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The Impact of Ultra-Processed Foods on the Risk of Developing Chronic Diseases

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Abstract

This study aims to comprehensively assess the impact of ultra-processed foods (UPFs) on the risk of non-communicable chronic diseases by integrating epidemiological, clinical, and experimental data. The relevance of this research is underscored by the persistently high proportion of UPFs in population diets and the established link between diet and 74% of global mortality from chronic diseases. The novelty of the work lies in the combined application of quintile analyses of dietary intake, meta-analytic reviews, and biomedical trials: from evaluation of nutrient profiles and micronutrient composition to clinical randomized controlled trials demonstrating excess energy intake and glycemic spikes on UPF diets, as well as mechanisms of inflammation, oxidative stress, and epigenetic alterations. Key findings indicate that increased UPF consumption more than doubles the proportion of free sugars and saturated fats while reducing fiber and micronutrients, leading to hyperpalatability, caloric overconsumption, and pronounced postprandial glycemic and lipid peaks. These metabolic perturbations are reinforced by low-grade inflammation, gut dysbiosis, and telomere shortening, which, in turn, translate into an elevated population-level risk of obesity, type 2 diabetes, cardiovascular disease, and various cancers. Consequently, restructuring the food system—limiting UPF availability and promoting whole-plant diets—is imperative for chronic disease prevention. This article will benefit dietitians, epidemiologists, and public health specialists who are developing primary prevention strategies.

Keywords: *Ultra-Processed Foods; Chronic Diseases; Metabolic Syndrome; Diet; Epidemiology.*

INTRODUCTION

Non-communicable chronic diseases now define the global mortality profile, claiming 41 million lives annually—74% of all deaths worldwide—with 17 million occurring before age 70, predominantly in low- and middle-income countries [1]. Among modifiable risk factors, diet remains paramount: the GBD 2019 project attributes 7.9 million deaths and 187.7 million DALYs each year to dietary imbalances, chiefly excessive sodium intake and insufficient whole grains and fruits [2].

In this context, analysis by degree of industrial processing has gained prominence. The NOVA classification assigns to Group 4 (ultra-processed foods, UPFs) those formulations composed of refined ingredients subjected to physicochemical modifications, with added emulsifiers, sweeteners, colorants, and flavorings; intact food matrices are disrupted to create hyper-palatable, shelf-stable products containing minimal whole-food inputs [3].

Over the past two decades, UPF consumption has risen steadily. In the United States, NHANES data show increased calories from UPFs from 53.5% in 2001–2002 to 57.0% in 2017–2018 [4]. In the United Kingdom, national surveys report 56.8% of daily energy from UPFs, accounting for two-thirds of free sugar intake [5]. Middle-income countries exhibit similar trends: in Brazil, UPFs' share of household food purchases grew from 12.6% (2002–2003) to 18.4% (2017–2018) [6]. Thus, the global dietary shift from minimally processed foods toward UPFs underpins the pathophysiology of the ongoing chronic disease pandemic.

MATERIALS AND METHODOLOGY

This investigation of UPF impact on chronic disease risk draws on 32 sources, including WHO and GBD reports [1, 2], the NOVA classification [3], national nutrition surveys in the US (NHANES) [4], UK [5], and Brazil [6], clinical randomized trials [12–14], meta-analyses [8, 19–22], and animal models [17]. The theoretical framework is established by the GBD 2019 dietary risk overview [2] and the concept of ultra-processed matrices with disrupted food structures and additives [3].

Methodologically, thestudycombined:

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- Comparative consumption analysis: The assessment of UPF share in diets based on NHANES [4], Rauber et al. in the UK [5], and Louzada et al. in Brazil [6] revealed a global increase to 57.0%, 56.8%, and 18.4% of energy, respectively.
- Systematic nutrient-profile review: Aggregation of data from Martínez Steele et al. [7] and Martini et al. [8] on added sugars and fats in UPFs, and predicted glycemic indices of breakfast cereals from Rytz et al. [9], demonstrating more than a two-fold rise in free sugar share with increased UPF consumption.
- Content analysis of cross-sectional surveys: Quintile analyses of UPF intake among Tunisian schoolchildren [10] and Brazilian adults [11] show shifts in macro- and micronutrient composition (increased saturated/trans fats, reduced fiber, and potassium).

