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Ultra-Processed Foods and Metabolic Syndrome: An Analytical Appraisal of Epidemiological, Experimental and Mechanistic Evidence, with Reference to the South Asian Context

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Abstract

Background: *Metabolic syndrome (MetS) is a cluster of central adiposity, dysglycaemia, atherogenic dyslipidaemia and raised blood pressure that sharply increases cardiovascular and diabetes risk. Ultra-processed foods (UPFs), defined by the NOVA classification as formulations of industrial ingredients and additives, now supply a large and rising share of energy worldwide. Whether UPFs contribute causally to MetS, or merely mark a poor diet, remains contested.*

Objective: *To appraise critically the evidence linking UPF consumption to MetS and its components, to compare findings across study designs, populations and analytical perspectives, and to identify what can and cannot be concluded.*

Methods: *A structured narrative review, informed by PRISMA 2020 and PRISMA-ScR reporting principles, of systematic reviews, umbrella reviews, prospective cohorts, cross-sectional studies, a controlled feeding trial and mechanistic studies. Evidence was organised by design and weighed against Bradford Hill-type considerations.*

Results: *Umbrella and meta-analytic evidence consistently links higher UPF intake to obesity, type 2 diabetes, cardiovascular disease, and mortality. Studies directly on MetS are fewer and mostly cross-sectional, but they point the same way. A randomised crossover feeding trial shows that UPF diets increase energy intake and weight gain independent of macronutrient matching. Mechanistic studies suggest roles for energy density, eating rate, glycaemic load, additive-driven microbiome disruption, and food-environment factors. Critics rightly note residual confounding, definitional heterogeneity, and the lack of long-term trials.*

Conclusion: *The convergence of epidemiological, experimental and mechanistic evidence supports a plausible causal contribution of UPFs to MetS, with uncertainty about which attributes drive harm. Policy action is justified on precautionary grounds, particularly in transitioning economies such as India, while research on MetS-specific outcomes and low- and middle-income populations is urgently needed.*

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Keywords: *ultra-processed foods; NOVA classification; metabolic syndrome; obesity; type 2 diabetes; nutrition transition; South Asia; narrative review*

1. Introduction

Metabolic syndrome is not a single disease. It is a clinical constellation of interrelated abnormalities that together mark a state of cardiometabolic vulnerability. The harmonised definition requires any three of five criteria: raised waist circumference (with population-specific thresholds), elevated triglycerides, reduced HDL cholesterol, elevated blood pressure, and raised fasting glucose [1]. Earlier consensus criteria differ in detail but share the same pathophysiological core of visceral adiposity and insulin resistance [2]. Roughly a quarter of adults worldwide are estimated to be affected, and prevalence has risen in step with urbanisation and the globalisation of dietary patterns [3].

The burden is especially acute in India. Recent national estimates from the ICMR-INDIAB study place the number of people with diabetes at around 100 million, with hundreds of millions affected by hypertension and abdominal obesity [4]. Wider analyses of India's disease burden and epidemiological transition describe a country in which non-communicable diseases (NCDs) are displacing infectious disease as the dominant cause of morbidity [5,6]. Asian populations also develop cardiometabolic disease at lower body mass indices and smaller waist circumferences than European populations, which makes dietary drivers of adiposity unusually consequential in this region [7].

Against this background, attention has turned to the industrial transformation of food. Ultra-processed products have become dominant in many national food supplies, displacing traditional dishes and minimally processed staples [8]. Sales-based analyses show the fastest expansion of these products in middle-income countries, driven by transnational food corporations, urban retail change, and aggressive marketing [9]. The NOVA system classifies foods by the nature, extent and purpose of processing rather than by nutrient content alone. Group 4, the ultra-processed category, comprises industrial formulations typically made from substances extracted from foods (such as modified starches, protein isolates and hydrogenated fats), together with cosmetic additives that make the product hyperpalatable, durable and convenient [10]. The framework rests on a deliberate conceptual shift. It proposes that the processing itself, and not only the nutrients it delivers, is a relevant exposure for health [11]. This contrasts with traditional dietary philosophies, such as the sattvic principles of the yogic tradition, which prioritise fresh, seasonal and lightly prepared foods [12].

