

REVIEW ARTICLE

FOODBORNE ILLNESSES, FOOD POISONING, ENVIRONMENTAL CONTAMINANTS AND FOOD SAFETY IN THE WORLD: A COMPREHENSIVE MINI REVIEW

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ABSTRACT

Food safety remains an absolute cornerstone of global public health and socioeconomic stability. Unsafe food causes an estimated 866 million illnesses and 1.52 million deaths globally each year, resulting in an annual economic loss of US\$ 310 billion across the globe. The main objective of this mini-review is to synthesize current knowledge on foodborne pathogens, food additives, heavy metals, microplastics, emerging anthropogenic hazards, and advances in diagnostic methods. A total of 1,500 research articles retrieved from Google Scholar, Web of Science, ScienceDirect, ResearchGate, and PubMed were systematically reviewed to compile this comprehensive review. Globally, an estimated 866 million (1 in 9 people) individuals experience illnesses annually from consuming contaminated food, resulting in approximately 1.52 million preventable deaths. This comprehensive mini-review paper critically evaluates major bacterial, viral, parasitic, chemical hazards and environmental contaminants. Furthermore, it provides a multidimensional analysis of emerging chemical threats, including microplastics in food systems, heavy metal contamination, and pesticide and fertilizer residues, as well as their potential associations with chronic kidney disease of unknown etiology (CKDu). Finally, the paper explores state-of-the-art diagnostic paradigms under a unified One Health framework proposed and implemented by the WHO and FAO. This review may support the food industry and policymakers in adopting modern technologies within the One Health framework to reduce the risk of foodborne illnesses and non-communicable diseases, such as diabetes, hypertension, and cancer.

Keywords: CKDu, Contaminated foods, Foodborne pathogens, Heavy metals, Microplastics, Pesticide residues

INTRODUCTION

Global burden of foodborne diseases

Access to safe, bio-secure, and nutritionally adequate food is an essential determinant of human life and sustained metabolic health (World Health Organization [WHO], 2026). Despite rapid industrial modernization, advanced cold-chain infrastructures, and stringent legislative regulatory mandates, unsafe food represents a persistent threat to global clinical public health. The global food matrix has evolved into a highly complex, interconnected network where a single localized contamination node can rapidly cascade into a transcontinental epidemiological emergency (Azigi and Adrah, 2025). According to core epidemiological matrices validated by the

WHO and the Foodborne Disease Burden Epidemiology Reference Group (FERG), approximately 866 million people fall ill each year following the ingestion of contaminated food (WHO, 2026).

This staggering incidence generates an annual mortality toll of roughly 1.52 million individuals and precipitates the loss of 33 million Disability-Adjusted Life Years (DALYs) worldwide (WHO, 2026; WHO, 2024). The demographic distribution of this public health crisis is profoundly asymmetrical. Pediatric populations under five years of age disproportionately shoulder 40% of the aggregate foodborne disease burden, suffering 125,000 annual fatalities (WHO, 2024). Unsafe food perpetuates a devastating biological cycle of gas-

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trointestinal infection, malabsorption, acute malnutrition, and immunological stunting, which exacerbates infant mortality rates across fragile ecosystems.

Economically, the impacts are deeply destabilizing to national architecture. A World Bank report estimated that productivity losses and healthcare costs associated with foodborne diseases impose a substantial economic burden on low- and middle-income countries (LMICs) (WHO, 2026). Moreover, the macroeconomic burden of foodborne diseases severely strains municipal healthcare frameworks, restricts agricultural trade opportunities, undermines domestic tourism, and hinders broader socioeconomic development (WHO, 2024).