RESULTS AND DISCUSSION

Industrial processing profoundly alters food nutrient profiles, creating matrices high in refined carbohydrates, fats, and additives and deficient in fiber and micronutrients. UPFs supply 57.9% of daily energy in the average American diet and 89.7% of energy from added sugars; as UPF share rises from 15% to 75%, energy from free sugars doubles from 8.5% to 19% of calories [7, 8]. In the UK, UPFs account for 64.7% of free sugar intake [5]. High glycemic potential arises from sucrose: industrial breakfast cereals' average predicted glycemic index is 68 (range 52–82), akin to white bread [9].

Excess saturated and trans fats mark the lipid component of UPFs. Among Tunisian children aged 3–9, UPFs provide 11.4% of calories from saturated fats and 0.15% from trans fats, contributing 29% and 48.4% of these fats in the diet [10]. In Brazil, moving from the lowest to highest UPF consumption quintile increased saturated fat from 7.9% to 11.5% of energy and trans fats from 0.8% to 1.9%. At the same time, energy density and free sugars also rose [11] (Table 1).

Table 1. Averages of nutritional indicators from food consumption by strata of the Brazilian population aged 10 years or over, referring to quintiles of ultra-processed food consumption [11]

Indicador	Quintile of ultra-processed food consumption (% of total energy)				
	Q1	Q2	Q3	Q4	Q5
Total energy (kcal/d)	1707.9	1794.4	1841.0	1920.4	2066.8
Energy density (kcal/g)[a]	1.5	1.5	1.6	1.7	1.9
Percentage contribution to the total energy of:					
Protein	19.3	18.2	17.3	16.3	14.8
Carbohydrate	56.7	56.5	56.2	56.1	55.6
Free sugar	10.9	13.1	15.0	17.6	20.2
Total fat	23.8	25.4	26.8	28.1	30.4
Saturatedfat	7.9	8.5	9.1	10.0	11.5
Trans fat	0.8	1.3	1.5	1.7	1.9
Nutrientdensity:					
Fiber (g/1,000 kcal)	13.0	11.9	11.3	10.3	8.9
Sodium (g/1,000 kcal)	1.9	1.8	1.7	1.7	1.6
Potassium (mg/1,000 kcal)	1414.2	1347.8	1309.7	1230.6	1074.6

UPFs exhibit low nutrient density: fiber falls from 13.0 to 8.9 g/1 000 kcal and potassium from 1414 to 1075 mg/1 000 kcal between extreme quintiles, while free sugars and fats increase and vitamins A, C, D, E, and B decline [11]. A meta-analysis of 14 national surveys consistently shows that diets with up to 80% of calories from UPFs are characterized by excess sugars and saturated fats alongside deficits in protein, fiber, and key micronutrients. Thus, UPF composition fosters an environment conducive to obesity, type 2 diabetes, cardiovascular and oncological pathologies, making food system restructuring a priority for chronic disease prevention.

The pathogenic sequence linking UPFs to chronic disease begins with their high energy density, soft texture, and hyperpalatability, prompting overeating before satiety signaling. In a metabolic ward study [12], participants consumed 508 ± 106 kcal/day more and gained 0.9 ± 0.3 kg over 14 days on a UPF diet, whereas they lost the same amount on a whole-food diet. Rapidly digestible sugars and starches devoid of fiber and amylose produce steep glycemic peaks: an isocaloric "white bread + jam" breakfast (GI 80) doubled glucose excursions compared to whole-grain bread with peanut butter (Fig. 1) [13].