Two questions follow. First, is UPF consumption associated with MetS and its components, and how strong and consistent is the association? Second, does the evidence support causation, or is the signal explained by confounding, nutrient composition, or definitional artefact? This paper addresses both through a structured, multi-perspective appraisal.

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2. Methods

2.1 Review design and reporting framework

This work is a structured narrative review, informed by the PRISMA 2020 statement [13] and the PRISMA extension for scoping reviews (PRISMA-ScR) [14]. PRISMA-ScR is designed for mapping the breadth and character of a research field rather than pooling effect estimates, and methodological guidance for emerging researchers emphasises its value for transparent landscape mapping [15,16]. The present review does not claim to be an exhaustive systematic search. It is a purposive, critically weighted synthesis of the major evidence streams, and no quantitative record-screening flow diagram is reported because none was generated. This distinction is stated openly because transparency about method is itself a component of research integrity and originality [17].

2.2 Evidence streams and eligibility

Evidence was grouped into six streams: (i) umbrella reviews and meta-analyses; (ii) large prospective cohorts; (iii) cross-sectional studies of MetS or its components; (iv) experimental feeding trials; (v) mechanistic and animal or microbiome studies; and (vi) conceptual, critical and policy literature. Studies were considered if they operationalised UPF exposure through NOVA or an explicit NOVA-derived scheme, or if they examined a defining attribute of ultra-processing (additives, sweeteners, beverages) with metabolic outcomes. Contextual literature on disease burden, equity and health systems was included to interpret the evidence for the South Asian setting.

2.3 Appraisal approach

Each stream was appraised against Bradford Hill-type considerations: strength, consistency, temporality, dose-response, biological plausibility, experimental support, and coherence. Particular attention was paid to confounding, measurement error in dietary assessment, exposure definition, and generalisability. Effect sizes are reported as published by the original authors and are approximate; the original sources should be consulted for exact confidence intervals.

3. Results

3.1 Review-level evidence

Review-level evidence is the most informative starting point because it integrates many primary studies. An umbrella review of meta-analyses evaluated associations between UPF exposure and dozens of health outcomes involving close to ten million participants in total [18]. It graded the evidence for cardiovascular mortality, type 2 diabetes and common mental disorders as convincing or highly suggestive, and also found direct associations with all-cause mortality, obesity, and several other outcomes. The authors cautioned that most of the underlying data were observational and that causality could not be established from this literature alone [18].

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For MetS specifically, a systematic review and meta-analysis of observational studies reported that higher UPF intake was associated with higher odds of overweight, obesity, abdominal obesity and MetS, although the MetS estimate rested on a small number of cross-sectional studies [19]. A separate systematic review of epidemiological studies on UPF and health outcomes reached a similar directional conclusion across obesity, dyslipidaemia, hypertension and MetS, noting heterogeneity in exposure measurement [20]. A dose-response meta-analysis of observational data found a significant positive association between UPF and overweight or obesity [21], and a meta-analysis of longitudinal studies reported higher type 2 diabetes risk with higher UPF intake [22]. Dose-response analyses of adult mortality likewise found risk rising with intake [23].

Table 1. Summary of key review-level evidence on UPF and cardiometabolic outcomes

