



FROM TRADITIONAL PLATES TO PROCESSED DIETS: A DECADE OF CHANGING FOOD HABITS AND THEIR IMPACT ON OBESITY, HYPERTENSION, DIABETES, AND CARDIOVASCULAR DISEASE (2015–2026)

Tejaswini V. Nandi¹, Suhasini L. Kudupali² and Aishwarya P. Kalkoti^{*3}

¹KLE Society's G. I. Bagewadi Arts, Science and Commerce College, Nippani-591237, Karnataka, India.

²KLE Society's S. Nijalingappa College, Rajajinagar, Bengaluru-560010, Karnataka, India.

³Rajiv Gandhi University of Health Sciences, Bengaluru, Karnataka, India.

*Corresponding author E-mail: draishwarya.77.09@gmail.com

Received: 13 May 2026

Revised: 17 June 2026

Accepted: 09 July 2026

Published: 30 July 2026

DOI: <https://doi.org/10.5281/zenodo.21839572>

Abstract:

The global food environment has undergone a significant transformation between 2015 and 2026, with traditional dietary patterns increasingly replaced by diets rich in ultra-processed foods, refined carbohydrates, added sugars, unhealthy fats and sodium. Driven by urbanization, globalization, technological advancements and changing lifestyles, this nutritional transition has contributed to the rising prevalence of obesity, hypertension, type 2 diabetes mellitus and cardiovascular disease. This review critically examines the changing food habits during the past decade and their impact on cardiometabolic health. Evidence synthesized from recent scientific literature indicates that increased consumption of processed foods is consistently associated with excessive energy intake, insulin resistance, chronic inflammation, oxidative stress, endothelial dysfunction, lipid abnormalities and gut microbiota alterations. In contrast, traditional diets rich in minimally processed plant-based foods, dietary fibre and bioactive compounds remain strongly associated with improved metabolic health and reduced disease risk. Promoting healthier dietary patterns through nutrition education, supportive public health policies and sustainable food systems is essential for reducing the growing burden of diet-related non-communicable diseases worldwide.

Keywords: Dietary Transition, Traditional Diet, Ultra-Processed Foods, Nutrition Transition, Obesity, Hypertension, Type 2 Diabetes Mellitus, Cardiovascular Disease, Non-Communicable Diseases, Food Environment.

1. Introduction

Food patterns represent one of the most important modifiable determinants of human health, influencing metabolism, immune function, disease development, and longevity. During the past decade (2015–2026), global dietary habits have undergone a major transformation, commonly referred to as the nutrition transition, characterized by the replacement of traditional dietary patterns with diets increasingly dominated by ultra-processed foods (UPFs), refined carbohydrates, added sugars, unhealthy fats, and sodium-rich convenience foods. This transition has been accelerated by urbanization, globalization of food systems, technological advancements, changing lifestyles, reduced time for home cooking, and expansion of commercial food environments. Although these developments have improved food accessibility and convenience, they have also contributed to the increasing burden of obesity, hypertension, type 2 diabetes mellitus (T2DM), and cardiovascular disease (CVD). [1–3]

Traditional dietary patterns across different regions, including Mediterranean, Asian, and other plant-based dietary models, have historically emphasized fruits, vegetables, legumes, whole grains, nuts, seeds, fermented foods, and minimally processed products. These dietary patterns provide high amounts of dietary fiber, antioxidants, phytochemicals, and unsaturated fatty acids while containing lower levels of refined sugars, unhealthy fats, and excess sodium. Evidence from epidemiological studies and clinical trials demonstrates that adherence to these dietary patterns is associated with improved metabolic health and reduced risk of obesity, cardiovascular events, diabetes, and premature mortality. [4,5]

In contrast, modern food systems have resulted in increased consumption of ultra-processed foods. According to the NOVA classification system, UPFs are industrial formulations produced mainly from extracted food substances, modified ingredients, and additives, with little resemblance to whole foods. Common examples include sugar-sweetened beverages, packaged snacks, processed meats, instant meals, confectioneries, fast foods, and commercially produced baked products. These foods are designed to enhance taste, convenience, affordability, and shelf stability, leading to their widespread incorporation into daily diets and displacement of nutrient-rich minimally processed foods. [6]

The increasing dependence on processed dietary patterns has been influenced by complex social, economic, and environmental factors. Urban lifestyles, increased workforce participation, changing household structures, digital food marketing, social media promotion, and online food delivery services have significantly altered food choices. Additionally, socioeconomic inequalities contribute to greater reliance on inexpensive energy-dense processed foods, increasing disparities in dietary quality and susceptibility to nutrition-related chronic diseases. [1,7]

The adverse health effects of processed diets extend beyond excessive calorie intake. High consumption of refined carbohydrates and added sugars contributes to hyperglycaemia, hyperinsulinemia, and insulin resistance, whereas excessive sodium intake promotes hypertension through fluid retention and vascular dysfunction. Diets rich in saturated and trans fats contribute to adverse lipid profiles and atherosclerotic processes. Furthermore, reduced intake of fiber, antioxidants, and bioactive plant compounds promotes oxidative stress, chronic low-grade inflammation, endothelial dysfunction, and metabolic disturbances that collectively increase the risk of obesity, T2DM, and CVD. [8–10]

Recent advances have highlighted the role of the gut microbiome as an important link between dietary patterns and metabolic health. Diets rich in plant-based foods support microbial diversity and production of beneficial

metabolites such as short-chain fatty acids, which regulate immune responses, maintain intestinal barrier function, and improve metabolic homeostasis. Conversely, UPF-rich diets are associated with reduced microbial diversity, altered microbial composition, increased intestinal permeability, and systemic inflammatory responses, potentially contributing to cardiometabolic dysfunction. [11]

The global impact of dietary transition is reflected in the rising prevalence of non-communicable diseases (NCDs). Obesity, hypertension, T2DM, and CVD remain among the leading causes of premature mortality and disability worldwide, placing substantial pressure on healthcare systems. Although genetic predisposition, aging, physical inactivity, and socioeconomic conditions influence disease risk, dietary quality remains one of the most important preventable determinants of long-term health outcomes. [2,12]

Despite extensive research on individual nutrients and specific food components, recent evidence highlights the need for a broader evaluation of overall dietary patterns, food processing levels, and their combined influence on major cardiometabolic diseases. Current literature increasingly recognizes that dietary patterns, rather than isolated nutrients alone, play a critical role in determining long-term metabolic health outcomes. However, further research is required to better understand the global shift from traditional dietary patterns toward processed food consumption and its implications across different populations and regions. [13–15]. A comprehensive understanding of this dietary transition is essential for developing effective prevention strategies that address both individual dietary behaviors and the broader food environment, including food accessibility, marketing practices, and policy-level interventions. [14,16] Therefore, this review examines the global transition from traditional diets to processed dietary patterns between 2015 and 2026, focusing on their relationship with obesity, hypertension, T2DM, and CVD, while discussing underlying biological mechanisms, regional variations, public health challenges, and future strategies for promoting healthier and sustainable dietary practices. [13–16]

2. Methodology

2.1 Study design and search strategy

This narrative review synthesized current evidence on the global transition from traditional dietary patterns to processed diets and their association with obesity, hypertension, type 2 diabetes mellitus (T2DM), and cardiovascular disease (CVD). Evidence from observational studies, randomized controlled trials, systematic reviews, meta-analyses, and reports from international health organizations was integrated to provide a comprehensive overview of dietary changes and their cardiometabolic consequences. [17]

A literature search was conducted for studies published between January 2015 and June 2026 using PubMed/MEDLINE, Scopus, Web of Science, Embase, Google Scholar, and Cochrane Library. Additional evidence was obtained from reports by the World Health Organization (WHO), Food and Agriculture Organization (FAO), Global Burden of Disease (GBD) Study, and major cardiovascular and diabetes organizations. Search terms included “dietary transition,” “traditional diet,” “ultra-processed foods,” “processed diet,” “obesity,” “hypertension,” “type 2 diabetes mellitus,” and “cardiovascular disease.” Reference lists of relevant articles were also screened. [18,19]

2.2 Eligibility criteria and data synthesis

Studies published in English that evaluated dietary patterns, ultra-processed food consumption, or nutrition transition in relation to obesity, hypertension, T2DM, CVD, or related metabolic outcomes were included. Original research articles, cohort studies, clinical trials, systematic reviews, meta-analyses, and authoritative reports were

considered eligible. Editorials, duplicate publications, conference abstracts, and studies unrelated to cardiometabolic outcomes were excluded. [18,20]

