition of prospective registration relative to its own implementation. In addition, eligibility refinement continued iteratively during the data extraction phase: as the PECO framework and outcome categories were finalized, several previously extracted articles were subsequently excluded for not meeting the revised eligibility criteria (e.g., reporting exposure or biomarker data without a health-related or health-relevant biological outcome). These exclusion decisions were based solely on adherence to the pre-specified eligibility criteria and were not influenced by the reported findings of individual studies. The final set of included studies reflects the fully revised PECO criteria described in the Methods section. These limitations are disclosed for transparency.

Implications

The findings of this review have implications for food safety monitoring and child health protection. Because children may be exposed to multiple chemicals through food, prevention and monitoring efforts should not focus exclusively on individual contaminants. Monitoring contaminants in foods frequently consumed by children, particularly among vulnerable groups, may help identify important sources of exposure that require control. Food safety education for parents, caregivers, and food providers may also contribute to reducing preventable exposure. These findings highlight the importance of integrating food contamination monitoring, dietary exposure considerations, and child health protection within a broader food safety framework.

CONCLUSIONS AND RECOMMENDATION

This systematic review indicates that dietary and food-related chemical exposures may be associated with a range of health-related outcomes in children, including impaired growth, neurodevelopmental and cognitive outcomes, altered pubertal development, cardiometabolic risk, dental outcomes, renal and oxidative-stress biomarkers, immune and gut microbiome responses, and acute clinical toxicity. The evidence encompassed multiple chemical groups, including mycotoxins, metals and metalloids, and endocrine-disrupting chemicals and plasticizers. However, the strength and consistency of the evidence varied across chemical-outcome relationships. Some associations were supported by several studies, whereas others were based on single-study evidence or showed inconsistent findings. Substantial heterogeneity in study populations and developmental stages, exposure assessment methods, chemical species and mixtures, outcome measures, and study designs further limits direct comparison and causal interpretation. Overall, the available

evidence supports associations between some dietary and food-related chemical exposures and specific health-related outcomes in children, but does not establish that dietary chemical exposures generally cause adverse health effects.

Future research should prioritize well-designed prospective studies with adequate sample sizes and broader representation of developmental periods to better assess temporal relationships between dietary and food-related chemical exposure and health outcomes. Repeated exposure assessment, including biomonitoring where feasible, could help capture changes in exposure over time and reduce reliance on single measurements. Standardized approaches integrating dietary assessment, food contaminant measurements, and biomonitoring are needed to improve comparability across studies. Where data permit, studies should examine dose-response relationships and relevant developmental windows while addressing important sources of bias, including confounding and reverse causality. Because children may be simultaneously exposed to multiple chemicals, mixture-based analytical approaches should also be expanded beyond single-chemical models and evaluated across multiple health outcomes. Finally, more prospective research is needed in low- and middle-income countries (LMICs), where dietary patterns, food contamination sources, exposure levels, and environmental conditions may differ from those in high-income settings. These approaches could strengthen the evidence base and improve understanding of the potential health implications of dietary and food-related chemical exposure during childhood.

DECLARATION

Ethics approval and consent to participate

This study is a systematic review that uses secondary data from published scientific articles; therefore, it does not directly involve participants and does not require ethical approval or consent to participate.

Consent for publication

Not applicable.

Availability of data and materials

All data supporting the findings of this study are derived from published scientific articles and are included in the References section.

Conflicts of interest Statement

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Authors' contributions

Delto Loisandro Tanesab and Ayu Windy Malisa Menno contributed equally to the conceptualization of the study, the development of the methodology, the literature search and selection, data extraction and analysis, the writing of the initial draft, manuscript revisions, and approval of the final version of the manuscript. Both authors are fully responsible for the entire content of the study.

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