METHODOLOGICAL OVERVIEW AND PRISMA FLOW

Methodological overview

The Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement offers an evidence-based minimum set of items aimed at helping authors report on systematic reviews and meta-analyses. This document outlines the standard 4-stage PRISMA structure tailored specifically for a comprehensive mini-review investigating the links between foodborne illnesses, heavy metals, microplastics, food additives, and Chronic Kidney Disease of Unknown Etiology (CKDu)

PRISMA flow

A total of 1,500 records were identified through database searches, including 260 from ScienceDirect, 400 from Google Scholar, 385 from PubMed, and 455 from ResearchGate. The search terms used included “foodborne illnesses,” “food poisoning,” “heavy metals,” “microplastics,” “food additives,” and “CKDu.” After removing duplicate records, 1,123 articles remained for title and abstract screening. Of these, 1,023 records were excluded because they were irrelevant to the study topic or did not match the required domain, were published in languages other than English, or consisted of editorials, opinion pieces, or non-peer-reviewed notes. Consequently, 100 full-text articles were as-

sessed for eligibility. Following full-text assessment, 44 articles were excluded because they lacked relevant data on the targeted contaminants or CKDu pathogenesis, had unavailable full texts or incomplete quantitative or qualitative data, or represented duplicate reports of the same cohort studies. Finally, 56 studies were included in the qualitative synthesis of the mini-review.

Key methodological guidelines for implementation

Database Search Query Construction: Group keywords using precise Boolean operators to balance sensitivity and specificity. For example: ("foodborne illnesses" OR "food poisoning") AND ("heavy metals" OR "microplastics" OR "food additives") AND "CKDu".

Duplicate Removal Protocol: Import initial search findings into reference management software (e.g., EndNote, Zotero, or Mendeley). Execute automated duplicate screening followed by manual title/author verification prior to initial screening.

Documenting Exclusion Criteria: Maintain an explicit, transparent audit trail detailing exact reasons for full-text article exclusion. Categorize exclusions (e.g., wrong clinical outcome, missing full text, inadequate toxicity data) to preserve rigor.

Taxonomy and pathophysiology of major foodborne pathogens

Foodborne illnesses are fundamentally infectious or toxicological syndromes triggered by a diverse spectrum of biological pathogens or persistent chemical compounds that infiltrate the human gastrointestinal tract via ingestible vehicles (WHO, 2026; WHO, 2024).

Bacterial hazards

Bacterial agents constitute the primary drivers of acute, severe foodborne hospitalizations and mortalities globally (WHO, 2026; WHO, 2024).

Salmonella enterica

The *Salmonella* genus, particularly non-typhoidal serovars such as *Salmonella enter-*

ica serovar Typhimurium and Enteritidis, displays exceptional epidemiological ubiquity due to its capacity to colonize a wide array of agricultural animal hosts (WHO, 2026). The primary biological reservoirs are avian poultry vectors and eggs (Mehta *et al.*, 2023). Upon ingestion, *Salmonella* utilizes its Salmonella Pathogenicity Islands (SPI-1 and SPI-2) along with Type III Secretion Systems (T3SS) to actively inject virulence effector proteins directly into human intestinal epithelial cells. These subvert host cytoskeletal machinery, driving cellular invasion, profound inflammatory enteritis, severe abdominal cramping, pyrexia, and systemic bacteremia in immunologically vulnerable individuals (WHO, 2026).

Campylobacter jejuni

Campylobacter species represent a leading bacterial cause of global diarrheal zoonoses (WHO, 2026). The primary transmission vectors include unpasteurized raw milk, undercooked poultry tissue, and contaminated open-source drinking water (WHO, 2026). Pathophysiologically, *C. jejuni* relies on its flagellar motility to penetrate the thick mucus layer of the human colon. It then secretes Cytotoxin Distending Toxin (CDT), which induces cell cycle arrest and apoptosis in host enterocytes. Autoimmune molecular mimicry between the lipooligosaccharides on the *C. jejuni* cell wall and human peripheral nerve gangliosides can trigger Guillain-Barré syndrome.

***Escherichia coli* (STEC/EHEC)**

Not all strains of *E. coli* are pathogenic. However, shiga toxin-producing *Escherichia coli* (STEC), alternatively referred to as Enterohemorrhagic *E. coli* (EHEC), features highly dangerous strains such as *E. coli* O157:H7 (Lim *et al.*, 2023). These strains harbor specialized virulence plasmids and lysogenic bacteriophages that encode potent Shiga toxins (Stx1 and Stx2). STEC binds tightly to host enterocytes using intimin proteins to induce attaching and effacing (A/E) lesions. The systemic translocation of Shiga toxins into the bloodstream leads to binding at globotriaosylceramide (Gb3) receptors, culminating in endothelial cell death, microvascular

thrombosis, hemolytic anemia, profound thrombocytopenia, and acute renal failure—a clinical triad diagnostic of Hemolytic Uremic Syndrome (HUS) (Zhan, 2025).

