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Review

Foodborne microbial diseases: pathogens, epidemiology, and prevention strategies

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Abstract: Foodborne illnesses represent a major global public health and economic challenge. According to WHO estimates for the year 2010, approximately 600 million people fell ill and 420,000 died as a result of contaminated food. This narrative review summarizes the main bacterial, viral and parasitic agents responsible for foodborne disease worldwide, with a particular focus on key bacterial pathogens (*Salmonella*, *Campylobacter*, pathogenic *Escherichia coli*, *Listeria monocytogenes*, *Staphylococcus aureus*, *Clostridium perfringens*, *Bacillus cereus* and others). For each pathogen group, we outline microbiology, epidemiology, pathogenesis, clinical manifestations and selected outbreak examples. The review further discusses the farm-to-fork continuum, microbiological risk assessment, and critical control points, emphasizing evidence-based prevention strategies such as Good Agricultural Practices, Good Manufacturing Practices, Hazard Analysis and Critical Control Points systems and consumer education. Emerging challenges—including antimicrobial resistance, climate change and globalization of food supply chains—are analysed within a One Health framework. This synthesis aims to inform food safety professionals, public health practitioners and policymakers in designing more effective interventions to reduce the global burden of foodborne diseases.

Keywords: foodborne diseases; foodborne pathogens; microbiological risk assessment; antimicrobial resistance; One Health; HACCP

1. Introduction

Foodborne diseases (FBDs) are illnesses that arise following ingestion of food or water contaminated with infectious agents (bacteria, viruses, parasites, prions) or toxic substances (microbial toxins, natural toxins, chemical contaminants) that reach the gastrointestinal tract and, in some cases, other organ systems (Riemann and Cliver, 2006).

From a public health and food safety perspective, foodborne diseases (FBDs) are commonly classified according to their underlying pathogenic mechanisms. In many cases, illness results from foodborne infections, which occur following the ingestion of viable pathogenic microorganisms, such as *Salmonella*, *Campylobacter*, *Listeria*, enteric viruses, and numerous parasites, that are capable of colonizing or invading the host. In other situations, disease is instead caused by foodborne intoxications, where pre-formed microbial toxins present in contaminated food are ingested, including the enterotoxins of *Staphylococcus aureus*, the neurotoxin of *Clostridium botulinum*, and the emetic toxin of *Bacillus cereus*. Between these two mechanisms lies foodborne toxicoinfection, in which consumption of large numbers of vegetative bacterial cells leads to their multiplication within the intestinal tract and subsequent in situ toxin production, as exemplified by *Clostridium perfringens*

type A and the diarrheal form of *Bacillus cereus* infection (Riemann and Cliver, 2006; Abebe *et al*., 2020). Together, these distinctions underscore that the clinical presentation and etiology of foodborne illness are fundamentally determined by whether disease arises from infection, intoxication, or toxicoinfection.

FBDs may present as mild, self-limited gastroenteritis or as invasive systemic disease (e.g., listeriosis, typhoid fever, brucellosis), neurological syndromes (botulism, prion diseases), chronic sequelae (reactive arthritis, Guillain–Barré syndrome, irritable bowel syndrome) or extra-intestinal parasitic disease (e.g., neurocysticercosis, toxoplasmosis) (Havelaar *et al*., 2015; Checkley *et al*., 2015).

In the Codex framework, hazards in food are broadly categorized as biological, chemical and physical. Biological hazards include bacteria, viruses, parasites and prions; chemical hazards include naturally occurring toxins (e.g., mycotoxins, marine biotoxins), process contaminants (e.g., acrylamide), industrial contaminants (e.g., dioxins), pesticide residues and veterinary drug residues; physical hazards include foreign materials such as glass, metal fragments and stones.

Although this review focuses on microbial hazards, chemical contaminants like aflatoxins and methylmercury also contribute measurably to the global burden of FBDs and are included in WHO FERG estimates (Gibb *et al*., 2015). For instance, a recent risk assessment of AFB1 in tomato and pepper pastes in Turkey highlighted the ongoing concern regarding dietary exposure to mycotoxins in processed foods (Aydemir Atasever *et al*., 2025).

In contrast to many bacterial hazards, enteric viruses do not cause visible spoilage or sensory changes in foods; instead, foods typically act only as passive vehicles for their transmission from infected hosts or contaminated environments.

Historically, many illnesses now recognized as foodborne were attributed to vague entities such as “ptomaine poisoning” or “miasma.” The late 19th and early 20th centuries saw the identification of specific aetiologic agents including *Salmonella enterica*, *Staphylococcus aureus*, *Clostridium botulinum* and *Escherichia coli*, and clarification that improper storage, processing and cooking of foods could facilitate their transmission (Riemann and Cliver, 2006).

Over the past decades, progress in refrigeration, pasteurization, canning, water treatment, sanitation and vaccination (e.g., against typhoid fever and hepatitis A) has substantially reduced some classical foodborne diseases in high-income settings (Havelaar *et al*., 2015). At the same time, new patterns of disease have emerged, linked to intensification of animal production, globalization of trade, industrial-scale processing, ready-to-eat (RTE) foods and changing consumer preferences for minimally processed or raw foods (Abebe *et al*., 2020; Hassan *et al*., 2023).

Contemporary food systems are characterized by long, complex supply chains that extend from primary production through processing, distribution and retail to the consumer. Contamination may occur at any point along this “farm-to-fork” continuum, and hazards can multiply or persist if control measures fail (Özlü and Atasever, 2018; Okafor, 2024).

Key features of contemporary food systems play a critical role in shaping the risk of foodborne diseases (FBDs). In particular, the intensification of animal production, often involving high stocking densities, promotes the maintenance and circulation of zoonotic pathogens such as *Salmonella*, *Campylobacter*, and Shiga toxin–producing *Escherichia coli* within animal reservoirs. These upstream risks are compounded by centralized, large-scale processing systems, in which a single contamination event can result in the extensive distribution of contaminated products before detection. At the same time, the globalization of trade in fresh produce and animal-derived foods enables foodborne pathogens to cross national borders with increasing speed and efficiency. Downstream in the food chain, the continued expansion of the ready-to-eat (RTE) and food service sectors has diminished the consumer’s role in applying final microbiological control steps, thereby shifting greater responsibility for risk management to food businesses and food handlers (EFSA and ECDC, 2021; Davydova *et al*., 2025). Collectively, these interrelated characteristics underscore the growing complexity and scale of foodborne disease risks in modern food systems.

These developments have driven a shift from purely end-product testing toward preventive, risk-based food safety systems such as HACCP and microbiological risk assessment, supported by improved surveillance and laboratory capacity (Havelaar *et al*., 2015).