Relevant data including study population, dietary exposure, assessment methods, outcomes, and major findings were extracted and synthesized narratively. Due to differences in dietary assessment methods and study populations, quantitative meta-analysis was not performed. Evidence from large cohort studies, clinical trials, and systematic reviews was prioritized. [17,21]

3. Global dietary transition (2015–2026): From traditional diets to ultra-processed foods

Human dietary patterns have undergone a substantial transformation over the past decade due to urbanization, globalization, economic development, and changes in food production systems. This nutrition transition has resulted in declining consumption of traditional minimally processed foods and increased reliance on ultra-processed foods (UPFs), refined carbohydrates, added sugars, and energy-dense convenience foods. These changes have occurred globally and have contributed to the rising burden of obesity and cardiometabolic diseases. [22,23]. Traditional dietary patterns, despite regional differences, are generally characterized by high consumption of vegetables, fruits, legumes, whole grains, nuts, seeds, and fermented foods. Diets such as the Mediterranean and traditional Asian dietary patterns provide abundant fiber, antioxidants, phytochemicals, and unsaturated fatty acids, which are associated with improved metabolic health, reduced inflammation, and lower risk of chronic disease. [24,25]

In contrast, modern diets increasingly contain UPFs, which are industrial formulations made from refined ingredients, additives, added sugars, sodium, and unhealthy fats. The NOVA classification system identifies products such as packaged snacks, sugar-sweetened beverages, processed meats, ready-to-eat meals, and commercially baked foods as UPFs. These foods are designed for convenience and high palatability, often resulting in increased energy intake and reduced dietary quality [26].

The expansion of UPFs has been driven by urban lifestyles, reduced time for home cooking, globalization of food markets, aggressive marketing, digital advertising, and increased availability of processed foods. Low- and middle-income countries are experiencing particularly rapid dietary changes, leading to a double burden of malnutrition where undernutrition and obesity coexist. [22,27]

Overall, the shift from traditional dietary patterns toward processed food environments represents a major nutritional change of the modern era. This transition has important implications for the development and prevention of obesity, hypertension, T2DM, and CVD. [22–27]

Table 1: Comparison of traditional and ultra-processed dietary patterns

Feature	Traditional Diets	Ultra-Processed Diets
Main foods	Fruits, vegetables, legumes, whole grains	Packaged foods, snacks, processed meals
Processing	Minimal	Extensive industrial processing
Fiber content	High	Low
Added sugars/sodium	Low	High
Bioactive compounds	Rich	Limited
Cardiometabolic effect	Generally protective	Increased disease risk

4. Biological mechanisms linking processed diets to cardiometabolic diseases

The relationship between processed dietary patterns and cardiometabolic diseases involves multiple interconnected biological pathways rather than a single mechanism. Diets high in ultra-processed foods (UPFs), refined carbohydrates, added sugars, sodium, and unhealthy fats promote metabolic disturbances through chronic inflammation, oxidative stress, insulin resistance, gut microbiota alterations, and vascular dysfunction, collectively increasing the risk of obesity, hypertension, type 2 diabetes mellitus (T2DM), and cardiovascular disease (CVD). [28,29]

4.1 Inflammation and oxidative stress

Chronic low-grade inflammation is a central pathway linking unhealthy dietary patterns with metabolic disease. Excess intake of refined carbohydrates, saturated fats, processed meats, and sugar-sweetened beverages promotes increased production of inflammatory mediators, including interleukin-6 (IL-6), tumor necrosis factor- α (TNF- α), and C-reactive protein (CRP). These inflammatory processes impair insulin signalling, promote adipose tissue dysfunction, and accelerate vascular injury. [30]

Processed diets also increase oxidative stress by promoting excessive reactive oxygen species (ROS) production while providing fewer antioxidant compounds compared with diets rich in fruits, vegetables, legumes, and whole grains. Oxidative damage contributes to endothelial dysfunction, pancreatic β -cell impairment, and progression of atherosclerosis. [31]

4.2 Insulin resistance and metabolic dysfunction

High consumption of refined carbohydrates and added sugars causes repeated glucose fluctuations and increased insulin demand. Over time, reduced insulin sensitivity develops, resulting in impaired glucose regulation and progression toward T2DM. Insulin resistance also promotes abnormal lipid metabolism, visceral adiposity, and metabolic syndrome, all of which increase cardiovascular risk. [32]

Energy-dense processed foods further contribute to obesity by promoting excessive calorie intake. Their low fiber content, high palatability, and rapid consumption reduce satiety signals and encourage overeating. Controlled feeding studies have demonstrated that ultra-processed diets can increase calorie intake and weight gain compared with minimally processed diets. [33]

4.3 gut microbiota dysbiosis

Dietary patterns strongly influence gut microbial composition and function. Traditional diets rich in fiber support beneficial bacteria that produce short-chain fatty acids (SCFAs), which improve intestinal barrier integrity, regulate immune responses, and enhance metabolic health. Conversely, UPF-rich diets with low fiber content and increased additives may reduce microbial diversity and promote intestinal permeability and metabolic endotoxemia. These alterations contribute to systemic inflammation, insulin resistance, obesity, and vascular dysfunction. [34,35]

4.4 Endothelial dysfunction and cardiovascular injury

High intake of sodium, saturated fats, and refined carbohydrates adversely affects vascular function by reducing nitric oxide availability, increasing oxidative stress, and promoting inflammation. Persistent endothelial dysfunction contributes to arterial stiffness, hypertension, and atherosclerotic plaque formation, increasing the risk of myocardial infarction and stroke. [36]

4.5 Integrated effects of processed diets

The mechanisms linking processed diets with chronic disease are highly interconnected. Excess energy intake promotes obesity, which amplifies inflammation and insulin resistance. Simultaneously, gut microbiota disturbances, oxidative stress, and vascular injury accelerate metabolic deterioration. Therefore, improving overall dietary quality rather than focusing on individual nutrients remains essential for preventing cardiometabolic diseases. [28–36]

Table 2: Major mechanisms linking ultra-processed diets with cardiometabolic disease

Mechanism	Dietary Factors	Major Consequences
Inflammation	Refined carbohydrates, processed fats, processed meats	Cytokine activation, metabolic dysfunction
Oxidative stress	High sugar intake, low antioxidants	Cellular injury, vascular damage
Insulin resistance	Sugary foods, refined carbohydrates	Hyperglycaemia, T2DM
Gut dysbiosis	Low fiber, food additives	Inflammation, metabolic endotoxemia
Endothelial dysfunction	Excess sodium, unhealthy fats	Hypertension, CVD

5. Processed dietary patterns and the global obesity epidemic

5.1 Global burden of obesity and dietary transition

Obesity has become one of the most significant public health challenges worldwide, with prevalence increasing across both high-income and low- and middle-income countries. The rapid rise in obesity over recent decades parallels the global shift toward energy-dense, highly processed dietary patterns and reduced consumption of minimally processed foods. Although genetic susceptibility influences individual risk, the magnitude and speed of the obesity epidemic indicate that environmental and dietary changes are major contributing factors. [37,38]

Excess adiposity is strongly associated with hypertension, insulin resistance, type 2 diabetes mellitus (T2DM), dyslipidaemia, cardiovascular disease (CVD), and reduced life expectancy. The increasing availability of ultra-processed foods (UPFs), combined with sedentary lifestyles, has created an obesogenic environment that promotes excessive energy intake and metabolic dysfunction. [37,39]

5.2 Ultra-processed foods and weight gain

UPFs contribute to obesity through multiple pathways, including increased energy density, reduced satiety, and altered appetite regulation. These foods are typically high in refined carbohydrates, added sugars, unhealthy fats, and sodium while being low in fiber and essential nutrients. Their soft texture, high palatability, and convenience facilitate rapid consumption and excessive calorie intake. [40]

Evidence from controlled feeding studies supports a direct relationship between UPF consumption and weight gain. Hall *et al.* demonstrated that individuals consuming an ultra-processed diet consumed substantially more calories and gained more weight compared with those consuming a minimally processed diet, despite similar macronutrient composition. [41]

Large prospective cohort studies and meta-analyses have further reported that higher UPF intake is associated with increased body mass index (BMI), abdominal obesity, and greater risk of developing obesity. Although observational studies may be influenced by residual confounding, the consistency of findings across different populations strengthens the evidence linking UPFs with adverse weight outcomes. [42,43]

5.3 Childhood obesity and food environment

The increasing consumption of processed foods among children and adolescents represents a major concern. Sugar-sweetened beverages, packaged snacks, fast foods, and processed breakfast products have increasingly replaced traditional meals, contributing to unhealthy dietary patterns early in life. Exposure to digital marketing, social media advertising, and targeted promotion of processed foods further influences children's food preferences and consumption behaviors. [44]

