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Review Article

Ultra-Processed Food Exposure from Childhood to Young Adulthood: A Systematic Review of Obesity Risk, Cardiometabolic Dysregulation, and Emerging Biomarkers

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Abstract: Ultra-processed foods (UPFs) have become an increasingly prominent component of dietary patterns worldwide, especially among children, adolescents, and young adults. Accumulating evidence links high UPF consumption to obesity and cardiometabolic disorders, yet findings remain fragmented across developmental stages and outcome domains. This systematic literature review aimed to synthesize evidence on associations between UPF exposure from childhood through young adulthood and (i) overweight and obesity trajectories, (ii) cardiometabolic dysregulation, and (iii) emerging biomarkers of early metabolic risk. This systematic review was carried out following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) framework. Peer-reviewed observational studies were identified through structured searches of Scopus and assessed for eligibility based on predefined criteria. Included studies primarily quantified UPF intake using the NOVA classification and reported obesity-related, cardiometabolic, or biomarker outcomes in youth and young adults. Due to heterogeneity in study design, populations, and exposure metrics, findings were synthesized narratively. Findings derived from both longitudinal and cross-sectional research consistently indicate that greater consumption of UPFs is linked to a higher likelihood of overweight and obesity across different stages of development, alongside adverse cardiometabolic profiles including impaired glucose homeostasis, dyslipidemia, inflammation, and altered appetite-regulating hormones. Emerging biomarkers, such as puberty sensitive indicators and nutritional status markers, suggested early biological perturbations linked to UPF intake. Structural determinants including food marketing, socioeconomic vulnerability, and food environments strongly shaped UPF exposure and modified health outcomes. Overall, UPF consumption across early life is consistently associated with metabolic risk, underscoring the need for life-course oriented, multi-level prevention strategies.

Keywords: cardiometabolic risk, children and adolescents, life-course approach, obesity, ultra-processed foods

1. INTRODUCTION

Ultra-processed foods (UPFs) have increasingly come to characterize modern food environments and dietary patterns globally. In the field of nutritional epidemiology, UPF intake is typically described using the NOVA framework, which classifies food items based on the level and intent of industrial processing involved. UPFs are generally defined as highly engineered products composed predominantly of processed ingredients, frequently incorporating technological additives such as stabilizers, flavor agents, and synthetic colorings aimed at enhancing taste, appearance, shelf stability, and ease of consumption. [1], [2]. Unlike minimally processed or whole foods, UPFs are engineered to be hyper-palatable, energy-dense, and easily consumable, characteristics that raise substantial concerns regarding their long-term health implications. Over the past two decades, UPF consumption has increased markedly across diverse geographic regions and socioeconomic contexts. Evidence indicates that this upward trend is observable not only in high-income countries but also

In low- and middle-income countries undergoing rapid shifts in nutritional patterns, growing UPF consumption [3], [4], [5]. Among children, adolescents, and young adults has been consistently linked with compromised diet quality. This includes elevated intake of added sugars, unhealthy fats, and sodium, alongside inadequate levels of fiber, vitamins, and key micronutrients. The widespread adoption of highly processed dietary habits signifies a profound shift in youth nutrition and presents major challenges for public health systems worldwide.

Across various regions, empirical evidence indicates that elevated UPF intake in young populations is associated with poor nutrient profiles and negative health consequences. Globally, diets abundant in UPFs tend to promote higher consumption of energy-dense macronutrients and reduced intake of protective dietary components, thereby increasing the risk of obesity and cardiometabolic diseases [6], [7], [8]. In Southern Europe, for instance, reduced adherence to traditional diets such as the Mediterranean diet has coincided with increased UPF intake, especially among younger generations. [9], [10]. Similar patterns have been documented in countries like Portugal, Italy, and Chile, where rising UPF consumption parallels worsening dietary quality in children and adolescents [11], [12]. Collectively, these findings highlight the broad and systemic impact of dietary transitions involving UPFs.

The significance of UPF consumption for public health is further emphasized by strong evidence linking its intake with excess weight and obesity in younger populations. Numerous observational studies have consistently shown that children and adolescents with higher UPF consumption levels are more likely to experience increased body weight and adiposity. For example, findings from the French NutriNet-Santé cohort revealed a positive correlation between UPF intake and elevated body mass index (BMI), along with a heightened risk of obesity [13], [14]. These outcomes are supported by systematic reviews that consolidate results from multiple populations, consistently reporting adverse anthropometric changes associated with high UPF consumption. [15], [16].

Adolescence constitutes a particularly sensitive life stage in the context of nutrition and health. During this period, dietary behaviors are frequently shaped by increased personal autonomy and inconsistent eating patterns, often resulting in greater intake of calorie-dense, nutrient-deficient products, notably ultra-processed foods (UPFs). Research suggests that adolescents with elevated UPF consumption tend to exhibit suboptimal diet quality and adverse metabolic profiles, raising serious concerns about long-term health outcomes [17], [18]. Empirical data from Brazil, for instance, has identified associations between the extent of UPF intake and several metabolic health indicators in adolescent groups, underscoring the likelihood of early metabolic dysregulation during this stage of development [19], [20]. Given adolescents' increased susceptibility to environmental and social influences, these insights reinforce the urgency of implementing public health interventions to reduce UPF consumption among this age group [18], [21].