It is crucial to distinguish “hazard” (a biological, chemical or physical agent with the potential to cause harm) from “risk” (the probability and severity of adverse health effects as a function of hazard presence and exposure).

Because many foodborne pathogens are zoonotic and influenced by environmental conditions, FBD prevention is inherently a One Health issue, requiring coordinated action across human, animal and environmental health sectors (Abebe *et al*., 2020; EFSA and ECDC, 2021; Velazquez-Meza *et al*., 2022).

This narrative review is based on literature retrieved from PubMed, Scopus and Web of Science. Searches were performed between 2000 and 2025 using combinations of the keywords “foodborne disease(s)”, “foodborne infection”, “foodborne pathogens”, “microbiological risk assessment”, “burden of disease”, “antimicrobial resistance”, “One Health”, “foodborne parasites”, “norovirus”, “climate change” and the names of specific pathogens and food vehicles. Priority was given to reports from FAO, WHO, WOAH and Codex Alimentarius, major burden-of-disease studies (including the WHO Foodborne Disease Burden Epidemiology Reference Group estimates), recent systematic reviews and large outbreak investigations. Additional references were identified from the reference lists of key articles and relevant guidelines. The review focuses on hazards of global importance and is not intended to be an exhaustive catalogue of all reported foodborne pathogens or national outbreaks. Pathogens were selected based on global burden, outbreak frequency, severity of outcomes, and relevance to contemporary food systems.

2. Global burden and epidemiology

2.1. Measuring the burden of foodborne disease

Traditional indicators of foodborne disease burden, such as outbreak counts and reported cases in surveillance systems, capture only a limited proportion of the true burden, as many infections are mild, self-limiting, or never laboratory-confirmed, a pattern often described as the “iceberg phenomenon” (Scallan *et al*., 2011). In response to these limitations, the Foodborne Disease Burden Epidemiology Reference Group (FERG) developed a comprehensive methodological framework to estimate the global and regional burden of foodborne diseases. The methodology employed by FERG integrated multiple complementary evidence streams to generate robust burden estimates, systematically combining data from extensive reviews of the scientific literature on disease incidence, exposure, and etiologic fractions with empirical data from national surveillance systems and burden-of-disease studies. To address critical data gaps, particularly in regions with limited surveillance capacity, the approach was further supplemented by structured expert elicitation. These diverse inputs were ultimately synthesized using the standardized metric of disability-adjusted life years (DALYs), which integrates both fatal outcomes, expressed as years of life lost, and non-fatal health consequences, expressed as years lived with disability, into a single coherent measure of disease burden (Havelaar *et al*., 2015).

2.2. Global and regional patterns

The WHO estimates that, in 2010, the 31 priority foodborne hazards caused 600 million illnesses and 420,000 deaths worldwide, corresponding to 33 million DALYs (Havelaar *et al*., 2015). These estimates refer to the global burden in 2010 and are commonly interpreted as representing the burden occurring in a typical recent year in the absence of major secular changes.

The global burden of foodborne disease is profoundly and unevenly distributed across regions. The highest rates of disease, expressed as disability-adjusted life years (DALYs) per 100,000 population, are observed in low-income settings, particularly in sub-Saharan Africa, South-East Asia, and parts of the Eastern Mediterranean. These elevated burdens largely reflect persistent structural challenges, including unsafe water supplies, inadequate sanitation, undernutrition, and limited capacity to control zoonotic pathogens and foodborne parasites. By contrast, the WHO European Region exhibits the lowest estimated per-capita burden. Nevertheless, this lower relative burden should not be interpreted as trivial, as contaminated food is still estimated to cause more than 23 million illnesses and approximately 5,000 deaths each year in the region, highlighting that foodborne disease remains a substantial public health concern even in settings with advanced food safety systems (WHO Regional Office for Europe, 2017).

In high-income settings, diarrhoeal pathogens such as norovirus, nontyphoidal *Salmonella* and *Campylobacter* dominate in case counts, whereas in many low- and middle-income settings, parasites (e.g., *Toxoplasma gondii*, *Taenia solium*, *Echinococcus* spp.) and chemical contaminants (e.g., aflatoxins) contribute substantially to deaths and DALYs (Havelaar *et al*., 2015).

2.3. Contributions of bacteria, viruses and parasites

A global synthesis of foodborne disease epidemiology indicates that bacterial pathogens account for approximately two-thirds of all cases, with viruses and parasites responsible for most of the remaining burden (Havelaar *et al*., 2015; Abebe *et al*., 2020; Okafor, 2024). Viral agents, particularly norovirus and hepatitis A virus, are major causes of acute gastroenteritis worldwide, with norovirus alone estimated to cause approximately 700 million cases annually (Carlson *et al*., 2024; Zhu *et al*., 2025). Historically, rotaviruses have contributed substantially to severe diarrhoeal disease and hospital admissions among infants and young children, especially in low- and middle-income countries prior to the widespread introduction of vaccination programmes.

In contrast, bacterial pathogens—including nontyphoidal *Salmonella*, *Campylobacter*, Shiga toxin–producing *Escherichia coli* (STEC), *Listeria monocytogenes*, and *Staphylococcus aureus*—are more frequently associated with severe clinical outcomes, including hospitalization and mortality, across many regions (Scallan *et al*., 2011; Abebe *et al*., 2020; Hassan *et al*., 2023). Although parasitic infections such as toxoplasmosis, cysticercosis, echinococcosis, and cryptosporidiosis account for a smaller proportion of total cases, they are often characterized by high severity and long-term sequelae, resulting in a disproportionate contribution to the overall burden measured in disability-adjusted life years (DALYs) (Havelaar *et al*., 2015).

These global patterns are mirrored in national-level estimates. In the United States, for instance, Scallan *et al*. (2011) estimated that 31 major domestically acquired foodborne pathogens cause approximately 9.4 million illnesses, 56,000 hospitalizations, and 1,351 deaths annually. While norovirus accounted for 58% of all illnesses, severe outcomes were driven primarily by bacterial and parasitic pathogens, with nontyphoidal *Salmonella* responsible for 35% of hospitalizations and *Toxoplasma gondii* (24%) and *Listeria monocytogenes* (19%) accounting for a substantial proportion of deaths.

Recent EU One Health zoonoses reports similarly identify campylobacteriosis and salmonellosis as the first and second most frequently reported zoonoses, with STEC infections, yersiniosis and listeriosis also important; listeriosis and West Nile virus infections show the highest hospitalisation rates among zoonoses (EFSA and ECDC, 2024). For example, surveillance data from both Europe and North America consistently rank campylobacteriosis among the most frequently notified bacterial enteric infections, with substantial numbers of hospitalizations recorded each year.