Reference	Design	Principal outcomes assessed	Direction and approximate result	Main caveat
Lane et al. [18]	Umbrella review of meta-analyses	32 health parameters (mortality, cardiometabolic, mental, respiratory)	Higher UPF intake linked to higher cardiovascular mortality, type 2 diabetes and common mental disorders (convincing or highly suggestive evidence)	Predominantly observational; heterogeneity in exposure definition
Pagliai et al. [19]	Systematic review and meta-analysis	Overweight, obesity, abdominal obesity, MetS, lipid and glycaemic markers	Positive associations; MetS odds roughly 1.8-fold in pooled cross-sectional data	Few MetS studies; cross-sectional designs
Chen et al. [20]	Systematic review	Obesity, hypertension, dyslipidaemia, MetS, cancer, mortality	Predominantly positive associations	Variable exposure measurement
Askari et al. [21]	Meta-analysis	Overweight and obesity	Higher intake associated with higher risk; dose-response pattern	Residual confounding
Delpino et al. [22]	Meta-analysis of longitudinal studies	Type 2 diabetes	Higher UPF intake associated with approximately 40% higher risk (highest vs lowest)	Self-reported diet; reverse causation less likely but possible
Suksatan et al. [23]	Dose-response meta-analysis	Adult all-cause mortality	Risk rises with increasing UPF intake	Mortality is a distal outcome; MetS not examined

Effect sizes are approximate and should be verified against the original publications.

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3.2 Prospective cohort evidence

Prospective cohorts address temporality, a key criterion for causal inference. In the SUN cohort in Spain, participants in the highest quartile of UPF consumption had a substantially higher risk of developing overweight or obesity over follow-up than those in the lowest quartile [24]. The same cohort later reported higher all-cause mortality with higher UPF intake, with each additional daily serving linked to an increase in risk [25]. In the French NutriNet-Santé cohort of over 100,000 adults, a 10% increase in the UPF proportion of the diet was associated with an approximately 12% higher risk of cardiovascular disease overall, with elevated coronary and cerebrovascular risks [26]. In the Framingham Offspring Study, each daily UPF serving was associated with a modestly higher risk of incident cardiovascular disease, and the authors reported a graded association with other cardiometabolic risk factors [27]. In three large US cohorts of health professionals, UPF intake was prospectively associated with higher type 2 diabetes risk, although subgroups of UPF were not uniformly associated, which signals that the category is heterogeneous [28].

Mortality analyses add convergent but indirect support. A French cohort of adults over 45 reported higher mortality with each 10% increment in UPF share [29], and analyses of the US NHANES III dataset found a similar pattern [30]. The Italian Moli-sani study also reported higher all-cause and cardiovascular mortality at higher UPF intake, and it examined whether the association was explained by nutritional quality [31]. Because MetS is a precursor state to many of these endpoints, these findings are consistent with an upstream effect on metabolic health, though they do not measure MetS itself.

Direct evidence on MetS is thinner. Early work in Brazilian adolescents found that higher UPF consumption was associated with greater odds of MetS in a cross-sectional design [32]. The scarcity of long-term prospective MetS endpoints is therefore a genuine evidence gap, and it should not be obscured by the abundance of evidence on adjacent outcomes.

3.3 Experimental evidence

Observational data cannot exclude confounding, so controlled feeding studies carry disproportionate weight. In a randomised, inpatient, crossover trial of twenty adults, participants consumed an ultra-processed diet and an unprocessed diet for two weeks each, with meals matched for presented calories, sugar, fibre, fat and macronutrients [33]. During the UPF phase participants ate roughly 500 kcal more per day, gained approximately 0.9 kg, and showed an unfavourable shift in some appetite-related hormones. During the unprocessed phase they lost a similar amount of weight [33]. The study was short and small, and it cannot test MetS directly. It remains the strongest design-level evidence that UPF attributes can promote excess energy intake independent of the nutrient profile as conventionally measured.

3.4 Nutrient profile and dietary context

Descriptive analyses show why UPFs are so often associated with poorer metabolic health. In the UK, higher UPF consumption was linked to diets higher in free sugars, total and saturated fat, and sodium, and lower in fibre and protein [34]. In the United States, UPFs supplied well

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over half of daily energy and the large majority of added sugars [35]. Sugar-sweetened beverages, an archetypal UPF, were meta-analytically associated with a higher risk of both MetS and type 2 diabetes [36]. Together these findings make it difficult to separate the effect of processing from the effect of nutrient composition, which is the central analytical problem addressed in the Discussion.