Childhood obesity is particularly important because excess weight during early life significantly increases the likelihood of adult obesity and related metabolic diseases. Therefore, interventions targeting food environments, school nutrition, and healthy dietary habits during childhood are essential components of obesity prevention strategies. [44,45]

5.4 Socioeconomic and environmental determinants

Obesity is influenced not only by individual choices but also by broader social and environmental factors. In many populations, UPFs are cheaper, more accessible, and more heavily marketed than fresh and minimally processed foods. Limited availability of healthy foods, economic constraints, urbanization, and reduced opportunities for home cooking contribute to inequalities in dietary quality. [46]

The modern food environment has therefore shifted dietary behaviour from traditional patterns toward commercially driven consumption. Addressing obesity requires population-level strategies, including improved food policies, nutrition education, healthier food environments, and promotion of minimally processed dietary patterns. [46,47]

Table 3: Representative evidence linking ultra-processed foods with obesity

Study	Design	Main Findings
Hall <i>et al.</i> (2019)	Randomized controlled trial	Ultra-processed diet increased calorie intake and weight gain
NutriNet-Sant� cohort	Prospective cohort	Higher UPF intake associated with increased obesity risk
Meta-analyses of cohort studies	Systematic evidence	UPF consumption linked with higher BMI and adiposity

5.5 Summary

The global obesity epidemic has developed alongside the widespread replacement of traditional diets with ultra-processed dietary patterns. Evidence from experimental studies, prospective cohorts, and systematic reviews indicates that higher UPF consumption promotes excessive calorie intake, weight gain, and obesity risk. Reducing reliance on processed foods while improving access to nutrient-rich minimally processed foods represents a key strategy for obesity prevention. [37–47]

6. Dietary transition and hypertension: Impact of processed diets on blood pressure

6.1 Global burden of hypertension and dietary change

Hypertension remains one of the leading preventable causes of cardiovascular disease (CVD), stroke, chronic kidney disease, and premature mortality worldwide. The increasing prevalence of elevated blood pressure over the past decade has occurred alongside major dietary changes, particularly the replacement of traditional diets with sodium-rich, energy-dense, and highly processed foods. This transition has contributed significantly to the

growing global burden of hypertension, especially in populations undergoing rapid urbanization and nutritional change. [48,49]

Unlike traditional dietary patterns rich in fruits, vegetables, legumes, and whole foods, modern processed diets commonly contain excessive sodium, refined carbohydrates, unhealthy fats, and fewer protective nutrients such as potassium and dietary fiber. These dietary changes promote obesity, inflammation, endothelial dysfunction, and metabolic disturbances that collectively increase blood pressure. [50]

6.2 Sodium, potassium imbalance, and vascular dysfunction

Excessive sodium intake is one of the strongest dietary determinants of hypertension. Processed foods such as packaged snacks, processed meats, instant meals, sauces, and fast foods contribute substantially to population sodium exposure. High sodium intake promotes fluid retention, increased vascular resistance, impaired nitric oxide availability, and activation of neurohormonal pathways involved in blood pressure regulation. [51]

At the same time, reduced consumption of potassium-rich foods due to declining intake of fruits, vegetables, and legumes has altered the sodium-potassium balance. Higher potassium intake improves vascular function, promotes sodium excretion, and is associated with lower blood pressure levels. Therefore, hypertension risk is influenced not only by excessive sodium intake but also by the overall deterioration of dietary quality. [52]

6.3 Ultra-processed foods and hypertension risk

Recent evidence suggests that the relationship between ultra-processed foods (UPFs) and hypertension extends beyond sodium content alone. UPFs frequently contain refined carbohydrates, unhealthy fats, additives, and low levels of protective nutrients, contributing to obesity, insulin resistance, systemic inflammation, and vascular injury. These combined effects increase susceptibility to elevated blood pressure. [53]

Large observational studies have consistently reported associations between higher UPF consumption and increased hypertension risk. Conversely, dietary patterns emphasizing fruits, vegetables, whole grains, legumes, and low-fat dairy products, such as the DASH diet, have demonstrated significant reductions in blood pressure in randomized clinical trials. [54,55]

6.4 prevention strategies

Prevention of diet-related hypertension requires population-level approaches, including sodium reduction, improved food labelling, regulation of unhealthy food marketing, and increased availability of affordable healthy foods. At the individual level, adoption of minimally processed dietary patterns rich in plant-based foods remains a cornerstone strategy for maintaining healthy blood pressure. [56]

Table 4: Representative evidence linking dietary patterns with hypertension

Study	Design	Main Findings
DASH Trial	Randomized trial	DASH diet significantly reduced blood pressure
INTERSALT Study	International study	Higher sodium intake associated with increased blood pressure
NutriNet-Santé Cohort	Prospective cohort	Higher UPF intake associated with greater hypertension risk
PURE Study	Multinational cohort	Higher-quality diets associated with improved cardiovascular outcomes

6.5 Summary

The global dietary transition has contributed to rising hypertension prevalence through increased sodium intake, reduced potassium consumption, and greater reliance on ultra-processed foods. Evidence supports shifting dietary patterns toward minimally processed foods rich in fruits, vegetables, whole grains, and legumes as an effective strategy for blood pressure prevention and cardiovascular protection. [48–56]

7. Dietary transition and type 2 diabetes mellitus: The role of ultra-processed diets

7.1 Global burden and dietary drivers of T2DM

Type 2 diabetes mellitus (T2DM) has emerged as one of the fastest-growing non-communicable diseases worldwide. The rapid increase in diabetes prevalence during recent decades parallels the widespread adoption of processed dietary patterns, increasing obesity rates, and declining physical activity. Although genetic susceptibility influences individual risk, environmental and dietary changes remain major contributors to the global diabetes epidemic. [57,58]

The transition from traditional diets rich in whole grains, legumes, fruits, and vegetables toward diets dominated by refined carbohydrates, sugar-sweetened beverages, and ultra-processed foods has substantially altered glucose metabolism and increased insulin resistance. [59]

7.2 Processed diets, insulin resistance, and glucose dysregulation

Ultra-processed diets contribute to T2DM through multiple interconnected pathways, including excessive intake of refined carbohydrates, added sugars, unhealthy fats, and reduced dietary fiber. Rapidly absorbed carbohydrates produce repeated glucose and insulin spikes, eventually leading to impaired insulin sensitivity and progressive pancreatic β -cell dysfunction. [60]

Sugar-sweetened beverages represent a major dietary risk factor because they provide high amounts of rapidly available sugars with minimal satiety. Prospective cohort studies and meta-analyses consistently demonstrate that frequent consumption of these beverages is associated with increased risk of obesity and T2DM. [61]

7.3 Ultra-processed foods, obesity, and diabetes risk

Obesity represents the strongest metabolic link between processed diets and T2DM. Excess visceral adiposity promotes inflammation, altered adipokine secretion, increased free fatty acid release, and impaired insulin signalling. Consequently, processed dietary patterns create a cycle of weight gain, insulin resistance, and progressive glucose intolerance. [62]

Large cohort studies, including NutriNet-Santé, UK Biobank, and other international investigations, consistently demonstrate that higher UPF consumption is associated with increased incidence of T2DM, even after adjustment for lifestyle factors and total energy intake. [63]

7.4 Gut microbiota and food processing

Dietary transition also influences glucose metabolism through alterations in gut microbiota. Traditional diets rich in fiber promote beneficial microbial populations that produce short-chain fatty acids, which support insulin sensitivity and intestinal barrier function. In contrast, UPFs are typically low in fiber and may contain additives that contribute to microbial imbalance, inflammation, and metabolic dysfunction. [64]

Although microbiome research is still developing, current evidence supports gut dysbiosis as an important pathway linking processed diets with impaired glucose regulation. [65]

7.5 Prevention and future directions

Prevention of T2DM requires strategies that improve overall dietary quality rather than focusing on individual nutrients alone. Increased consumption of whole grains, legumes, fruits, vegetables, nuts, and minimally processed foods, combined with reduced intake of UPFs and sugar-sweetened beverages, has consistently demonstrated metabolic benefits. [66]

Future research should focus on standardized definitions of UPFs, improved dietary assessment methods, long-term intervention trials, and integration of nutritional science with precision medicine approaches. [67]

Table 5: Representative Evidence Linking Dietary Patterns with T2DM

Study	Design	Main Findings
NutriNet-Santé Cohort	Prospective cohort	Higher UPF intake associated with increased T2DM risk
UK Biobank	Cohort study	Processed food consumption linked with metabolic risk
Nurses' Health Study	Prospective cohort	Healthy dietary patterns reduced diabetes incidence
Whole-grain meta-analyses	Systematic evidence	Higher whole-grain intake associated with lower diabetes risk