2.4. Vulnerable populations

Burden-of-disease analyses highlight that children under five years carry about 40% of FBD DALYs, despite representing a much smaller fraction of the global population (Havelaar *et al*., 2015). Young children, pregnant women, older adults and immunocompromised individuals are particularly susceptible to severe disease and adverse outcomes from *Salmonella*, *Campylobacter*, STEC, *Listeria*, norovirus and many parasitic infections (Farber and Peterkin, 1991; Checkley *et al*., 2015).

Social and economic determinants strongly shape exposure and resilience. Populations living in poverty, informal settlements or rural areas with limited access to safe water, sanitation and healthcare often experience higher incidence and worse outcomes from FBDs (Havelaar *et al*., 2015; Okafor, 2024). Occupational exposure among food handlers, slaughterhouse workers, veterinarians and agricultural labourers also increases risk.

2.5. Underreporting, attribution and emerging data sources

Because many cases are mild, self-treated or never tested, underreporting and underdiagnosis remain major challenges. Multipliers are often applied to reported case counts to estimate the true burden, but uncertainty remains high (Havelaar *et al*., 2015).

A central epidemiological challenge in food safety is source attribution, defined as the process of identifying the specific foods, animal reservoirs, or environmental transmission routes responsible for human infections. Addressing this challenge requires the integration of multiple complementary methodological approaches. Classical epidemiological strategies include outbreak-based attribution, in which cases are assigned to food categories implicated in investigated outbreaks, as well as analytical designs such as case–control and case–case studies. Within risk assessment frameworks, comparative exposure assessment and structured expert elicitation are commonly used to estimate the relative contributions of different sources. At the molecular level, microbial subtyping methods, including serotyping and pulsed-field gel electrophoresis, have long supported source attribution efforts; however, these approaches are increasingly being augmented, and in many contexts replaced, by whole-genome sequencing of clinical isolates combined with systematic sampling from potential reservoirs. This integration provides high-resolution evidence for linking human infections to specific sources and transmission pathways (Havelaar *et al*., 2015; Davydova *et al*., 2025).

Emerging data sources—such as syndromic surveillance, prescription data, consumer complaint systems and wastewater-based monitoring—may complement traditional notification-based systems to improve early detection of FBD events and refine burden estimates (Hassan *et al*., 2023; Carlson *et al*., 2024).

2.6. Surveillance systems and outbreak investigation

Effective control of foodborne diseases relies on multi-layered surveillance systems, which are implemented in many countries. These systems typically integrate multiple complementary components to detect, monitor, and investigate threats across the farm-to-fork continuum. A core element is the passive notification of laboratory-confirmed human infections, including salmonellosis, listeriosis, and Shiga toxin–producing *Escherichia coli*

(STEC). To improve the accuracy of incidence estimates, this information is often supplemented by sentinel or active surveillance networks, such as FoodNet in the United States. In parallel, dedicated outbreak surveillance systems compile data from epidemiological cluster investigations to identify implicated foods and transmission pathways. Beyond human health surveillance, specific monitoring programmes target zoonotic and foodborne pathogens in animal populations, food products, and environmental reservoirs, thereby supporting a comprehensive One Health surveillance framework (CDC, 2023; EFSA and ECDC, 2024).

A standard outbreak investigation proceeds through verification of the outbreak, development of a case definition, case finding, descriptive epidemiology (time–place–person), hypothesis generation, analytic studies (e.g., cohort or case–control), and microbiological investigation of clinical, food and environmental specimens, followed by implementation of control measures (Riemann and Cliver, 2006; Heymann, 2015).

3. Microbial food safety risk assessment

3.1. Risk analysis framework

Microbiological risk assessment (MRA) provides a structured and systematic approach for estimating the likelihood and severity of adverse health effects resulting from pathogenic microorganisms or their toxins in foods, thereby supporting evidence-based risk management decisions, including the establishment of microbiological criteria, performance objectives, and HACCP-based control measures. Within the broader tripartite risk analysis framework, which encompasses risk assessment, risk management, and risk communication, MRA constitutes the core scientific component underpinning food safety decision-making. As defined within the Codex Alimentarius system, MRA is a science-based process consisting of four interrelated steps: hazard identification, hazard characterization, including dose–response assessment, exposure assessment, and risk characterization (CAC/GL 30–1999). Together, these components provide a qualitative or quantitative basis for estimating public health risks associated with foodborne hazards and for transparently linking scientific evidence to risk management actions.

3.2. Hazard identification

The initial and foundational step in microbiological risk assessment is hazard identification, which involves the systematic compilation and evaluation of scientific evidence to determine whether a specific microorganism or toxin present in food is capable of causing adverse effects in humans. This process draws on multiple lines of evidence, including epidemiological data from outbreaks and sporadic cases, findings from experimental infection studies and dose–response assessments, information on ecological reservoirs and the ability of the agent to survive, grow, or produce toxins in food matrices, as well as established knowledge of virulence factors and pathogenicity mechanisms (Riemann and Cliver, 2006). A well-established example is the hazard identification of *Listeria monocytogenes* in ready-to-eat (RTE) chilled foods, which has identified this pathogen as a serious hazard, particularly for pregnant women and immunocompromised individuals. This conclusion reflects its capacity to grow at refrigeration temperatures, persist in food processing environments, and cause invasive listeriosis, a disease associated with high hospitalization and case-fatality rates (Farber and Peterkin, 1991).

3.3. Hazard characterization and dose–response

Hazard characterization, the second component of microbiological risk assessment, describes the nature and severity of adverse health effects associated with a given pathogen or toxin and establishes the corresponding dose–response relationship. This relationship links the ingested dose to the probability and severity of illness, providing a critical basis for estimating health risks. For microbial hazards, the development of dose–response models is inherently complex and must account for multiple interacting determinants. These include pathogen-specific characteristics, such as virulence factors and toxin production; host-related factors, including age, immune status, pregnancy, and underlying disease; the route and frequency of exposure; and the temporal course of infection, distinguishing between acute disease and chronic sequelae. Through this detailed characterization, risk assessors can evaluate potential health outcomes across different population groups and exposure scenarios.

Various mathematical forms—exponential, Beta–Poisson and others—have been used to describe dose–response relationships for pathogens such as *Salmonella* spp., *Campylobacter jejuni*, STEC, *L. monocytogenes* and norovirus (Havelaar *et al*., 2015). For some pathogens (e.g., *L. monocytogenes*), models may incorporate age-specific or risk-group–specific parameters to capture the markedly higher susceptibility of vulnerable groups (Farber and Peterkin, 1991; Radoshevich and Cossart, 2018). The concept of a minimum infectious dose—ranging from only a few viral particles for some enteric viruses to very high cell numbers for certain

toxin-forming bacteria—thus represents a critical bridge between hazard characterization and exposure assessment.

