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FOOD PRESERVATIVES AND HUMAN HEALTH: MOLECULAR MECHANISMS, CLINICAL CONSEQUENCES, AND PUBLIC HEALTH IMPLICATIONS: A NARRATIVE REVIEW

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ABSTRACT

Introduction: Food preservatives are widely used in modern food systems to prevent microbial spoilage and extend shelf life. However, increasing evidence suggests that their long-term consumption may have implications for human health, particularly in relation to chronic non-communicable diseases. Common preservatives, including sodium benzoate, sulfites, and nitrate/nitrite compounds, may disrupt redox homeostasis, induce oxidative stress, alter gut microbiota, and activate pro-inflammatory pathways such as nuclear factor kappa B (NF- κ B).

Aim of the Study: This narrative review aims to synthesize current evidence on the biological effects of selected food preservatives, focusing on molecular mechanisms, clinical relevance, and public health implications.

Materials and Methods: A narrative literature review was conducted using PubMed, Scopus, and Web of Science. Additional sources included reports from international organizations. Keywords such as “food preservatives,” “oxidative stress,” “gut microbiota,” and “inflammation” were used. Peer-reviewed articles published mainly between 2010 and 2026 were included.

Results: Evidence indicates that preservatives may increase reactive oxygen species production and impair antioxidant defenses, leading to oxidative stress and cellular damage. They may also alter gut microbiota composition, reduce microbial diversity, and impair intestinal barrier function, promoting systemic inflammation. Activation of inflammatory pathways, including NF- κ B signaling, has been reported. Epidemiological studies suggest an association between high intake of preservative-rich ultra-processed foods and an increased risk of metabolic disorders, cardiovascular diseases, and cancer.

Conclusion: Food preservatives may affect human health through interconnected mechanisms involving oxidative stress, gut dysbiosis, and inflammation. These findings highlight the need for integrative risk assessment approaches and dietary strategies reducing ultra-processed food consumption.

KEYWORDS

Food Preservatives, Oxidative Stress, Gut Microbiota, Inflammation, Metabolic Disorders, Public Health

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Introduction

The increasing global demand for processed and long-shelf-life food products has led to the widespread use of food preservatives as essential components of modern food systems (World Health Organization, 2023). These substances play a critical role in preventing microbial contamination, reducing food waste, and ensuring food safety across complex distribution networks. Consequently, exposure to food preservatives has become continuous and population-wide, particularly in industrialized societies characterized by a high intake of ultra-processed foods.

Despite their technological benefits and regulatory approval within established safety limits, growing scientific attention has been directed toward the potential biological effects of food preservatives beyond their intended function. Increasing evidence suggests that certain preservatives may interfere with fundamental cellular processes, including redox homeostasis and inflammatory signaling pathways. These disturbances are increasingly being investigated as potential contributors to the pathogenesis of chronic non-communicable diseases.

From a biochemical perspective, preservatives such as sodium benzoate, sulfites, and nitrate/nitrite compounds have been associated with increased production of reactive oxygen species and alterations in endogenous antioxidant defense systems (Minamiyama et al., 2021; Asejeje et al., 2023). These effects may contribute to oxidative stress, a mechanism implicated in cellular damage, genomic instability, and activation of pro-inflammatory pathways, including nuclear factor kappa B (NF- κ B) (Soliman et al., 2022). In parallel, emerging research indicates that food preservatives may influence the composition and metabolic activity of

the gut microbiota, contributing to dysbiosis, impaired intestinal barrier function, and systemic low-grade inflammation (Zhou et al., 2023; Singh et al., 2024).

These molecular and physiological alterations may have clinical relevance, as they have been associated with metabolic disorders and other chronic conditions (Dai et al., 2024; Lane et al., 2024). Importantly, these effects should be interpreted in the context of long-term, low-dose exposure and cumulative intake within complex dietary patterns.

The research problem addressed in this study concerns the need to better understand the relationship between commonly used food preservatives and human health, particularly in terms of their underlying molecular mechanisms and broader clinical and public health implications. Therefore, the aim of this narrative review is to critically synthesize current scientific evidence regarding the biological effects of selected food preservatives, with a focus on mechanistic pathways, clinical relevance, and implications for population health.

Methodology

This study was designed as a narrative literature review aimed at synthesizing current scientific evidence on the effects of food preservatives on human health, with particular emphasis on molecular mechanisms and their clinical and public health implications.