Childhood, adolescence, and young adulthood are widely recognized as critical periods for dietary exposures with long-lasting implications for metabolic health. During these life stages, individuals experience rapid physical growth, hormonal changes, and neurodevelopmental maturation, while simultaneously establishing eating behaviors that often persist into adulthood [22], [23]. Psychosocial pressures during adolescence such as peer influence, academic stress, and heightened digital media exposure play a significant role in shaping dietary behaviors [24], [25]. These factors often lead to increased reliance on ultra-processed foods (UPFs) and contribute to unhealthy weight trajectories that may persist into adulthood. Neurobiological mechanisms are also critically involved in dietary regulation during adolescence. The adolescent brain is particularly sensitive to reward-based stimuli, especially from energy-dense and highly palatable foods. This elevated reward sensitivity may enhance preferences for UPFs and reinforce habitual consumption behaviors [26], [27]. When coupled with pervasive marketing strategies aimed at young consumers, this developmental vulnerability increases the risk of establishing long-term dietary patterns that promote excessive caloric intake and subsequent weight gain [15], [18]. The convergence of neurobiological predispositions, psychosocial influences, and commercial marketing highlights the significance of shaping early-life dietary environments to promote healthier long-term outcomes.

Several biological and behavioral pathways have been suggested to explain the link between UPF consumption and the development of obesity and cardiometabolic disorders. From a biological perspective, UPFs are typically energy-dense with low satiety potential, and their nutrient profile is often characterized by high levels of added sugars and unhealthy fats, while being deficient in dietary fiber, which may impair satiety regulation, accelerate eating speed, and lead to excessive consumption [28], [29]. Additionally, UPFs may include substances such as phthalates and bisphenol A, which have been associated with metabolic dysfunction and increased obesity risk due to hormonal disruption and altered energy metabolism [28]. On the behavioral side, UPF intake is frequently tied to modern lifestyle habits, such as dependence on convenience foods and exposure to pervasive food marketing. Intensive promotional efforts targeting children and adolescents further shape dietary preferences and normalize frequent intake of nutrient-poor, energy-dense products [30], [31], [32].

The prolonged impact of UPF intake beginning in early life is particularly concerning due to consistent evidence showing the persistence of obesity and cardiometabolic risk factors from childhood into adulthood. Longitudinal studies indicate that many children who are obese continue to be obese during adolescence and into later life, which contributes to sustained elevated risks for metabolic conditions [33]. This continuity of obesity presents a major concern for public health. Prolonged obesity from youth into adulthood has been linked to higher likelihood of chronic diseases including type 2 diabetes, cardiovascular disease, and hypertension [34]. As a result, early-life dietary exposures especially high UPF intake are key prevention targets. Reducing UPF intake during childhood and adolescence is essential to prevent long-term metabolic disorders and decrease the overall burden of non-communicable diseases [10], [29], [35].

In this context, it is essential to comprehensively synthesize existing research on UPF exposure spanning from childhood to early adulthood. Although current literature offers meaningful insights into the relationship between UPF intake, obesity, and metabolic health, findings remain fragmented across age categories, outcome variables, and methodological approaches. This review aims to consolidate recent evidence linking UPF consumption to obesity risk, metabolic dysfunction, and early biomarkers across critical developmental stages [4]. Through a life-course perspective and consideration of biological, behavioral, and contextual factors, this synthesis seeks to advance current understanding, highlight existing research gaps, and guide future public health efforts aimed at improving dietary behaviors in younger populations.

2. MATERIALS AND METHODS

This systematic literature review (SLR) followed internationally accepted protocols for evidence synthesis in the field of nutritional epidemiology. The approach emphasized transparency, reproducibility, and methodological rigor in the processes of identifying, evaluating, and integrating data on ultra-processed food (UPF) exposure and its association with obesity-related and cardiometabolic outcomes across the developmental stages from childhood to young adulthood.

2.1. Search Strategy

The development of the review protocol and reporting was guided by the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, which offer a structured and standardized model for conducting rigorous and transparent evidence synthesis [36]. The literature search was implemented using Scopus as the primary database. The search strategy was structured using Boolean logic (AND/OR) to target three main concept blocks: (1) ultra-processed foods; (2) age groups (children, adolescents, youth); and (3) obesity or cardiometabolic outcomes. The specific

Scopus search formula used for titles, abstracts, and keywords was as follows: TITLE-ABS-KEY (("ultra processed" OR ultraprocessed) AND adolescent AND (obesity OR overweight OR BMI OR "metabolic syndrome")). Additional search filters were applied to include peer-reviewed original research articles, written in English, and published within the defined time window of 2021–2025. The literature search was conducted using the Scopus database, selected due to its broad multidisciplinary coverage, extensive indexing of peer-reviewed journals in nutrition, public health, and epidemiology, and inclusion of international publications relevant to ultra-processed food research. Scopus provides comprehensive citation tracking and advanced search functionalities, which facilitate systematic identification of studies across diverse geographic regions and study designs. Furthermore, the reference lists of all included studies were manually examined to retrieve additional relevant publications that might not have been captured through the initial database search, in accordance with best practice guidelines for systematic literature reviews in nutrition science [34].

2.2. Eligibility Criteria

Eligibility criteria were determined in advance to ensure uniformity and relevance in study selection. Studies were included if they met the following conditions: (1) utilized an observational design (cross-sectional or longitudinal); (2) involved populations of children, adolescents, or young adults; (3) clearly measured UPF intake, preferably using the NOVA classification system, which categorizes foods into four groups according to the nature, extent, and purpose of industrial processing. These groups include: (1) unprocessed or minimally processed foods (e.g., fresh fruits, vegetables, grains, milk, and meat); (2) processed culinary ingredients (e.g., oils, butter, sugar, and salt); (3) processed foods (e.g., canned vegetables, cheeses, freshly baked bread); and (4) ultra-processed foods, defined as industrial formulations made mostly or entirely from substances derived from foods and additives, typically containing little or no intact whole food and designed to be hyper-palatable, convenient, and ready-to-consume or a comparable processing-based scheme; (4) reported at least one outcome related to obesity or cardiometabolic health, such as body mass index (BMI), overweight/obesity prevalence, or indicators of metabolic syndrome [14], [35]. Exclusion criteria encompassed: (1) studies that lacked a clear assessment of UPF intake; (2) research focusing only on adult populations beyond the target age range; (3) articles not addressing obesity or cardiometabolic-related outcomes; (4) non-original publications (e.g., commentaries, editorials, or conference abstracts); (5) studies with insufficient methodological quality or inaccessible full-text versions.

