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ULTRA-PROCESSED FOODS, MICROBIAL DIVERSITY, AND INTESTINAL HEALTH: A FOOD SCIENCE PERSPECTIVE

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Abstract

Over the last two decades, ultra-processed foods (UPFs) have become the staple of the diets of most people in the world today, and the question one now has to ask is: what is happening to the trillions of microbes in the human gut? This review compiled the evidence that has been published mainly between 2022 and 2026 regarding the association between UPF consumption and intestinal microbial diversity and gut health. From the NOVA classification system, the review will examine how the processing of food in industrial settings, whereby dietary fibre is removed, combined with the inclusion of emulsifiers, non-nutritive sweeteners and other synthetic additives, affects the gut ecosystem in several distinct, but interlinked, ways. The results from the cohort studies, randomised feeding trials and animal models suggest a fairly consistent pattern: higher intake of UPF is linked to lower levels of microbial diversity, fewer Faecalibacterium prausnitzii and Akkermansia muciniphila, a thinner protective mucus layer, and a more permeable gut lining. The mechanism of these changes is linked to low-grade inflammation and to increased risk of metabolic syndrome, inflammatory bowel disease, colorectal cancer and mood disorders via the gutbrain axis. Early life seems to be a very sensitive window, with breastfeeding providing some protection against UPF-induced dysbiosis in infants. The review ends with an analysis of the identified gaps in the existing body of literature and recommendations for future uses of processed foods in food science, such as how they can be formulated to better fit into the gut microbiome.

1. INTRODUCTION

In the past few decades, the eating habits of the human population have been more different than in any similar period before. Packaged snacks, ready meals, sugary drinks, and reconstituted meat products are becoming more common on supermarket shelves in Lahore, London and Los Angeles. They're typically lumped together as a single term: ultra-processed food (UPF). According to the NOVA classification system, a UPF is not simply a food that has been processed almost all food is processed to some degree, even a bag of frozen peas or a loaf of home-baked bread. Rather, UPFs are industrial products formulated primarily from food ingredients that are extracted or refined including starches, sugars, oils, and protein isolates, mixed with a long list of additives such as colours, emulsifiers, flavour enhancers and preservatives, many of which would not be found in a home kitchen (Juul et al., 2022).

The health impacts of such a diet are clearly known increased total energy consumption, increased added sugar and salt and significantly less fibre than a diet based on minimally processed foods (Dicken & Batterham, 2022). However, nutrition and food science scientists have grown more curious about the second, less apparent question. What happens to the bacterial, archaeal, fungal and viral community in the human gut when the food entering the gut contains no fibre and synthetic additives? In general, this ecosystem known as the gut microbiota is not an inactive participant in digestion. It breaks down fibre which human enzymes can't, trains and calibrates the immune system, creates some vitamins and sends messages to the brain via hormones, immune signals and microbial metabolites. It has now been established that a dysbiotic gut microbiota (one that is lost in diversity, or one that has the wrong balance of microbial species) is associated with nearly every major non-communicable disease including obesity, type 2 diabetes, inflammatory bowel disease and depression (Whelan et al., 2024).

This article gathers some of the most recent scientific, nutritional and microbiological information (largely from the last four years) and addresses three related questions. First, what is it about UPFs composition and physical structure that might disrupt the gut microbiota? Second, what do the facts, as they are presented in human cohorts, controlled feeding trials, and animal models, tell us about the direction and consistency of these effects? Thirdly, what implications does this have in practice for the health of the intestinal tract and what can food science do about it? As far as possible the intention throughout was to write a review that could be of value to a food science audience, but not so demanding as to be inaccessible to a clinician or a public health practitioner or a concerned member of the public who does not have a microbiological background.