A literature search was conducted using electronic databases, including PubMed, Scopus, and Web of Science. Additional sources included reports and publications from international organizations such as the World Health Organization (WHO). The search strategy was based on combinations of keywords such as “food preservatives”, “oxidative stress”, “gut microbiota”, “inflammation”, “metabolic disorders”, and “food additives”.

The selection of studies was guided by predefined inclusion criteria, which comprised peer-reviewed articles published in English that addressed the biological effects of commonly used food preservatives, including sodium benzoate, sulfites, and nitrate/nitrite compounds. Both experimental and clinical studies, as well as systematic reviews and meta-analyses, were included. Particular emphasis was placed on recent publications (primarily from 2010 onwards), with priority given to studies published between 2020 and 2026.

The selection process was narrative and interpretative rather than systematic. Studies were chosen based on their relevance to the research objectives, scientific quality, and contribution to understanding key mechanistic pathways, including oxidative stress, gut microbiota alterations, and inflammatory signaling.

Exclusion criteria included studies not directly related to human health, articles lacking methodological rigor, and publications focusing exclusively on technological or industrial aspects of food preservation without addressing biological effects.

Given the narrative nature of this review, no formal systematic review protocol (e.g., PRISMA) or quantitative meta-analysis was applied. Instead, emphasis was placed on integrating mechanistic insights with clinical and epidemiological evidence in order to provide a comprehensive and interdisciplinary perspective on the topic.

Results

1. Food Preservatives and Oxidative Stress

A growing body of evidence suggests that commonly used food preservatives may disrupt cellular redox homeostasis. Substances such as sodium benzoate, sulfites, and nitrite-derived compounds have been shown to increase the intracellular production of reactive oxygen species (ROS), including superoxide anion, hydrogen peroxide, and hydroxyl radicals (Minamiyama et al., 2021; Soliman et al., 2022). Excessive accumulation of ROS may lead to oxidative stress, defined as an imbalance between pro-oxidant and antioxidant systems.

At the molecular level, oxidative stress induced by food preservatives may result in lipid peroxidation, particularly affecting polyunsaturated fatty acids within cellular membranes. This process generates secondary reactive products such as malondialdehyde and 4-hydroxynonenal, which can further damage cellular structures (Minamiyama et al., 2021). Additionally, oxidative modifications of proteins may alter enzymatic activity and structural integrity, while oxidative DNA damage, including strand breaks and base modifications, may contribute to mutagenesis and carcinogenesis (Mafe & Büsselberg, 2025).

Mitochondria appear to be a potential target of preservative-induced oxidative stress. Experimental evidence suggests that preservative exposure may impair mitochondrial function, including alterations in energy metabolism and membrane potential (Minamiyama et al., 2021). These alterations may further amplify ROS generation, creating a self-perpetuating cycle of oxidative damage.

Exposure to preservatives has also been associated with disruption of mitochondrial-related processes, leading to increased ROS formation (Soliman et al., 2022). In this context, mitochondrial dysfunction may contribute to cellular injury and apoptosis-related processes. Chronic activation of these mechanisms may contribute to tissue damage, particularly in metabolically active organs such as the liver, brain, and intestinal epithelium (Minamiyama et al., 2021).

Furthermore, exposure to food preservatives has been associated with decreased activity of endogenous antioxidant defense systems, including superoxide dismutase (SOD), catalase, and glutathione peroxidase. Depletion of reduced glutathione (GSH), a key intracellular antioxidant, has also been reported (Asejeje et al., 2023). Since GSH is essential for maintaining cellular redox balance, its reduction may further increase susceptibility to oxidative injury.

Importantly, certain preservatives such as sodium benzoate have been shown to exacerbate oxidative stress and inflammatory responses, particularly under conditions of pre-existing physiological stress, such as lipopolysaccharide-induced inflammation (Asejeje et al., 2023). Similarly, nitrite exposure has been linked to oxidative imbalance through modulation of key regulatory pathways, including the Nrf2/HO-1 and NF- κ B signaling pathways (Soliman et al., 2022).

Emerging evidence also indicates that preservatives can induce oxidative stress responses at the microbial level, suggesting that oxidative mechanisms may contribute not only to host cellular damage but also to alterations in microbial physiology (Holden et al., 2025).

Overall, these findings suggest that oxidative stress may represent an important mechanistic pathway through which food preservatives exert biological effects and may contribute to broader pathological processes, including inflammation and carcinogenesis (Mafe & Büsselberg, 2025).