2.3. Screening and Selection Process

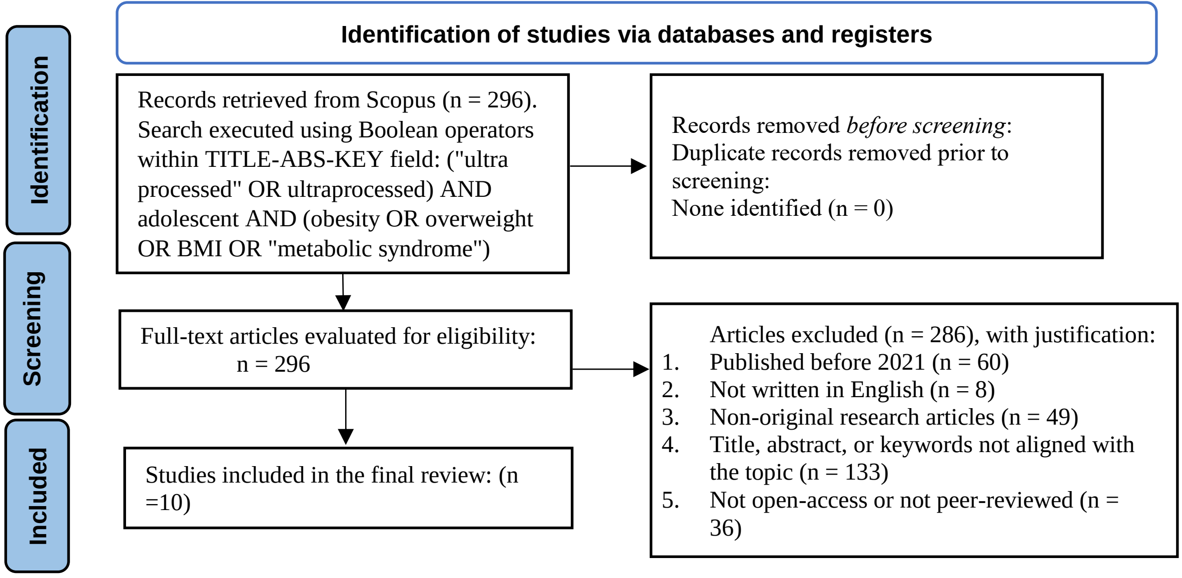

Figure 1. PRISMA 2020-based flow diagram depicting the process of study identification, screening, and inclusion within this systematic review

The process of study selection adhered to the PRISMA 2020 reporting standards to enhance methodological transparency. A total of 296 records were retrieved from the Scopus database and subsequently imported into a reference management tool. Duplicate entries were assessed, though none were identified for removal. Screening occurred in two sequential phases: first, a review of titles and abstracts, followed by full-text evaluation, both guided by predefined eligibility criteria. Any disagreements encountered during the screening process were addressed through consensus among reviewers, and reasons for study exclusion were documented accordingly. Ultimately, ten studies fulfilled the inclusion criteria and were retained for the final synthesis. A visual overview of this selection process is presented in the PRISMA flowchart (Figure 1).

2.4 Quality Assessment and Synthesis Approach

To evaluate methodological rigor and potential bias, all included observational studies were appraised using well-established tools commonly used in nutritional epidemiology. These included the Newcastle-Ottawa Scale (NOS) and relevant checklists from the Joanna Briggs Institute (JBI). These instruments facilitated structured evaluation of study design, exposure and outcome assessments, and confounding control, thereby enhancing the reliability of the synthesized findings [37]. Due to the variability in study characteristics, such as population demographics, UPF exposure definitions, and outcome indicators, a narrative synthesis was employed as the primary strategy for integrating findings. When feasible, comparisons were drawn across developmental stages, geographic regions, dietary assessment methodologies, and levels of statistical adjustment for confounding. Specific emphasis was placed on identifying sources of heterogeneity, including age group, sex, socioeconomic status, and the operational definition of UPF intake. This analytical approach aligns with previously published systematic literature reviews that have investigated dietary exposures and cardiometabolic outcomes among youth populations [34], [35].

3. RESULTS AND DISCUSSION

Based on the predefined Boolean search strategy, a total of 296 records were initially identified in the Scopus database, focusing on ultra-processed foods, adolescents, obesity, and cardiometabolic outcomes. After removing duplicates, 231 unique records proceeded to the title and abstract screening phase. Among them, 221 articles were excluded due to eligibility criteria. Ten eligible articles underwent full-text review and were all included in the final qualitative synthesis. These studies were categorized into four thematic areas: (1) obesity trajectories (Section 3.1), (2) cardiometabolic dysregulation (3.2), (3) emerging biomarkers (4.3), and (4) structural and behavioral determinants of UPF intake (3.4), forming the basis of the narrative synthesis (see Figure 1).

3.1. UPF Exposure and Overweight/Obesity Trajectories Across Development

This section compiles findings regarding the link between UPF consumption and overweight/obesity progression from childhood to young adulthood, based on the studies included in Table 1. The body of evidence reveals a consistent trend in which elevated UPF intake is associated with increased total and central adiposity, higher BMI, greater waist circumference, increased body fat percentage, reduced lean mass proportion, and a heightened risk of developing overweight and obesity across various developmental stages and populations.