7.6 Summary

The global rise in T2DM closely parallels the transition from traditional dietary patterns to ultra-processed diets. Processed foods promote insulin resistance, obesity, inflammation, and metabolic dysfunction, whereas diets emphasizing minimally processed plant-based foods improve glucose regulation and reduce diabetes risk. Addressing dietary transition remains essential for controlling the future global diabetes burden. [57–67]

8. Dietary transition and cardiovascular disease: From processed diets to atherosclerosis

8.1 Dietary transition and cardiovascular risk

Cardiovascular disease (CVD) remains the leading cause of global mortality, with coronary artery disease, stroke, and heart failure contributing substantially to premature death and disability. Despite advances in prevention and treatment, CVD burden continues to rise, particularly in populations undergoing rapid nutritional transition. The replacement of traditional dietary patterns with processed, energy-dense, and nutrient-poor foods has emerged as a major modifiable determinant of cardiovascular risk. [68–74]

Processed dietary patterns influence cardiovascular health through interconnected mechanisms, including dyslipidaemia, hypertension, obesity, insulin resistance, endothelial dysfunction, oxidative stress, inflammation, and altered gut microbial metabolism. Therefore, dietary quality and food processing represent important determinants of long-term cardiovascular outcomes. [69–75]

8.2 Processed diets, atherosclerosis, and metabolic dysfunction

Atherosclerosis is the primary pathological process underlying most cardiovascular events. Diets rich in saturated fats, trans fats, refined carbohydrates, processed meats, and ultra-processed foods promote endothelial dysfunction, LDL cholesterol oxidation, inflammatory activation, and plaque progression, increasing the risk of myocardial infarction and stroke. [70–76]

Processed dietary patterns contribute to unfavourable lipid profiles by increasing LDL cholesterol and triglycerides while reducing HDL cholesterol. Conversely, replacement of saturated fats with unsaturated fats from nuts, seeds, fish, and plant oils improves lipid metabolism and reduces cardiovascular risk. [71–77]

Frequent intake of processed meats, refined sugars, and sugar-sweetened beverages further increases cardiovascular risk through sodium overload, oxidative stress, insulin resistance, obesity, hypertriglyceridemia, and vascular inflammation. [72–78]

8.3 Dietary patterns and cardiovascular outcomes

Evidence from large cohort studies and randomized trials consistently demonstrates that dietary patterns emphasizing minimally processed foods are associated with improved cardiovascular outcomes. Mediterranean and DASH dietary patterns, characterized by high consumption of fruits, vegetables, legumes, whole grains, nuts, fish, and healthy fats, improve blood pressure, lipid profiles, endothelial function, and inflammatory status while reducing cardiovascular events. [73–79]

In contrast, Western dietary patterns dominated by ultra-processed foods, refined carbohydrates, processed meats, and sugar-sweetened beverages are associated with increased risks of coronary artery disease, stroke, and heart failure. The cardioprotective effects of healthy dietary patterns result from their combined influence on metabolic regulation, vascular function, and inflammation rather than the effect of individual nutrients alone. [74–80]

8.4 Public health implications for cardiovascular prevention

Reducing the global burden of cardiovascular disease requires population-level strategies that improve food environments and promote healthier dietary choices. Effective interventions include sodium reduction programs, elimination of industrial trans fats, front-of-package nutrition labelling, taxation of sugar-sweetened beverages, regulation of unhealthy food marketing, and improved availability of affordable nutritious foods. [75–81]

Promotion of culturally appropriate dietary patterns based on minimally processed foods should remain central to cardiovascular prevention. Early dietary interventions during childhood and adolescence may provide long-term benefits by reducing the development of obesity, hypertension, diabetes, and cardiovascular disease later in life. [76–82]

8.5 critical appraisal

The relationship between dietary patterns and cardiovascular disease is supported by evidence from randomized clinical trials, prospective cohort studies, systematic reviews, and mechanistic investigations. However, limitations include reliance on self-reported dietary assessments, residual confounding, differences in food classification systems, and challenges in accurately measuring long-term dietary exposure. [77–83]

Despite these limitations, the consistency of findings across diverse populations strongly supports the role of improved dietary quality as a key strategy for cardiovascular disease prevention. [78–84]

9. Discussion

The evidence synthesized in this review demonstrates that the global transition from traditional dietary patterns to ultra-processed food consumption represents a major contributor to the increasing burden of obesity, hypertension, type 2 diabetes mellitus, and cardiovascular disease. Rather than acting through isolated pathways, unhealthy dietary patterns promote interconnected metabolic disturbances involving excess energy intake, inflammation, oxidative stress, insulin resistance, endothelial dysfunction, and altered lipid metabolism. [79–85]

A major advancement in nutritional science has been the recognition that overall dietary patterns and food processing are more important determinants of health than individual nutrients alone. The harmful effects of ultra-processed foods are likely related to their combined characteristics, including high energy density, poor nutrient quality, reduced fiber content, altered food structure, and presence of industrial additives. [80–86]

The relationship between diet and cardiometabolic disease is further influenced by socioeconomic factors, urbanization, food accessibility, and changing lifestyle behaviors. Low- and middle-income countries are particularly affected by rapid nutrition transition, where increasing consumption of processed foods often occurs alongside persistent nutritional inequalities. Therefore, effective prevention strategies must consider cultural, economic, and environmental factors. [81–87]

Although current evidence strongly supports the association between processed dietary patterns and chronic disease, several research gaps remain. Future studies should improve objective dietary assessment methods, standardize ultra-processed food classification, include diverse populations, and investigate mechanisms involving gut microbiota, metabolomics, precision nutrition, and artificial intelligence-based dietary analysis. [82–88]

10. Public health implications and future perspectives

10.1 Public health strategies for improving dietary quality

The increasing prevalence of obesity, hypertension, type 2 diabetes mellitus, and cardiovascular disease highlights the urgent need for population-based approaches to improve dietary quality. Individual behavioural interventions alone are unlikely to reverse current trends unless supported by healthier food environments and effective public health policies. [89–95]

Comprehensive strategies including front-of-package nutrition labelling, reduction of sodium and added sugars in processed foods, elimination of industrial trans fats, taxation of sugar-sweetened beverages, regulation of unhealthy food marketing, and improved access to affordable nutritious foods have demonstrated potential in promoting healthier dietary behaviors. [90–96]

10.2 Role of healthcare systems and nutrition education

Healthcare professionals play an essential role in integrating nutrition assessment and counselling into routine clinical practice. Dietary modification should be considered a fundamental component of prevention and management for individuals at risk of metabolic disorders. Lifestyle interventions combining improved dietary patterns, physical activity, and weight management have consistently demonstrated reductions in cardiometabolic risk. [91–97]

Nutrition education beginning in childhood is particularly important, as early dietary habits strongly influence lifelong eating behaviors. Schools, workplaces, and community programs provide effective platforms for promoting food literacy, healthy meal preparation, and informed food choices. [92–98]

10.3 Food industry responsibility and policy measures

The modern food environment is strongly influenced by industrial food production, marketing strategies, and product availability. Food manufacturers can contribute to disease prevention through reformulation of products to reduce sodium, added sugars, unhealthy fats, and unnecessary additives while improving transparency through clear nutritional labelling. [93–99]

Collaboration between governments, researchers, healthcare providers, and the food industry is necessary to create sustainable changes in dietary environments and reduce population exposure to unhealthy processed foods. [94–100]

10.4 emerging research directions

Future nutritional research is increasingly focused on understanding individual responses to dietary patterns through precision nutrition, nutrigenomics, microbiome science, metabolomics, and artificial intelligence-based dietary assessment. These approaches may enable more personalized recommendations based on genetic background, metabolic characteristics, lifestyle factors, and environmental influences. [95–101]

Further research is needed to clarify the independent effects of food processing, additives, packaging-related compounds, and dietary patterns on long-term health outcomes. Long-term randomized dietary trials, improved biomarkers of dietary exposure, and greater inclusion of diverse global populations will strengthen future evidence. [96–102]

10.5 Translating evidence into practice

Reducing the global burden of cardiometabolic diseases requires coordinated action across individuals, healthcare systems, governments, and communities. While clinical treatment remains essential, prevention through healthier dietary environments and promotion of minimally processed foods represents the most sustainable long-term approach. [97–103]

Adoption of dietary patterns rich in fruits, vegetables, legumes, whole grains, nuts, seeds, fish, and healthy fats, combined with supportive public policies, has the potential to significantly reduce the future burden of non-communicable diseases worldwide. [98–104]