2. Defining Ultra-Processed Foods: The NOVA Framework and Food Matrix Disruption

The majority of food classification systems employed in nutrition science classify foods according to the amount of fat, sugar, or salt they contain. Throughout the literature reviewed here, foods are instead clustered according to their type, degree and purpose of processing. It categorises all foods and drinks into four groups, as summarised in Table 1: unprocessed or minimally processed foods; processed culinary ingredients (such as oils, butter, table sugar); processed foods, which are simple combinations of Group 1 and Group 2 foods (such as canned vegetables, cheese, freshly made bread); and ultra-processed foods, those that are industrial formulations and contain ingredients rarely used in home cooking (Ozkan et al., 2025).

Table 1. The NOVA food classification framework

NOVA Group	Description	Representative examples
1	Unprocessed or minimally processed foods: edible parts of plants or animals altered only by removal, drying, chilling, freezing, or fermentation without added substances.	Fresh or frozen vegetables and fruit, plain yoghurt, eggs, grains, fresh meat and fish

2	Processed culinary ingredients: substances derived from Group 1 foods or from nature by processes such as pressing, refining, or milling, used to prepare and season Group 1 foods.	Vegetable oils, butter, table sugar, salt, honey
3	Processed foods: relatively simple products made by adding Group 2 ingredients to Group 1 foods, typically using methods such as canning, bottling, or baking.	Canned vegetables in brine, cheese, freshly made bread, smoked fish
4	Ultra-processed foods: industrial formulations made mostly from substances extracted or synthesised from foods, containing little or no intact Group 1 food, plus additives with cosmetic or functional purposes.	Packaged snacks, sugary breakfast cereals, carbonated soft drinks, instant noodles, reconstituted meat and fish products, mass-produced packaged bread

The key factor for the gut is not only the unfamiliar additives, but also what food scientists refer to as the food matrix. Even though all three can be considered loosely as apple products, they are nutritionally very different to each other (whole apple, apple purée and an apple flavoured snack bar). The apple's cell structure, the structure of its fibre network and polyphenols contained in apple plant cell walls are preserved. This architecture is generally lost during ultra-processing, allowing rapid absorption of sugars in the small intestine, and limiting the amount of fermentable substrate that reaches the colon (Whelan et al., 2024). The result is that the microorganisms at the end of the digestive system (the ones that produce the short chain fatty acids) are starved of their favourite fuel before the food that were eaten in the UPF diet has even finished digesting.

3. The Human Gut Microbiome: A Primer on Diversity and Function

The human colon is home to an estimated 38 trillion microbial cells, which is approximately the same number as human cells, mainly represented by four phyla: *Pseudomonadota* (formerly Proteobacteria), *Actinomycetota* (formerly Actinobacteria), *Bacteroidota* (formerly Bacteroidetes), and *Bacillota* (formerly Firmicutes). These large groups contain hundreds of different species that vary significantly in their abundance among individuals depending on the genetic differences, geographic location, age, medication and, most importantly, diet (Wang, 2024).

There are two similar but different ideas that are repeated throughout this review. Alpha diversity refers to the variety of microbial taxa in an individual's gut, and the evenness of their distribution; commonly reported using Shannon diversity or Simpson's index. Beta diversity refers to the differences between different people's microbial communities, however. Generally, the more alpha diverse a system is, the more resilient and stable it will be and the more it can stand up to disturbances, such as a course of antibiotics, travellers' diarrhoea or a night without sleep, without reverting to a less functional state. On the other hand, a less diverse microbiota is likely to be dominated by fewer generalist or opportunistic taxa that are not as efficient at doing the jobs that the community collectively needs to do (Zhang et al., 2023).

Of these jobs, the fermentation of dietary fibre into short-chain fatty acids (SCFAs), primarily acetate, propionate and butyrate, is one of the most impactful on intestinal health. Butyrate is the preferred fuel for the cells lining the colon, propionate is delivered to the liver to regulate glucose and lipid metabolism, and acetate is widely distributed and can be a signalling molecule in peripheral tissues and even the brain (Tan et al., 2023). SCFAs also help to preserve the integrity of the gut's mucus layer and its single-cell epithelial lining, which is the physical barrier between the trillions of microbes and the bloodstream. If that barrier is breached a state that is sometimes called a “leaky gut” a variety of bacterial components can

enter the circulation and lead to low-grade, chronic inflammation, which is linked to a very broad spectrum of downstream conditions (Whelan et al., 2024).