Longitudinal data provide strong support for a temporal association between UPF exposure and subsequent weight gain. In a large cohort study from Germany, Figueiredo Barata et al. (2025) tracked 4,762 children aged 3–17 years over approximately 11 years and identified a clear dose–response pattern linking higher UPF intake to increased risk of later overweight and obesity. Specifically, each 10% increment in UPF intake was associated with a 12% increase in odds of becoming overweight (OR 1.12; 95% CI 1.03–1.23) Furthermore, children in the highest quartile of UPF consumption were significantly more likely to develop overweight than those in the lowest quartile (OR 1.40; 95% CI 1.05–2.11). Obesity risk was more than doubled in the third quartile (OR 2.32; 95% CI 1.38–3.88), and remained significantly elevated in the top quartile (OR 1.74; 95% CI 1.06–2.84) [38]. These findings align with life-course epidemiology models, which posit that early and sustained exposure to energy-dense diets during critical developmental periods can program long-term obesity risk through cumulative and pathway effects.

Cross-sectional studies conducted among children and adolescents further support these findings, despite their inherent limitations in establishing causality. In Jordan, Al Hourani et al. (2025) found that UPFs contributed to roughly 40% of children's daily caloric intake, with higher intake levels being modestly but significantly associated with increased waist circumference ($r = 0.119$; $p = 0.005$). Although the strength of this association was relatively small, it highlights the considerable role that UPFs play in overall energy consumption and their potential link to central fat accumulation a key indicator of cardiometabolic risk in youth populations [39]. Similarly, González-Rodríguez et al. (2025) demonstrated that Spanish children and adolescents aged 6–15 years living in socioeconomically vulnerability tended to consume higher amounts of UPFs, exhibited reduced alignment with Mediterranean dietary patterns, and demonstrated less favorable body composition metrics, including elevated BMI [40]. These findings underscore the interplay between social vulnerability, food processing level, and obesity outcomes emphasizing that UPF exposure is not an isolated factor but rather part of broader dietary and contextual influences, as discussed in Section 3.3.

Further insights from clinical and high-risk adolescent populations add depth to our understanding of UPF exposure in relation to body composition beyond BMI. Neres et al. (2025), in a study involving Brazilian adolescents diagnosed with obesity, observed that reduced intake of minimally processed foods coupled with greater reliance on UPFs was associated with increased body fat percentage and decreased lean mass proportion. In addition, neuroendocrine factors involved in appetite regulation were found to be relevant; specifically, agouti-related peptide (AgRP) was identified as a significant predictor of UPF consumption scores ($\beta = 0.30$; $p = 0.04$), regardless of age, adiposity, or presence of binge eating tendencies [41]. These outcomes suggest a potential interaction between UPF intake and biological pathways affecting appetite and body composition, in line with theories of food matrix and metabolic programming presented earlier in Section 3.1.

The transition to young adulthood represents a particularly sensitive period in which UPF intake may play a role in shaping obesity patterns. A large observational study involving 2,512 university students in Turkey, conducted by Eroğlu et al. (2025), identified a statistically significant though modest positive association between UPF intake scores and body mass index (BMI) ($r = 0.101$) Although the correlation was relatively weak, it co-occurred with clusters of maladaptive behaviors such as night eating syndrome and digital dependence, both of which were also linked to elevated BMI [42]. These findings point to the possibility that UPF consumption in early adulthood may be embedded within a broader spectrum of behavioral risk factors.

Collectively, the studies presented in Table 1 offer consistent support for a link between elevated UPF intake and negative weight-related health indicators from early childhood through young adulthood. The strength of the evidence is greatest for longitudinal findings demonstrating incident overweight and obesity, while cross-sectional studies consistently reveal associations with BMI, waist circumference, and unfavorable body composition. Importantly, the magnitude and expression of these associations appear to be modified by developmental stage, socioeconomic context, and behavioral factors, underscoring the complexity of UPF obesity relationships. These findings are theoretically coherent with life course epidemiology and food processing frameworks, which posit that early exposure to highly processed, energy-dense foods can exert lasting effects on metabolic regulation and obesity risk.

Table 1. Evidence Map for Theme 1 UPF Exposure and Overweight/Obesity Trajectories across Development

Author, Year	Country	Population	Study Design	Outcome	Main Finding	Covariates
Figueiredo Barata et al., 2025	Germany, national cohort (KiGGS)	Children & adolescents 3–17 y, n = 4,762, 11 y follow-up	Longitudinal cohort, FFQ-based diet	Incident overweight, incident obesity	10% UPF → overweight OR 1.12 (95% CI 1.03–1.23), Q4 vs Q1 overweight OR 1.49 (1.05–2.11), obesity OR up to 2.32	Age, sex, SES, physical activity, baseline BMI-z
Al Hourani et al., 2025	Jordan	Children/ adolescents, overweight 23.9%, obesity 13.3%	Cross-sectional, 24-h recall	BMI-z, WC, WHtR	UPF 40% energy, WC correlated with UPF (r = 0.119, p = 0.005)	Limited details reported
González-Rodríguez et al., 2025	Spain, urban	Children/ adolescents 6–15 y, n = 280	Cross-sectional	BMI, body composition	Higher UPF intake among vulnerable youth, higher BMI, poorer body composition	Multivariate incl. SES
Neres et al., 2025	Brazil, clinical	Adolescents with obesity n = 96	Cross-sectional, FFQ	Body fat %, lean mass %	Higher UPF linked to ↑ body fat, ↓ lean mass; AgRP predicts UPF score (β = 0.30)	Age, body fat, binge eating
Eroğlu et al., 2025	Turkey, university	Young adults, n = 2,512, mean age 21 y	Cross-sectional survey	BMI	UPF score correlated with BMI (r = 0.101)	Multivariable behavioral models

In conclusion, the collective findings within this theme suggest that exposure to UPFs is positively linked to greater adiposity and elevated risk of obesity, with the most compelling support derived from longitudinal data beginning in childhood and continuing into adolescence and early adulthood. These observations establish a crucial empirical basis for subsequent sections addressing cardiometabolic dysregulation and related physiological mechanisms.