2. Effects on Gut Microbiota and Intestinal Barrier Function

Recent studies have highlighted the potential impact of food preservatives and additives on the gut microbiota, a complex and dynamic ecosystem that plays a fundamental role in host metabolism, immune regulation, and intestinal barrier integrity (Li, Qu, et al., 2024; Singh et al., 2024). Due to their antimicrobial properties, preservatives may non-selectively affect both pathogenic and beneficial microbial populations, potentially leading to microbial imbalance and alterations in diversity (Zhou et al., 2023).

Alterations in microbial composition, including shifts in dominant bacterial taxa and decreased microbial diversity, have been associated with exposure to food additives (Bevilacqua et al., 2025; Seto et al., 2025). In particular, reductions in beneficial bacteria involved in the production of short-chain fatty acids (SCFAs), such as butyrate-producing species, have been reported (Li, Qu, et al., 2024; Singh et al., 2024). SCFAs are essential for maintaining intestinal epithelial integrity, modulating immune responses, and regulating metabolic pathways.

Among SCFAs, butyrate plays a particularly important role as the primary energy source for colonocytes and a key regulator of mucosal homeostasis. A reduction in butyrate-producing bacteria may be associated with impaired epithelial regeneration, weakened intestinal barrier function, and a greater propensity for inflammatory responses (Seto et al., 2025). Furthermore, SCFAs are involved in metabolic regulation through signaling pathways associated with G-protein-coupled receptors and epigenetic mechanisms, linking gut microbiota composition with systemic metabolic health (Li, Qu, et al., 2024).

Disruption of the gut microbiota may also lead to increased intestinal permeability, commonly referred to as “leaky gut.” This condition is characterized by impaired tight junction function and may facilitate the translocation of microbial components, including lipopolysaccharides (LPS), into systemic circulation (Besedin et al., 2024). The presence of LPS in the bloodstream has been associated with chronic low-grade inflammation and metabolic disturbances.

The integrity of the intestinal barrier depends on the coordinated function of tight junction proteins such as occludin, claudins, and zonula occludens proteins. Food preservatives may influence these structures both directly and indirectly through alterations in microbial composition and metabolite production (Zhou et al., 2023; Seto et al., 2025). Disruption of tight junction integrity facilitates the entry of luminal antigens and bacterial endotoxins into the circulation, thereby linking gut-level disturbances with systemic inflammatory responses.

In addition, changes in microbial metabolism may affect bile acid transformation, energy harvesting, and host signaling pathways. Emerging evidence suggests that food additives may indirectly influence the microbiota–gut–brain axis, potentially affecting neurological and behavioral processes through microbial metabolites and neuroactive compounds (Song et al., 2023).

Importantly, the effects of food preservatives on gut microbiota are context-dependent and may vary depending on dietary patterns, host factors, and interactions with other food additives (Bevilacqua et al., 2025). Individuals consuming diets rich in ultra-processed foods are particularly exposed to cumulative effects of multiple additives, which may act synergistically to disrupt microbial homeostasis (Singh et al., 2024).

Taken together, these findings suggest that food preservatives may influence gut microbiota composition and function and may potentially contribute to impaired intestinal barrier integrity as well as systemic metabolic and inflammatory effects. This highlights the role of the gut microbiota as a central mediator of the biological effects of dietary additives on human health.

3. Activation of Inflammatory Pathways

The interplay between oxidative stress and gut dysbiosis may contribute to the activation of inflammatory signaling pathways. One of the proposed mechanisms involves activation of nuclear factor kappa B (NF- κ B), a transcription factor that regulates the expression of numerous pro-inflammatory genes and is considered a key link between cellular stress, immune activation, and chronic disease development (Soliman et al., 2022).

Activation of NF- κ B is associated with increased production of pro-inflammatory mediators such as tumor necrosis factor alpha (TNF- α), interleukin-1 beta (IL-1 β), interleukin-6 (IL-6), and, in some contexts, dysregulation of anti-inflammatory cytokines such as interleukin-10 (IL-10) (Abd-Elhakim et al., 2020). These cytokines may promote a sustained inflammatory response which, when persistent, may contribute to tissue injury, immune dysregulation, and progression of chronic metabolic and inflammatory disorders.

Experimental evidence indicates that long-term exposure to common food preservatives may alter cytokine profiles and induce immunological alterations. In animal studies, chronic exposure to multiple food preservatives has been associated with modulation of TNF- α , interferon gamma, IL-1 β , IL-6, and IL-10, suggesting a potential shift toward a pro-inflammatory state (Abd-Elhakim et al., 2020). These findings support the view that inflammation may represent not only a secondary consequence of oxidative injury, but also an important component of preservative-related biological responses.

