

Ultra-Processed Food Consumption and Its Association with Obesity, Type 2 Diabetes, and Cardiovascular Disease: A Narrative Review

Miftahul Jannah^{1*}, Wahyu Aulia Hasibuan², Prananingrum Kinasih³, Adelia Paradya Zetta⁴

¹Faculty of Health and Science, Universitas Internasional Batam, Indonesia

²Faculty of Health and Science, Universitas Internasional Batam, Indonesia

³Faculty of Health and Science, Universitas Internasional Batam, Indonesia

⁴Faculty of Health and Science, Universitas Internasional Batam, Indonesia

*Corresponding Author: miftahul.jannah@uib.ac.id

History of Article

Submitted : February 3rd, 2026

Revised : March 17th, 2026

Accepted : March 31st, 2026

Published : June 3rd, 2026

DOI : 10.37253/nurish.v1i2.12445

ABSTRACT

Background: The global food supply has shifted markedly toward industrially formulated, ready-to-eat products classified as ultra-processed foods (UPF) under the NOVA system. Concurrently, the burden of obesity, type 2 diabetes, and cardiovascular disease (CVD) continues to rise in both high-income and low- and middle-income countries, including Indonesia. This narrative review synthesizes current epidemiological and experimental evidence on the association between UPF consumption and the incidence of obesity, type 2 diabetes, and CVD, and examines the underlying biological mechanisms and policy responses.

Methods: A non-systematic literature search was conducted in PubMed, Web of Science, and Google Scholar using combinations of the terms “ultra-processed food,” “NOVA classification,” “obesity,” “type 2 diabetes,” and “cardiovascular disease,” prioritizing systematic reviews, meta-analyses, prospective cohort studies, and randomized controlled trials published within the last ten years.

Results: Prospective cohort studies and meta-analyses consistently show that higher UPF intake is associated with increased risk of overweight, obesity, type 2 diabetes, dyslipidemia, hypertension, and incident cardiovascular events. A landmark inpatient randomized controlled trial demonstrated that ad libitum UPF diets causally increase energy intake and body weight compared with minimally processed diets. Proposed mechanisms include hyperpalatability, low satiety, rapid ingestion rate, altered food matrices, additive exposure, and gut microbiome disruption.

Conclusion: The cumulative evidence supports a consistent, biologically plausible, and partly causal relationship between UPF consumption and major non-communicable diseases. Multisectoral policies, including front-of-package warning labels, fiscal measures, and marketing restrictions, are recommended to curb UPF consumption and its downstream cardiometabolic burden.

Keywords: cardiovascular disease; non-communicable disease; obesity; type 2 diabetes; ultra-processed food

A. BACKGROUND

Over the past four decades, global diets have undergone a profound transformation characterized by the displacement of fresh and minimally processed foods with industrially manufactured products designed for convenience, palatability, and long shelf life⁽¹⁾. To capture this shift, the NOVA classification system grouped foods into four categories according to the extent and purpose of industrial processing: unprocessed or minimally processed foods, processed culinary ingredients, processed foods, and ultra-processed foods (UPF)^(1,2). UPF are formulations made mostly or entirely from substances derived from foods and additives, combined through industrial techniques such as extrusion, moulding, and pre-frying, and typically contain colourants, emulsifiers, flavour enhancers, and non-sugar sweeteners rarely found in home cooking^(1,2). Common examples include carbonated soft drinks, packaged snacks, instant noodles, reconstituted meat products, flavoured yogurts, and ready-to-heat meals.

The proliferation of UPF in the global food system has been driven primarily by economic considerations within the food industry, including affordability, aggressive marketing, and the expansion of supermarkets and transnational food and beverage corporations into low- and middle-income countries (LMICs)⁽³⁾. In many countries, UPF now contribute more than half of total daily energy intake, although the proportion remains comparatively lower in parts of Asia, including Indonesia, where minimally processed staples still dominate the diet⁽³⁾. Nevertheless, the trajectory of UPF sales growth in Southeast Asia closely mirrors patterns observed in countries that later experienced sharp increases in obesity and non-communicable disease (NCD) prevalence⁽³⁾.