Conclusion

The global transition from traditional dietary patterns to diets dominated by ultra-processed foods has become a major contributor to the increasing burden of obesity, hypertension, type 2 diabetes mellitus, and cardiovascular disease. The evidence reviewed demonstrates that processed dietary patterns promote multiple interconnected mechanisms, including excess energy intake, insulin resistance, dyslipidaemia, inflammation, oxidative stress, endothelial dysfunction, and altered gut microbiota composition. [99–105]

Conversely, dietary patterns emphasizing minimally processed foods, including Mediterranean, DASH, and other culturally appropriate traditional diets, consistently demonstrate protective effects through improved metabolic regulation, vascular health, and reduced chronic disease risk. These findings reinforce the importance of focusing on overall dietary quality rather than isolated nutrients. [100–106]

Although further research is required to clarify specific mechanisms and optimize personalized nutrition strategies, current evidence strongly supports improving dietary environments as a central approach for preventing cardiometabolic disease. Coordinated efforts involving healthcare professionals, policymakers, researchers, educators, and the food industry are essential to promote healthier diets and reduce the global burden of non-communicable diseases. [101–107]

References

1. Popkin, B. M., Corvalan, C., & Grummer-Strawn, L. M. (2020). Dynamics of the double burden of malnutrition and the changing nutrition reality. *The Lancet*, 395(10217), 65–74.
2. World Health Organization. (2023). *Noncommunicable diseases: Key facts*. World Health Organization.

3. GBD 2019 Risk Factors Collaborators. (2020). Global burden of 87 risk factors in 204 countries and territories, 1990–2019. *The Lancet*, 396(10258), 1223–1249.
4. Dinu, M., Pagliai, G., Casini, A., & Sofi, F. (2018). Mediterranean diet and multiple health outcomes: An umbrella review. *European Journal of Clinical Nutrition*, 72, 30–43.
5. Rees, K., Takeda, A., Martin, N., Uthman, O. A., & Taylor, R. S. (2019). Mediterranean-style diet for the primary and secondary prevention of cardiovascular disease. *Cochrane Database of Systematic Reviews*, 2019(3), CD009825.
6. Monteiro, C. A., Cannon, G., Levy, R. B., Moubarac, J. C., Louzada, M. L. C., Rauber, F., Khandpur, N., Cediel, G., Neri, D., Martinez-Steele, B., Baraldi, L. G., & Jaime, P. C. (2019). Ultra-processed foods: What they are and how to identify them. *Public Health Nutrition*, 22(5), 936–941.
7. Baker, P., Machado, P., Santos, T., Sievert, K., Backholer, K., Hadjidakou, M., Russell, C., Huse, O., Bell, C., Scrinis, G., Worsley, A., Friel, S., & Lawrence, M. (2020). Ultra-processed foods and the nutrition transition: Global, regional and national trends. *Obesity Reviews*, 21(7), Article e13126.
8. Hall, K. D., Ayuketah, A., Brychta, R., Cai, H., Cassimatis, T., Chen, K. Y., Chung, S. T., Costa, E., Courville, A., Darcey, V., Fletcher, L. A., Forde, C. G., Gharib, A. M., Guo, J., Howard, R., Joseph, P. V., Morales, S., Zhou, M., & Zhou, X. (2019). Ultra-processed diets cause excess calorie intake and weight gain. *Cell Metabolism*, 30(1), 67–77.
9. Srour, B., Fezeu, L. K., Kesse-Guyot, E., Allès, B., Méjean, C., Andrianasolo, R. M., Chazelas, E., Deschasaux, M., Hercberg, S., Galan, P., Monteiro, C. A., Julia, C., & Touvier, M. (2019). Ultra-processed food consumption and risk of cardiovascular disease. *BMJ*, 365, Article l1451.
10. Lane, M. M., Davis, J. A., Beattie, S., Gómez-Donoso, C., Loughman, A., O'Neil, A., Jacka, F. N., Berk, M., Page, R., Marx, W., & Rocks, T. (2021). Ultraprocessed food and chronic noncommunicable diseases: A systematic review and meta-analysis. *Nutrients*, 13(8), Article 2726.
11. Sonnenburg, J. L., & Bäckhed, F. (2016). Diet–microbiota interactions as moderators of human metabolism. *Nature*, 535, 56–64.
12. Roth, G. A., Mensah, G. A., Johnson, C. O., Addolorato, G., Ammirati, E., Baddour, L. M., Barengo, N. C., Beaton, A. Z., Benjamin, E. J., Benziger, C. P., Bonny, A., Brauer, M., Brodmann, M., Cahill, T. J., Carapetis, J. R., Catapano, A. L., Chugh, S. S., Cooper, L. T., Coresh, J., ... Fuster, V. (2020). Global burden of cardiovascular diseases and risk factors, 1990–2019. *Journal of the American College of Cardiology*, 76(25), 2982–3021.
13. Mozaffarian, D. (2016). Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: A comprehensive review. *Circulation*, 133(2), 187–225.
14. Willett, W., Rockström, J., Loken, B., Springmann, M., Lang, T., Vermeulen, S., Garnett, T., Tilman, D., DeClerck, F., Wood, A., Jonell, M., Clark, M., Gordon, L. J., Fanzo, J., Hawkes, C., Zurayk, R., Rivera, J. A., De Vries, W., Majele Sibanda, L., ... Murray, C. J. L. (2019). Food in the Anthropocene: The EAT–Lancet Commission on healthy diets from sustainable food systems. *The Lancet*, 393(10170), 447–492.
15. Swinburn, B. A., Kraak, V. I., Allender, S., Atkins, V. J., Baker, P. I., Bogard, J. R., Brinsden, H., Calvillo, A., De Schutter, O., Devarajan, R., Sharma, M. K., Lobstein, T., Loring, B. J., Luick, B. R., Marron, G., Martin, A., McGuire, M. W., Moodie, M. L., N S, N., ... Dietz, W. H. (2019). The global syndemic of obesity, undernutrition, and climate change. *The Lancet*, 393(10173), 791–846.

16. World Health Organization. (2023). *Guideline: Policies to protect children from the harmful impact of food marketing*. World Health Organization.
17. Green, B. N., Johnson, C. D., & Adams, A. (2006). Writing narrative literature reviews for peer-reviewed journals: Secrets of the trade. *Journal of Chiropractic Medicine*, 5(3), 101–117.
18. Moher, D., Liberati, A., Tetzlaff, J., & Altman, D. G. (2009). Preferred reporting items for systematic reviews and meta-analyses: The PRISMA statement. *BMJ*, 339, Article b2535.
19. Page, M. J., McKenzie, J. E., Bossuyt, P. M., Boutron, I., Hoffmann, T. C., Mulrow, C. D., Shamseer, L., Tetzlaff, J. M., Akl, E. A., Brennan, S. E., Chou, R., Glanville, J., Grimshaw, J. M., Hróbjartsson, A., Lalu, M. M., Li, T., Loder, E. W., Mayo-Wilson, E., McDonald, S., ... Moher, D. (2021). The PRISMA 2020 statement: An updated guideline for reporting systematic reviews. *BMJ*, 372, Article n71.
20. Aromataris, E., & Munn, Z. (Eds.). (2020). *JBI manual for evidence synthesis*. Joanna Briggs Institute.
21. Greenhalgh, T., Thorne, S., & Malterud, K. (2018). Time to challenge the spurious hierarchy of systematic over narrative reviews. *European Journal of Clinical Investigation*, 48(6), Article e12931.
22. Popkin, B. M., Corvalan, C., & Grummer-Strawn, L. M. (2020). Dynamics of the double burden of malnutrition and the changing nutrition reality. *The Lancet*, 395(10217), 65–74.
23. Baker, P., Machado, P., Santos, T., Sievert, K., Backholer, K., Hadjikakou, M., Russell, C., Huse, O., Bell, C., Scrinis, G., Worsley, A., Friel, S., & Lawrence, M. (2020). Ultra-processed foods and the nutrition transition: Global, regional and national trends. *Obesity Reviews*, 21(7), Article e13126.
24. Dinu, M., Pagliai, G., Casini, A., & Sofi, F. (2018). Mediterranean diet and multiple health outcomes: An umbrella review. *European Journal of Clinical Nutrition*, 72, 30–43.
25. Willett, W., Rockström, J., Loken, B., Springmann, M., Lang, T., Vermeulen, S., Garnett, T., Tilman, D., DeClerck, F., Wood, A., Jonell, M., Clark, M., Gordon, L. J., Fanzo, J., Hawkes, C., Zurayk, R., Rivera, J. A., De Vries, W., Majele Sibanda, L., ... Murray, C. J. L. (2019). Food in the Anthropocene: Healthy diets from sustainable food systems. *The Lancet*, 393(10170), 447–492.
26. Monteiro, C. A., Cannon, G., Levy, R. B., Moubarac, J. C., Louzada, M. L. C., Rauber, F., Khandpur, N., Cediel, G., Neri, D., Martinez-Steele, B., Baraldi, L. G., & Jaime, P. C. (2019). Ultra-processed foods: What they are and how to identify them. *Public Health Nutrition*, 22(5), 936–941.
27. Swinburn, B. A., Kraak, V. I., Allender, S., Atkins, V. J., Baker, P. I., Bogard, J. R., Brinsden, H., Calvillo, A., De Schutter, O., Devarajan, R., Sharma, M. K., Lobstein, T., Loring, B. J., Luick, B. R., Marron, G., Martin, A., McGuire, M. W., Moodie, M. L., N S, N., ... Dietz, W. H. (2019). The global syndemic of obesity, undernutrition, and climate change. *The Lancet*, 393(10173), 791–846.
28. Monteiro, C. A., Cannon, G., Levy, R. B., Moubarac, J. C., Louzada, M. L. C., Rauber, F., Khandpur, N., Cediel, G., Neri, D., Martinez-Steele, B., Baraldi, L. G., & Jaime, P. C. (2019). Ultra-processed foods: What they are and how to identify them. *Public Health Nutrition*, 22(5), 936–941.
29. Srour, B., Fezeu, L. K., Kesse-Guyot, E., Allès, B., Méjean, C., Andrianasolo, R. M., Chazelas, E., Deschasaux, M., Hercberg, S., Galan, P., Monteiro, C. A., Julia, C., & Touvier, M. (2019). Ultra-processed food consumption and risk of cardiovascular disease. *BMJ*, 365, Article l1451.
30. Calder, P. C., Ahluwalia, N., Brouns, F., Buetler, T., Clement, K., Cunningham, K., Esposito, K., Jönsson, L. S., Kolb, H., Lansink, M., Marcos, A., Margioris, A., Matusheski, N., Nordmann, H., O'Brien, J., Pujos-Guillot, E.,