4. Mechanistic Pathways Linking UPF Consumption to Microbial Diversity Loss

Food scientists may treat "ultra-processed foods" as a single exposure, but a close look at the list reveals at least four ways UPFs disrupt the gut ecosystem, some of which overlap: UPFs lack fibre, their barrier-disrupting effects are attributed to emulsifiers, their metabolic and microbial effects are ascribable to sweeteners, and their effects stem from miscellaneous additives, microparticles and nanoparticles. These routes converge at a shared ecological consequence reduced diversity and dysbiosis and then diverge again to form different clinical consequences (summarised in figure 1).

4.1 Fibre Deprivation and the Loss of Fermentative Capacity

The one most crucial food component for a healthy gut flora is dietary fibre and this is the component that is often lost or broken down during industrial processing. The process of refining, extracting juices and reconstituting plant-ingredient purees and concentrates in foods remove the plant cell-wall structures that would otherwise survive to the colon. The bacterial guilds that are specialised for fermenting polysaccharides are at a competitive disadvantage compared to the bacteria that can survive on host-derived mucus glycans and on simple sugars (Tan et al., 2023). As the substrate wears out over time, it's believed that it will help create a more limited and mucous-busting ecosystem, which has been referred to as the microbiota devouring its host's lining instead of eating its host's food.