3.4. Exposure assessment

Exposure assessment, the third component of microbiological risk assessment, aims to estimate the likely intake of a pathogen or toxin by the target consumer population. This step involves the systematic integration of information from multiple domains. It considers the occurrence and concentration of the hazard in raw materials and foods at different stages along the food chain, together with the effects of processing, storage, distribution, and preparation practices. These include factors such as cooking lethality, holding temperatures, cross-contamination, and typical consumer handling, all of which may reduce, amplify, or redistribute microbial hazards. Exposure estimates are further refined using data on food consumption patterns, encompassing the amounts and frequencies of foods consumed by different demographic groups. Importantly, exposure assessment must explicitly address variability and uncertainty across all inputs in order to characterize the resulting distribution of exposures and support robust risk characterization.

Exposure models may be deterministic or probabilistic (e.g., Monte Carlo simulation) and can range from simple screening-level assessments to complex, dynamic models of farm-to-fork pathways. Examples include models of *Salmonella* in eggs and broiler meat, *Campylobacter* in poultry, *L. monocytogenes* in RTE foods, and norovirus in shellfish and fresh produce (Havelaar *et al*., 2015).

3.5. Risk characterization

Risk characterization represents the final step of microbiological risk assessment and integrates the outputs of hazard characterization and exposure assessment to generate quantitative or qualitative estimates of public health risk. These estimates may be expressed in several complementary ways, including the probability of infection or illness per serving of a given food, the expected annual number of cases, hospitalizations, or deaths in a defined population, and the distribution of risk across different or vulnerable subpopulations. In addition, risk characterization provides a framework for evaluating the potential public health impact of specific control measures or interventions implemented along the food chain.

Risk characterization should transparently describe assumptions, data gaps, model structure and uncertainties, as these affect interpretation and subsequent risk management decisions.

3.6. Examples of microbiological risk assessment

The structured framework of microbiological risk assessment (MRA) has been applied in a number of influential assessments conducted by FAO/WHO and national authorities, demonstrating how scientific evidence can be translated into practical food safety policy. A joint FAO/WHO MRA on *Listeria monocytogenes* in ready-to-eat foods, for example, showed that the risk of invasive listeriosis is strongly influenced by initial contamination levels, product characteristics, and storage time and temperature, and that even low levels of contamination can pose a serious risk to susceptible populations. Similarly, MRAs addressing *Salmonella* in eggs and poultry have been used to quantitatively evaluate interventions such as on-farm vaccination, improved hygiene, refrigeration, and pasteurization, thereby supporting the establishment of performance objectives and microbiological criteria. In the case of *Campylobacter* in broiler chickens, risk models have assessed the population-level impact of carcass decontamination, on-farm biosecurity, freezing of highly contaminated carcasses, and consumer cooking practices, requiring detailed consideration of the pathogen's narrow optimal growth range and sensitivity to environmental stresses. Quantitative MRAs for Shiga toxin–producing *Escherichia coli* (STEC) in ground beef and leafy greens have likewise informed risk management decisions related to testing strategies, production practices, and cooking recommendations. Taken together, these examples illustrate how MRA can be used to evaluate the effects of changes at different points along the farm-to-fork continuum and to prioritize control measures based on their expected risk reduction and cost-effectiveness (Farber and Peterkin, 1991; Havelaar *et al*., 2015).

3.7. From risk assessment to risk management and microbiological criteria

Risk assessment provides the scientific basis for risk management, which encompasses the selection and implementation of appropriate control options to reduce foodborne risks. These options may include the establishment of microbiological criteria for specific pathogens in defined foods, the specification of process criteria such as validated time–temperature combinations, and the setting of performance objectives (POs) or food safety objectives (FSOs) that link control measures to acceptable levels of public health protection. Risk

management further involves the development, implementation, and verification of Hazard Analysis and Critical Control Point (HACCP) plans and prerequisite programmes, as well as effective communication with stakeholders, including industry, regulatory authorities, and consumers (CAC/GL 63-2007). Codex guidelines emphasize that microbiological criteria should be risk-based and scientifically justified and applied within an integrated risk management framework, rather than used as the sole determinant of food safety. In practice, this approach entails combining end-product testing with preventive hygiene measures, process control, and verification activities as part of a comprehensive food safety management system.

4. Major bacterial foodborne pathogens

Bacterial agents remain among the most important causes of foodborne disease worldwide. Reviews consistently identify nontyphoidal *Salmonella* spp., *Campylobacter* spp., pathogenic *E. coli*, *Listeria monocytogenes*, *Staphylococcus aureus*, *Clostridium perfringens* and *Bacillus cereus* as key zoonotic and foodborne pathogens (Abebe *et al*., 2020; Okafor, 2024; EFSA and ECDC, 2024).

4.1. Nontyphoidal *Salmonella* spp.

4.1.1. Reservoirs and transmission

Nontyphoidal *Salmonella enterica* serovars (e.g., Enteritidis, Typhimurium) colonize the intestinal tract of many animals—especially poultry, pigs, cattle and reptiles—and contaminate a wide variety of foods, including eggs, chicken, pork, beef, raw milk, fresh produce and low-moisture foods such as nuts and spices (Havelaar *et al*., 2015; Abebe *et al*., 2020).

Transmission occurs predominantly via ingestion of contaminated food or water, or through direct animal contact. Vertical transmission in poultry (e.g., *S. Enteritidis* in eggs) has been particularly important in human epidemics.

4.1.2. Clinical manifestations and burden

Nontyphoidal *Salmonella* usually causes acute self-limited gastroenteritis with fever, diarrhea and abdominal cramps, but can cause invasive disease and bacteremia, especially in infants, older adults and immunocompromised individuals (Franco *et al*., 2007; Fabrega and Vila, 2013). Invasive nontyphoidal *Salmonella* is a significant cause of bloodstream infection and death in parts of sub-Saharan Africa, often associated with HIV infection, malaria and malnutrition (Havelaar *et al*., 2015).

Scallan *et al*. (2011) estimated that nontyphoidal *Salmonella* causes about 1 million illnesses, 19,000 hospitalisations and 378 deaths per year in the US, representing a major contributor to foodborne disease burden. Large national and multi-country outbreaks linked to eggs, broiler meat, pork and low-moisture products such as nuts and powdered foods illustrate how contamination at centralized processing plants can expose thousands of consumers across wide geographic areas.

4.1.3. Control strategies

Control spans farm-to-fork: on-farm biosecurity and vaccination, feed control, hygiene at slaughter and processing, strict temperature control, consumer education on cooking and cross-contamination, and risk-based microbiological criteria for *Salmonella* in meat and eggs (EFSA and ECDC, 2024). In traditional dairy products like Civil cheese (produced in Erzurum, Turkiye), parameters such as milk acidity and salting technique are critical control points that directly affect microbiological safety and quality.

4.2. *Campylobacter* spp.