In addition to generalized immune activation, specific preservatives appear capable of inducing organ-specific inflammatory responses. Sodium benzoate, for example, has been shown to promote pancreatic inflammation and β -cell apoptosis, partially through mechanisms linked to protein benzylation and inflammatory signaling (Li, Zhang, et al., 2024). Similarly, sodium benzoate exposure in animal models has been associated with increased inflammatory cytokine expression together with enhanced oxidative stress and biochemical disturbances, supporting a close interaction between redox imbalance and inflammation (Khan et al., 2022).

Another important mechanism by which preservatives may contribute to inflammation is through disruption of intestinal barrier integrity and subsequent translocation of microbial products into the circulation. Increased systemic exposure to bacterial components such as lipopolysaccharides may activate toll-like receptor-dependent signaling pathways and amplify downstream inflammatory cascades, thereby linking gut dysbiosis with chronic low-grade inflammation (Besedin et al., 2024; Seto et al., 2025).

The inflammatory impact of food additives should also be interpreted in the broader context of dietary patterns. Diets rich in ultra-processed foods may expose individuals to multiple additives that collectively influence oxidative and inflammatory pathways, potentially enhancing low-grade systemic inflammation over time (Singh et al., 2024; Bevilacqua et al., 2025). This is particularly relevant in relation to metabolic syndrome, obesity, and chronic gastrointestinal disorders, in which inflammation is a key pathogenic mechanism.

Overall, the available evidence suggests that activation of inflammatory pathways represents a potential biological consequence of food preservative exposure. Through modulation of cytokine networks, redox-sensitive signaling pathways, and gut-derived immune activation, preservatives may contribute to chronic inflammatory states with clinically relevant systemic effects.

4. Specific Preservatives and Their Biological Effects

Different classes of food preservatives exhibit distinct biological activities, although many of their effects converge on common pathways such as oxidative stress, inflammation, and alterations in gut microbiota composition.

Sodium benzoate

Sodium benzoate is one of the most widely used food preservatives and has been widely studied for its biological effects. Experimental evidence indicates that sodium benzoate exposure may increase the production of reactive oxygen species, reduce antioxidant defense capacity, and induce mitochondrial dysfunction (Asejeje et al., 2023; Khan et al., 2022). These effects are often accompanied by increased levels of inflammatory cytokines and biochemical markers of tissue damage.

In addition to systemic oxidative and inflammatory effects, sodium benzoate has been associated with organ-specific effects in experimental studies. Experimental studies have suggested that sodium benzoate may induce pancreatic inflammation and β -cell apoptosis, suggesting potential implications for metabolic regulation and glucose homeostasis (Li, Zhang, et al., 2024). Furthermore, sodium benzoate exposure has been linked to alterations in gut microbiota composition and host physiological responses, indicating that its biological effects may extend beyond direct cellular toxicity (Xiao et al., 2023).

Neurobiological effects have also been reported. Experimental models suggest that sodium benzoate may affect neurotransmitter balance and induce behavioral changes, including anxiety-like responses, potentially through oxidative and neuroinflammatory mechanisms (Gaur et al., 2018; Walczak-Nowicka & Herbet, 2022). These findings highlight the possibility that certain preservatives may exert systemic effects involving both peripheral and central pathways.

Sulfites

Sulfites are commonly used as preservatives in processed foods and beverages and have been associated with adverse reactions in susceptible individuals. Clinical and epidemiological data indicate that sulfite exposure may trigger hypersensitivity reactions, including bronchoconstriction and, in rare cases, anaphylaxis (Reus et al., 2000; Zhang et al., 2014). These effects are particularly relevant in individuals with asthma or sulfite sensitivity.

At the molecular level, sulfites may contribute to oxidative stress through interactions with cellular thiol groups and disruption of redox balance. Additionally, dietary exposure assessments suggest that intake levels may vary significantly across populations, raising concerns about cumulative exposure in certain dietary patterns (Lien et al., 2016). Although sulfites are generally considered safe within regulated limits, their potential to induce both oxidative and immune-mediated responses highlights the importance of individual susceptibility.