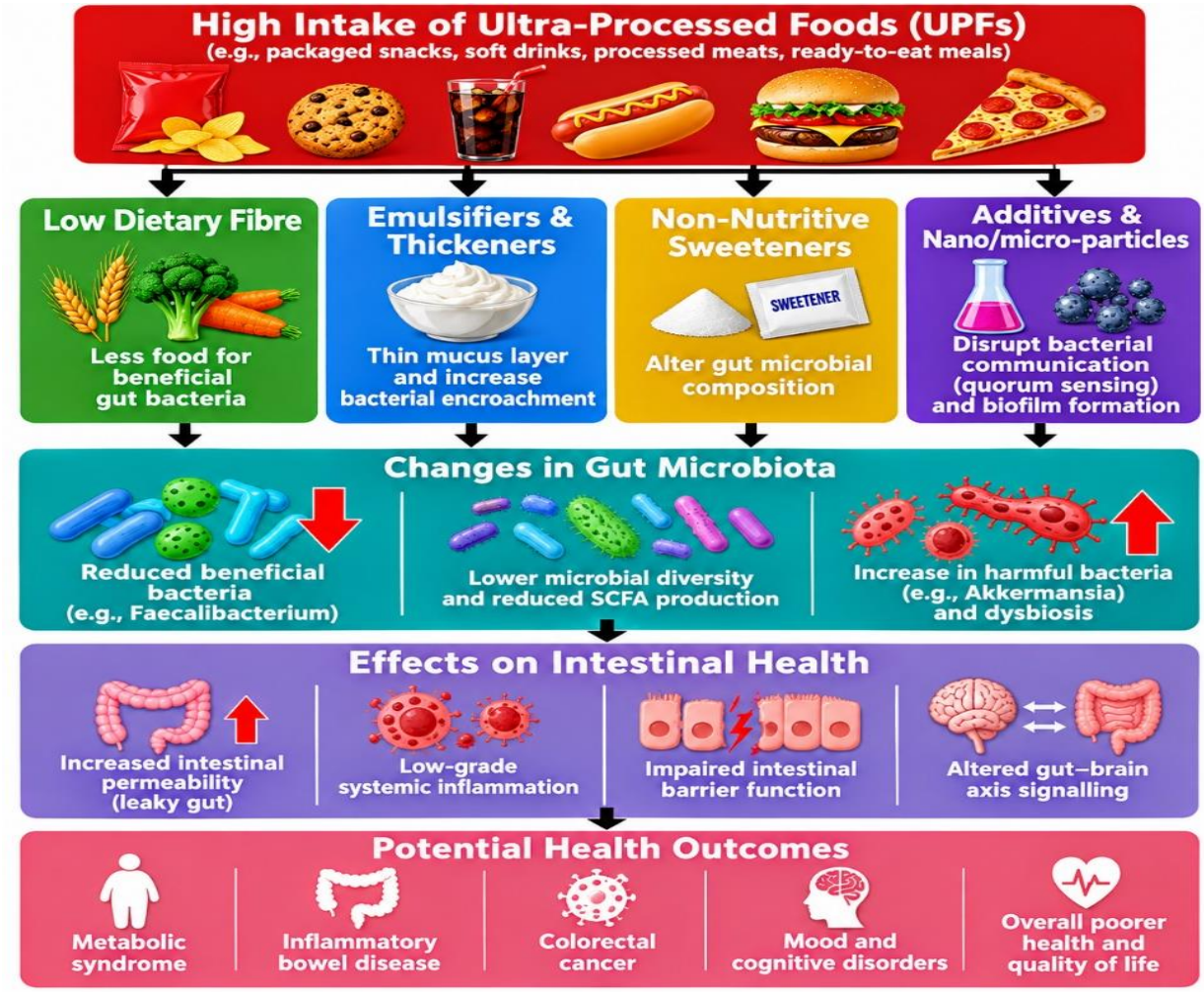


Figure 1: Mechanistic pathway linking ultra-processed food intake to reduced microbial diversity and impaired intestinal health.

4.2 Emulsifiers and Mucus Barrier Disruption

A big portion of UPF products contain emulsifiers like carboxymethylcellulose (CMC), polysorbate 80, carrageenan and various mono- and diglycerides to help keep ingredients like oil and water mixed and combined, and to extend shelf life and enhance texture. A landmark random controlled feeding study in humans showed that dietary CMC intake led to a decrease in gut microbiota diversity and increased the presence of bacteria in the normally sterile inner mucus layer, as well as changes in host metabolome indicative of early low-grade inflammation (Chassaing et al., 2022). Likewise, in human mouse models, the combination of emulsifiers induced metabolic disturbance and increased intestinal dysbiosis (Panyod et al., 2024) and when mothers consumed emulsifiers during pregnancy, their offspring's metabolic and even neuropsychological development was affected, as gut microbiota of both mother and infant changed (Milà-Guasch et al., 2023). In a related line of evidence, the occurrence of diet-induced chronic exposure to emulsifiers has been shown to accelerate the onset of auto-immune diseases in a genetically susceptible mouse model of type 1 diabetes (Delaroque & Chassaing, 2024) and a large-scale human cohort study showed that higher self-reported dietary intakes of emulsifiers, based on detailed composition data from packaged products, were correlated with increased risk of incident type 2 diabetes (Salame et al., 2024). This collection of literature represents one of the more mechanistically well-supported links between a specific class of UPF additive and microbiota mediated harm as reviewed by De Siena et al. (2022).

4.3 Non-Nutritive Sweeteners and Microbial Dysbiosis

Non-nutritive sweeteners like sweetners such as sucralose, saccharin, aspartame, and acesulfame potassium have become very common additives, as many of these products are formulated to be “low sugar” or “diet” versions of conventional products. Metabolisable or not, by definition, these compounds are far from biologically inert once they reach the colon. A human trial study that was widely reported in the media showed that non-nutritive sweeteners could impact glucose tolerance in a highly person-dependent fashion, with some individuals benefiting and others suffering, depending on the individual's gut microbiome (Suez et al., 2022). Human faecal inoculation of controlled bioreactor systems has demonstrated varying effects on microbial diversity and network structure, with some sweeteners having more disruptive effects on microbial structure than others, such as saccharin and sucralose, respectively, compared to others, such as xylitol (Kidangathazhe et al., 2025). The mechanistic reviews paint a cohesive picture—exposure to sweeteners has been associated with disruption of the gut barrier, shifts in bile acid metabolism, and alterations in SCFA levels, in various experimental systems (Conz et al., 2023; Feng et al., 2024). However, there is also emerging and, somewhat surprisingly, evidence that NNS might facilitate the transfer of ARGs between members of the gut microbial community, not just diversity metrics (Yu et al., 2023). Although not definitive, human cohort data supporting a microbiota-mediated pathway exist for sweetener intake and cardiovascular disease and type 2 diabetes (Debras et al., 2022, 2023) and recent narrative reviews warn of inconsistent and sometimes conflicting findings between sweetener types (Ghusn et al., 2023; Kossiva et al., 2024; Shaher et al., 2023).