4.2.1. Epidemiology and reservoirs

Campylobacter jejuni and *C. coli* are now among the most commonly reported bacterial causes of gastroenteritis in many high-income countries. The EU Zoonoses reports have repeatedly identified campylobacteriosis as the leading zoonosis reported in humans since 2005 (EFSA and ECDC, 2024).

Poultry flocks are major reservoirs; *Campylobacter* colonizes the intestinal tract of chickens without causing disease, leading to high contamination rates of broiler carcasses at slaughter. Other reservoirs include ruminants and pets, and transmission may occur via contaminated milk and water. *C. jejuni* is a spiral, motile and microaerophilic organism that grows best at 42–43 °C under reduced oxygen tension, is unable to ferment carbohydrates, and relies mainly on amino acids as energy sources, features that help explain its adaptation to the intestinal tract of warm-blooded animals.

4.2.2. Disease and sequelae

Illness typically presents as acute watery or bloody diarrhea, abdominal cramps and fever, with an incubation period of 2–5 days. Although usually self-limited, *Campylobacter* infection is associated with several post-infectious sequelae, notably Guillain–Barré syndrome, reactive arthritis and irritable bowel syndrome (Dasti *et al*., 2010; Havelaar *et al*., 2015). In addition to sporadic cases associated with poultry, important outbreaks have been linked to unpasteurised milk and inadequately treated drinking water, underscoring the close connections between food and water safety in campylobacteriosis epidemiology. The combination of a very low infectious dose and the potential for serious post-infectious sequelae such as Guillain–Barré syndrome and reactive arthritis underscores the disproportionate public health impact of campylobacteriosis relative to its often self-limiting clinical course.

4.2.3. Control and challenges

Control focuses on reducing flock colonization (biosecurity, feed and water treatment, potential vaccination), improving hygiene at slaughter and processing, and ensuring adequate cooking and separation of raw poultry in the kitchen. No fully effective on-farm intervention has yet been widely implemented, and fluoroquinolone resistance in *Campylobacter* is a major concern (EFSA and ECDC, 2021, 2024).

4.3. Pathogenic *Escherichia coli*

4.3.1. Pathotypes and reservoirs

While *Escherichia coli* is a normal commensal inhabitant of the intestinal tract of humans and warm-blooded animals, several distinct pathogenic pathotypes are well-established causes of diarrhoeal disease. These include Shiga toxin–producing *E. coli* (STEC, also referred to as enterohaemorrhagic *E. coli* or EHEC), enterotoxigenic *E. coli* (ETEC), which is a major cause of travellers' diarrhoea and childhood diarrhoea in low-income settings, as well as enteropathogenic *E. coli* (EPEC), enteroaggregative *E. coli* (EAEC), and enteroinvasive *E. coli* (EIEC) (Riemann and Cliver, 2006). Among these pathotypes, STEC—including serotype O157:H7 and numerous non-O157 serogroups—represents a particular concern in the context of foodborne transmission. Cattle are the principal reservoir for STEC, although other ruminants may also harbour these strains. Human infection most commonly occurs through the consumption of contaminated foods, such as undercooked ground beef, raw milk, unpasteurised juices, and fresh produce, as well as through exposure to contaminated recreational water and person-to-person transmission (Havelaar *et al*., 2015).

4.3.2. Clinical features and complications

STEC infection ranges from mild diarrhea to hemorrhagic colitis. A proportion of cases, especially in children, progress to hemolytic uremic syndrome (HUS) with acute kidney injury, hemolytic anemia and thrombocytopenia, often requiring dialysis and intensive care (Riemann and Cliver, 2006).

Major outbreaks—such as the 2011 O104:H4 outbreak in Europe linked to fenugreek sprouts—illustrate the potential of STEC to cause large, severe foodborne events associated with non-traditional vehicles like sprouts and leafy greens (Havelaar *et al*., 2015). These events demonstrate that leafy greens, sprouts and other minimally processed plant-based foods can serve as high-impact vehicles for STEC, even in high-income countries with advanced food control systems.

4.3.3. Prevention

Control measures include farm-level interventions to reduce shedding in cattle, hygienic slaughter and processing, "cook, chill, separate" messages for consumers, and stringent hygiene for foods eaten raw (e.g., salads, sprouts). For STEC, antibiotic therapy is generally avoided because of concern about increased toxin release (Heymann, 2015).

4.4. *Listeria monocytogenes*

4.4.1. Ecology and food vehicles

Listeria monocytogenes is an environmental, psychrotrophic, Gram-positive bacterium capable of growth at refrigeration temperatures and survival in processing environments, including biofilms on equipment and surfaces (Farber and Peterkin, 1991; Naik *et al*., 2023).

High-risk foods include RTE refrigerated foods such as soft cheeses, deli meats, smoked fish, pâtés and prepackaged salads. Because it can grow gradually during storage, small initial contamination can lead to high levels at the end of shelf life.

4.4.2. Disease and high-risk groups

Listeriosis mainly affects pregnant women, neonates, older adults and immunocompromised individuals, causing severe outcomes: septicemia, meningitis, encephalitis and fetal loss. Outbreaks associated with contaminated RTE meats, soft cheeses and produce have high case-fatality rates (around 20–30%) (Farber and Peterkin, 1991; EFSA and ECDC, 2024; Atasever, 2025a). Recurrent outbreaks associated with ready-to-eat meats, soft cheeses made from unpasteurised milk and prepackaged salads, often with high case-fatality rates, have driven increasingly stringent regulatory expectations for environmental monitoring and product testing in high-risk food sectors.

4.4.3. Regulatory approaches

Because of its severity and growth at refrigeration temperatures, many countries apply stringent microbiological criteria for *L. monocytogenes* in RTE foods (e.g., absence in 25 g or ≤100 CFU/g throughout shelf life) and require food business operators to implement environmental monitoring, hygienic design and strict temperature control (Farber and Peterkin, 1991).

4.5. *Staphylococcus aureus*

S. aureus is a common commensal of human skin and nasal mucosa. Certain strains produce heat-stable enterotoxins in food subject to temperature abuse. Because these toxins resist usual cooking temperatures, reheating does not prevent disease once they have formed (Kadariya *et al*., 2014; Abebe *et al*., 2020).

Staphylococcal food poisoning is characterized by rapid onset (1–6 h) of nausea, vomiting, abdominal cramps and sometimes diarrhea, typically resolving within 24–48 h. Common vehicles are high-protein RTE foods requiring extensive handling and storage at ambient temperatures, such as cream-filled pastries, salads and sliced meats (Kadariya *et al*., 2014). Control hinges on exclusion of ill food workers, good hand hygiene, minimizing bare-hand contact and strict temperature control.