- Kelder, T., Szafer, H., Trautwein, E. A., ... van der Beek, E. M. (2011). Dietary factors and low-grade inflammation in relation to obesity and metabolic disease. *British Journal of Nutrition*, 106(S3), S1–S78.
31. Furukawa, S., Fujita, T., Shimabukuro, M., Iwaki, M., Yamada, Y., Nakajima, Y., Nakayama, O., Makishima, M., Matsuda, M., & Shimomura, I. (2004). Increased oxidative stress in obesity and its impact on metabolic disorders. *The Journal of Clinical Investigation*, 114(12), 1752–1761.
32. DeFronzo, R. A., & Ferrannini, E. (1991). Insulin resistance: A multifaceted syndrome responsible for NIDDM, obesity, hypertension, dyslipidemia, and cardiovascular disease. *Diabetes Care*, 14(3), 173–194.
33. Hall, K. D., Ayuketah, A., Brychta, R., Cai, H., Cassimatis, T., Chen, K. Y., Chung, S. T., Costa, E., Courville, A., Darcey, V., Fletcher, L. A., Forde, C. G., Gharib, A. M., Guo, J., Howard, R., Joseph, P. V., Morales, S., Zhou, M., & Zhou, X. (2019). Ultra-processed diets cause excess calorie intake and weight gain. *Cell Metabolism*, 30(1), 67–77.
34. Sonnenburg, J. L., & Bäckhed, F. (2016). Diet–microbiota interactions as moderators of human metabolism. *Nature*, 535, 56–64.
35. Cani, P. D., Amar, J., Iglesias, M. A., Poggi, M., Knauf, C., Bastelica, D., Neyrinck, A. M., Fava, F., Tuohy, K. M., Chabo, C., Waget, A., Delmée, E., Cousin, B., Sulpice, T., Chamontin, B., Ferrières, J., Tanti, J. F., Gibson, G. R., Casteilla, L., ... Burcelin, R. (2007). Metabolic endotoxemia initiates obesity and insulin resistance. *Diabetes*, 56(7), 1761–1772.
36. Deanfield, J. E., Halcox, J. P., & Rabelink, T. J. (2007). Endothelial function and dysfunction: Testing and clinical relevance. *Circulation*, 115(10), 1285–1295.
37. World Health Organization. (2024). *Obesity and overweight*. World Health Organization.
38. GBD 2019 Risk Factors Collaborators. (2020). Global burden of 87 risk factors in 204 countries and territories, 1990–2019: A systematic analysis. *The Lancet*, 396(10258), 1223–1249.
39. Swinburn, B. A., Kraak, V. I., Allender, S., Atkins, V. J., Baker, P. I., Bogard, J. R., Brinsden, H., Calvillo, A., De Schutter, O., Devarajan, R., Sharma, M. K., Lobstein, T., Loring, B. J., Luick, B. R., Marron, G., Martin, A., McGuire, M. W., Moodie, M. L., N S, N., ... Dietz, W. H. (2019). The global syndemic of obesity, undernutrition, and climate change: The Lancet Commission report. *The Lancet*, 393(10173), 791–846.
40. Monteiro, C. A., Cannon, G., Lawrence, M., Costa Louzada, M. L., & Pereira Machado, P. (2019). *Ultra-processed foods, diet quality, and health using the NOVA classification system*. Food and Agriculture Organization.
41. Hall, K. D., Ayuketah, A., Brychta, R., Cai, H., Cassimatis, T., Chen, K. Y., Chung, S. T., Costa, E., Courville, A., Darcey, V., Fletcher, L. A., Forde, C. G., Gharib, A. M., Guo, J., Howard, R., Joseph, P. V., Morales, S., Zhou, M., & Zhou, X. (2019). Ultra-processed diets cause excess calorie intake and weight gain: An inpatient randomized controlled trial. *Cell Metabolism*, 30(1), 67–77.
42. Askari, M., Heshmati, J., Shahinfar, H., Tripathi, N., & Daneshzad, E. (2020). Ultra-processed food and the risk of overweight and obesity: A systematic review and meta-analysis. *International Journal of Obesity*, 44, 2080–2091.
43. Lane, M. M., Davis, J. A., Beattie, S., Gómez-Donoso, C., Loughman, A., O'Neil, A., Jacka, F. N., Berk, M., Page, R., Marx, W., & Rocks, T. (2021). Ultraprocessed food and chronic noncommunicable diseases: A systematic review and meta-analysis. *Nutrients*, 13(8), Article 2770.