Non-communicable diseases, particularly obesity, type 2 diabetes mellitus, and cardiovascular disease (CVD), remain the leading causes of morbidity and mortality worldwide. A growing body of observational and experimental research has implicated UPF consumption as a modifiable dietary driver of these conditions, independent of conventional nutrient profiles such as energy density, sugar, sodium, and saturated fat content⁽⁴⁻⁶⁾. However, the NOVA concept and its clinical relevance remain debated, with critics arguing that processing markers are poorly defined and that observed associations may be confounded by overall diet quality⁽⁷⁾. Given the magnitude of the public health stakes and the persistence of scientific debate, a synthesis of the current evidence is warranted. This narrative review aims to: (1) describe the definition and mechanisms by which UPF may influence cardiometabolic health; (2) summarize epidemiological evidence linking UPF consumption to obesity, type 2

diabetes, and CVD; (3) contextualize these findings within the Indonesian and broader Asian food environment; and (4) discuss policy approaches to reduce UPF consumption.

B. METHODS

This article was prepared as a narrative literature review rather than a formal systematic review. A literature search was conducted in PubMed/MEDLINE, Web of Science, and Google Scholar for articles published between 2016 and 2026, using combinations of the keywords “ultra-processed food,” “NOVA classification,” “obesity,” “overweight,” “type 2 diabetes,” “cardiovascular disease,” “cardiometabolic risk,” “metabolic syndrome,” and “Indonesia.” Priority was given to systematic reviews and meta-analyses of prospective cohort studies, randomized controlled trials, and large nationally representative surveys, supplemented by key mechanistic and policy-oriented publications. Articles in the form of conference abstracts, non-peer-reviewed commentaries, and studies unavailable in full text were excluded. Findings were narratively synthesized and organized thematically around disease outcomes, biological mechanisms, regional epidemiology, and policy response, consistent with the scope of a literature-based review article.

C. RESULT AND DISCUSSION

1. Defining Ultra-Processed Foods and Proposed Mechanisms of Harm

Under the NOVA framework, UPF are distinguished from merely “processed” foods (e.g., canned vegetables, cheese, freshly baked bread) by their formulation from cosmetic additives and substances of exclusive industrial use, such as hydrogenated oils, modified starches, protein isolates, and high-fructose corn syrup, combined in ways that imitate or enhance sensory qualities while bearing little resemblance to the original whole food^(1,2). Several converging mechanisms have been proposed to explain why UPF consumption may promote excess energy intake and cardiometabolic dysfunction. First, UPF are typically energy-dense, soft in texture, and rapidly ingestible, which reduces oro-sensory exposure time and blunts satiety signalling^(8,9). Second, their hyperpalatable combinations of sugar, fat, and sodium can override homeostatic appetite regulation and engage neural reward pathways in a manner similar to substances of abuse^(8,9). Third, altered food matrices—where the structural integrity of whole foods has been destroyed through extrusion or refining—accelerate gastric emptying and glycaemic response while diminishing dietary fibre and micronutrient content^(1,8). Finally, chronic exposure to certain emulsifiers and other additives has been linked in preclinical and limited human studies to alterations in gut microbiota composition and low-

grade systemic inflammation, both implicated in the pathogenesis of insulin resistance and atherosclerosis^(8,9).

The most direct causal evidence to date comes from an inpatient randomized controlled crossover trial in which 20 weight-stable adults were fed ad libitum diets matched for presented calories, energy density, macronutrients, sugar, sodium, and fibre, alternating between an ultra-processed and a minimally processed diet for two weeks each⁽⁹⁾. Participants spontaneously consumed approximately 500 kcal/day more on the ultra-processed diet, with increased intake of carbohydrate and fat, and gained on average 0.9 kg of body weight, while losing a similar amount on the unprocessed diet⁽⁹⁾. This trial provided the first experimental evidence that UPF can causally increase energy intake and weight gain independent of conventional nutrient composition, lending biological plausibility to the much larger body of observational findings described below.