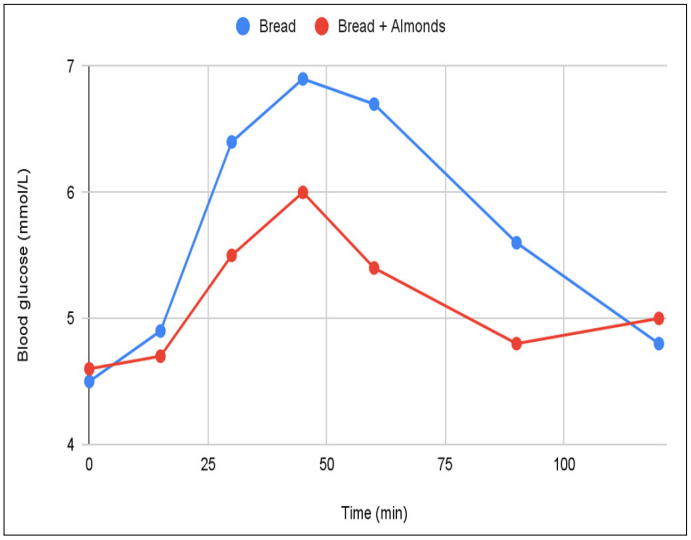

Fig. 1. Almonds Reduce Post-Prandial Glucose [13]

An excessive intake of industrial fats further exacerbates postprandial lipemia: following a mixed-fat test meal, the incremental area under the triglyceride curve reached 1.6 mmol·h/L with olive oil versus 0.72 mmol·h/L with butter, thus doubling the atherogenic burden on the endothelium [14]. Recurrent glycemic and lipid excursions accelerate the development of insulin resistance and endothelial dysfunction.

Persistent metabolic surges fuel low-grade inflammation: in the study by Lane et al. [15], each 100 g/day increase in UPF consumption was associated with a 4% rise in high-sensitivity C-reactive protein (95% CI 2.1–5.9%), independent of BMI. Concurrently, oxidative stress develops: among elderly patients with metabolic syndrome, those in the highest UPF consumption quartile exhibited a 24% decrease in superoxide dismutase activity (from 180.1 to 136.6 pkat/L) and a 27% increase in myeloperoxidase (from 53.3 to 67.6 μkat/mL), thereby promoting lipid peroxide damage and NF-κB activation [16].

A critical link is intestinal dysbiosis. In a murine model, administration of only 1% carboxymethylcellulose or polysorbate-80 in drinking water for 12 weeks reduced the bacterial–epithelial distance by more than threefold, increased microbial mucosal adhesion, and elevated serum flagellin and lipopolysaccharide titers, precipitating metabolic syndrome and colitis [17]. Enhanced intestinal permeability ("leaky gut") permits translocation of bacterial PAMPs and dietary advanced glycation end-products into the circulation, thereby amplifying systemic inflammation.

Chronic exposure to pro-oxidants and inflammatory signals induces epigenetic alterations. In the study by Alonso-Pedrero et al. [18], individuals in the highest UPF consumption quartile had nearly twice the odds of shortened telomeres (< 20th percentile) (OR 1.82; 95% CI 1.05–3.22). Thus, overeating and macronutrient "spikes" trigger metabolic and vascular derangements reinforced by inflammation, oxidative stress, microbiota shifts, and epigenetic modifications; each of these links is tied to the characteristic processing methods and composition of UPFs, explaining their cumulative contribution to the pathogenesis of obesity, type 2 diabetes, atherosclerosis, and cancer.