4.4 Other Additives, Colourants, and Micro- and Nanoparticles

In addition to emulsifiers and sweeteners, UPFs typically include one or more of the following ingredients: synthetic colourants, preservatives, flavour enhancers, and in more recent times, food-grade micro and nanoparticles like titanium dioxide, and silicon dioxide, which are used to whiten, anti-cake or otherwise stabilise a product. Several of these can change gut microbial composition, boost gut permeability, and trigger a low-grade inflammation of the gut, although the evidence base is lower for most of the individual additives than that of emulsifiers and sweeteners (Whelan et al., 2024). One of the most crucial, yet underappreciated arguments found in this literature is that real-world UPFs seldom consist of a single additive; a typical packaged snack may include an emulsifier, a sweetener, a colourant and a preservative in a single product, with the additive mixtures that consumers are exposed to rarely having been tested as combination products, and only as single additives.

5. Evidence from Human and Animal Studies

This review brings together the mechanistic threads of Section 4; Table 2 summarises a representative selection of the studies reviewed including cross-sectional human surveys, a birth cohort, a randomised controlled-feeding trial, in vitro bioreactor studies and rodent models. Despite the diverse microbial taxa reported in each study based on the population studied, sequencing technique, and measurement of UPF exposure, there is a general direction of effect across this heterogeneous evidence base.

Table 2. Selected studies examining ultra-processed food intake and gut microbial or intestinal health outcomes (20222025)

Study	Design / population	Exposure	Key finding relevant to gut microbial diversity / health
Atzeni et al. (2022)	Cross-sectional; older adults with overweight/obesity and metabolic syndrome, Spain	Habitual UPF intake (NOVA)	Higher UPF consumption associated with distinct gut microbiota composition and lower abundance of several health-associated taxa
Chassaing et al. (2022)	Randomised controlled-feeding trial, healthy adults	Dietary emulsifier (carboxymethylcellulose)	Reduced microbial diversity, mucus-layer encroachment, and metabolomic changes consistent with early inflammation
Faggiani et al. (2025)	Birth cohort (MINA-Brazil), > 700 infants	UPF intake in the first year of life	Lower Bifidobacterium and higher potentially pathogenic taxa in non-breastfed infants consuming UPFs; breastfeeding attenuated the effect
Panyod et al. (2024)	Mouse model	Common dietary emulsifier mixtures	Promoted intestinal microbiota dysbiosis alongside metabolic disorders
Milà-Guasch et al. (2023)	Mouse model, maternal exposure	Maternal emulsifier consumption	Altered offspring metabolic and neuropsychological outcomes, linked to microbiota transmission
Suez et al. (2022)	Randomised human trial	Non-nutritive sweeteners	Individually variable, microbiota-dependent effects on glucose tolerance
Kidangathazhe et al. (2025)	In vitro bioreactor, human faecal inocula	Five common artificial sweeteners	Differential, sweetener-specific reductions in microbial diversity and network stability
Wang et al. (2022)	Three prospective US cohorts (~206,000 adults)	Habitual UPF intake	Higher UPF intake associated with increased colorectal cancer risk in men
Shu et al. (2023)	Systematic review and meta-analysis	UPF intake across pooled studies	Confirmed a positive association between UPF consumption and colorectal cancer risk
Spiller et al. (2025)	Critical review	UPF intake and gut microbiota	Consistent association between high UPF intake and dysbiosis, reduced SCFA output, and barrier disruption relevant to inflammatory bowel disease
Whelan et al. (2024)	Comprehensive narrative review	UPFs and food additives broadly	Synthesised mechanistic and epidemiological evidence linking UPFs and additives to gut

			dysbiosis, permeability, and gut disease
Ozkan et al. (2025)	Cross-sectional, Turkish adults	NOVA-classified processed food intake	Higher processed/ultra-processed intake associated with elevated cardiometabolic risk scores