3.2. Cardiometabolic Dysregulation Linked to Ultra-Processed Foods

This section presents evidence connecting UPF intake to disruptions in cardiometabolic regulation during adolescence and young adulthood, relying solely on studies listed in Table 2 and the theoretical frameworks discussed earlier. Overall, the literature shows that frequent UPF consumption is correlated with impaired glucose control, adverse lipid and inflammatory markers, and disrupted metabolic and neuroendocrine regulation, reinforcing its plausible role in early-onset cardiometabolic conditions.

Longitudinal research involving young adults has yielded strong evidence for a time-linked association between UPF intake and impaired glucose metabolism. A prospective study of 85 individuals aged 17 to 22 years with prior experiences of overweight or obesity conducted by Li et al. (2025) found that changes in UPF consumption were significantly related to glucose regulation markers. A 10% increase in UPF intake from baseline was associated with a 51% rise in the likelihood of developing prediabetes (OR 1.51; 95% CI 1.04–2.31), and a more than twofold increase in the risk of impaired glucose tolerance (OR 2.58; 95% CI 1.43–5.85). In addition, higher initial UPF consumption levels were predictive of greater insulin levels two hours after testing and larger insulin AUC values during oral glucose tolerance tests [43]. These outcomes align with life-course models, highlighting the impact of prolonged intake of ultra-processed, calorie-dense products on insulin sensitivity and early metabolic disturbances.

Cross-sectional analyses utilizing a nationally representative sample drawn from the Canadian Health Measures Survey provide further support for these associations across a wider cardiometabolic risk profile. Baric et al. (2025) assessed adults aged 19 to 79 years and documented a clear gradient in cardiometabolic risk that corresponded with increasing UPF intake. Participants in the highest UPF consumption quartiles displayed significantly elevated body mass index and waist circumference, along with a range of adverse metabolic markers, such as higher diastolic blood pressure, increased glycated hemoglobin (HbA1c), fasting insulin, triglycerides, and low-grade inflammation biomarkers including C-reactive protein (CRP) and and white blood cell concentrations continued to show significant associations even after adjusting for fruit and vegetable intake, suggesting that the metabolic consequences of UPF intake may extend beyond dietary quality alone. These findings are aligned with theoretical frameworks discussed earlier in Section 3, which propose that industrial food processing may exert distinct metabolic effects beyond nutrient composition [44].

Further evidence from adolescent clinical samples provides insight into the physiological pathways linking UPF intake with cardiometabolic disturbances. In a cross-sectional analysis involving 96 Brazilian adolescents with obesity, Neres et al. (2025) reported that dietary profiles characterized by elevated UPF consumption and reduced intake of minimally processed foods were linked to disruptions in appetite-regulating neuroendocrine pathways. Specifically, agouti-related peptide (AgRP) a hormone that stimulates appetite and affects energy balance was found to be a significant predictor of UPF intake scores ($\beta = 0.30$; $p = 0.04$), even after controlling for age, adiposity levels, and symptoms of binge eating [41]. Additionally, lower intake of unprocessed foods correlated with increased circulating levels of ghrelin and higher body fat percentage accompanied by reduced lean mass (fat-free mass). While these markers are not classical indicators of cardiometabolic health, they reflect upstream mechanisms through which UPFs may promote excess energy intake, increased adiposity, and early metabolic disturbances, in line with models discussed in Section 3.1.

Collectively, the studies summarized in Table 2 present consistent evidence linking UPF consumption to cardiometabolic dysfunction, including impaired glucose regulation, altered lipid metabolism, inflammation, and neuroendocrine imbalances. The strongest evidence emerges from longitudinal data linking increases in UPF intake to incident prediabetes and impaired glucose tolerance in young adulthood, while cross-sectional population studies demonstrate consistent associations with established cardiometabolic risk markers. Although causality cannot be definitively established due to the predominance of observational designs, the coherence of findings across developmental stages, outcome measures, and biological pathways strengthens the argument that UPF exposure contributes meaningfully to early cardiometabolic risk. These findings highlight the critical need to intervene on UPF intake during early developmental periods in order to halt the advancement from mild metabolic disruptions to clinically evident cardiometabolic conditions.

In summary, evidence from this theme indicates that UPF exposure is consistently associated with early cardiometabolic dysregulation, spanning impaired glucose metabolism, inflammatory activation, dyslipidemia, and neuroendocrine alterations. These findings provide a critical bridge between observed obesity trajectories and the mechanistic pathways through which UPFs may influence long-term cardiometabolic health.

Table 2. Evidence linking ultra-processed food (UPF) exposure with cardiometabolic dysregulation and related biomarkers (childhood to young adulthood)

Study (Author, Year)	Country	Design, Population	Cardiometabolic	Key results	Covariates
Li et al., 2025	United States; Meta-AIR (Children's Health Study)	Longitudinal, young adults with history of overweight/ obesity (n = 85; 17–22 y)	Prediabetes, impaired glucose tolerance, 2-h insulin, insulin AUC	10% UPF → prediabetes OR 1.51 (95% CI 1.04–2.31), impaired glucose tolerance OR 2.58 (95% CI 1.43–5.85), higher baseline UPF → ↑ 2-h insulin & AUC	Demographics, physical activity, total energy intake
Baric et al., 2025	CanadaCHMS 2016–2019	Cross-sectional, adults 19–79 y (n = 6,517)	BMI, WC, diastolic BP, HbA1c, fasting insulin, triglycerides, CRP, WBC	Higher UPF quartiles → ↑ BMI, WC, BP, HbA1c, insulin, TG, CRP, WBC; associations partly attenuated after F&V adjustment	Sociodemographic & lifestyle variables, sensitivity incl. F&V intake
Neres et al., 2025	Brazil, clinical setting	Cross-sectional, adolescents with obesity (n = 96)	Ghrelin, leptin, NPY, AgRP; MCH; α-MSH, body composition	AgRP predicts UPF score ($\beta = 0.30$, $p = 0.04$); lower unprocessed intake → ↑ ghrelin	Age, body fat, binge eating symptoms

Abbreviations: UPF, ultra-processed food; OR, odds ratio; CI, confidence interval; AUC, area under the curve; BMI, body mass index; WC, waist circumference; BP, blood pressure; HbA1c, glycated hemoglobin; CRP, C-reactive protein; WBC, white blood cell count; SES, socioeconomic status; F&V, fruit and vegetable intake; NPY, neuropeptide Y; AgRP, agouti-related peptide; MCH, melanin-concentrating hormone; α-MSH, alpha-melanocyte-stimulating hormone.

