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Review Article

Ultra-Processed Foods and Preventive Ayurveda: Effects on Gut Microbiota, Metabolism and Mental Health

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Abstract

Background and Objectives: Ultra-processed foods (UPFs) now form a substantial part of many diets. High intake has been associated with obesity, type 2 diabetes, cardiovascular disease and common mental health problems. This review examines these findings alongside the preventive dietary principles of Ayurveda.

Methods: A narrative search of PubMed, Scopus, Web of Science and Google Scholar was conducted for literature on UPFs, gut microbiota, metabolic disease, mental health and the gut-brain axis. Classical Ayurvedic sources were reviewed for *Ahara*, *Agni*, *Ama*, *Pathya*, *Dinacharya* and related concepts.

Results: The most consistent evidence links greater UPF exposure with excess energy intake, weight gain and cardiometabolic risk. Associations with depression, anxiety and adverse sleep outcomes are also reported, although reverse causation and residual confounding remain possible. Low fibre intake, reduced food-matrix integrity and selected additives

may influence microbial metabolism and intestinal barrier regulation, but direct human microbiome evidence is still limited.

Conclusions: Preventive Ayurveda supports a practical response based on fresh and minimally processed foods, appropriate quantity, regular meal timing, digestive suitability, physical activity and adequate sleep. Ayurvedic concepts can guide prevention, but they should not be presented as direct biological equivalents of dysbiosis, insulin resistance or neuroinflammation.

Keywords: *ultra-processed foods; Ayurveda; gut microbiota; metabolic health; mental health*

Introduction

Modern food processing ranges from useful practices such as cooking, fermentation, pasteurization and freezing to the manufacture of highly formulated products. The NOVA classification uses the nature, purpose and extent of processing to distinguish unprocessed or minimally processed foods from ultra-processed formulations ^[1,2]. Typical UPFs include sugar-sweetened drinks, packaged snacks,

confectionery, instant foods, reconstituted meat products and many ready-to-eat meals. The category is broad, and products within it do not all have the same nutrient profile or health effect.

Concern about UPFs is not based only on sugar, fat or salt. Food structure, energy density, eating rate, fibre content, palatability and displacement of whole foods may act together. In a controlled inpatient crossover trial, 20 adults consumed more energy and gained weight during an ultra-processed diet than during a matched unprocessed diet [3]. At population level, a 2024 umbrella review covering 45 pooled analyses and almost 9.9 million participants linked greater UPF exposure with adverse outcomes across cardiometabolic, mental health and mortality domains, while also showing that certainty varied considerably by outcome [4].

Ayurveda places everyday food behaviour at the centre of health preservation. The *Charaka Samhita*, *Sushruta Samhita* and *Ashtanga Hridaya* discuss the quality, quantity, preparation, timing and suitability of food, as well as daily and seasonal routines [5-9]. These ideas offer a useful preventive framework for the current food environment. They should, however, remain conceptually distinct from modern biomedical terms. *Ama* is not dysbiosis, *Agni Mandya* is not insulin resistance, and *Medovaha Srotodushti* is not metabolic syndrome.

This review therefore asks a practical question: how can current evidence on UPFs, gut health, metabolism and mental well-being be interpreted alongside preventive Ayurvedic principles without forcing one knowledge system into the terminology of the other?

Aim

To critically review the effects of ultra-processed food consumption on gut microbiota, metabolic health and mental well-being, and to examine their relevance to preventive Ayurvedic dietary and lifestyle principles.

Literature Search Strategy

A narrative review was undertaken. PubMed, Scopus, Web of Science and Google Scholar were searched using combinations of “ultra-processed food,”

“NOVA classification,” “gut microbiota,” “gut microbiome,” “intestinal barrier,” “food additives,” “obesity,” “type 2 diabetes,” “cardiovascular disease,” “depression,” “anxiety,” “mental health” and “gut-brain axis.” Priority was given to systematic reviews, meta-analyses, prospective cohorts, controlled feeding studies and major mechanistic reviews published in English. Classical Ayurvedic sources and established translations were examined for *Ahara*, *Agni*, *Ama*, *Ahara Vidhi*, *Pathya-Apathya*, *Viruddha Ahara*, *Dinacharya*, *Ritucharya* and *Swasthavritta*. Studies were included when they addressed the three prespecified domains or helped explain a plausible pathway. Papers lacking a clear connection to these domains were excluded. Because the exposures and outcomes were heterogeneous, findings were synthesized narratively rather than pooled again.