Nitrates and nitrites

Nitrates and nitrites represent another important class of food preservatives with complex biological effects. Under physiological conditions, these compounds contribute to nitric oxide production, which plays a role in vascular regulation and cellular signaling (Hord et al., 2009; Hord, 2011). However, under certain conditions, particularly in acidic environments or during high-temperature food processing, they may participate in the formation of N-nitroso compounds, some of which are considered potentially carcinogenic.

Epidemiological evidence suggests possible associations between dietary exposure to nitrites and nitrates, particularly from processed foods, and increased risk of certain cancers, including colorectal cancer (Chazelas et al., 2022; Sokal-Dembowska et al., 2025). These associations may involve mechanisms such as DNA damage, nitrosative stress, and chronic inflammation.

At the same time, it is important to emphasize that nitrates derived from natural food sources, such as vegetables, may exert protective effects due to the presence of antioxidants and other bioactive compounds (Hord et al., 2009). This dual role highlights the importance of dietary context in determining the overall health impact of nitrate and nitrite exposure.

Integrated perspective on preservative exposure

The biological effects of food preservatives are influenced by multiple factors, including dose, duration of exposure, dietary context, and individual susceptibility. Importantly, real-world dietary exposure rarely involves a single preservative in isolation. Individuals consuming diets rich in ultra-processed foods are typically exposed to multiple additives simultaneously, raising the possibility of additive or synergistic interactions.

Such combined effects remain insufficiently characterized in current toxicological frameworks, which are largely based on single-compound evaluation. Emerging evidence suggests that cumulative exposure to

multiple preservatives may enhance oxidative stress, inflammatory responses, and other adverse biological effects, thereby potentially increasing long-term health risks (Simmons et al., 2014; Hasenböhler et al., 2026).

These findings underscore the need for a more comprehensive approach to evaluating the biological effects of food preservatives, taking into account not only individual compounds but also their combined impact within complex dietary patterns.

5. Clinical and Public Health Implications

The molecular mechanisms associated with food preservative exposure, particularly oxidative stress, gut microbiota alterations, and chronic low-grade inflammation, may have clinical relevance and may contribute to the development of several non-communicable diseases.

Metabolic disorders

A growing body of evidence suggests that food preservatives and other dietary additives may be associated with metabolic disorders, including obesity, insulin resistance, and type 2 diabetes. Experimental studies indicate that sodium benzoate may impair insulin signaling and induce metabolic dysfunction at the cellular level (Sharma et al., 2026). These effects may be mediated by oxidative stress, mitochondrial dysfunction, and inflammatory activation.

Epidemiological data further support this association. A large prospective cohort study (NutriNet-Santé) reported an association between higher exposure to preservative food additives and increased risk of type 2 diabetes (Hasenböhler et al., 2026). These findings suggest that long-term dietary exposure to preservatives may contribute to metabolic dysregulation at the population level, although causality remains to be clarified.

Additionally, broader analyses of food additives and contaminants have proposed multiple mechanisms linking dietary exposure to obesity, including endocrine disruption, mitochondrial impairment, and altered energy metabolism (Simmons et al., 2014). Together, these mechanisms provide a biologically plausible link between preservative intake and metabolic disease development.

Cardiovascular and systemic effects

Oxidative stress and chronic inflammation are central to the pathogenesis of cardiovascular diseases. Preservative-induced redox imbalance and inflammatory activation may be linked to processes such as endothelial dysfunction, lipid oxidation, and impaired vascular signaling.

Alterations in gut microbiota composition may further contribute to cardiovascular risk through mechanisms involving metabolic endotoxemia and immune activation (Besedin et al., 2024). These findings highlight the interconnected nature of metabolic and cardiovascular pathways influenced by dietary exposures.

Cancer risk

The potential role of food preservatives in carcinogenesis has been increasingly investigated. Mechanistically, oxidative stress, DNA damage, chronic inflammation, and the formation of reactive compounds such as N-nitroso derivatives may contribute to tumor development.

Large-scale epidemiological studies provide emerging evidence supporting these associations. Data from the NutriNet-Santé cohort indicate a possible association between higher intake of preservative food additives and increased cancer risk (Hasenböhler et al., 2026). Similarly, nitrite exposure from processed foods has been linked to increased cancer risk, particularly in the gastrointestinal tract (Chazelas et al., 2022).

Meta-analyses and systematic reviews further suggest that high consumption of ultra-processed foods, which are major sources of preservatives, is associated with an increased risk of gastrointestinal cancers and other chronic diseases (Meine et al., 2024; Lane et al., 2024). While causality remains difficult to establish, the consistency of epidemiological findings combined with mechanistic evidence supports the plausibility of these associations.