Accumulated epidemiological evidence corroborates the mechanistic chain linking UPFs to disrupted energy homeostasis, inflammation, and dysbiosis. In the most extensive umbrella review to date—covering six systematic reviews and 97 primary studies—the highest versus lowest quintile of UPF intake was associated with a 55% greater likelihood of obesity (OR 1.55; 95% CI 1.36–1.77); in dose–response analyses, each additional 10% of energy from UPFs increased obesity risk by 7% (OR 1.07; 1.03–1.11). A prospective study of 22,659 UK Biobank participants similarly found that high UPF consumption conferred a 1.79-fold higher risk of incident obesity (95% CI 1.42–2.25) over a median follow-up of 4.9 years, underscoring the clinical relevance of these effects in real-world populations. For metabolic syndrome—encompassing abdominal obesity, insulin resistance, dyslipidemia, and hypertension—the same umbrella review reported a relative risk of 1.25 (95% CI 1.09–1.42) in the highest UPF consumers, with moderate between-study heterogeneity. These findings quantitatively translate the experimentally observed hyperpalatability and postprandial peaks into a population-level impact: dietary shifts toward UPFs measurably exacerbate the obesity epidemic and hasten the emergence of metabolic syndrome criteria [19].

Even more compelling evidence exists for type 2 diabetes. The study by Souza et al. [20] showed that individuals in the top UPF consumption quintile had a significantly higher risk of incident diabetes compared with those in the bottom quintile; a linear dose–response indicated that each additional 10% of energy from UPFs was associated with a 13% increase in diabetes risk. Sensitivity analyses within these cohorts ruled out substantial confounding by follow-up duration, geography, or dietary assessment methods, strengthening the causal inference. Thus, the combination of chronic glycemic and lipid excursions, systemic inflammation, and microbiota alterations demonstrated in experimental models receives robust epidemiological validation, explaining the role of UPFs in the global rise of type 2 diabetes.

Cohort and meta-analytic data further demonstrate that dietary shifts toward UPFs convert the described mechanisms of dysmetabolism and inflammation into concrete cardiovascular risk. In three large US prospective cohorts, the highest versus lowest quintile of UPF intake was linked to an 11% increase in overall cardiovascular disease risk (HR 1.11; 95% CI 1.06–1.16) and a 16% increase in coronary heart disease risk (HR 1.16; 1.09–1.24); stroke risk exhibited a more negligible but directionally consistent effect (HR 1.04; 0.96–1.12). A systematic review of 22 prospective studies confirmed a linear relationship: high UPF consumption raised cardiovascular disease risk by 17%, coronary heart disease by 23%, and stroke by 9% [21].

Oncological outcomes exhibit a similar dose–response pattern. Fiolet et al. [22] reported that UPF intake was associated with higher overall cancer risk (n = 2,228 cases; RR per 10% energy increase 1.12; 95% CI 1.06–1.18) and breast cancer risk (n = 739 cases; RR 1.11; 1.02–1.22). For colorectal cancer—pathogenetically linked to dysbiosis and prolonged oxidative stress of the intestinal mucosa—a pooled analysis of HPFS and NHS (including NHS II) showed that men in the highest UPF quintile had a 29% greater risk (aHR 1.29; 1.08–1.53), whereas no association was observed in women, highlighting sex differences in colonic reactivity to dietary emulsifiers and nitrosamines [23].

Collectively, these epidemiological results synthesize the pathophysiological sequence: frequent intake of hyperpalatable, energy-dense UPFs accelerates the atherosclerotic continuum and increases the likelihood of both hormonally driven and dysbiosis-related malignancies, making UPF reduction a key priority for cardiovascular and cancer prevention.

Cohort data on dementia mirror the neurovascular and metabolic mechanisms described above: a systematic review of ten observational studies involving 867,316 participants found that high versus low UPF consumption increased all-cause dementia risk by 44% (RR 1.44; 95% CI 1.09–1.90), with high between-study heterogeneity but preserved dose–response linearity after exclusion of lower-quality studies. Given that neurodegeneration is exacerbated by systemic inflammation and endothelial dysfunction, this quantitative link with UPF intake underscores the etiological significance of diet [24].

Recent data reveal a comparable threat for Parkinson's disease. In a prospective cohort of nearly 43 000 adults followed for a median of 26 years, consumption of ≥ 11 UPF servings per day was associated with a 2.5-fold higher likelihood of exhibiting three or more prodromal parkinsonism signs (REM sleep behavior disorder, depression, hyposmia, etc.) versus < 3 servings, after adjustment for age, smoking, and physical activity. This finding emphasizes that even preclinical stages of neurodegeneration are sensitive to dietary processing [25].