6. Consequences for Intestinal Health: Barrier Function, Inflammation, and Disease Risk

6.1 Gut Barrier Integrity and Low-Grade Endotoxemia

The gut contains a huge number of bacteria, which are physically separated from the bloodstream by the single-cell epithelial lining of the gut, the mucus layer and tight junction proteins between neighbouring cells. The following mechanisms, listed in Section 4, all have a common downstream effect: an increased permeability or "leakiness" of the intestinal barrier that is due to: fibre deprivation, mucus-layer thinning due to emulsifiers, and sweetener-driven changes in handling of bile acids. If this barrier is breached, bacterial cell-wall components, such as lipopolysaccharide, enter into systemic circulation in small but chronic amounts resulting in a state of metabolic endotoxemia, which leads to the constant activation of the innate immune system (Whelan et al., 2024).

6.2 Inflammatory Bowel Disease

The inflammatory bowel disease (IBD) - Crohn's disease and ulcerative colitis - has experienced a dramatic increase in incidence in newly industrialised countries over the last few decades, and the connection between the spread of the modern diet rich in foods that contain UPF and inflammatory bowel diseases has been so closely linked that it has been added to the scientific list of watch lists. The recent critical reviews suggest that high intake of UPF is linked to intestinal dysbiosis characterised by decreased microbial diversity, low production of SCFAs, compromised barrier integrity and disrupted protective mucus layer which all point to abnormal, self-sustaining immune activation characteristic of IBD (Spiller et al., 2025). Similarly, a separate narrative review concludes that there is consistent evidence of association of higher UPF intake with an increased risk of developing Crohn's disease, whereas the association between higher UPF intake and the risk of developing ulcerative colitis appears to be less consistent and comparatively weak (Choi & Moon, 2025). While this is a challenge for translating these mechanistic findings to humans for definitive causal claims, this is the biological plausibility of the associations as a set of culprit ingredients being identified in the pre-clinical setting: emulsifiers, carrageenan, maltodextrin, and microparticles in the diet have been found to disrupt epithelial integrity and activate innate immune pathways. (Whelan et al., 2024).

6.3 Colorectal Cancer

Results from a large prospective study that tracked over 200,000 US health professionals over 2030 years indicated that men in the highest quintile of UPF consumption were significantly more likely to develop CRC than men in the lowest quintile, whereas there was no significant association among women; processed meats, sugar-sweetened beverages and ready-to-eat foods were among the food subgroups most strongly linked to CRC, but the authors could not entirely account for this gender difference (Wang et al., 2022). A subsequent systematic review and meta-analysis of several cohort studies found an overall positive association between UPF intake and colorectal cancer risk (Shu et al., 2023), while a large multinational European cohort found that UPF consumption was associated with the increased risk of multiple co-occurring chronic diseases, including colorectal cancer and cardiometabolic diseases (Cordova et al., 2023). Butyrate levels, which are normally anti-inflammatory and even tumour-suppressive to colonic epithelial cells, are reduced; and there is a shift towards bacterial taxa that can convert bile acids to pro-carcinogenic secondary metabolites, a pathway that has been mechanistically linked to the production of some dietary emulsifiers (De Siena et al., 2022).

