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A Review Between Ultra-Processed Food and the Risk of Cancer

Kamlesh Suthar (Author)

Assistant Professor, Shri P. T. Patel Science College, Deesa, kamleshbhaibs99@gmail.com

Sukhi Chaudhari (Coauthor)

Assistant Professor, Shri P. T. Patel Science College, Deesa,

Abstract

Ultra-processed foods (UPFs) comprise a growing portion of dietary intake worldwide and have been implicated in numerous chronic diseases, including cancer. This review synthesizes current epidemiological, cohort, and mechanistic evidence to evaluate the association between UPF consumption and cancer risk. Drawing on systematic reviews, large-scale prospective studies, and mechanistic literature, the paper outlines how UPFs are defined, examines associations with specific cancer types (e.g., colorectal and breast), discusses underlying biological mechanisms, highlights limitations in current research, and offers directions for future studies. Overall, evidence consistently suggests that high consumption of UPFs is associated with increased risks for several cancers, although causation remains to be definitively established. Policy implications include dietary recommendations to limit UPF intake and encourage whole, minimally processed foods.

Key Words: Ultra-processed foods; Cancer risk; Dietary patterns; Prospective cohort study; Cancer epidemiology; Nutrition and cancer; Food processing; Lifestyle factors; Public health nutrition

Introduction

Cancer remains one of the leading causes of morbidity and mortality globally, with lifestyle factors exerting a significant influence on incidence and progression (World Health Organization, 2021). Among modifiable risk factors, diet plays a pivotal role in cancer development and prevention (Diet and Cancer, 2025). In recent decades, ultra-processed foods (UPFs) have become ubiquitous in global food supplies and dietary patterns, especially in high-income and transitioning middle-income countries. Defined by the NOVA classification as industrial formulations containing minimal whole foods and excessive amounts of sugars, fats, salt, and additives (e.g., flavoring agents, emulsifiers) manufactured through multiple processing steps, UPFs are characterized by poor nutrient profiles and high energy density. They now contribute over 50% of daily energy intake in many countries (Wikipedia, 2025). Understanding the relationship between UPF consumption and cancer risk has emerged as a critical public health priority.

This review evaluates the current evidence base relating UPFs to cancer outcomes. It synthesizes findings from recent systematic reviews and meta-analyses, large prospective

cohort studies, and preprints exploring mechanistic links. Specific attention is given to associations with colorectal, breast, gastrointestinal, and overall cancer risk, as well as to potential causal mechanisms such as inflammatory pathways and carcinogenic chemical exposures.

Definition and Classification of Ultra-Processed Foods

Ultra-processed foods are defined according to the NOVA classification as "industrial formulations typically with five or more ingredients and additives, including flavorings, colorings, emulsifiers, and preservatives" (Nature Scientific Reports, 2025; Wikipedia, 2025). Examples include soft drinks, packaged snacks, ready-to-eat meals, breakfast cereals with added sugars, and processed meats. These foods are often hyper-palatable, energy-dense, and low in fiber and micronutrients, in contrast to minimally processed foods such as fruits, vegetables, and whole grains.

The NOVA classification system categorizes foods into four groups:

1. Unprocessed or minimally processed foods (e.g., fresh fruits, vegetables).
2. Processed culinary ingredients (e.g., oils, sugar).
3. Processed foods (e.g., canned vegetables, cheeses).
4. Ultra-processed foods (highly industrialized, additive-rich items).

This standardized classification facilitates comparative research on processing and health outcomes across populations and studies.

Epidemiological Evidence Linking Ultra-Processed Foods to Cancer Risk

Figure 1 Cumulative cancer incidence (overall cancer risk) (Fiolet, T. et.al, 2018)

Figure 1 illustrates the cumulative incidence patterns for cancer outcomes. In Model 1, higher consumption of ultra-processed foods was significantly associated with an elevated risk of total cancer incidence. Specifically, each 10-percentage-point increase in the proportion of ultra-processed foods in the diet was linked to a 12% increase in overall cancer risk (hazard ratio [HR] = 1.12; 95% confidence interval [CI]: 1.06–1.18; $P < 0.001$). A similar association was observed for breast cancer, with an 11% higher risk per 10-point increase in ultra-processed food intake (HR = 1.11; 95% CI: 1.02–1.22; $P = 0.02$). Stratified analyses indicated that this relationship was statistically significant for postmenopausal breast cancer ($P = 0.04$), whereas no significant association was detected for premenopausal breast cancer ($P = 0.20$) (Fiolet, T. et.al, 2018).