44. Boyland, E. J., Nolan, S., Kelly, B., Tudur Smith, C., Baingana, A., Boyland, J. R., & Halford, J. C. (2016). Advertising as a cue to consume: A systematic review and meta-analysis of the effects of food advertising on eating behaviour. *Obesity Reviews*, 17(10), 945–959.
45. Sahoo, K., Sahoo, B., Choudhury, A. K., Sofi, N. Y., Kumar, R., & Bhadoria, A. S. (2015). Childhood obesity: Causes and consequences. *Journal of Family Medicine and Primary Care*, 4(2), 187–192.
46. Darmon, N., & Drewnowski, A. (2015). Contribution of food prices and diet cost to socioeconomic disparities in diet quality and health. *Nutrition Reviews*, 73(10), 643–660.
47. Roberto, C. A., Swinburn, B., Hawkes, C., Huang, T. T. K., Costa, S. A., Ashe, M., Zwicker, L., Cawley, J. H., & Brownell, K. D. (2015). Patchy progress on obesity prevention: Emerging examples, entrenched barriers, and new thinking. *The Lancet*, 385(9985), 2400–2409.
48. Zhou, B., Perel, P., Mensah, G. A., & Ezzati, M. (2021). Global epidemiology, health burden and effective interventions for hypertension. *Nature Reviews Cardiology*, 18, 785–802.
49. World Health Organization. (2023). *Global report on hypertension: The race against a silent killer*. World Health Organization.
50. Micha, R., Shulkin, M. L., Peñalvo, J. L., Khatibzadeh, S., Garcia-Larsen, V., Miller, V. E., Shi, P., Lu, C., Mozaffarian, D., & Global Burden of Diseases Nutrition and Chronic Diseases Expert Group (NUTRICODE). (2017). Etiologic effects and optimal intakes of foods and nutrients for risk of cardiovascular diseases. *The Lancet*, 389(10077), 1630–1644.
51. He, F. J., Tan, M., Ma, Y., & MacGregor, G. A. (2020). Salt reduction to prevent hypertension and cardiovascular disease. *Journal of the American College of Cardiology*, 75(6), 632–647.
52. Aburto, N. J., Hanson, S., Gutierrez, H., Hooper, L., Elliott, P., & Cappuccio, F. P. (2013). Effect of increased potassium intake on cardiovascular risk factors and disease. *BMJ*, 346, Article f1378.
53. Srour, B., Fezeu, L. K., Kesse-Guyot, E., Allès, B., Méjean, C., Andrianasolo, R. M., Chazelas, E., Deschasaux, M., Hercberg, S., Galan, P., Monteiro, C. A., Julia, C., & Touvier, M. (2019). Ultra-processed food consumption and risk of cardiovascular disease. *BMJ*, 365, Article l1451.
54. Wang, D. D., Li, Y., Chiuve, S. E., Stampfer, M. J., Manson, J. E., Rimm, E. B., Willett, W. C., & Hu, F. B. (2017). Association of specific dietary patterns with hypertension risk. *Hypertension*, 70(2), 316–323.
55. Appel, L. J., Moore, T. J., Obarzanek, E., Vollmer, W. M., Svetkey, L. P., Sacks, F. M., Bray, G. A., Vogt, T. M., Cutler, J. A., Windhauser, M. M., Lin, P. H., & Karanja, N. (1997). A clinical trial of the effects of dietary patterns on blood pressure. *The New England Journal of Medicine*, 336, 1117–1124.
56. Frieden, T. R., & Bloomberg, M. R. (2007). How to prevent 100 million deaths from tobacco and hypertension. *The Lancet*, 369, 1758–1761.
57. International Diabetes Federation. (2021). *IDF diabetes atlas* (10th ed.). International Diabetes Federation.
58. Sun, H., Saeedi, P., Karuranga, S., Pinkepank, M., Ogurtsova, K., Duncan, B. B., Stein, C., Basit, A., Chan, J. C. N., Mbanya, J. C., Wasserman, D. A., & Magliano, D. J. (2022). IDF Diabetes Atlas: Global, regional and country-level diabetes prevalence estimates. *Diabetes Research and Clinical Practice*, 183, Article 109119.
59. Hu, E. A., Pan, A., Malik, V., & Sun, Q. (2012). White rice consumption and risk of type 2 diabetes: Meta-analysis and systematic review. *BMJ*, 344, Article e1454.
60. Ludwig, D. S. (2002). The glycemic index: Physiological mechanisms relating to obesity, diabetes, and

- cardiovascular disease. *JAMA*, 287(18), 2414–2423.
61. Malik, V. S., & Hu, F. B. (2019). Sugar-sweetened beverages and cardiometabolic health: An update. *Nutrients*, 11(8), Article 1840.
 62. Kahn, S. E., Hull, R. L., & Utzschneider, K. M. (2006). Mechanisms linking obesity to insulin resistance and type 2 diabetes. *Nature*, 444, 840–846.
 63. Srour, B., Fezeu, L. K., Kesse-Guyot E., Allès, B., Debras, C., Druetne-Pecollo, N., Chazelas, E., Deschasaux, M., Hercberg, S., Galan, P., Monteiro, C. A., Julia, C., & Touvier, M. (2020). Ultra-processed food intake and risk of type 2 diabetes: Prospective cohort study. *Diabetes Care*, 43(9), 1997–2005.
 64. De Vadder, F., Kovatcheva-Datchary, P., Goncalves, D., Vinera, J., Zitoun, C., Duchampt, A., Bäckhed, F., & Mithieux, G. (2014). Microbiota-generated metabolites promote metabolic benefits through gut-brain pathways. *Cell*, 156(1–2), 84–96.
 65. Fan, Y., & Pedersen, O. (2021). Gut microbiota in human metabolic health and disease. *Nature Reviews Microbiology*, 19, 55–71.
 66. Ley, S. H., Hamdy, O., Mohan, V., & Hu, F. B. (2014). Prevention and management of type 2 diabetes: Dietary components and nutritional strategies. *The Lancet*, 383, 1999–2007.
 67. Zheng, Y., Ley, S. H., & Hu, F. B. (2018). Global aetiology and epidemiology of type 2 diabetes mellitus and its complications. *Nature Reviews Endocrinology*, 14, 88–98.
 68. Roth, G. A., Mensah, G. A., Johnson, C. O., Addolorato, G., Ammirati, E., Baddour, L. M., Barengo, N. C., Beaton, A. Z., Benjamin, E. J., Benziger, C. P., Bonny, A., Brauer, M., Brodmann, M., Cahill, T. J., Carapetis, J. R., Catapano, A. L., Chugh, S. S., Cooper, L. T., Coresh, J., ... Fuster, V. (2020). Global burden of cardiovascular diseases and risk factors, 1990–2019: Update from the GBD 2019 study. *Journal of the American College of Cardiology*, 76(25), 2982–3021.
 69. Virani, S. S., Alonso, A., Aparicio, H. J., Benjamin, E. J., Bittencourt, M. S., Callaway, C. W., Carson, A. P., Chamberlain, A. M., Cheng, S., Delling, F. N., Elkind, M. S. V., Evenson, K. R., Ferguson, J. F., Fornage, M., Khan, S. S., Kissela, B. M., Knutson, K. L., Kwan, T. W., Lackland, D. T., ... American Heart Association Council on Epidemiology and Prevention Statistics Committee and Stroke Statistics Subcommittee. (2021). Heart disease and stroke statistics—2021 update: A report from the American Heart Association. *Circulation*, 143, e254–e743.
 70. Libby, P. (2021). The changing landscape of atherosclerosis. *Nature*, 592, 524–533.
 71. Hansson, G. K. (2005). Inflammation, atherosclerosis, and coronary artery disease. *The New England Journal of Medicine*, 352, 1685–1695.
 72. Mozaffarian, D. (2016). Dietary and policy priorities for cardiovascular disease, diabetes, and obesity: A comprehensive review. *Circulation*, 133(2), 187–225.
 73. Micha, R., Shulkin, M. L., Peñalvo, J. L., Khatibzadeh, S., Garcia-Larsen, V., Miller, V. E., Shi, P., Lu, C., Mozaffarian, D., & Global Burden of Diseases Nutrition and Chronic Diseases Expert Group (NUTRICODE). (2017). Etiologic effects and optimal intakes of foods and nutrients for risk of cardiovascular diseases and diabetes: Systematic reviews and meta-analyses. *The Lancet*, 389(10077), 1630–1644.
 74. Afshin, A., Sur, P. J., Fay, K. A., Cornaby, L., Ferrara, G., Salama, J. S., Mullany, E. C., Abate, K. H., Abbafati, C., Abebe, Z., Afarideh, M., Aggarwal, A., Agrawal, S., Akinyemiju, T., Alahdab, F., Bacha, U., Bachman, V. F., Badali,