4. Mechanistic Considerations

Biological plausibility is supported through several non-exclusive pathways.

Energy density, texture and eating rate. Compared with minimally processed foods, UPFs tend to be softer, more energy-dense, and quicker to eat. These properties are associated with lower satiety and higher glycaemic responses [37]. If the structure of the food matrix is destroyed by industrial processing, nutrients are absorbed faster, which may stress insulin secretion over time.

Additives and the gut microbiome. In mice, common dietary emulsifiers altered the gut microbiota, promoted low-grade inflammation and induced features of MetS including adiposity and insulin resistance [38]. Non-caloric artificial sweeteners were shown to induce glucose intolerance via microbiota changes in animal experiments and in a small human component of the same work [39]. In the NutriNet-Santé cohort, higher intake of certain emulsifiers was associated with cardiovascular outcomes [40]. These are promising mechanistic leads, but translation from rodent models and observational additive exposure to human MetS remains incomplete. Broader reviews of the Western diet–microbiome–host axis link low-fibre, high-additive eating patterns to metabolic disease [41].

Hyperpalatability and reward. UPFs are engineered to be maximally appealing. The conceptual debate about whether they are "addictive" is unresolved, and the term should be used cautiously, but the underlying reward-driven overconsumption is behaviourally plausible and is supported by the trial above [33].

5. Discussion

5.1 Is the association causal? A Hill-type appraisal

Strength and consistency. Associations with obesity, diabetes and cardiovascular outcomes are of moderate size but remarkably consistent across countries, cohorts and designs [18,22,26]. Consistency across populations with different confounding structures weakens, though it does not eliminate, the confounding explanation.

Temporality. Prospective cohorts establish that exposure precedes outcomes [24,26,28], which counters simple reverse causation, although people with early metabolic disturbance might shift their diets before diagnosis.

Dose-response. Monotonic gradients are reported in several analyses [21,23,25].

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Experimental evidence. The controlled feeding trial supports a causal effect on energy intake and weight, the core driver of MetS [33], but it is short, small and does not measure MetS.

Plausibility and coherence. Mechanistic leads are credible but incompletely validated in humans [37–41].

On this appraisal, a causal contribution is plausible and probable, but not proven for MetS as a distinct endpoint.

5.2 The principal scientific controversy: processing versus nutrients

The most substantive critique holds that UPF merely proxies for high sugar, saturated fat, and sodium, and low fibre, so that nutrient-based guidance would suffice. Critics argue that the NOVA category is broad, that it groups nutritionally diverse products (for example, a fortified wholegrain cereal and a sugary soda), and that inter-rater classification can be inconsistent [42]. Proponents counter that nutrient-based approaches have not arrested the global rise in metabolic disease, that cohort associations persist after adjustment for nutrient quality in several studies, and that the feeding trial matched nutrients yet still produced overeating [33,43]. A balanced reading is that both explanations are partly right: nutrient profile explains a substantial portion of harm, but processing-specific attributes appear to add independent risk. This is why the debate is better framed as complementary than as adversarial [43].

5.3 Comparative analysis across evidence types

Table 2. Comparative appraisal of evidence streams linking UPF to MetS

Evidence stream	Representative sources	Strength for causal inference	Principal limitation	Contribution to MetS question
Umbrella / meta-analytic	[18–23]	Moderate (consistency across studies)	Inherits bias of primary observational studies	Establishes breadth and direction of association
Prospective cohorts	[24–28]	Moderate to high for temporality	Self-reported diet; residual confounding	Supports adiposity, diabetes and CVD pathways to MetS
Cross-sectional MetS studies	[19,32]	Low	Cannot establish temporality	Direct but weak evidence on MetS
Controlled feeding trial	[33]	High for mechanism of intake	Small, 2 weeks, not MetS endpoints	Demonstrates processing-linked overeating
Mechanistic / microbiome	[38–41]	Low in humans (hypothesis-generating)	Rodent models; extrapolation	Provides biological plausibility
Conceptual/critical	[42,43]	Not applicable	Debate over definition	Frames interpretation and policy

The table shows a typical pattern for nutritional epidemiology: no single stream is decisive, but the independent streams converge. Triangulation of imperfect designs with different biases is more persuasive than any one design taken alone.