3.3. Emerging Biomarkers and Early-Warning Indicators Associated with Ultra-Processed Food Exposure

This section integrates findings on novel and non-traditional biomarkers that may act as early indicators of metabolic disturbances linked to UPF intake in adolescence and early adulthood. Drawing upon studies summarized in Table 3 and underpinned by life-course and food-processing frameworks described in Sections 1 and 3, this body of evidence emphasizes the potential for UPF exposure to affect biological pathways related to puberty, appetite regulation, and nutritional balance before clinical cardiometabolic conditions arise.

Biomarkers sensitive to pubertal timing further demonstrate the nuanced and age-specific nature of UPF-related effects. Based on data sourced from the Chilean Growth and Obesity Longitudinal Cohort, Devoto et al. (2025) investigated how UPF intake during early puberty correlates with breast tissue development, evaluating breast density four years post-menarche in a sample of 330 adolescent girls. Breast density, assessed via dual-energy X-ray absorptiometry (DXA) using %FGV and AFGV, has been proposed as a biomarker of future breast cancer risk. Overall, the analysis revealed no clear association between UPF intake and breast density outcomes. Stratified analyses revealed stage-specific effects: among girls evaluated during the follicular phase of their menstrual cycles, those in the second quartile of UPF intake (based on percentage of grams consumed) demonstrated significantly greater %FGV ($\beta \approx 0.12$; 95% CI 0.01–0.22) and higher AFGV ($\beta \approx 0.25$; 95% CI 0.07–0.43) relative to those in the lowest intake group [45]. These results emphasize the relevance of developmental timing and hormonal conditions in shaping the relationship between UPF exposure and emerging metabolic biomarkers, in line with life-course epidemiology.

Hormonal regulators of appetite offer additional insight into potential biomarkers linking UPF intake to future metabolic dysregulation. In a clinical cohort of 96 Brazilian adolescents with obesity, Neres et al. (2025) explored the relationship between UPF intake and circulating appetite-regulating hormones, including ghrelin, leptin, neuropeptide Y (NPY), AgRP, MCH, and α-MSH. Dietary data were collected via a NOVA-classified food frequency questionnaire, with intake levels expressed as annualized scores for both unprocessed and ultra-processed items. Results indicated that adolescents with lower intake of unprocessed foods exhibited significantly elevated ghrelin levels ($p = 0.023$) and poorer body composition. Furthermore, AgRP was identified as a key predictor of higher UPF scores (β = 0.30; p = 0.04), regardless of age, fat mass, or binge eating symptoms [41]. These quantitative findings suggest that UPF exposure may be intertwined with dysregulation of appetite signaling pathways that promote increased energy intake, providing a plausible mechanistic bridge between dietary processing and obesity trajectories discussed in earlier sections.

Biomarkers of nutritional status provide further evidence regarding how UPF-centric dietary patterns may affect early metabolic vulnerability, especially during the transition from adolescence to early adulthood. In a sample of first-year university students (n = 142; average age ≈ 20 years), Fedde et al. (2025) compared vegan, vegetarian, and omnivorous groups and found that UPFs made up nearly half of daily energy intake (49%), irrespective of dietary group. Assessment of holotranscobalamin and ferritin levels representing B12 and iron status revealed no significant difference in prevalence across diet groups, likely influenced by supplementation. Still, dietary analysis showed that UPFs were consistently lower in protein, fiber, and essential micronutrients compared to less processed foods [21]. These findings position nutritional biomarkers as early signals of potential metabolic compromise, particularly when UPF-heavy diets displace nutrient-dense foods during critical life transitions.

In sum, the findings in Table 3 reinforce that UPF exposure correlates with a spectrum of emerging biological indicators, including puberty-related changes, appetite dysregulation, and poor nutritional profiles. While not clinical endpoints themselves, these biomarkers offer important insight into early biological alterations linked to UPF intake during adolescence and early adulthood.

Table 3. Novel Biomarkers and Preliminary Indicators Linked to Ultra-Processed Food (UPF) Intake during Adolescence and Early Adulthood

Study (Author, Year)	Biomarker domain	Population & setting	Biomarker measurement	Main findings	Confounders
Devoto et al., 2025	Puberty-sensitive breast density (%FGV, AFGV)	Chile, Growth and Obesity Cohort Study, adolescent girls (n = 330)	DXA assessment 4 years post-menarche; stratified by menstrual cycle phase	No overall association, follicular phase Q2 vs Q1: ↑ %FGV (β ≈ 0.12; 95% CI 0.01–0.22), ↑ AFGV (β ≈ 0.25; 95% CI 0.07–0.43)	Birth weight, BMI, physical activity, tobacco use, energy intake, maternal factors
Neres et al., 2025	Appetite-regulating hormones (ghrelin, leptin, NPY, AgRP, MCH, α-MSH)	Brazil, adolescents with obesity (n = 96)	Serum biomarkers, body composition; BES	Lower unprocessed intake → ↑ ghrelin (p = 0.023), AgRP predicts UPF score (β = 0.30; p = 0.04)	Age, body fat, binge-eating symptoms
Fedde et al., 2025	Nutritional-status biomarkers (holotranscobalamin, ferritin)	University students, mean age 20 y (n = 142)	Blood biomarkers, dietary comparisons	Low B12 and ferritin prevalence similar across diets, UPFs lower in protein, fiber, micronutrients	Descriptive comparisons