Taken together, the evidence demonstrates that ultra-processed dietary matrices extend beyond metabolic disorders to directly participate in the pathogenesis of dementia, Parkinsonism, and depression via convergent pathways of hyperglycemia, lipotoxicity, systemic inflammation, and dysbiosis, thereby broadening the spectrum of clinical outcomes preventable through a whole-food diet.

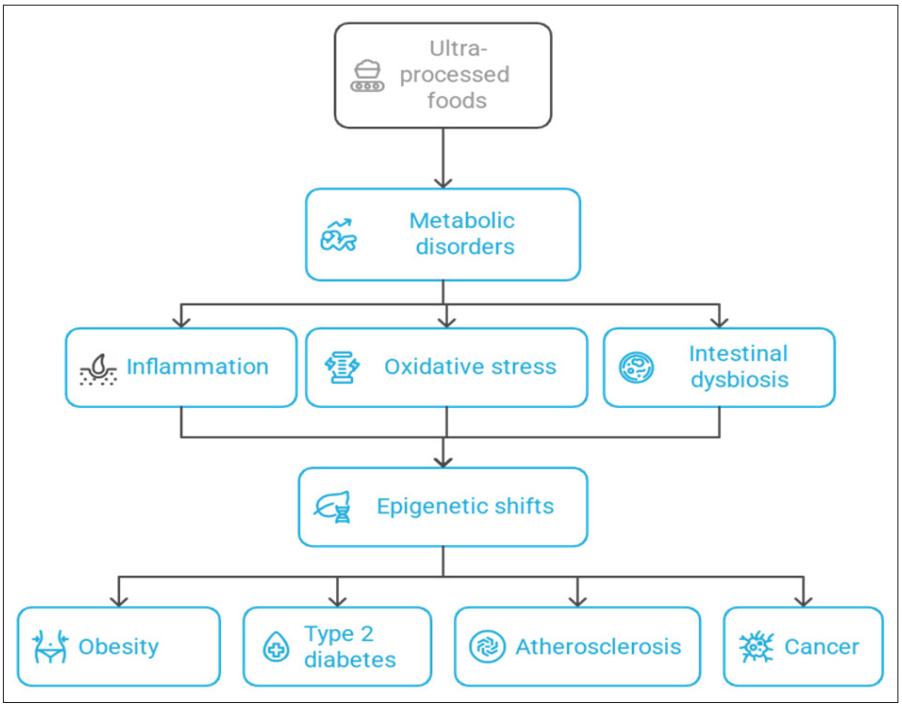

Fig. 2. Systematization of the impact of ultra-processed foods on health (compiled by author)

A whole-food, plant-based (WFPB) diet emphasizes vegetables, fruits, whole grains, legumes, nuts, and seeds, with minimal or no animal-derived or industrially processed ingredients. The Healthful Plant-Based Diet Index (hPDI) quantifies diet quality by assigning positive scores to whole-plant groups and negative scores to refined starches, sugary beverages, and animal products. This distinction is crucial, as different plant-based diets yield divergent metabolic and epidemiological disease profiles.

UK Biobank prospective data show that high hPDI adherence reduces all-cause mortality by 16% (HR 0.84; 95% CI 0.78–0.91), CVD risk by 8% (HR 0.92; 0.86–0.99), and cancer incidence by 7% (HR 0.93; 0.88–0.99) comparing extreme quartiles [26]. These figures quantitatively contrast WFPB with the UPF-dominated pattern described above. Fiber and phytonutrients mediate WFPB benefits: each additional 7 g/day of fiber associates with a 9% reduction in CVD risk [27]. Polyphenols, carotenoids, and isothiocyanates further

activate Nrf2-dependent detoxification genes, mitigating oxidative stress and inflammation, mechanisms absent in refined "plant" junk foods.