5.4 Psychosocial and structural perspectives

A narrow biomedical framing misses the social conditions under which UPFs are chosen. Three perspectives merit attention, with the caveat that the supporting literature is largely theoretical or indirect and these should be treated as hypotheses for empirical testing rather than established mechanisms.

Digital attention, stress and eating. Research on digital reward loops and adolescent social media use describes how engineered engagement design shapes behaviour and neurochemical reward [44], and related work on doomscrolling documents mental fatigue under constant crisis media [45]. If stress, cognitive overload and screen-based eating displace mindful meal habits, this could plausibly intersect with hyperpalatable UPF consumption, although direct evidence in this area is limited. The marketing environment also matters: sociological critiques of influencer culture and self-branding describe how commercial persuasion is embedded in everyday social media [46], and this is relevant to the promotion of branded snack and beverage products to young consumers.

Time poverty and precarious work. Sociological study of gig work describes precarious labour and the erosion of stable routines [47]. Irregular hours and low incomes reduce the time and resources available for cooking, which makes ready-to-eat products structurally attractive. This is an inference from labour sociology rather than a demonstrated dietary pathway in the cited work.

Stress physiology and lifestyle-based modulation. Reviews on psychoneuroimmunology describe how stress affects immune and inflammatory function [48], and work on the vagus nerve describes parasympathetic regulation through yogic practice [49]. Studies on telomere biology explore cellular ageing effects of lifestyle [50], and a meta-analysis of clinical studies addresses yoga and cardiovascular health [51]. A further review addresses yoga therapy and depression [52], a condition bidirectionally linked with both poor diet and MetS. Constitution-based approaches that integrate Ayurveda and yoga likewise offer a culturally embedded framework for personalised lifestyle guidance [53]. These sources do not test UPF directly; they suggest that dietary change may be most effective when paired with stress management and physical activity, a point consistent with the multifactorial nature of MetS. Because these particular reports are not randomised trials of UPF reduction, they should be read as complementary context.

Consumer demand for less-processed food. Comparative work on consumer behaviour in organic food markets documents growing demand for foods perceived as natural and less processed [54]. This offers a market-side counterweight to the expansion of UPFs but also illustrates the equity issue, since such products are often costlier.

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5.5 Equity, health systems and policy

UPF-related risk is socially patterned. Socio-legal analysis of health disparities in Punjab highlights structural inequities and legislative gaps in access to equitable healthcare [55], and the same structural logic applies to food environments, where low-income households face cheaper, more aggressively marketed UPFs and fewer fresh-food options. Analysis of digital health equity across socioeconomic groups emphasises that technological solutions can widen gaps if access is unequal [56], which is a caution for any plan to deliver dietary counselling or MetS screening through digital channels. A policy-legal perspective on health legislation argues that statute can strengthen national health systems [57]; in the UPF context, candidate instruments include front-of-pack warning labels, restrictions on marketing to children, fiscal measures on sugary beverages, and public procurement standards. The evidence base for such measures comes mainly from outside India, and their effectiveness in Indian settings requires evaluation.

Health technology may also support implementation. Reviews of artificial intelligence in diagnostic medicine point to prospects for earlier risk identification [58], and analysis of patient engagement in AI-driven systems highlights both promise and design challenges [59]. In principle, such tools could support personalised dietary risk scoring for MetS, but their validation for this purpose remains to be demonstrated. Evidence-based environmental design and educational interventions in healthcare settings have also been proposed as levers for improving outcomes [60], and effective leadership in health organisations is a practical precondition for implementing preventive nutrition programmes [61].