Gastrointestinal and immune-related conditions

Food preservatives may also contribute to gastrointestinal disorders through their effects on gut microbiota and intestinal barrier function. Disruption of microbial balance and increased intestinal permeability may contribute to chronic inflammation and immune dysregulation.

Recent studies have linked food additives with inflammatory bowel disease and metabolic syndrome, suggesting that dietary exposures may play a role in the pathogenesis of these conditions (Besedin et al., 2024; Singh et al., 2024). In addition, long-term exposure to preservatives has been associated with altered cytokine profiles, including increased levels of pro-inflammatory mediators such as TNF- α , IL-6, and IL-1 β (Abd-Elhakim et al., 2020).

These findings indicate that preservatives may not only act as chemical stressors but also modulate immune responses, potentially contributing to chronic inflammatory diseases.

Population-level exposure and public health relevance

From a public health perspective, the widespread consumption of ultra-processed foods represents a major source of preservative exposure. Global dietary trends indicate that a substantial proportion of daily caloric intake in many populations is derived from such foods, leading to chronic and cumulative exposure.

Umbrella reviews and meta-analyses have reported associations between high intake of ultra-processed foods and increased risk of obesity, cardiovascular disease, cancer, and mental health disorders (Dai et al., 2024; Lane et al., 2023; Bhavé et al., 2024). These findings suggest that preservative exposure should be considered within the broader context of dietary patterns and lifestyle factors.

Importantly, certain population groups may be particularly vulnerable, including children, individuals with metabolic disorders, and those with pre-existing gastrointestinal or inflammatory conditions. Variability in genetic background, microbiota composition, and metabolic capacity may further influence individual susceptibility.

6. Dietary Exposure and Real-World Context of Preservative Intake

An essential aspect in interpreting the biological effects of food preservatives is the context of real-world dietary exposure. While experimental studies typically investigate isolated compounds under controlled conditions, human exposure occurs within complex dietary patterns involving multiple food additives consumed simultaneously.

In modern food systems, preservatives are predominantly found in ultra-processed foods, including processed meats, packaged snacks, ready-to-eat meals, and sugar-sweetened beverages. These products are widely consumed in many populations, contributing to widespread and repeated exposure (World Health Organization, 2023; Dai et al., 2024). As a result, dietary intake of preservatives appears to be widespread and, in many populations, may be chronic and cumulative.

Epidemiological evidence indicates that a substantial proportion of total daily energy intake in many populations is derived from ultra-processed foods, which represent an important source of food additives, including preservatives (Mambrini et al., 2023). High consumption of such foods has been associated with increased risks of obesity, metabolic disorders, cardiovascular diseases, and cancer (Dai et al., 2024; Lane et al., 2024).

Importantly, real-life exposure rarely involves a single additive. Individuals consuming ultra-processed diets are typically exposed to multiple preservatives alongside other additives such as emulsifiers, artificial sweeteners, and colorants. The combined or synergistic effects of these compounds remain incompletely understood and are not adequately captured in traditional toxicological assessments, which generally evaluate single substances in isolation (Simmons et al., 2014; Bevilacqua et al., 2025).

Another important factor is interindividual variability in dietary patterns and susceptibility. Children and adolescents often exhibit higher relative intake of ultra-processed foods in relation to body weight, which may lead to relatively higher exposure to preservatives (Lane et al., 2024). Similarly, individuals with limited access to fresh foods or those relying heavily on convenience-based diets may experience disproportionately higher intake levels.

The food matrix may also modulate the biological effects of preservatives. Interactions between preservatives and other dietary components such as fats, proteins, fiber, and antioxidants can influence their absorption, metabolism, and biological activity. For instance, dietary antioxidants may partially counteract oxidative stress induced by certain preservatives, whereas high-fat diets may exacerbate inflammatory responses and metabolic disturbances.

Furthermore, host-related factors including gut microbiota composition, metabolic status, and genetic background may influence individual responses to preservative exposure. Emerging evidence suggests that interactions between dietary additives and the gut microbiome represent a key determinant of health outcomes, highlighting the importance of individualized susceptibility (Singh et al., 2024; Seto et al., 2025).

From a public health perspective, these observations highlight potential limitations of current risk assessment models based on acceptable daily intake (ADI), which may not fully reflect cumulative exposure, additive interactions, and long-term dietary patterns. According to the World Health Organization (2023), food additives are regulated within established safety frameworks; however, these frameworks may not fully capture real-life exposure scenarios.