Listeria monocytogenes

Listeria monocytogenes is a psychrotrophic, ubiquitously distributed environmental pathogen capable of actively proliferating at refrigeration temperatures (4 °C). As a result, this ability makes it a persistent threat within ready-to-eat (RTE) meat formulations and unpasteurized soft cheeses (Jayamanne and Samarajeewa, 2011; WHO, 2026). Its case-fatality rate is uniquely high, ranging between 20% and 30% among highly vulnerable demographics (Jayamanne and Samarajeewa, 2001; WHO, 2026). It produces listeriolysin O (LLO) and phospholipases to escape phagosomal vacuoles, replicating directly within the host cytoplasm and breaching intestinal, blood-brain, and placental barriers.

Staphylococcus aureus

S. aureus acts as a major foodborne hazard primarily through the intoxication pathway, rather than active *in vivo* infection. It is frequently introduced into the food matrix by asymptomatic human food handlers carrying the bacteria within their nasal passages (Pattabhiramaiah and Mallikarjunaiah, 2023). When food matrices are subjected to prolonged temperature abuse, *S. aureus* proliferates and synthesizes structurally stable staphylococcal enterotoxins (SEs; including SEA, SEB, and SED), which function as potent superantigens, triggering a massive release of pro-inflammatory cytokines that induce rapid, projectile emesis.

Viral and parasitic hazards

Beyond bacterial agents, viral and parasitic pathogens contribute heavily to the global foodborne disease burden (Table 1). Foodborne viruses are microscopic pathogens that contaminate food or water and cause illness when ingested. Unlike bacteria, which can multiply in food under the right conditions, viruses cannot reproduce outside a living host; they simply use food as a vehicle for transportation.

Key characteristics and transmission

The primary culprits are Norovirus and Hepatitis A that are the most common and impactful foodborne viruses globally (Table 1). Further, it takes only a tiny number of viral particles to cause severe illness, making them exceptionally contagious. Contamination typically happens when an infected food handler

touches food without proper hand hygiene, or through contaminated irrigation water used on crops like berries and leafy greens. Many foodborne viruses can survive on surfaces, endure freezing temperatures, and resist mild acidic environments like the human stomach (WHO, 2024).

Table 1: Viruses and parasites that cause foodborne illnesses in the world

Pathogen Class	Representative Organism	Primary Food Vehicles	Key Pathological Impact
Virus	Norovirus	Bivalve mollusks, fresh salads, contaminated drinking water	Acute, hyper-contagious gastroenteritis, rapid outbreaks.
Virus	Hepatitis A Virus (HAV)	Contaminated water, raw shellfish, handled RTE items	Hepatic targeting, acute hepatocellular inflammation, jaundice.
Parasite	<i>Toxoplasma gondii</i>	Undercooked porcine/ovine meats, unwashed produce	Tissue cyst encystment, congenital ocular/neurological damage.
Parasite	<i>Cryptosporidium parvum</i>	Wastewater-irrigated produce, contaminated drinking water	Oocyst-mediated infection, severe, profuse watery diarrhea.
Parasite	<i>Taenia solium</i>	Undercooked or contaminated pork products	Intestinal taeniasis; larval migration causing neurocysticercosis.

Source: WHO (2026)

Major illnesses caused

Norovirus, which is often called the "stomach flu," causes sudden acute gastroenteritis, leading to severe vomiting, watery diarrhea, nausea, and stomach cramps. It spreads rapidly in closed environments like restaurants, cruise ships, and schools. On the other hand, Hepatitis A, a liver infection, causes fatigue, nausea, abdominal pain, dark urine, and jaundice (yellowing of the skin and eyes). Unlike Norovirus, it has a much longer incubation period (weeks) and can be prevented by a vaccine.