3.4. Determinants and Pathways: Food Environment, Marketing, Social Vulnerability, and Behavioral Mediators

This section consolidates evidence on the broader contextual factors and mechanisms influencing UPF consumption and its related consequences for obesity and cardiometabolic health, relying solely on the studies listed in Table 4 and earlier conceptual discussions (Sections 1 and 3). Rather than isolating biological responses, this synthesis reveals how environmental, social, and behavioral conditions shape dietary decisions throughout childhood and adolescence, thereby supporting a life-course perspective on UPF intake.

Commercial forces, particularly food advertising, appear to be a key contributor to UPF consumption among youth. In a nationwide content analysis of food and drink advertisements broadcast on Kenyan television, Wanjohi et al. (2025) recorded 3,700 advertisements across three national stations. Of these, 36% promoted foods and drinks, with a striking 94.7% categorized as ultra-processed. On average, children viewed roughly 2.8 UPF-related ads per hour, and nearly 96% of such ads were shown during peak viewing times. Qualitative data indicated high brand recall among children and minimal parental concern about advertising exposure. Though direct links to health outcomes weren't evaluated, the data strongly suggest a marketing environment that reinforces UPF consumption through early social exposure.

Socioeconomic disadvantage is another major structural factor influencing dietary exposure and obesity related health outcomes [46]. In a study by González-Rodríguez et al. (2025), Spanish children and adolescents aged 6–15 from disadvantaged socioeconomic backgrounds reported increased UPF consumption, higher food insecurity, and notably, lower adherence to the Mediterranean dietary pattern. These eating behaviors were associated with increased BMI and poorer body composition outcomes compared to their non-disadvantaged peers. Although effect sizes were not fully reported, the consistent co-occurrence of vulnerability, elevated UPF intake, and obesity-related outcomes underscores the role of structural inequality in shaping dietary exposures [40]. These findings align with theoretical perspectives discussed in Section 3, which emphasize that food processing and dietary quality are deeply embedded within broader social and economic contexts rather than being solely individual choices.

Behavioral and psychological mechanisms further influence the link between ultra-processed food (UPF) intake and outcomes related to body weight. In a large study of 2,512 university students in Turkey, Eroğlu et al. (2025) identified a clustering of UPF consumption with maladaptive behaviors, including night eating syndrome and digital addiction. UPF consumption scores were positively correlated with BMI ($r = 0.101$), and higher night eating severity was also associated with higher BMI and greater digital addiction. Multivariable analyses suggested that UPF intake contributed to variance in both digital addiction and night eating behaviors, highlighting how modern lifestyle factors such as screen time, sleep disruption, and irregular eating patterns may reinforce UPF consumption and energy imbalance during young adulthood. These findings provide behavioral context for the persistence of UPF-heavy diets beyond adolescence, as predicted by life-course epidemiology frameworks [42].

Dietary context and food substitution patterns also shape UPF exposure, particularly among young adults adopting plant-forward diets. Fedde et al. (2025) investigated first-year university students and found that UPFs accounted for nearly half of daily caloric intake, independent of diet type (vegan, vegetarian, or omnivorous). Although nutritional biomarkers such as No significant differences were found in holotranscobalamin and ferritin levels significantly across dietary categories, potentially explained by supplement use dietary analyses revealed that UPFs and Alternatives based on plant sources tended to have reduced protein levels, fiber, and key micronutrients compared with less processed foods [7]. These findings suggest a substitution pathway whereby well-intentioned dietary shifts may inadvertently increase reliance on UPFs, potentially undermining nutritional quality and reinforcing metabolic risk over time.

Finally, broader sociodemographic patterning of UPF consumption provides important contextual insight for interpreting youth-focused findings. Using nationally representative Canadian data, Baric et al. (2025) showed that higher UPF consumption clustered with male sex, lower income, and lower educational attainment. Although this study focused on adults aged 19–79 years, it demonstrated that UPF intake is socially patterned and intertwined Lifestyle patterns marked by low physical activity and sedentary time [44]. These patterns inform youth research by highlighting the need to account for socioeconomic and demographic confounding when assessing UPF health relationships across the life course.

Taken together, the studies summarized in Table 4 reveal that UPF exposure is shaped by a complex interplay of commercial pressures, social vulnerability, behavioral clustering, and dietary substitution. Quantitative indicators such as the dominance of UPFs in food advertising (94.7% of food ads), substantial energy contribution from UPFs (49% of total intake), and measurable correlations between UPF intake and BMI (r = 0.101) demonstrate that these determinants operate at both population and individual levels. These findings reinforce the argument that effective interventions must extend beyond individual behavior change to address structural and environmental drivers of UPF consumption, particularly during critical developmental periods emphasized in Sections 1 and 3.