5.6 The South Asian context

Three features make the Indian setting analytically distinctive. First, the epidemiological transition is compressed: infectious and undernutrition-related burdens coexist with rapidly rising NCDs [5,6]. Second, South Asian phenotypes are prone to central adiposity and insulin resistance at lower BMI, so the same UPF exposure may have disproportionate metabolic consequences [7]. Third, the food system is in flux. Traditional diets based on pulses, whole grains, vegetables and home-cooked meals are being supplemented, and in urban areas partly replaced, by packaged snacks, sweetened beverages, and reformulated staples [9,12]. Most of the high-quality UPF evidence derives from Brazil, France, Spain, the United Kingdom and the United States, so its transportability to Indian populations is assumed rather than established. Prospective Indian cohorts with NOVA-based dietary assessment and MetS endpoints are a priority.

6. Critical Appraisal and Limitations

Limitations of the evidence base. (a) Dietary data in cohorts are self-reported using food frequency questionnaires, which are poorly suited to capturing degree of processing, generating misclassification. (b) Residual confounding by socioeconomic status, physical activity, smoking and overall health consciousness is likely despite adjustment. (c) Health-conscious behaviours cluster, so UPF avoidance may mark a generally healthier lifestyle. (d) The NOVA Group 4 category is heterogeneous, and null or inverse associations for some subgroups (for

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example, certain breads or cereals) remain to be explained [28,42]. (e) There are no long-term randomised trials with MetS endpoints, and the only controlled feeding trial is brief [33]. (f) Industry-funded research and conflict-of-interest dynamics may influence the framing of the nutrient-versus-processing debate and merit scrutiny in both directions.

Limitations of this review. This is a structured narrative review rather than an exhaustive systematic review, so selection bias is possible. No new meta-analysis was performed, and effect sizes are cited approximately from the original reports. Several psychosocial and structural sources are conceptual or indirect, and their use is explicitly hypothetical. Publication bias, which tends to favour positive associations, cannot be excluded. A full PRISMA-compliant systematic review with a registered protocol, dual screening and risk-of-bias assessment would be the logical next step [13,14].

7. Implications and Recommendations

For clinical practice. Clinicians managing or screening for MetS should include a brief assessment of dietary processing, such as frequency of packaged snacks, sweetened beverages and ready meals, alongside conventional counselling on energy, sugar and fat. Combined dietary and lifestyle approaches, including physical activity and stress management, are biologically coherent with the multifactorial nature of MetS [48–53].

For public health policy. Given consistent associations, a credible mechanism and an experimental signal, precautionary action is defensible: front-of-pack labelling, marketing restrictions for children, fiscal measures on sugar-sweetened beverages [36], and school and institutional food standards. Legal frameworks can be a vehicle for such measures [57], and equity should be an explicit design criterion [55,56].

For research. Priorities include: prospective cohorts in India and other low- and middle-income countries with MetS endpoints; longer randomised feeding trials with metabolic biomarkers; better exposure instruments for processing; mechanistic human studies on emulsifiers and sweeteners [38–40]; and studies disentangling nutrient composition from processing attributes [43].

8. Conclusion

The evidence reviewed here shows a coherent picture. Higher UPF consumption is associated with obesity, type 2 diabetes, cardiovascular disease and mortality across populations and designs [18,22,26], with directly relevant but thinner evidence for MetS itself [19,32]. A controlled feeding trial demonstrates that UPF attributes can drive overeating independent of macronutrient matching [33], and mechanistic work on texture, additives and the microbiome supplies plausible pathways [37–41]. The main scientific disagreement, whether processing adds risk beyond nutrient profile, is better understood as a question of degree than of kind [43]. Residual confounding, definitional heterogeneity and a shortage of long-term trials prevent a definitive causal conclusion for MetS, but they do not justify inaction, particularly in transitioning economies where structural inequities amplify exposure [5,6,55]. The most defensible position is evidence-informed precaution: reduce population exposure to ultra-

processed products through regulation and food-environment reform, while building the regionally specific, methodologically rigorous research needed to settle what remains uncertain.

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