Prevention and control

Strict handwashing by food handlers is the single most effective defense against contamination. Safe Sourcing: Using clean, unpolluted water for washing produce and harvesting shellfish only from approved, monitored waters. While viruses are hardy, cooking foods to proper internal temperatures and thoroughly reheating leftovers can reduce risk, though some viruses still withstand moderate heat.

Anthropogenic and emerging chemical threats in food systems

In addition to the common foodborne diseases,

the world has observed several emerging threats to food safety for the last few decades and those are discussed in detail below.

Food additives

The global surge in the consumption of ultra-processed foods (UPFs) has fundamentally altered modern dietary patterns. Beyond their poor macronutrient profiles, UPFs are uniquely characterized by the ubiquitous inclusion of food additives, substances added intentionally to improve shelf life, appearance, texture, and palatability (Bevilacqua *et al.*, 2024; Rondinella *et al.*, 2025). Although regulatory bodies like the European Food Safety Authority (EFSA) and the U.S. Food and Drug Administration (FDA) historically recognized these compounds as safe within Acceptable Daily Intake (ADI) thresholds, emerging clinical and epidemiological research suggests prolonged, cumulative exposure to specific food additives may pose substantial risks to human health (Atary-Sheetryt *et al.*, 2026; Meijer, 2026).

Gut dysbiosis and intestinal barrier disruption

One of the primary mechanisms through which food additives negatively impact human health is by altering the delicate ecosystem of the gut microbiota. Emulsifiers (e.g., carboxymethyl cellulose, polysorbate-80) and thickeners (e.g., carrageenan) are widely used to maintain structural uniformity in processed foods, but recent evidence indicates they can directly degrade the protective mucous layer of the gastrointestinal tract (Rondinella *et al.*, 2025; Spiller, 2025). This degradation increases intestinal permeability, colloquially referred to as "Increased Intestinal Permeability" or "leaky gut," allowing pro-inflammatory microbial components to enter the systemic circulation (Rondinella *et al.*, 2025; Anyimadu *et al.*, 2026).

Furthermore, industrial thickeners and stabilizers have been shown to induce distinct taxonomic shifts in the gut, diminishing populations of beneficial, short-chain fatty acid (SCFA)-producing bacteria while encouraging the overgrowth of pathobionts (Bevilacqua *et al.*, 2024; Spiller, 2025). This persistent microbial imbalance observed in human clinical trials is strongly implicated in the rising incidence of chronic gastrointestinal pathologies, particularly Inflammatory Bowel Disease (IBD) and irritable bowel syndrome (Singh, 2025; Spiller, 2025).

Metabolic syndrome and obesity

The correlation between highly processed diets and the metabolic syndrome pandemic has increasingly been linked to non-nutritive additives. Artificial sweeteners (e.g., acesulfame potassium, aspartame, cyclamate) and certain chemical preservatives (e.g., sodium propionate) have traditionally been utilized as inert tools for weight management. However, recent cohort studies reveal a dose-dependent association between high exposure to these additives and increased Body Mass Index (BMI), insulin resistance, and visceral adiposity (Atary-Sheetryt *et al.*, 2026). Rather than acting as inert substitutes, these compounds can trigger pro-inflammatory pathways and alter glucose homeostasis, ultimately compounding metabolic dysfunction and elevat-

ing the long-term risk of type 2 diabetes (Atary-Sheetryt *et al.*, 2026; Meijer, 2026).

Carcinogenicity and systemic inflammatory risks

Beyond metabolic and gastrointestinal disturbances, the long-term oncogenic potential of industrial preservatives remains a critical focus of epidemiological oversight. High dietary intakes of specific non-antioxidant preservatives, notably nitrites and nitrates commonly found in processed meats, exhibit strong statistical associations with elevated cancer risks, particularly breast, prostate, and overall colorectal cancers, as demonstrated by Hasenböhler (2026) and Meijer (2026). While individual additive exposures rarely exceed established toxicological limits daily, the concurrent consumption of multiple synthetic agents introduces a potential "cocktail effect" or mixture toxicity that may exacerbate systemic inflammation and cellular oxidative stress over a lifetime (Meijer, 2026).