4.6. *Clostridium perfringens*

C. perfringens type A forms heat-resistant spores that survive cooking and germinate in large, bulk-cooked dishes—such as institutional roasts, stews and gravies—when cooled slowly and held for long periods at warm temperatures (Grass *et al*., 2013). *C. perfringens* food poisoning therefore exemplifies a toxicoinfection, in which large numbers of vegetative cells are ingested with food and subsequently sporulate and produce enterotoxin in the intestine.

Illness is a toxicoinfection: ingestion of large numbers of vegetative cells is followed by sporulation and enterotoxin production in the intestine, leading to abdominal cramps and diarrhea 8–16 h after ingestion. Outbreaks are often associated with large-scale catering and institutional food service where time–temperature control failed.

Prevention focuses on rapid cooling in shallow containers, hot-holding above 60°C, reheating to adequate temperatures, and careful management of leftovers.

4.7. *Bacillus cereus*

B. cereus spores are common in the environment and in starchy foods such as rice, pasta and sauces. Two distinct syndromes are recognized: an emetic syndrome associated with pre-formed cereulide toxin in food, and a diarrheal syndrome associated with enterotoxin production in the intestine (Bottone, 2010).

The emetic syndrome follows consumption of cooked rice or starchy foods held at room temperature for several hours, with sudden onset of vomiting; the diarrheal form is linked to a broader range of foods and presents with watery diarrhea and abdominal cramps after a longer incubation (Riemann and Cliver, 2006). Prevention relies on rapid cooling, refrigeration, and avoidance of prolonged holding in the temperature “danger zone.” Classical B. cereus outbreaks therefore often involve cooked rice, pasta or other starchy dishes that have been cooled slowly and held at ambient temperature, allowing spores to germinate and heat-stable emetic toxin to accumulate despite subsequent reheating.

4.8. *Yersinia enterocolitica* and other enteropathogens

Y. enterocolitica is a psychrotrophic enteric pathogen, with pigs as an important reservoir. It can grow at refrigeration temperatures and is associated with undercooked pork, raw milk and tofu, causing yersiniosis characterized by fever, abdominal pain and diarrhea; in older children and adults, pseudoappendicitis may occur.

Other bacterial enteropathogens relevant to FBD include *Shigella* spp., *Vibrio* spp. (especially *V. parahaemolyticus* and *V. vulnificus*), *Brucella* spp., *Mycobacterium bovis* and *Clostridium botulinum*, each with specific reservoirs, vehicles and clinical patterns (Abebe *et al*., 2020; Baker-Austin *et al*., 2018). Although *Brucella* spp. do not multiply in foods, they can survive for prolonged periods in raw milk, fresh cheeses and other dairy products, making these commodities important vehicles in regions where animal brucellosis remains endemic and pasteurisation is not consistently applied. An overview of selected major bacterial and viral foodborne pathogens, their typical reservoirs, key food vehicles, incubation periods and severe outcomes is provided in Table 1.

Table 1. Selected major bacterial and viral foodborne pathogens, typical reservoirs, key food vehicles, approximate incubation periods and major severe outcomes.

Pathogen	Main reservoir(s)	Key food vehicles	Typical incubation period*	Major severe outcomes
Nontyphoidal *Salmonella* spp.	Poultry, pigs, cattle, reptiles	Eggs, poultry meat, pork, beef, raw milk, fresh produce, nuts, spices	6–72 h	Bacteremia, invasive disease in infants and immunocompromised
Campylobacter jejuni / C. coli	Poultry, ruminants, pets	Poultry meat, raw milk, contaminated water	2–5 days	Guillain–Barré syndrome, reactive arthritis
Shiga toxin–producing *E. coli* (STEC)	Cattle and other ruminants	Undercooked ground beef, raw milk, sprouts, leafy greens, juices	2–10 days	Hemorrhagic colitis, haemolytic uraemic syndrome (HUS)
Listeria monocytogenes	Environment, livestock, food plants	RTE meats, soft cheeses, smoked fish, prepacked salads	3–70 days	Septicaemia, meningitis, fetal loss
Staphylococcus aureus	Humans (skin, nose), animals	RTE foods handled extensively, cream-filled pastries, sliced meats	1–6 h	Acute vomiting, dehydration
Clostridium perfringens	Environment, animals, humans	Bulk-cooked meat dishes, stews, gravies	8–16 h	Severe diarrhoea, abdominal cramps
Bacillus cereus (emetic/diarrhoeal)	Environment, cereals, spices	Cooked rice and pasta, sauces, mixed dishes	0.5–6 h / 8–16 h	Acute vomiting (emetic), watery diarrhoea (diarrhoeal)
Norovirus	Humans	RTE foods, fresh produce, bivalve shellfish, contaminated water/ice	12–48 h	Acute gastroenteritis, dehydration
Hepatitis A virus	Humans	Bivalve shellfish, fresh produce, contaminated water	15–50 days	Acute hepatitis, liver failure (rare)

*Typical incubation periods are broad ranges and may vary according to dose, host factors and specific strains.

5. Viral foodborne diseases

From a practical standpoint, food-related enteric viruses can be grouped into (i) gastroenteritis viruses such as noroviruses, rotaviruses, enteric adenoviruses, astroviruses and sapoviruses, (ii) enterically transmitted hepatitis viruses (hepatitis A and E) and (iii) other enteric viruses that may cause neurological or systemic disease, including certain enteroviruses.

5.1. Norovirus

5.1.1. Virology, classification and genomic diversity

Noroviruses (NoVs) are small, non-enveloped viruses with a single-stranded, positive-sense RNA genome of approximately 7.5–7.7 kb, classified within the family Caliciviridae (Carlson *et al*., 2024). The genome typically contains three open reading frames (ORFs): ORF1 encodes a polyprotein that is cleaved into non-structural proteins, including the RNA-dependent RNA polymerase (RdRp); ORF2 encodes the major capsid protein VP1; and ORF3 encodes the minor structural protein VP2 (Koo *et al*., 2010).

Noroviruses are divided into at least 10 genogroups (GI–GX), of which GI, GII, GIV, GVIII and GIX infect humans, and are further subdivided into numerous genotypes based on capsid sequence diversity (Carlson *et al*., 2024). Historically, GII.4 variants have dominated global epidemics, although other lineages such as GII.17 and GII.2 have periodically become prominent in certain regions, illustrating dynamic genotype turnover (Gao *et al*., 2025).

Substantial genetic and antigenic diversity, together with host factors such as histo-blood group antigen (HBGA) expression that influence susceptibility, complicates vaccine development and interpretation of seroepidemiological data (Koo *et al*., 2010; Tan and Jiang, 2024). These virological features help explain why norovirus causes repeated infections throughout life despite the development of strain-specific immunity.