Taken together, these findings suggest that the health effects of food preservatives may not be fully understood without considering the broader dietary context. The interaction between multiple additives, dietary patterns, and host-specific factors represents a complex system that requires a multidisciplinary approach in both research and risk assessment.

Discussion

1. Integration of Molecular Mechanisms

The findings presented in this review suggest that the biological effects of food preservatives may be interpreted within an integrated mechanistic framework involving oxidative stress, gut microbiota alterations, and chronic low-grade inflammation. These processes are not independent but rather form an interconnected network that may contribute to long-term health effects associated with preservative exposure.

Oxidative stress appears to be a central mechanism linking preservative exposure to cellular dysfunction. Experimental studies have demonstrated that commonly used preservatives can increase reactive oxygen species (ROS) production and impair antioxidant defense systems, leading to redox imbalance and cellular damage (Minamiyama et al., 2021; Asejeje et al., 2023). This oxidative environment may further activate redox-sensitive signaling pathways, including nuclear factor kappa B (NF- κ B), thereby promoting inflammatory responses (Soliman et al., 2022).

At the same time, alterations in gut microbiota composition represent an important complementary mechanism. Food additives, including preservatives, may reduce microbial diversity and disrupt beneficial bacterial populations, leading to dysbiosis (Zhou et al., 2023; Singh et al., 2024). These changes are associated with impaired intestinal barrier function and increased permeability, facilitating the translocation of microbial components such as lipopolysaccharides into systemic circulation.

The interaction between oxidative stress and gut dysbiosis may amplify inflammatory signaling, creating a self-reinforcing cycle of metabolic and immune dysfunction. This integrated perspective supports the view that preservatives may act not only as isolated chemical stressors but also as modulators of complex biological systems.

2. Clinical Relevance and Disease Associations

The molecular mechanisms discussed in this review may have important implications for human health, particularly in relation to chronic non-communicable diseases. Oxidative stress and chronic inflammation are well-established contributors to metabolic disorders, cardiovascular diseases, and cancer.

Recent epidemiological evidence strengthens the relevance of these mechanisms. Prospective cohort data from the NutriNet-Santé study indicate that higher intake of preservative food additives has been associated with increased risk of type 2 diabetes and cancer (Hasenböhler et al., 2026). Similarly, high consumption of ultra-processed foods, a major source of preservatives, has been linked to a broad range of adverse health outcomes, including obesity, cardiovascular disease, and gastrointestinal cancers (Dai et al., 2024; Lane et al., 2024; Meine et al., 2024).

In the gastrointestinal context, disruption of gut microbiota and barrier function may contribute to inflammatory bowel disease and metabolic endotoxemia, further supporting the role of preservatives in immune and metabolic dysregulation (Besedin et al., 2024; Seto et al., 2025).

Although causality remains difficult to establish due to the observational nature of most studies, the convergence of mechanistic and epidemiological evidence supports the hypothesis that food preservatives may contribute to chronic disease development.

3. Role of Dietary Patterns and Cumulative Exposure

An important insight emerging from this review is that the health effects of food preservatives may not be fully understood without considering real-world dietary patterns. In contrast to controlled experimental conditions, human exposure is characterized by chronic, low-dose intake of multiple additives within complex food matrices.

Ultra-processed foods represent the primary source of preservative exposure and are consumed at high levels in many populations. These dietary patterns are associated with cumulative exposure to numerous additives, raising concerns about additive or synergistic effects (Bevilacqua et al., 2025; Simmons et al., 2014).

Current toxicological assessments are largely based on acceptable daily intake (ADI) values for individual compounds, which may not adequately reflect combined exposure or long-term dietary habits. This limitation suggests that existing risk assessment frameworks may underestimate the potential biological impact of food preservatives.

Furthermore, individual susceptibility varies depending on factors such as genetic background, metabolic status, and gut microbiota composition. This variability highlights the need for a more personalized approach to dietary risk assessment and reinforces the importance of considering host–environment interactions.

4. Methodological Limitations of Current Evidence

Despite increasing research interest, several limitations should be considered when interpreting the available evidence on food preservatives and human health.

First, a substantial proportion of mechanistic data is derived from *in vitro* and animal studies, which may not fully replicate human physiology. Differences in metabolism, exposure levels, and experimental conditions limit the direct translation of these findings to clinical settings (Minamiyama et al., 2021).