Microplastics in food systems

The contamination of the biosphere by plastic debris has emerged as a major ecological crisis, transitioning directly into agricultural and marine food chains (Alimi *et al.*, 2018). Microplastics (MPs), defined as plastic particles smaller than 5 mm, enter food systems via primary manufacturing source paths or secondary environmental fragmentation (Eerkes-Medrano *et al.*, 2022). In marine environments, bivalve mollusks, crustaceans, and predatory fish ingest MPs, mistaking them for planktonic food resources (Carbery *et al.*, 2021). Consequently, these particles translocate across the intestinal epithelium into the systemic circulation and the muscular tissues of seafood species consumed by humans (Savinelli *et al.*, 2023).

Terrestrial food systems are equally vulnerable. MPs accumulate in agricultural soils through the application of sewage sludge, plastic mulching films, and contaminated irrigation water (Rillig *et al.*, 2022). Recent studies have confirmed that sub-micrometer plastics and nanoplastics can be absorbed through the root systems of staple crop plants via crack-entry modes, subsequently translocating

to edible vegetative organs and fruits (Li *et al.*, 2023).

Beyond physical tissue irritation, MPs function as toxicological vectors. Due to their hydrophobic surfaces and high surface-area-to-volume ratios, they actively adsorb persistent organic pollutants (POPs) and heavy metals from the surrounding environment (Wang *et al.*, 2021). When ingested, the low pH of the human gastric environment triggers the desorption and bioavailability of these bound chemical hazards, alongside intrinsic plastic additives like phthalates and bisphenol A (BPA) (Amelia *et al.*, 2021). Intracellularly, MPs induce chronic inflammation, generate reactive oxygen species (ROS), disrupt gut microbiota homeostasis, and may breach the blood-brain barrier, leading to systemic neurodevelopmental and metabolic anomalies (Prata *et al.*, 2020).

Heavy metal contamination in foods

Industrial discharge, unregulated mining activities, and the application of low-grade chemical fertilizers introduce heavy metals, specifically Arsenic (As), Cadmium (Cd), Lead (Pb), and Methylmercury (MeHg) into soil matrices and aquatic ecosystems (WHO, 2024; Taylor and Post, 2024). These elemental contaminants are non-biodegradable and undergo significant bioaccumulation and biomagnification up the trophic pyramid.

Staple cereal matrices, particularly rice (*Oryza sativa*), are highly efficient at hyper-accumulating inorganic arsenic and cadmium from contaminated agricultural soils and groundwater (Taylor and Post, 2024). Lead contamination is frequently driven by atmospheric deposition from industrial smelting and legacy leaded gasoline residues, which adhere to the surfaces of leafy vegetables and enter root crops (Zhuang *et al.*, 2021). In aquatic systems, predatory marine species like tuna and swordfish biomagnify methylmercury within their lipid-dense muscular tissues, posing severe clinical risks to human consumers (Tchounwou *et al.*, 2022).

Biochemically, heavy metals exert intense toxicity at the cellular level. They deplete en-

dogenous intracellular glutathione pools, generate damaging hydroxyl radicals, and directly bind to functional sulfhydryl (-SH) groups on essential enzymatic structures (WHO, 2024). This molecular subversion manifests clinically as severe neurotoxicity (particularly MeHg and Pb in pediatric populations), irreversible renal tubular necrosis (Cd), bone demineralization, and an elevated incidence of various visceral malignancies (WHO, 2024).

Pesticide and fertilizer residues in foods

The intensive prioritization of crop yield maximization has catalyzed the widespread application of synthetic pesticides and chemical fertilizers globally (Damalas and Eleftherohorinos, 2021). Dietary exposure to pesticide residues comprising organophosphates, carbamates, pyrethroids, and organochlorine compounds occurs primarily through the ingestion of fresh fruits, vegetables, and surface water supplies (Nicolopoulou-Stamati *et al.*, 2023).