Ultra-Processed Foods

Not every processed food is unhealthy. Processing can improve safety, digestibility and shelf life. UPFs are different because they are generally industrial formulations made largely from refined substances, additives and ingredients rarely used in ordinary kitchens, with relatively little intact food. Many are soft, convenient, rapidly eaten and strongly palatable. This combination can make passive overconsumption easier.

The effect of a UPF-rich diet is unlikely to arise from one ingredient alone. A refined food matrix can alter eating rate and the physiological handling of nutrients; low intact fibre can reduce fermentable substrates; high energy density can increase calorie intake; and frequent use can displace fruits, vegetables, pulses, whole grains and traditionally prepared meals. Table 1 separates these possible pathways from the strength of the inference.

Table 1: Features of UPF-rich dietary patterns and possible health pathways

Dietary feature	Possible pathway	Cautious interpretation
High energy density and rapid eating	Greater energy intake before satiety develops	Supported by controlled feeding evidence[3]
Refined starches and added sugars	Larger glycaemic exposure in	Effect depends on the whole diet and host factors

	susceptible individuals	
Low intact fibre	Fewer substrates for microbial fermentation	Microbiome response varies between individuals ^[10,11]
Low plant diversity	Reduced diversity of food-derived substrates	Often reflects displacement of whole foods
Selected emulsifiers or sweeteners	Possible effects on microbiota, mucus and metabolic signalling	Compound-, dose- and model-specific ^[12-14]
High sodium in some products	Greater sodium exposure	Not a property of every UPF
Convenience and intense palatability	Frequent snacking and distracted eating	Strongly influenced by the food environment

Gut Microbiota and Intestinal Health

Diet is one of the main modifiable influences on the intestinal microbiota. Recent reviews describe plausible links between UPF-rich dietary patterns, altered microbial metabolism and gut disease, but they also stress the limited number and methodological variability of direct human studies^[10,11]. The microbiome is shaped by age, geography, medicines, antibiotics, physical activity and the rest of the diet, so no single microbial pattern can currently be labelled a universal “UPF microbiome.”

Food additives are an important mechanistic area. Certain emulsifiers altered microbiota composition and inflammatory responses in animal models, and ex vivo work found changes in human microbial composition and gene expression^[12,13]. A frequently cited study also linked non-nutritive sweeteners with glucose intolerance through microbiota-dependent mechanisms^[14]. These observations justify further research, but they cannot be applied to every additive or ordinary human exposure without compound-specific clinical evidence.

The broader dietary pattern may be more important than any single additive. Industrialized diets commonly contain less diverse plant material than traditional dietary patterns^[15]. Fermentable fibres support microbial metabolism and the production of short-chain fatty acids, including butyrate, which

contributes to colonocyte energy supply and intestinal barrier regulation^[16,17]. Western dietary patterns may disturb this host-microbe relationship through several interacting changes^[18]. A proposed sequence—reduced fibre and plant diversity, altered metabolites, impaired barrier regulation and low-grade inflammation—is biologically plausible, but it remains a hypothesis rather than a proven pathway for all UPFs.

Metabolic Health

The evidence linking UPFs with metabolic outcomes is more developed than the microbiome evidence. Systematic reviews have repeatedly reported associations with obesity, type 2 diabetes, cardiovascular disease and mortality^[19-21]. The interpretation is strengthened by the controlled feeding trial, but most long-term data remain observational.

In three large US cohorts, higher total UPF intake was associated with a greater risk of type 2 diabetes, and a meta-analysis within the same study found a dose-response relationship^[22]. Cardiovascular evidence includes a dose-response meta-analysis and pooled findings from large perspective cohorts^[23,24]. Prospective studies from Spain and France also reported higher all-cause mortality among people with greater UPF consumption^[25,26], while the NutriNet-Santé cohort linked UPF exposure with incident cardiovascular disease^[27]. Cancer evidence is less consistent, although an early NutriNet-Santé analysis reported an association with overall cancer risk^[28].