2. UPF Consumption and Obesity

A systematic review of 17 prospective cohort studies found substantial agreement that higher UPF consumption was associated with increased incidence of general and abdominal obesity, with eight studies specifically evaluating weight gain outcomes and a smaller subset confirming associations with central adiposity⁽⁵⁾. A subsequent systematic review and meta-analysis of prospective cohort studies pooling data on diabetes, hypertension, dyslipidaemia, and obesity similarly reported that individuals in the highest UPF consumption category had a significantly elevated relative risk of developing obesity compared with those in the lowest category, although the certainty of evidence was rated as low to moderate using the NutriGrade scoring system owing to heterogeneity in dietary assessment methods⁽⁶⁾. An earlier meta-analysis encompassing 43 observational studies likewise concluded that UPF intake was associated with a higher risk of overweight and obesity, alongside other non-communicable disease outcomes, reinforcing the consistency of this association across populations and dietary assessment tools^(7,10).

The body of evidence reviewed here demonstrates a consistent and biologically plausible association between UPF consumption and increased risk of obesity, type 2 diabetes, and cardiovascular disease. The convergence of large-scale prospective cohort data, systematic reviews and meta-analyses, and at least one inpatient randomized controlled trial strengthens confidence that this relationship reflects more than confounding by overall diet quality or socioeconomic status^(5,6,9). Mechanistically, the hyperpalatability, energy density, low satiating capacity, and altered food matrix characteristic of UPF provide a coherent explanation for

excess energy intake, while additive exposure and gut microbiome disruption offer plausible pathways linking UPF to chronic low-grade inflammation and insulin resistance^(8,9).

Most epidemiological evidence remains observational and subject to residual confounding, recall bias inherent to dietary assessment tools such as food frequency questionnaires, and heterogeneity in how UPF exposure is quantified across studies^(5,6). Critics of the NOVA classification have further argued that processing-based categories are imprecisely defined and may misclassify foods with substantially different nutritional profiles into the same group, complicating causal inference⁽⁴⁾. The number of randomized controlled trials remains small, with the landmark inpatient feeding trial limited by its short duration, small sample size, and tightly controlled clinical setting that may not generalize to free-living populations⁽⁹⁾. Future research employing longer-duration controlled feeding trials, objective biomarkers of UPF exposure, and harmonized exposure metrics across cohorts would substantially strengthen the evidence base.

3. UPF Consumption and Type 2 Diabetes Mellitus

Multiple prospective cohort studies included in recent systematic reviews have linked higher UPF intake with incident type 2 diabetes, with effect estimates persisting after adjustment for body mass index and total energy intake, suggesting that processing-related characteristics may contribute to glycaemic dysregulation beyond simple caloric excess^(5,6). Proposed contributory pathways include the high glycaemic index and glycaemic load of many UPF products, rapid absorption kinetics due to the loss of the food matrix, and the promotion of visceral adiposity and insulin resistance through chronic overconsumption^(8,10). While the number of cohort studies specifically isolating diabetes incidence remains smaller than those addressing obesity, the directionality and consistency of findings across European, North American, and Asian cohorts strengthen the inference of a genuine association^(6,10).

4. UPF Consumption and Cardiovascular Disease

The NutriNet-Santé prospective cohort study, following more than 100,000 French adults, reported that a 10 percentage point increase in the proportion of UPF in the diet was associated with a significantly higher risk of overall, coronary heart, and cerebrovascular disease, independent of established dietary and lifestyle confounders⁽¹¹⁾. Subsequent systematic reviews and meta-analyses have corroborated this association, reporting that higher UPF consumption was linked to increased incidence of hypertension and dyslipidaemia, two principal modifiable risk factors for CVD, although the absolute number of studies addressing hard cardiovascular endpoints remains more limited than those addressing obesity^(5,6). A 2023 commentary in the BMJ emphasized that the convergence of mechanistic plausibility, dose-

response relationships, and replication across independent cohorts justifies treating UPF reduction as a public health priority for cardiometabolic disease prevention, even as debate continues regarding the precise causal pathways⁽¹²⁾.