5.1.2. Global burden and epidemiology

Norovirus is now recognized as one of the leading causes of acute gastroenteritis worldwide and the single most important cause of foodborne illness in many countries (Scallan *et al*., 2011; Carlson *et al*., 2024). The epidemiological significance and control challenges of foodborne viruses, including norovirus, have also been emphasized in regional contexts (Atasever *et al*., 2015). Global modeling suggests that norovirus is responsible for approximately 685–700 million cases of acute gastroenteritis annually, with an estimated 136,000–278,000 deaths, most of which occur in low- and middle-income countries and among young children and older adults (Carlson *et al*., 2024; Zhu *et al*., 2025).

From a foodborne perspective, norovirus accounts for roughly half of all foodborne illness outbreaks in the United States and a substantial proportion in Europe and other regions (Scallan *et al*., 2011; EFSA and ECDC, 2021; CDC, 2024). Verhoef *et al*. (2015) estimated that about 14% of all norovirus outbreaks are primarily foodborne**,** although secondary person-to-person and environmental transmission frequently amplifies and prolongs outbreaks (Silverberg, 2018).

Seasonality is well documented: norovirus activity peaks during cooler months (“winter vomiting disease”) in temperate climates, while tropics may show more complex or year-round patterns (Carlson *et al*., 2024). Outbreaks commonly occur in semi-closed settings—nursing homes, hospitals, schools, cruise ships and military barracks—where crowding and shared facilities facilitate rapid spread (Koo *et al*., 2010; Tan, 2024).

Recent data from several surveillance systems, including wastewater monitoring, suggest recurrent winter surges following relaxation of COVID-19 control measures, reflecting a rebound in norovirus transmission after pandemic-related non-pharmaceutical interventions (Carlson *et al*., 2024; Zhu *et al*., 2025).

5.1.3. Transmission routes and foodborne vehicles

Norovirus is characterized by highly efficient transmission through multiple, interconnected routes, including direct person-to-person spread via the faecal–oral route or exposure to infectious vomitus, indirect transmission through contaminated surfaces, fomites, and aerosols, as well as foodborne and waterborne pathways (Koo *et al*., 2010; Silverberg, 2018). From a food safety perspective, transmission is most commonly associated with foods that do not undergo a further microbial inactivation step after contamination. Key vehicles therefore include ready-to-eat foods, such as sandwiches and salads, contaminated by infected food handlers; fresh produce, including leafy greens and soft fruits, contaminated through polluted irrigation water or infected harvesters; and bivalve molluscan shellfish harvested from contaminated waters and consumed raw or lightly cooked (CDC, 2024; FDA, 2025). In addition, inadequate water treatment and distribution systems facilitate transmission through drinking water and ice (Gao *et al*., 2025). Together, these features underpin the exceptional transmissibility of norovirus and explain its dominant role as a cause of foodborne gastroenteritis worldwide.

Noroviruses are excreted at very high titers (up to 10^{11} genome copies per gram of stool) and exhibit exceptional environmental stability, remaining infectious for weeks on surfaces and surviving freezing and mild heating (Carlson *et al*., 2024). Infectious dose experiments and outbreak back-calculations suggest that as few as 10–100 virions may cause disease (Koo *et al*., 2010). These features explain the high secondary attack rates and the frequent observation of multiple, overlapping transmission pathways in outbreaks.

5.1.4. Pathogenesis and clinical manifestations

Norovirus infection primarily targets the small intestine**,** where the virus infects mature enterocytes and possibly immune cells in the lamina propria (Carlson *et al*., 2024). Histological changes include blunting of villi, crypt hyperplasia and infiltration of lamina propria with mononuclear cells; however, major structural damage is usually limited and reversible (Koo *et al*., 2010). Malabsorption, impaired brush border enzyme activity and secretory mechanisms contribute to watery diarrhea.

The incubation period ranges from 12 to 48 hours**,** followed by abrupt onset of vomiting, watery diarrhea, abdominal cramps, nausea, low-grade fever, headache and myalgia (CDC, 2024). Symptoms typically last 1–3 days in immunocompetent hosts, but illness may be prolonged in infants, older adults and immunocompromised patients, who may also experience complications such as dehydration, acute kidney injury and, rarely, death (Carlson *et al*., 2024; Tan, 2024).

Chronic norovirus infection has been increasingly recognized in solid-organ transplant recipients, patients with primary immunodeficiency and individuals receiving chemotherapy, leading to months of persistent diarrhea, weight loss and viral shedding (Carlson *et al*., 2024). Such chronic shedders may contribute to evolution of novel variants under immune pressure.

5.1.5. Immunity, reinfection and vaccines

Protective immunity to norovirus is incomplete and strain-specific**.** Experimental human challenge studies indicate that prior infection with a given strain confers short-term protection (few months to a couple of years), but heterologous protection is limited and reinfections with antigenically distinct variants are common throughout life (Koo *et al*., 2010; Carlson *et al*., 2024). The role of mucosal IgA, serum neutralizing antibodies and cellular responses in protection remains an active area of research (Tan and Jiang, 2024).

Multiple vaccine candidates are under development, including virus-like particle (VLP) formulations, nanoparticle vaccines and vector-based approaches. Early-phase clinical trials have demonstrated immunogenicity and partial protection against homologous challenge, but the high antigenic diversity of circulating strains and lack of clear correlates of protection complicate design of broadly protective vaccines (Tan and Jiang, 2024; Carlson *et al*., 2024). Target populations include young children, older adults, immunocompromised patients and residents of long-term care facilities.

5.1.6. Diagnosis and laboratory methods

Routine diagnosis in clinical and outbreak settings relies on RT-qPCR detection of norovirus RNA in stool samples, vomitus, environmental swabs, foods and water (Verhoef *et al*., 2015). Multiplex molecular panels for acute gastroenteritis often include norovirus GI and GII targets. Antigen-based rapid tests are available but have lower sensitivity and are generally used for screening rather than definitive diagnosis (Carlson *et al*., 2024).

Molecular genotyping based on capsid and/or polymerase sequences supports surveillance of circulating variants, outbreak linkage, source attribution and evaluation of vaccine escape. Metagenomic sequencing and environmental monitoring (e.g., sewage, shellfish, irrigation water) are increasingly used to track norovirus circulation at the community level (Carlson *et al*., 2024; Gao *et al*., 2025).

5.1.7. Outbreak investigation and food industry implications

Epidemiological investigations of foodborne norovirus outbreaks typically converge on a characteristic triad of evidence. First, case interviews frequently identify a clear point-source exposure, such as a shared meal at a restaurant or catered event, or the consumption of a widely distributed contaminated product, including frozen berries or shellfish. Second, analytical epidemiological studies, such as cohort or case–control designs, demonstrate a strong association between illness and the consumption of a specific food item. Third, laboratory confirmation is obtained through the detection of norovirus RNA in clinical specimens from cases, in implicated food samples, or in environmental swabs, with molecular typing revealing matching genotypes across these matrices. Together, these elements provide a robust and coherent basis for attributing outbreaks to specific food vehicles (EFSA and ECDC, 2021).