Several pathways may act together: higher energy intake, rapid eating, low satiety, refined carbohydrates, weight gain, excess sodium and poorer overall dietary quality. Physical inactivity, sleep, socioeconomic conditions, genetics and adiposity remain important co-determinants. UPFs should therefore be understood as one part of an obesogenic and metabolically adverse environment, not as the sole cause of diabetes or cardiovascular disease.

Mental Health and the Gut-Brain Axis

Mental health has become a major area of UPF research. A systematic review and meta-analysis of observational studies found greater UPF exposure associated with depression and anxiety-related outcomes [29]. A separate dose-response meta-analysis of 260,385 adults reported a higher risk of depression, although the anxiety estimate was not statistically conclusive [30]. Prospective cohorts in Spain and France have also linked greater UPF intake with later depressive symptoms [31,32].

The gut and brain communicate through neural, endocrine, immune and metabolic pathways. Major reviews describe the microbiota-gut-brain axis as a bidirectional network rather than a single pathway [33,34]. Inflammation, stress physiology and microbial metabolites may participate in depression, but the evidence is still evolving [35,36]. Other reviews likewise emphasize that altered microbiota can be relevant without being sufficient to explain a complex mental disorder [37,38].

Causality is especially difficult here. Depression, chronic stress or poor sleep may increase reliance on convenient foods, while socioeconomic circumstances can influence both diet and mental health. Dietary improvement has reduced depressive symptoms in a randomized trial, but that finding concerns an overall dietary intervention rather than UPF withdrawal alone [39]. Current mechanistic reviews describe interacting pathways involving inflammation, oxidative stress, the microbiota, hypothalamic-pituitary-adrenal signalling and neuroplasticity [40]. A balanced conclusion is therefore that the association is credible, probably bidirectional and not explained by one microbial signature.

Preventive Ayurvedic Interpretation

Ayurveda does not assess food only by its nutrient content. *Ahara* is considered in relation to quality, quantity, preparation, combination, timing, season, digestive capacity and the condition of the person. *Agni* represents a broad functional capacity for digestion and assimilation; it should not be reduced to gastric acid, enzymes or the microbiome. *Ama* is a

classical construct linked with inadequate processing and disturbed function, but it has no established single molecular equivalent.

The useful comparison is at the level of preventive reasoning. Both modern nutrition and Ayurveda recognize that the effect of food depends on repeated habits, digestive and metabolic context, and the wider lifestyle. *Ahara Vidhi* adds attention to how food is eaten: suitable quantity, appropriate timing, proper preparation, hunger and undistracted eating. These principles are relevant in a setting where soft packaged foods can be eaten quickly, late at night, while viewing screens or repeatedly between meals.

Ayurvedic descriptions of excessive nourishment, inactivity and excess sleep also provide a preventive framework for considering *Medo Dhatu* disturbance. The framework is not identical to obesity, yet both perspectives recognize the interaction of diet, activity and metabolism. Mental well-being is similarly approached through a combination of food, sleep, routine, sensory regulation and behaviour. It is more defensible to say that habitual dependence on nutritionally poor, highly palatable products conflicts with this broader preference for appropriate and consciously consumed food than to label every UPF as *Tamasika Ahara*.

Table 2: Conceptual links with preventive Ayurveda and their limits

Contemporary issue	Ayurvedic concept	Interpretive boundary
Digestion and metabolic processing	<i>Agni</i>	Broader than enzymes, acid or microbiota
Consequences of inadequate processing	<i>Ama</i>	No validated single molecular equivalent
Appropriate dietary choice	<i>Pathya Ahara</i>	Individualized and context-dependent
Unsuitable habitual diet	<i>Apathya Ahara</i>	Not synonymous with UPFs
Metabolic excess and adiposity	<i>Medo Dushti / Medovaha Srotodushti</i>	Not identical to obesity or metabolic syndrome
Manner and timing of eating	<i>Ahara Vidhi</i>	Behavioural framework, not a processing classification
Regular daily routine	<i>Dinacharya</i>	Extends beyond metabolic prevention
Seasonal adaptation	<i>Ritucharya</i>	Has no single modern dietary variable

A Practical Preventive Model

The most useful approach is *Pathya* rather than fear or absolute prohibition. Not every packaged item must be eliminated, and occasional intake is different from allowing UPFs to become the foundation of the diet. The main goal is replacement: increase minimally processed food diversity and gradually reduce habitual dependence on sugar-sweetened drinks, packaged snacks and ready-to-eat products.