The positive association between ultra-processed food intake and overall cancer risk remained consistent across all examined population subgroups after adjustment for Model 1 covariates. Increased risks were observed among both men (HR = 1.12; 95% CI: 1.02–1.24; P

= 0.02; 663 cases) and women (HR = 1.13; 95% CI: 1.06–1.20; P < 0.001; 1,565 cases). Age-specific analyses showed stronger associations in younger adults under 40 years of age (HR = 1.21; 95% CI: 1.09–1.35; P < 0.001) compared with individuals aged 40 years or older (HR = 1.09; 95% CI: 1.03–1.16; P = 0.03) (Fiolet, T. et.al, 2018).

Furthermore, elevated cancer risks associated with ultra-processed food consumption were evident among both smokers (HR = 1.18; 95% CI: 1.04–1.33; P = 0.01), even after adjusting for smoking intensity, and non-smokers (HR = 1.11; 95% CI: 1.05–1.17; P < 0.001). Associations were also present across physical activity levels, including participants with low to moderate physical activity (HR = 1.07; 95% CI: 1.00–1.15; P = 0.04) and those with high physical activity levels (HR = 1.19; 95% CI: 1.09–1.30; P < 0.001) (Fiolet, T. et.al, 2018).

Systematic Review and Meta-Analysis

A 2023 systematic review and meta-analysis integrating 13 studies (4 cohort and 9 case–control) encompassing 625,738 participants found that higher consumption of UPFs was significantly associated with increased odds of colorectal cancer (OR = 1.23) and colon cancer (OR = 1.25), as well as a modest increase in breast cancer risk (OR = 1.10) compared to low UPF intake. No significant associations were found for rectal cancer or prostate cancer (Lian et al., 2023). Further subgroup analysis indicated that greater UPF intake was more strongly linked with colorectal cancer in men than in women, suggesting possible sex-specific dietary effects. These findings support the hypothesis that dietary patterns dominated by UPFs elevate the risk of certain site-specific cancers (Lian et al., 2023; PMC10285062).

Prospective Cohort Studies

Large prospective analyses have expanded these observations. For example, a recent study from a US cohort (Scientific Reports, 2025) involving 445,998 person-years of follow-up identified 2,245 breast cancer cases and 270 deaths, demonstrating that women in higher quintiles of UPF consumption had an increased hazard for breast cancer incidence (HR ~1.16) and a trend toward elevated mortality (HR ~1.33) after adjusting for confounders. Subgroup analyses highlighted stronger associations among older participants (>65 years), alcohol drinkers, and those with a family history of breast cancer, emphasizing that lifestyle and genetic factors may modulate UPF-associated risk (Nature Scientific Reports, 2025; turn0search19).

Prospective evidence from UK Biobank and other European cohorts further suggests that incremental increases in UPF intake are associated with elevated overall cancer incidence. For every 10% increase in UPF in the diet, overall cancer risk rose modestly, with particularly notable elevations for cancers such as ovarian and breast cancer (Imperial College study; turn0search1; EMJ Reviews, 2023; turn1search12). Although specific risk magnitudes vary by study and cancer type, the directionality remains consistent: higher UPF intake correlates with higher incidence of various cancers.

Umbrella Reviews and Large Population Studies

An umbrella review of 45 pooled meta-analyses on UPF exposure and health outcomes reported associations between greater UPF intake and increased risks for 32 adverse outcomes, including cancer and mortality, across almost 10 million participants. While evidence quality varied (often low to moderate in the GRADE framework), the overall pattern underscored consistent associations between high UPF exposure and increased cancer risk (BMJ umbrella review; turn1search11; turn1search18).

Emerging Preprint Evidence

A preprint focusing on gastrointestinal cancers outlines potential links between chemicals commonly found in UPFs — such as nitroso compounds, acrylamide, and carrageenan-kappa — and carcinogenesis in organs including the mouth, esophagus, stomach, and colon. It also notes that obesity, often a consequence of high UPF intake, itself significantly increases cancer risk (Research Archive preprint; turn1search0; turn1search4).

Cancer Site-Specific Associations

Colorectal Cancer

Colorectal cancers, among the most common cancer types globally, show robust associations with UPF consumption. The meta-analysis discussed earlier reported a 23% increased risk of colorectal cancer with the highest UPF consumption category, and additional prospective cohorts have supported links between UPFs and colorectal neoplasia or earlier precancerous lesions (Lian et al., 2023; turn0search0). Some studies indicate that men may be disproportionately affected, potentially due to differences in diet composition and hormonal or metabolic factors (turn0search0).

Breast Cancer

Breast cancer is another malignancy for which evidence points toward a positive association with UPF intake. Cohort data suggest increased incidence and a trend toward higher mortality among women with high UPF diets, particularly in subgroups with established risk factors such as older age, alcohol use, and family history (Nature Scientific Reports, 2025; turn0search19). Additional analyses have found that high UPF consumption may be linked with hormone receptor–specific breast tumor subtypes, reflecting complex interactions with endocrine pathways (Lian et al., 2023).