Table 4. Determinants and pathways Food environment, marketing, social vulnerability, and behavioral mediators shaping UPF exposure

Study (Author, Year)	Determinant type	Region	Population	UPF-related outcome	Health outcome link	Key pathway insights	Policy
Wanjohi et al., 2025	Commercial determinants (TV marketing)	Kenya, national television	Children/ adolescents (9–18 y), parents	94.7% of food ads were UPFs, 2.8 ads/channel/hour, 96% at peak hours	Not directly measured	High exposure → brand recall → preference → consumption	Restrict unhealthy food advertising; nutrition education
González-Rodríguez et al., 2025	Socioeconomic vulnerability & food insecurity	Spain, urban	Children/ adolescents 6–15 y (n = 280)	Higher UPF intake among vulnerable youth	Higher BMI; poorer body composition	Structural vulnerability → UPF reliance → obesity risk	Equity-focused dietary interventions
Eroğlu et al., 2025	Behavioral/ psychosocial (digital addiction, night eating)	Turkey, university	Young adults, n = 2,512	UPF score correlated with BMI (r = 0.101)	Higher BMI, night eating severity	Behavioral clustering reinforces UPF intake	Screen-time and sleep interventions
Fedde et al., 2025	Dietary substitution (plant-based patterns)	University transition	Young adults; n = 142	UPF 49% of energy across diets	Nutritional biomarkers (B12, ferritin)	Substitution toward UPFs reduces diet quality	Guidance on minimally processed foods
Baric et al., 2025	Sociodemographic patterning	Canada, national survey	Adults 19–79 y, n = 6,517	Higher UPF intake in lower SES groups	Cardiometabolic risk clustering	Structural confounding of UPF exposure	Population-level policy actions

In summary, evidence from this theme demonstrates that UPF consumption is not merely an individual dietary choice but the product of interlocking commercial, social, and behavioral systems that shape exposure from childhood through young adulthood. Addressing these determinants is therefore essential for reducing obesity and as well as long-term cardiometabolic health risks throughout different life stages.

This systematic literature review provides converging proof that regular intake of ultra-processed foods (UPFs) is strongly linked to obesity andcardiometabolic risk across childhood, adolescence, and young adulthood. Findings synthesized from Tables 1–4 indicating that greater UPF intake correlates with elevated levels of adiposity, unfavorable body composition, impaired glucose homeostasis, inflammatory activation, and early biological perturbations, despite heterogeneity in populations and study designs. These results align with prior systematic reviews and cohort studies reporting robust connections involving UPF intake and obesity, abdominal adiposity, diabetes, and cardiometabolic risk across the life course [14], [28], [34], [47], [48].

Across developmental stages, the evidence suggests that UPFs may influence health through multiple, interrelated pathways. Longitudinal data in youth indicate that incremental increases in UPF intake translate into higher odds of incident overweight and obesity, while findings from young adulthood link UPF exposure to prediabetes and impaired glucose tolerance. These patterns are biologically plausible given the nutritional and chemical composition of UPFs, which commonly contain excessive added sugars, harmful fats, and synthetic additives that promote inflammation, insulin resistance, and metabolic dysregulation [1], [49], [50], [51]. Emerging biomarker evidence further suggests that UPFs may disrupt appetite regulation and developmental processes, providing early signals of metabolic vulnerability before overt disease manifests.

Nevertheless, heterogeneity across studies warrants careful interpretation. Sex-specific effects have been reported, with some evidence indicating stronger associations between UPF intake and obesity indicators among women than men [52], [53]. Variability in dietary assessment methods, including reliance on food frequency questionnaires and self-reported intake, may also contribute to inconsistent effect estimates and exposure misclassification [54], [55]. Moreover, socioeconomic context emerges as a critical modifier: structural vulnerability, food insecurity, and differential food environments shape UPF exposure and may confound or amplify observed associations [56], [57], [58].

Methodological limitations remain a central challenge in UPF research. The predominance of cross-sectional designs limits causal inference, while the relative scarcity of long-term cohort studies in children and adolescents constrains understanding of life-course effects [59], [60]. Inadequate consideration of socioeconomic and environmental determinants may further bias estimates and obscure structural drivers of UPF consumption [61], [62].

Importantly, evidence from Table 4 highlights that UPF consumption is embedded within broader commercial and social systems. Aggressive marketing, particularly toward youth, and food environments characterized by high UPF availability disproportionately affect socioeconomically disadvantaged populations, reinforcing dietary inequities and metabolic risk [63], [64], [65], [66]. Addressing UPF-related health burdens therefore requires policy and environmental interventions that extend beyond individual behavior change.

Future research should prioritize longitudinal, developmentally sensitive studies that integrate dietary processing, nutrient quality, biomarkers, and contextual determinants. Evaluating the impact of marketing regulations, food environment reforms, and equity-oriented nutrition policies will be essential to inform effective strategies for lowering UPF intake and minimizing its prolonged health impacts [3], [67].

4. CONCLUSION

This systematic review offers a comprehensive life-course overview, linking ultra-processed food (UPF) exposure from childhood through young adulthood to elevated obesity risk, disruptions in cardiometabolic regulation, and early indicators of metabolic imbalance. Across varied populations and research designs, increased UPF intake consistently correlates with greater adiposity, less favorable body composition, impaired glucose control, heightened inflammation, and changes in hormones related to appetite and metabolism.

The findings emphasize the critical role of early-life dietary habits, particularly during periods of high metabolic plasticity like childhood and adolescence. Longitudinal data highlight a clear link between higher UPF intake and increased risk of overweight and obesity, while cross-sectional and mechanistic studies point to early metabolic dysfunctions that precede disease onset. Rather than reflecting solely personal dietary choices, UPF consumption is shaped by broader structural factors such as aggressive marketing, socioeconomic vulnerability, and food environments that perpetuate dietary inequalities and long-term health disparities.

Despite a growing body of research, significant limitations remain. The reliance on cross-sectional designs, inconsistency in dietary assessment methods, and lack of longitudinal studies in younger populations constrain the ability to infer causality. Future research should standardize UPF exposure assessment, prioritize long-term studies across key developmental phases, and incorporate biomarker data within broader contextual frameworks. Alongside research efforts, public health strategies should address food marketing, improve food system equity, and reduce social disparities to curb obesity and cardiometabolic risks throughout life. Reducing UPF intake in early life may be essential to preventing non-communicable diseases and supporting long-term metabolic well-being from childhood into adulthood.

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