6.4 Metabolic Syndrome and Cardiometabolic Disease

A connection between the consumption of UPF and gut dysbiosis and cardiometabolic disease is perhaps the most well-studied of these. A cross-sectional study of older people with established metabolic syndrome revealed specific associations between microbiota shifts and increased UPF consumption, and a systematic review of prospective studies identified a relationship between long-term UPF consumption and the incidence of obesity and a cluster of cardiometabolic risk markers in adults (Atzeni et al., 2022; Mambrini et al., 2023). Large US cohort analyses have associated UPF consumption with an increased risk for cardiovascular disease specifically, and a meta-analysis of prospective studies showed that processed and ultra-processed food intake, as determined by the NOVA classification system, was associated with increased cardiometabolic risk scores (Mendoza et al., 2024; Ozkan et al., 2025). This picture is further complicated by two large-scale French cohort studies; high consumption of artificial sweeteners was linked to greater cardiovascular disease risk (Debras et al., 2022) and high consumption of emulsifiers, as assessed from detailed information on packaged food composition, was found to be independently associated with incident type 2 diabetes risk (Salame et al., 2024). While none of these cohort analyses can conclusively establish a causal relationship between UPF or additives consumption and CMB, the epidemiological signal is robust and consistent with the mechanistic and animal evidence reviewed in Section 4, thus a microbiota mediated pathway is a key driver of explanation.

6.5 The GutBrain Axis and Mental Health

Mood and cognitive changes may be the more obvious and debated effect of UPF-induced gut dysbiosis. A bidirectional communication system exists between the gut and the brain, known as the gutbrain axis, with reciprocal links occurring via the vagus nerve, immune signalling and microbial metabolites such as SCFAs and neurotransmitter precursors. A focused review of this literature demonstrated that the combination of UPF-associated dysbiosis, diminished barrier integrity and low-grade systemic inflammation is a plausible biological pathway through which diet quality could be linked to depressive symptomology, and that prospective cohort studies support a link between UPF intake and increasing incidence of depressive symptoms over time (Song et al., 2023). A comprehensive review of dietmicrobialomebrain relationship also found that diet patterns rich in UPFs, saturated fat and low fibre are consistently linked to gut dysbiosis, and dysfunction of gut barrier and an increased risk of cognitive difficulties, anxiety and depression (Uţu et al., 2026); and a complementary narrative review showed that diet patterns that promote microbial diversity, such as the Mediterranean diet, could be an adjunct strategy to improve mental health (Patil & Mehdi, 2025).

7. Vulnerable Populations: Early Life and Childhood Exposure

There is no period in human life when gut microbial diversity is more important than infancy. The first year to two years of life are the most critical times in which the gut microbiota is seeded from essentially a sterile state and then evolves into a relatively stable community that will persist, albeit with some changes, for decades, and these are closely linked to immune and metabolic programming. The finding in the MINA-Brazil birth cohort is of particular interest from a public health perspective: children who had consumed UPFs in the first year of life, who were not breastfed, exhibited a microbiota profile that was less favourable, characterized by having lower levels of Bifidobacterium and higher levels of taxa that are generally linked to obesity or gastrointestinal disease in childhood and adulthood; breastfeeding had only a partial (but significant) protective effect (Faggiani et al., 2025).

The simple take-home message is that although the biology behind the research may be complex, it's not complicated that more than 80% of the children surveyed in this cohort had already eaten UPFs well before the recommended age. Because it is challenging to change established, low-diversity microbiota at an older age, the evidence provided here supports a nuanced approach to considering early-life diet quality in clinical counselling and food policy considerations, which is echoed in several of the overarching reviews presented in Section 6 (Choi & Moon, 2025; Whelan et al., 2024).

8. From Food Science to Food Design: Toward Gut-Friendly Reformulation

None of this evidence suggests that the industrial food category is incompatible with a healthy gut flora it suggests that the specific formulations chosen today are the issue, and the choice of formulation in principle is within the control of food scientists and food manufacturers. The above literature indicates some practical directions.