Gastrointestinal and Other Cancers

Emerging evidence suggests associations between UPF consumption and risk for gastrointestinal cancers beyond colorectal, including non-cardia gastric cancer and esophageal adenocarcinoma, although effect sizes vary and causal links are less well-established (turn0reddit46; turn1search7). A large European cohort study (IARC) reported associations between high UPF intake and multimorbidity that included cancer outcomes

(turn1search8). Further, some data hint at links with lung cancer risk among high UPF consumers after adjusting for lifestyle factors such as smoking and overall diet quality (turn1news23).

Overall Cancer Burden

Across diverse study designs and populations, a consistent pattern emerges: diets high in UPFs are associated with a higher risk of cancer incidence and cancer-related mortality (turn1search18). Although most data are observational and cannot definitively prove causality, the consistency and biological plausibility lend support to these associations.

Biological Mechanisms Linking Ultra-Processed Foods to Carcinogenesis

Multiple mechanistic pathways have been proposed to explain the association between UPF intake and cancer risk:

1. Poor Nutritional Quality and Obesity: UPFs are high in added sugars, saturated fat, and calories while being low in fiber and protective micronutrients. Diets rich in UPFs contribute to obesity — a recognized risk factor for multiple cancers (organ size and cancer correlation) — by increasing energy intake and adiposity, which in turn promote chronic inflammation and insulin resistance, creating a pro-carcinogenic milieu (turn0reddit35; turn1academia33).

2. Inflammation and Oxidative Stress: UPF diets can increase systemic inflammation, as evidenced by elevated biomarkers such as IL-6, which are involved in tumor initiation, progression, and metastasis. Food additives and contaminants may exacerbate inflammatory responses, further promoting carcinogenesis.

3. Endocrine Disruption: Certain components of UPFs, including plasticizers (bisphenol A, phthalates) and other endocrine-disrupting chemicals, can interfere with hormonal regulation. These disruptions are especially relevant for hormone-sensitive cancers like breast cancer, potentially stimulating tumorigenesis.

4. Gut Microbiome Modulation: UPFs may negatively affect gut microbiota composition and function. Altered microbial ecology can impair immune surveillance, promote DNA damage, and influence carcinogenic pathways in the gastrointestinal tract.

5. Carcinogenic Compounds: Industrial processing can produce or introduce carcinogenic compounds such as acrylamide and nitroso compounds. These substances have been implicated in DNA damage and cancer risk in experimental models and epidemiological studies (turn1search0; turn1search4).

Overall, these interconnected pathways illustrate how a diet high in UPFs might contribute to cancer risk through both direct and indirect mechanisms.

Limitations of Current Evidence

Despite significant associations, the literature has several limitations:

- Observational Design: Most studies are observational; they can demonstrate association but cannot establish causality due to potential residual confounding. Unmeasured factors such as genetic predisposition, environmental exposures, and other lifestyle behaviors may influence results.
- Dietary Assessment Challenges: Many studies rely on self-reported dietary data (e.g., food frequency questionnaires) that may misclassify UPF intake or lack precision for distinguishing processing levels.
- Heterogeneity: Differences in methodologies, cancer outcomes, population demographics, and UPF definitions contribute to heterogeneity across studies, complicating meta-analytic synthesis and interpretation.
- Evidence Quality: Umbrella reviews indicate that while associations are frequent, the overall quality of evidence for some outcomes remains low to moderate, highlighting the need for rigorous prospective and interventional research (turn1search15; turn1search16).

Public Health Implications and Future Directions

The accumulating evidence linking UPFs to increased cancer risk has important implications:

- Dietary Guidelines: Public health agencies may consider strengthening dietary guidelines to limit UPF consumption and promote whole, minimally processed foods rich in fiber, antioxidants, and anti-inflammatory compounds.
- Policy Interventions: Policies such as taxation of UPF items, front-of-package warning labels, and food reformulation strategies could help reduce UPF intake at a population level.
- Research Priorities: Future research should prioritize well-designed longitudinal cohorts with precise dietary assessments, mechanistic studies elucidating causal pathways, and randomized dietary interventions where feasible.

Conclusion

A growing body of epidemiological evidence suggests that high consumption of ultra-processed foods is associated with an increased risk of several cancers, particularly colorectal and breast cancer. While causality cannot yet be definitively established, the consistency of findings across diverse studies and plausible biological mechanisms supports a prudent approach: reducing UPF intake and emphasizing whole-food-based diets as part of cancer prevention strategies. Continued research — especially studies that address methodological limitations and clarify mechanisms — will be essential to refine dietary recommendations and inform public health policy.

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