However, veganism per se is not protective. Replacing 10% of energy from plant UPFs with minimally processed plant foods lowered CVD risk by 7% and mortality by 13%, whereas high plant UPF intake increased these outcomes by 5% and 12%, respectively [28]. Thus, the preventive value of WFPB lies in substituting whole plants for UPFs—including vegan burgers, desserts, and sweetened drinks—rather than merely excluding animal products.

Market analyses confirm that many "vegan" meat, cheese, and dessert analogues more closely resemble UPFs than whole plants. In Australia/New Zealand, the average plant burger contained five additives and 23% of the daily sodium limit per serving, while saturated fat content rivaled traditional meat products, despite higher fiber [29]. Portuguese supermarket surveys of 407 vegan items found 71.4% of cheese substitutes exceeded 1.5 g salt/100 g, and half of the desserts contained ≥ 5 g saturated fat per 100 g [30].

Epidemiology underscores that processing degree outweighs plant-based status: replacing 10% UPF energy with minimally processed plant foods reduced CVD risk by 7%, whereas high plant UPF intake increased CVD mortality by 15% [31]. Micronutrient deficits also emerge: vitamin B_{12} < 156 pmol/L affected 52% of vegans versus 1% of omnivores; while meat analogues are fortified, cheese substitutes and desserts are not, exacerbating B_{12} deficiency and hyperhomocysteinemia. Iron bioavailability is similarly compromised by non-heme iron and phytate content, raising latent anemia prevalence among UPF-oriented vegans [32].

Thus, the distinction between healthful WFPB and vegan UPF diets lies not in animal product exclusion but in salt, saturated fats, additives, and micronutrient density. A shift toward isolate-based burgers and coconut-oil cheeses sustains or elevates CVD risk and micronutrient deficiencies. In contrast, whole-plant emphasis, with targeted fortification, remains the sole evidence-based chronic disease prevention strategy.

CONCLUSION

As a result of the foregoing analysis of the role of ultra-processed foods in the development of chronic pathology, it has been determined that technologically modified food matrices—enriched in refined carbohydrates, trans and saturated fats, emulsifiers, colorants, and flavorings—exhibit low nutritional value and elevated hyperpalatability. The high energy density and rapid digestibility of such products lead to excessive caloric intake and persistent postprandial glycemic and lipid "spikes," which promote the development of insulin resistance, endothelial dysfunction, and chronic low-grade inflammation. These metabolic disturbances, reinforced by gut dysbiosis and oxidative stress, form a pathophysiological chain that underlies the increased population-level risks of obesity, type 2 diabetes mellitus, cardiovascular diseases, and various neoplastic conditions.

Epidemiological studies confirm a dose–response relationship between the share of ultra-processed foods in the diet and the incidence of adverse outcomes: each additional 10% of energy derived from such foods is associated with a 7% increase in obesity risk, a 13% increase in type 2 diabetes risk, and comparable increases in cardiovascular events and certain cancers. Populations with consistently high consumption of ultra-processed foods are particularly vulnerable, exhibiting higher morbidity and mortality rates compared to groups favoring minimally processed foods.

In contrast, a whole-food plant-based diet—centered on vegetables, fruits, whole grains, legumes, nuts, and seeds, and excluding ultra-processed ingredients—demonstrates pronounced protective effects. Its high content of dietary fiber, antioxidants, and phytonutrients contributes to normalization of glycemia, reduction of lipotoxicity and systemic inflammation, maintenance of a healthy microbiota, and minimization of epigenetic damage. It is important to emphasize that the benefits of a WFPB diet derive not merely from the exclusion of animal-derived foods but from the emphasis on intact plant components, in contrast to ultra-processed bars, burgers, and desserts.

Accordingly, an effective strategy for chronic disease prevention must include restructuring the food environment: reducing the availability and appeal of ultra-processed foods, promoting consumption of whole-plant foods, and educating the public on the distinction between healthful plant-based diets and their ultra-processed counterparts. Only such an approach will reduce the global burden of noncommunicable diseases and improve population health.

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