Advice should also recognize cost, time, work schedules, cooking facilities and food access. A recommendation that cannot be followed in daily life is unlikely to produce lasting prevention. Table 3 translates the integrative principles into practical, non-punitive actions.

Table 3: Practical preventive actions for reducing habitual UPF dependence

Area	Practical action	Preventive rationale
Food base	Build most meals from vegetables, fruits, pulses, whole grains, nuts, seeds and suitable animal foods	Restores intact food structure, fibre and dietary diversity
Replacement	Replace one frequently consumed UPF at a time with a feasible home-prepared option	Supports sustainable change rather than rigid prohibition
Quantity	Serve an appropriate portion and pause before taking more	Supports satiety and the Ayurvedic emphasis on <i>Matra</i>
Meal rhythm	Prefer regular meals and reduce continuous snacking	Links modern meal regularity with <i>Ahara Vidhi</i>
Eating behaviour	Eat seated, slowly and with attention rather than while working or viewing screens	May reduce rapid, distracted consumption
Lifestyle	Combine dietary change with activity, adequate sleep and stress management	Reflects <i>Dinacharya</i> and multiple metabolic determinants
Individualization	Adapt advice to tolerance, age, season, disease status and resources	Preserves the practical meaning of <i>Pathya</i> and <i>Satmya</i>

Discussion

The strongest current evidence concerns cardiometabolic outcomes. Repeated associations across cohorts and meta-analyses, together with one tightly controlled feeding study, support a genuine concern about diets dominated by UPFs. Even so, the

category is heterogeneous. Some products classed as ultra-processed have more favourable nutrient profiles than others, and high intake often travels with low whole-food intake, socioeconomic disadvantage, physical inactivity and poor sleep. These factors complicate causal interpretation.

The microbiome provides a plausible bridge between diet, metabolism and the brain, but it is currently the least settled part of the argument. Direct human studies are fewer, methods vary and a microbial change may be a marker rather than the cause of poor dietary quality. The responsible conclusion is not that UPFs invariably cause dysbiosis, but that low-fibre, low-diversity dietary patterns and selected additives deserve closer study.

Preventive Ayurveda contributes most clearly at the behavioural and person-centred level. *Agni* draws attention to digestive suitability; *Pathya* supports appropriate selection; *Ahara Vidhi* emphasizes quantity, timing and attention; and *Dinacharya* places diet within sleep, activity and daily routine. This framework can strengthen health counselling without claiming that classical texts described NOVA categories, microbiome sequencing or modern metabolic biomarkers.

Research Gaps and Future Directions

Future studies should separate UPF subgroups, use repeated and validated dietary measurements, and account for overall diet quality and social determinants. Longer controlled feeding studies are needed, although they are difficult and costly. Microbiome research should combine metagenomics with microbial metabolites, intestinal barrier markers and carefully documented additive exposure rather than reporting taxonomic abundance alone.

For integrative research, a useful design would compare a standard minimally processed healthy diet with an individualized Ayurvedic preventive programme. Outcomes could include percentage energy from UPFs, body weight, waist circumference, fasting glucose and insulin, HbA1c, lipids, inflammatory markers, short-chain fatty acids, validated gastrointestinal symptoms, depression, anxiety, sleep and quality of life. Ayurvedic variables such as meal timing, tolerance and clinically assessed

digestive pattern should be defined in advance and measured consistently. Such work would test practical interventions without forcing classical concepts into artificial biomedical equivalents.

Conclusion

Greater consumption of ultra-processed foods is consistently associated with obesity, type 2 diabetes, cardiovascular outcomes, premature mortality and common mental health problems, although the certainty and causal strength differ between outcomes. The gut microbiota may participate through changes in fibre exposure, microbial metabolism, intestinal barrier regulation and selected additives, but direct causal pathways in humans remain incompletely established.

Preventive Ayurveda offers a realistic complementary response: make fresh and minimally processed foods the basis of daily meals, use appropriate quantity, eat regularly and attentively, consider digestive suitability, remain physically active and protect sleep. Its value lies in strengthening everyday prevention and individualization, not in equating *Agni*, *Ama* or *Dosha-Dhatu* balance with specific modern biomarkers.

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