Keeping the food matrix intact, or minimally disrupted, rather than breaking it down to individual starches, sugars and fibres seems to be more important than simply meeting a target fibre content on the nutrition label; whole or less disrupted plant cell structures release fermentable content more slowly and in a form which supports a wider variety of fermenting bacteria (Whelan et al., 2024; Zhang et al., 2023). Second, reformulating products to employ less emulsifier content in total or less disruptive stabilising methods (when practicable) or at least to test emulsifier mixtures as they are actually used rather than as single individual products alone would close an important evidence gap and likely minimize harm (Chassaing et al., 2022; De Siena et al., 2022). Third, as reviewed in the sweetener literature, a "one size fits all" recommendation is unlikely to be effective, as the metabolic response to a specific sweetener seems to depend on an individual's microbiota composition, suggesting a more long-term application of microbiome-informed personalised nutrition, although not yet routine practice (Suez et al., 2022; Kidangathazhe et al., 2025).

Last, but not least, the additive combination problem discussed in Section 4.4 is not given sufficient attention from a regulatory and a food-industry point of view. The current knowledge base to date with respect to safety has been compiled from a series of individual additive tests, and the findings from these tests may underestimate the effects of the additive combinations found in real UPFs as they typically contain multiple classes of additives. Mixture-level testing of common additive mixtures would be a step forward for food safety science in this area, if encouraged or mandated.

9. Critical Appraisal and Research Gaps

The evidence presented here comes with a number of caveats that limit the extent to which it can be pushed. In most human studies that have investigated the relationship between UPF intake and microbiota composition, reverse causation and residual confounding by other lifestyle factors (such as physical activity, socioeconomic status or overall diet quality) cannot be ruled out (Atzeni et al., 2022; Ozkan et al., 2025). UPF intake is typically estimated based on self-reported dietary recall or food-frequency questionnaires, and then classified by the use of NOVA which has subjective judgement calls at its border and is applied inconsistently across studies, making direct comparisons of effect sizes challenging (Dicken & Batterham, 2022).

Methodologically, studies differ widely in how the microbiota itself is characterised, from the older amplicon sequencing of 16S rRNA sequences, which only detect taxa at the genus level, to the newer shotgun metagenomic sequencing, which can detect taxa at the species level and even the strain level, and measure the functional genes present. This variability can make it challenging to interpret why some taxa, like Blautia, have variable results and whether this is biological or due to sequencing depth and analytical pipeline (Faggiani et al., 2025). Randomised controlled feeding trials of the type performed by Chassaing et al. (2022), which directly manipulate UPF or intake of specific additives in human subjects, are still relatively scarce, costly, and are short-term by necessity, and so can only tell part of the story of the decades-long exposure most people experience.

There are three research priorities that will be addressed in the future. More long-term human feeding studies, preferably using realistic combinations of additives instead of single isolated chemicals, would add much to the causal inference. More extensive birth cohorts with a broader spatial and temporal scope would help shed light on the persistence of early-life microbiota disruption. Lastly, better communication between the food scientists, who have more intimate knowledge of the process and formulation involved, and the microbiome scientists who know the ecological and functional implications, would facilitate the testing of new food formulations for their impact on gut health, rather than assuming they are positive based on a decrease in sugar or salt on the label.

10. Conclusion

This review collates evidence primarily from the last four years of food science, nutrition and microbiology research and provides a fairly cohesive picture. The removal of fibre, the addition of emulsifiers, non-nutritive sweeteners and other synthetic additives in ultra-processed foods have a cumulative effect that can lead to a loss of microbial diversity in the gut, a decrease in beneficial producers of short-chain-fatty-acids, the thinning of the protective mucus layer and an increase in intestinal permeability. These alterations are mechanistically and epidemiologically linked to a plethora of downstream conditions, such as metabolic syndrome, inflammatory bowel disease, colorectal cancer, and mood disturbances via the gutbrain axis, and also early life emerges as an exceptionally delicate time for enduring disruption. None of this evidence indicates that food processing is the culprit; canning, freezing, and fermenting are all ways of processing foods that result in better or even preserved nutritional value. More specifically, it is the mix of destruction of the matrix and loading of the additives that is the concern with the ultra-processed category, as it is presently formulated. This is a challenge and an opportunity for food science, too: in principle, the same industrial methods that have been applied to the development of UPF products could be applied to products that are designed to be made from the ground up to support gut microbial ecology, rather than to treat it as an afterthought, and which are minimised from disruptive combinations of additives.

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