5. Regional Context: Indonesia and the Asia-Pacific

Within Indonesia, food systems have undergone rapid transformation alongside urbanization, rising incomes, and the expansion of modern retail and transnational food and beverage corporations across the wider Asia-Pacific region^(3,13). Analysis of the 2014 Jakarta Individual Food Consumption Survey found that UPF contributed approximately 19.5% of total food weight and 15.7% of daily energy intake, a lower proportion than typically reported in high-income countries but indicative of an accelerating transition⁽¹⁴⁾. A qualitative study among urban households in Yogyakarta similarly found that, despite generally high nutritional awareness, ultra-processed and high-fat, high-salt, high-sugar products were consumed regularly, driven primarily by affordability, convenience, and time constraints associated with participation in the modern workforce⁽¹⁵⁾. These findings are consistent with the broader Asia-Pacific nutrition transition, in which non-communicable diseases related to diet now coexist with persisting undernutrition and stunting in the same populations^(13,15), underscoring the relevance of UPF-related research to Indonesia's evolving public health agenda.

6. Policy Responses to Reduce UPF Consumption

Several countries have implemented regulatory measures explicitly targeting UPF and nutrients of concern. Peru's mandatory front-of-package warning label (FOPWL) policy, evaluated through longitudinal analysis of top-selling processed and ultra-processed products, was associated with measurable reformulation of the food supply, including reductions in the median sugar content of beverages and saturated fat content of foods, and a corresponding decline in the proportion of products requiring a warning label⁽¹⁶⁾. Comparable fiscal and labelling policies have been adopted in Mexico, Chile, and Colombia, often combining taxation on energy-dense products with octagonal “high in” warning labels and restrictions on marketing to children^(12,16). These experiences suggest that combined regulatory approaches—rather than isolated interventions—are more likely to shift the food environment at a population level, offering potential models for Indonesia and other Asia-Pacific nations seeking to curb the growing burden of UPF-related non-communicable disease^(12,13).

From a public health perspective, the precautionary principle supports action even amid residual scientific uncertainty, particularly given the rapidly rising burden of obesity and NCDs in transitioning economies such as Indonesia^(3,13,14). The Indonesian food environment is at a critical juncture: while UPF currently contribute a smaller proportion of energy intake than in

high-income countries, the trajectory of urbanization, retail modernization, and changing household dynamics closely parallels patterns observed elsewhere prior to sharp increases in obesity prevalence^(3,13,15). Multisectoral interventions combining front-of-package warning labelling, fiscal measures, marketing restrictions, and nutrition education—as demonstrated in Latin American contexts—represent a feasible and evidence-informed pathway for Indonesian policymakers, clinicians, and public health practitioners to mitigate the future cardiometabolic burden associated with UPF consumption^(12,16).

D. CONCLUSION

Current epidemiological and experimental evidence supports a consistent association between higher ultra-processed food consumption and increased risk of obesity, type 2 diabetes, and cardiovascular disease, with biological plausibility reinforced by mechanistic studies on satiety, palatability, and gut-brain signalling, and by at least one randomized controlled trial demonstrating a causal effect on energy intake and weight gain. In the Indonesian and wider Asia-Pacific context, the accelerating availability and affordability of ultra-processed foods amid ongoing urbanization signal a need for proactive rather than reactive public health response. Multisectoral policies including front-of-package warning labels, fiscal measures on energy-dense products, restrictions on marketing to children, and strengthened nutrition education should be prioritized and adapted to the local food environment. Future research should focus on longer-term controlled feeding trials, standardized UPF exposure metrics, and locally generated cohort data to further clarify causal pathways and inform context-specific interventions.