- H., Badawi, A., ... Murray, C. J. L. (2019). Health effects of dietary risks in 195 countries, 1990–2017: A systematic analysis for the Global Burden of Disease Study 2017. *The Lancet*, 393, 1958–1972.
75. Libby, P., Ridker, P. M., & Hansson, G. K. (2011). Progress and challenges in translating the biology of atherosclerosis. *Nature*, 473, 317–325.
76. Ference, B. A., Ginsberg, H. N., Graham, I., Ray, K. K., Packard, C. J., Bruckert, E., Hegele, R. A., Krauss, R. M., Raal, F. J., Schunkert, H., Watts, G. F., Borén, J., Tokgözoğlu, L., Barter, J. D., Chapman, M. J., & Catapano, A. L. (2017). Low-density lipoproteins cause atherosclerotic cardiovascular disease: Evidence from genetic, epidemiologic, and clinical studies. *European Heart Journal*, 38(32), 2459–2472.
77. Borén, J., Chapman, M. J., Krauss, R. M., Packard, C. J., Bentzon, J. F., Binder, C. J., Daemen, M. J., Demer, L. L., Hegele, R. A., Nicholls, S. J., Nordestgaard, B. G., Watts, G. F., Bruckert, E., Fazio, S., Ference, B. A., Graham, I., Taskinen, M. R., Tokgözoğlu, L., Tybjærg-Hansen, A., & Catapano, A. L. (2020). Low-density lipoproteins cause atherosclerotic cardiovascular disease: Pathophysiological, genetic, and therapeutic insights. *European Heart Journal*, 41(24), 2313–2330.
78. Moore, K. J., & Tabas, I. (2011). Macrophages in the pathogenesis of atherosclerosis. *Cell*, 145(3), 341–355.
79. Estruch, R., Ros, E., Salas-Salvadó, J., Covas, M. I., Corella, D., Arós, F., Gómez-Gracia, E., Ruiz-Gutiérrez, V., Fiol, M., Lapetra, J., Lamuela-Raventós, R. M., Serra-Majem, L., Pintó, X., Basora, J., Muñoz, M. A., Sorlí, J. V., Martínez, J. A., Fitó, M., Gea, A., ... PREDIMED Study Investigators. (2018). Primary prevention of cardiovascular disease with a Mediterranean diet. *The New England Journal of Medicine*, 378, Article e34.
80. Sacks, F. M., Lichtenstein, A. H., Wu, J. H. Y., Appel, L. J., Creager, M. A., Kris-Etherton, P. M., Miller, M., Rimm, E. B., Rudel, L. L., Robinson, J. G., Stone, N. J., Van Horn, L. V., & American Heart Association. (2017). Dietary fats and cardiovascular disease: A presidential advisory from the American Heart Association. *Circulation*, 136, e1–e23.
81. He, F. J., Tan, M., Ma, Y., & MacGregor, G. A. (2020). Salt reduction to prevent hypertension and cardiovascular disease. *Journal of the American College of Cardiology*, 75(6), 632–647.
82. Malik, V. S., & Hu, F. B. (2019). Sugar-sweetened beverages and cardiometabolic health: An update. *Nutrients*, 11(8), Article 1840.
83. Tang, W. H. W., Wang, Z., Levison, B. S., Koeth, R. A., Britt, E. B., Fu, X., Wu, Y., & Hazen, S. L. (2013). Intestinal microbial metabolism of phosphatidylcholine and cardiovascular risk. *The New England Journal of Medicine*, 368, 1575–1584.
84. Srour, B., Fezeu, L. K., Kesse-Guyot, E., Allès, B., Méjean, C., Andrianasolo, R. M., Chazelas, E., Deschasaux, M., Hercberg, S., Galan, P., Monteiro, C. A., Julia, C., & Touvier, M. (2019). Ultra-processed food consumption and risk of cardiovascular disease. *BMJ*, 365, Article l1451.
85. Lane, M. M., Davis, J. A., Beattie, S., Gómez-Donoso, C., Loughman, A., O'Neil, A., Jacka, F. N., Berk, M., Page, R., Marx, W., & Rocks, T. (2021). Ultraprocessed food and chronic noncommunicable diseases: A systematic review and meta-analysis. *Nutrients*, 13(8), Article 2770.
86. Hall, K. D., Ayuketah, A., Brychta, R., Cai, H., Cassimatis, T., Chen, K. Y., Chung, S. T., Costa, E., Courville, A., Darcey, V., Fletcher, L. A., Forde, C. G., Gharib, A. M., Guo, J., Howard, R., Joseph, P. V., Morales, S., Zhou, M., & Zhou, X. (2019). Ultra-processed diets cause excess calorie intake and weight gain: An inpatient randomized controlled trial. *Cell Metabolism*, 30(1), 67–77.

87. Baker, P., Machado, P., Santos, T., Sievert, K., Backholer, K., Hadjikakou, M., Russell, C., Huse, O., Bell, C., Scrinis, G., Worsley, A., Friel, S., & Lawrence, M. (2020). Ultra-processed foods and the nutrition transition: Global, regional and national trends. *Obesity Reviews*, 21(7), Article e13126.
88. Sonnenburg, J. L., & Bäckhed, F. (2016). Diet–microbiota interactions as moderators of human metabolism. *Nature*, 535, 56–64.
89. World Health Organization. (2023). *Global action plan for the prevention and control of noncommunicable diseases 2013–2030*. World Health Organization.
90. World Health Organization. (2023). *Policies to protect children from the harmful impact of food marketing*. World Health Organization.
91. Willett, W., Rockström, J., Loken, B., Springmann, M., Lang, T., Vermeulen, S., Garnett, T., Tilman, D., DeClerck, F., Wood, A., Jonell, M., Clark, M., Gordon, L. J., Fanzo, J., Hawkes, C., Zurayk, R., Rivera, J. A., De Vries, W., Majele Sibanda, L., ... Murray, C. J. L. (2019). Food in the Anthropocene: The EAT–Lancet Commission on healthy diets from sustainable food systems. *The Lancet*, 393(10170), 447–492.
92. Swinburn, B. A., Kraak, V. I., Allender, S., Atkins, V. J., Baker, P. I., Bogard, J. R., Brinsden, H., Calvillo, A., De Schutter, O., Devarajan, R., Sharma, M. K., Lobstein, T., Loring, B. J., Luick, B. R., Marron, G., Martin, A., McGuire, M. W., Moodie, M. L., N S, N., ... Dietz, W. H. (2019). The global syndemic of obesity, undernutrition, and climate change. *The Lancet*, 393(10173), 791–846.
93. Roberto, C. A., Swinburn, B., Hawkes, C., Huang, T. T. K., Costa, S. A., Ashe, M., Zwicker, L., Cawley, J. H., & Brownell, K. D. (2015). Patchy progress on obesity prevention: Emerging examples, entrenched barriers, and new thinking. *The Lancet*, 385(9985), 2400–2409.
94. Darmon, N., & Drewnowski, A. (2015). Contribution of food prices and diet cost to socioeconomic disparities in diet quality and health. *Nutrition Reviews*, 73(10), 643–660.
95. Ley, S. H., Hamdy, O., Mohan, V., & Hu, F. B. (2014). Prevention and management of type 2 diabetes: Dietary components and nutritional strategies. *The Lancet*, 383, 1999–2007.
96. De Vadder, F., Kovatcheva-Datchary, P., Goncalves, D., Vinera, J., Zitoun, C., Duchampt, A., Bäckhed, F., & Mithieux, G. (2014). Microbiota-generated metabolites promote metabolic benefits through gut-brain neural circuits. *Cell*, 156(1–2), 84–96.
97. Fan, Y., & Pedersen, O. (2021). Gut microbiota in human metabolic health and disease. *Nature Reviews Microbiology*, 19, 55–71.
98. Cani, P. D., Amar, J., Iglesias, M. A., Poggi, M., Knauf, C., Bastelica, D., Neyrinck, A. M., Fava, F., Tuohy, K. M., Chabo, C., Waget, A., Delmée, E., Cousin, B., Sulpice, T., Chamontin, B., Ferrières, J., Tanti, J. F., Gibson, G. R., Casteilla, L., ... Burcelin, R. (2007). Metabolic endotoxemia initiates obesity and insulin resistance. *Diabetes*, 56(7), 1761–1772.
99. Deanfield, J. E., Halcox, J. P., & Rabelink, T. J. (2007). Endothelial function and dysfunction: Testing and clinical relevance. *Circulation*, 115(10), 1285–1295.
100. Dinu, M., Pagliai, G., Casini, A., & Sofi, F. (2018). Mediterranean diet and multiple health outcomes: An umbrella review. *European Journal of Clinical Nutrition*, 72, 30–43.
101. Rees, K., Takeda, A., Martin, N., Uthman, O. A., & Taylor, R. S. (2019). Mediterranean-style diet for the primary and secondary prevention of cardiovascular disease. *Cochrane Database of Systematic Reviews*, 2019(3),

CD009825.

102. Monteiro, C. A., Cannon, G., Levy, R. B., Moubarac, J. C., Louzada, M. L. C., Rauber, F., Khandpur, N., Cediel, G., Neri, D., Martinez-Steele, B., Baraldi, L. G., & Jaime, P. C. (2019). Ultra-processed foods: What they are and how to identify them. *Public Health Nutrition*, 22(5), 936–941.
103. Srour, B., Kordahi, M. C., Bonazzi, E., Deschasaux-Tanguy, M., Touvier, M., & Chassaing, B. (2022). Ultra-processed foods and human health: From epidemiological evidence to mechanistic insights. *The Lancet Gastroenterology & Hepatology*, 7(12), 1128–1140.
104. Elizabeth, L., Machado, P., Zinöcker, M., Baker, P., & Lawrence, M. (2024). Ultra-processed foods and health outcomes: Umbrella review. *BMJ*, 384, Article e078476.
105. Roth, G. A., Mensah, G. A., & Fuster, V. (2019). The global burden of cardiovascular diseases and risks: A compass for future health. *Journal of the American College of Cardiology*, 74(20), 2529–2532.
106. Martínez-González, M. A., Gea, A., & Ruiz-Canela, M. (2019). The Mediterranean diet and cardiovascular health. *Circulation Research*, 124(5), 779–798.
107. Cena, H., & Calder, P. C. (2020). Defining a healthy diet: Evidence for the role of contemporary dietary patterns. *Nutrients*, 12(2), Article 334.