Organophosphate pesticides, such as chlorpyrifos and malathion, act as potent neurotoxins by phosphorylating the active site of the acetylcholinesterase (AChE) enzyme within synaptic junctions (Casida and Durkin, 2022). This precipitates a toxic overaccumulation of acetylcholine, triggering acute cholinergic crisis, respiratory depression, and long-term neurodevelopmental deficits in children (Kim *et al.*, 2021). Chronic dietary ingestion of organochlorine residues, which exhibit structural persistence and accumulate within human adipose tissues, is clinically linked to endocrine disruption, reproductive failures, and elevated risks of hormone-dependent breast and prostate cancers (Mnif *et al.*, 2021).

Simultaneously, the overapplication of nitrogenous and phosphatic fertilizers reconfigures the chemical equilibrium of agricultural ecosystems (Savci, 2022). Excessive nitrate (NO_3^-) residues leach into groundwater and accumulate within leafy green matrices (Spalding *et al.*, 2023). Upon human consumption, salivary and gastric enzymes reduce nitrates to reactive nitrites (NO_2^-) (Ward *et al.*, 2022). These species oxidize the ferrous iron (Fe^{2+}) in functional hemoglobin into ferric iron (Fe^{3+}), generating methemoglobin,

which is incapable of binding oxygen (methemoglobinemia or "blue baby syndrome") (Avery, 1999). Furthermore, nitrites react with secondary amines in the stomach to synthesize highly carcinogenic N-nitroso compounds (Mostafa *et al.*, 2024).

Chronic Kidney Disease of unknown etiology (CKDu) due to environmental contaminants

Over the past few decades, a devastating epidemiological crisis identified as chronic kidney disease of Unknown Etiology (CKDu) has emerged in distinct agricultural regions globally, most notably within the North Central Province of Sri Lanka, the Pacific coast of Central America (Mesoamerican Nephropathy), and parts of India (Wijkström *et al.*, 2020). Unlike conventional chronic kidney disease, CKDu progresses covertly in young, male agricultural laborers who lack typical clinical preconditions such as diabetes mellitus or chronic hypertension (Wanigasuriya, 2022). Comprehensive epidemiological and toxicological analyses strongly point toward chronic, low-dose dietary exposure to synergistic chemical contaminant matrices within food and water supply frameworks as the primary causative driver (Chandrajith *et al.*, 2021).

The pathophysiology of CKDu involves the concurrent ingestion of heavy metals (Cd, As, Pb), fluoride, and nephrotoxic pesticide residues (such as glyphosate and paraquat) via contaminated staple foods like locally grown rice, freshwater fish (e.g., *Tilapia*), and shallow well water (Jayatilake *et al.*, 2023). In these agricultural matrices, agrochemical residues act as chelating agents, binding tightly with heavy metals and hard-water ions (Ca^{2+} , Mg^{2+}) present in the geographic soil profile (Gunatilake *et al.*, 2024). This structural chelation prevents heavy metals from precipitating out of solution, thereby enhancing their stable delivery into the human gastrointestinal tract (Jayasinghe *et al.*, 2021). However, future research needs to focus on finding the exact mechanism of CKDu especially in the north-central and eastern provinces in Sri Lanka. Some scientists hypothesize that not only water but our day-to-day eating patterns

plays a major role in never-ending spread of CKDu in Sri Lanka and elsewhere (Jayamanne, 2026).

Upon absorption, these complexed chemical compounds translocate to the renal architecture, where they preferentially accumulate within the proximal convoluted tubules of the nephron (Redmon *et al.*, 2021). The highly concentrated environment of the renal tubule causes the complexes to dissociate, releasing free cadmium, arsenic, and pesticide molecules directly onto the tubular epithelial cells (Liyanage *et al.*, 2022). This induces severe, localized mitochondrial damage, triggers lipid peroxidation pathways, suppresses antioxidant enzyme production, and drives extensive tubulointerstitial nephritis and interstitial fibrosis (Senevirathna *et al.*, 2023). The insidious, asymptomatic progression of this foodborne toxicological syndrome eventually culminates in end-stage renal disease (ESRD), requiring renal replacement therapy or causing premature mortality (Wasana *et al.*, 2021).