Second, epidemiological studies are subject to confounding factors, including dietary habits, lifestyle variables, socioeconomic status, and co-exposure to multiple additives. These factors make it challenging to isolate the specific effects of preservatives from broader dietary patterns (Dai et al., 2024).

Third, heterogeneity in study design, exposure assessment, and outcome definitions complicates comparison across studies. In addition, many experimental studies use concentrations that exceed typical dietary exposure, raising questions about their relevance to real-world conditions.

Finally, the majority of existing research focuses on individual compounds, whereas real-life exposure involves complex mixtures of additives. The potential for additive or synergistic effects remains insufficiently explored and represents a critical gap in current knowledge.

5. Implications for Clinical Practice and Public Health

The findings of this review have important implications for both clinical practice and public health strategies.

From a clinical perspective, healthcare professionals should consider the potential role of ultra-processed foods and food additives as contributors to chronic disease risk. Dietary assessment may benefit from incorporating the degree of food processing alongside traditional nutritional parameters.

Reducing the consumption of ultra-processed foods and increasing the intake of minimally processed, nutrient-dense foods may represent a potential strategy to limit exposure to preservatives and mitigate their potential biological effects. Diets rich in fiber, antioxidants, and bioactive compounds may help counteract oxidative stress and support gut microbiota balance.

From a public health standpoint, the widespread and increasing consumption of ultra-processed foods represents a significant challenge. Current regulatory frameworks may need to be expanded to consider cumulative exposure, mixture effects, and long-term dietary patterns rather than focusing solely on individual additives.

These considerations are consistent with international food safety frameworks, which emphasize the need for continuous evaluation of food additive exposure in the context of evolving dietary patterns (World Health Organization, 2023).

Increased awareness among consumers, clinicians, and policymakers is essential to address the potential health implications of preservative exposure within modern food systems.

6. Future Research Directions

Future research should aim to bridge the gap between mechanistic findings and population-level health outcomes.

Well-designed longitudinal studies and randomized controlled trials are needed to further assess the long-term effects of preservative exposure. Integrative approaches combining multi-omics technologies, microbiome analysis, and metabolic profiling may provide deeper insights into the complex interactions between dietary additives and human biology.

Particular attention should be given to individual variability in response to preservative exposure, including genetic factors and microbiota composition. Such approaches may contribute to the development of personalized dietary recommendations and improved risk assessment models.

In addition, future studies should focus on the combined effects of multiple food additives and their interactions within realistic dietary contexts, which remain largely unexplored.

Conclusions

This narrative review highlights that food preservatives, although essential for ensuring food safety and extending shelf life, may have biological effects extending beyond their intended technological functions. The available evidence suggests that selected preservatives may interfere with key molecular pathways, particularly those related to oxidative stress, gut microbiota composition, and inflammatory signaling.

These mechanisms appear to be closely interconnected, although their relative contribution may vary depending on the preservative, exposure context, and host susceptibility. Oxidative stress, microbial dysbiosis, and chronic low-grade inflammation may be viewed as an interconnected mechanistic triad potentially involved in the development of non-communicable diseases, including metabolic disorders, cardiovascular diseases, and cancer. Experimental and epidemiological findings support the plausibility of these associations, although direct causal relationships in humans remain difficult to establish.

Importantly, the health impact of food preservatives should be considered within the context of real-world dietary exposure. In modern dietary patterns, preservatives are primarily consumed through ultra-processed foods, leading to chronic, low-dose, and cumulative exposure. This raises concerns regarding potential additive or synergistic effects, which are not fully captured by current risk assessment models based on individual compounds and acceptable daily intake (ADI) thresholds.

Furthermore, individual susceptibility to preservative-related effects may vary depending on factors such as genetic background, metabolic status, and gut microbiota composition. These considerations highlight limitations of traditional toxicological approaches and emphasize the need for more integrative and personalized risk assessment strategies.

From a public health perspective, reducing the consumption of ultra-processed foods and promoting dietary patterns based on minimally processed products may represent a potential strategy to limit preservative exposure and mitigate associated health risks.

Future research should focus on long-term human studies, improved exposure assessment, and integrative approaches combining molecular, clinical, and epidemiological data. In particular, greater attention should be given to the combined effects of multiple food additives and their interactions within realistic dietary environments.

In conclusion, while food preservatives remain indispensable components of modern food systems, their potential impact on human health warrants further investigation. A more comprehensive approach to food safety assessment integrating mechanistic insights, population-level data, and real-world exposure may be valuable for developing more accurate risk models and evidence-based dietary recommendations.

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