REFERENCES

1. Monteiro CA, Cannon G, Levy RB, Moubarac JC, Louzada ML, Rauber F, et al. Ultra-processed foods: what they are and how to identify them. *Public Health Nutr.* 2019;22(5):936-41.
2. Food and Agriculture Organization of the United Nations. *Ultra-Processed Foods, Diet Quality and Health Using the NOVA Classification System*. Rome: FAO; 2019.
3. Monteiro CA, Moubarac JC, Cannon G, Ng SW, Popkin B. Ultra-processed products are becoming dominant in the global food system. *Obes Rev.* 2013;14(Suppl 2):21-8.
4. Astrup A, Monteiro CA. Does the concept of “ultra-processed foods” help inform dietary guidelines, beyond conventional classification systems? *NO. Am J Clin Nutr.* 2022;116(6):1482-8.

5. Mambrini SP, Menichetti F, Ravella S, Pellizzari M, De Amicis R, Foppiani A, et al. Ultra-processed food consumption and incidence of obesity and cardiometabolic risk factors in adults: a systematic review of prospective studies. *Nutrients*. 2023;15(11):2583.
6. Vitale M, Costabile G, Testa R, D'Abbronzio G, Nettore IC, Macchia PE, et al. Ultra-processed foods and human health: a systematic review and meta-analysis of prospective cohort studies. *Adv Nutr*. 2024;15(1):100157.
7. Lane MM, Davis JA, Beattie S, Gómez-Donoso C, Loughman A, O'Neil A, et al. Ultraprocessed food and chronic noncommunicable diseases: a systematic review and meta-analysis of 43 observational studies. *Obes Rev*. 2021;22(3):e13146.
8. Dicken SJ, Batterham RL. Ultra-processed food and obesity: what is the evidence? *Curr Nutr Rep*. 2024;13:23-38.
9. Hall KD, Ayuketah A, Brychta R, Cai H, Cassimatis T, Chen KY, et al. Ultra-processed diets cause excess calorie intake and weight gain: an inpatient randomized controlled trial of ad libitum food intake. *Cell Metab*. 2019;30(1):67-77.
10. Pagliai G, Dinu M, Madarena MP, Bonaccio M, Iacoviello L, Sofi F. Consumption of ultra-processed foods and health status: a systematic review and meta-analysis. *Br J Nutr*. 2021;125(3):308-18.
11. Srour B, Fezeu LK, Kesse-Guyot E, Allès B, Méjean C, Andrianasolo RM, et al. Ultra-processed food intake and risk of cardiovascular disease: prospective cohort study (NutriNet-Santé). *BMJ*. 2019;365:11451.
12. Touvier M, da Costa Louzada ML, Mozaffarian D, Baker P, Juul F, Srour B. Ultra-processed foods and cardiometabolic health: public health policies to reduce consumption cannot wait. *BMJ*. 2023;383:e075294.
13. Baker P, Friel S, Schram A, Labonté R. Ultra-processed foods and the nutrition transition: global, regional and national trends, food systems transformations and political economy drivers. *Obes Rev*. 2020;21(12):e13126.
14. Setyowati D, Andarwulan N, Giriwono PE. Processed and ultraprocessed food consumption pattern in the Jakarta Individual Food Consumption Survey 2014. *Asia Pac J Clin Nutr*. 2018;27(4):840-7.
15. Colozza D. A qualitative exploration of ultra-processed foods consumption and eating out behaviours in an Indonesian urban food environment. *Nutr Health*. 2024;30(3):613-23.

16. Saavedra-Garcia L, Meza-Hernández M, Diez-Canseco F, Taillie LS. Reformulation of top-selling processed and ultra-processed foods and beverages in the Peruvian food supply after front-of-package warning label policy. *Int J Environ Res Public Health*. 2022;20(1):424.