Macro-environmental drivers and diagnostic surveillance methodologies

Climate change and supply chain globalization

Environmental climate change reconfigures the geographic boundaries and seasonal survival patterns of foodborne pathogens (WHO, 2024). Warmer coastal waters accelerate the proliferation of pathogenic *Vibrio* species in marine zones, while elevated ambient heat profiles accelerates the replication dynamics of *Salmonella* in open airfields (Azigi and Adrah, 2025). Severe flooding also overwhelms wastewater infrastructures, causing raw sewage to contaminate irrigation resources (Mehta *et al.*, 2023). Concurrently, the globalization of food supply chains means that a single localized contamination node can be rapidly distributed across multiple international borders within hours, making traceback investigations exceptionally difficult (WHO, 2024).

Advanced diagnostics and one health frameworks

To counter these evolving biological and chemical threats, food safety systems are

moving toward rapid, high-throughput technologies. Accordingly, whole-genome sequencing (WGS) and metagenomics allow for single-nucleotide resolution and enable single-nucleotide-resolution mapping of pathogens, enabling real-time tracebacks during international outbreaks (Tadesse and McDermott, 2025). Furthermore, the implementation of the WHO Global Strategy for Food Safety (2022–2030) demands a unified One Health framework that links human clinical medicine, veterinary surveillance, and environmental toxicology to mitigate risks before they infiltrate the human dietary matrix.

CONCLUSIONS AND FUTURE RECOMMENDATIONS

Over the past several decades, ensuring food safety has persisted as a complex, rapidly transforming global public health priority. Conventional sanitation protocols, although fundamental, are no longer capable of independently addressing the modern global food network, a system increasingly strained by anthropogenic climate change, widespread antimicrobial resistance, environmental pollution, and complex international trade routes. Furthermore, the pervasive bioaccumulation of microplastics, heavy metals (such as lead, cadmium, and arsenic), and residual chemical compounds from agrochemicals and fertilizers represents an escalating global health emergency. Concurrently, traditional biological agents, including foodborne viruses (such as Norovirus and Hepatitis A), pathogenic bacteria, toxigenic fungi, and parasites remain enduring scientific concerns. A major amplifier of this burden is the worldwide rise in antimicrobial resistance (AMR), driven by extensive antibiotic overuse in livestock and agriculture; notable examples include Methicillin-Resistant *Staphylococcus aureus* (MRSA) and extended-spectrum beta-lactamase (ESBL)-producing *Enterobacteriaceae*. Effectively mitigating the global morbidity and mortality associated with foodborne illnesses necessitates an active, sustained transition toward next-generation surveillance systems. Incorporating advanced diagnostic methodologies, such as high-throughput whole-genome sequencing (WGS), CRISPR-Cas-based molecular assays, and nanotechnology-driven elec-

trochemical biosensors into routine monitoring enable real-time pathogen identification and rapid intervention across the supply chain.

Contemporary peer-reviewed literature highlights a significant conceptual shift regarding the biological assessment and regulatory scrutiny of food additives. Although industrial additives remain indispensable for extending shelf life, mitigating spoilage, reducing food waste, and stabilizing food security, the ubiquitous consumption of synthetic emulsifiers, artificial colorants, and chemical preservatives in ultra-processed foods demands stricter health policies and modernized dietary recommendations. Comprehensive longitudinal cohort studies and multi-omics research are essential to systematically map the physiological mechanism by which chronic, low-dose additive exposure induces gut microbial dysbiosis, disrupts metabolic homeostasis, increases intestinal permeability, and triggers systemic low-grade inflammation. At the policy and governance scale, global adoption of the integrated One Health framework is imperative to disrupt transmission chains across human, animal, and environmental domains. By deploying robust, technologically resilient food safety infrastructure and expanding international scientific collaboration, the global community can strengthen supply chain integrity, shield vulnerable demographics, and substantially alleviate the preventable global socio-economic burden of foodborne diseases.

AUTHOR CONTRIBUTION

Conceptualization, literature survey, literature collection, data interpretation, writing the first draft, manuscript editing and reviewing: VSJ

DECLARATION OF CONFLICT OF INTEREST

The author confirms that they have no conflicts of interest to declare.

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