Although only a minority of norovirus outbreaks are purely foodborne, food service establishments and food processing environments are critical control points because contamination at these stages can seed large outbreaks that subsequently propagate via person-to-person spread (Silverberg, 2018; CDC, 2024). Regulatory agencies increasingly expect food businesses to incorporate norovirus-specific controls into their HACCP or food safety management systems, particularly for high-risk commodities such as bivalve shellfish and frozen berries (EFSA and ECDC, 2021; FDA, 2025).

5.1.8. Prevention and control

In the absence of a specific antiviral therapy for norovirus infection, clinical management is limited to supportive care, primarily oral or intravenous rehydration (Heymann, 2015). Prevention therefore depends on interrupting transmission through a multi-barrier approach. Key measures include the exclusion of symptomatic food handlers from work until at least 48 hours after symptom resolution, together with rigorous hand hygiene using soap and water, as alcohol-based hand sanitizers are less effective against norovirus. Environmental control requires thorough cleaning and disinfection of contaminated surfaces with chlorine-based or other virucidal agents. From a food safety perspective, adequate cooking of shellfish and avoidance of raw or lightly cooked oysters, particularly among vulnerable populations, are essential. At the broader system level, protection of shellfish harvesting areas and irrigation water from sewage contamination, combined with ongoing education

of food workers and the public regarding norovirus transmission and control, underpins comprehensive prevention strategies (Silverberg, 2018; CDC, 2024).

In the longer term, successful deployment of effective vaccines, improved environmental sanitation, and integrated surveillance that combines clinical, outbreak, wastewater and food-chain data will be essential to reduce the substantial global burden of norovirus disease (Carlson *et al*., 2024; Zhu *et al*., 2025).

5.2. Hepatitis A virus

Hepatitis A virus is a non-enveloped, single-stranded RNA virus with a very low infectious dose (on the order of tens of particles) and remarkable environmental stability, tolerating acidic conditions, refrigeration and freezing and even moderate heat treatments, which helps to explain its frequent association with raw or undercooked bivalve shellfish, fresh produce and contaminated drinking water.

5.3. Rotavirus

Although most rotavirus transmission is person-to-person rather than strictly foodborne, outbreaks linked to contaminated salads, fruits, cold dishes and drinking water indicate that foods and water can occasionally act as vehicles, particularly in settings with intense community circulation.

6. Parasitic foodborne diseases

6.1. Overview and global significance

Foodborne parasites have historically received less attention than bacterial and viral pathogens, but recent FAO/WHO initiatives and burden estimates highlight their significant and often underappreciated impact (Havelaar *et al*., 2015). A landmark FAO/WHO ranking of foodborne parasites identified *Taenia solium*, *Echinococcus granulosus*, *Echinococcus multilocularis*, *Toxoplasma gondii* and *Cryptosporidium* spp. among the highest-priority hazards globally, due to their severe outcomes and wide distribution.

Parasitic foodborne diseases differ fundamentally from acute bacterial and viral gastroenteritis in both their epidemiological patterns and clinical manifestations. A defining feature is their tendency to cause chronic or latent infections; parasites such as *Taenia solium*, *Toxoplasma gondii*, and *Fasciola* spp. may result in progressive conditions with long-term or lifelong health consequences, including neurocysticercosis, ocular toxoplasmosis, and chronic hepatic disease. Transmission dynamics are often complex, involving multiple intermediate and definitive hosts as well as environmentally persistent stages, which complicates prevention and control. Clinically, disease manifestations are frequently extra-intestinal, with involvement of organs such as the liver, central nervous system, or eyes. In addition, detection and source attribution are particularly challenging because infections may remain asymptomatic for extended periods, symptoms may emerge long after exposure, and routine diagnostic testing is often limited, thereby obscuring the causal link between illness and the original food vehicle (Dubey *et al*., 2020; Briciu *et al*., 2024).

6.2. Meat-borne parasites

6.2.1. *Taenia solium* and neurocysticercosis

Taenia solium (pork tapeworm) is transmitted when humans ingest larval cysticerci in undercooked pork, leading to intestinal taeniasis, or when they ingest eggs excreted by human tapeworm carriers, resulting in larval infection (cysticercosis) in various tissues (Garcia *et al*., 2014). When larvae invade the central nervous system, neurocysticercosis can cause seizures, headaches, hydrocephalus and other neurological deficits, and is a leading cause of acquired epilepsy in many endemic regions (Garcia *et al*., 2014; Havelaar *et al*., 2015).

Foodborne exposure to *T. solium* occurs through consumption of raw or undercooked pork from infected pigs, especially where free-range pigs have access to human feces and meat inspection is inadequate. Control requires a multi-pronged One Health strategy: improved sanitation, treatment of human tapeworm carriers, pig confinement, vaccination and/or anthelmintic treatment of pigs, and strengthened meat inspection (Garcia *et al*., 2014).

6.2.2. *Trichinella* spp.

Trichinella spp. are nematodes transmitted when humans consume raw or undercooked meat containing encysted larvae, historically associated with pork and game meat (Dubey *et al*., 2024). After ingestion, larvae mature in the intestine and newborn larvae migrate to striated muscles, causing myalgia, fever, facial edema and, in severe cases, myocarditis or encephalitis (Yu *et al*., 2025).

In many industrialized countries, implementation of controlled housing for pigs, feed controls and routine meat inspection has dramatically reduced classical pork-associated trichinellosis, shifting risk toward consumption of

inadequately cooked game (e.g., wild boar, bear) and traditional products from small-scale or backyard production (Dubey *et al*., 2024). Molecular diagnostic methods (PCR, LAMP, RPA) have improved detection of *Trichinella* in animals and food matrices, but routine surveillance still relies largely on digestion methods in slaughterhouses (Yu *et al*., 2025). Consequently, regulatory recommendations for meat-borne parasites emphasize thorough cooking to specified internal temperatures, deep freezing under defined time–temperature combinations and, where appropriate, sufficient salting or curing to inactivate tissue cysts and ensure product safety.

6.2.3. *Toxoplasma gondii*

Toxoplasma gondii is a globally distributed protozoan parasite with a complex life cycle. Felids, including domestic and wild cats, act as the definitive hosts and shed environmentally resistant oocysts in their feces. A wide range of warm-blooded animals, including major livestock species, serve as intermediate hosts in which tissue cysts are formed. Human infection occurs through several well-defined pathways, most commonly via the consumption of undercooked or raw meat containing tissue cysts, particularly pork, lamb, and goat. Infection may also result from the ingestion of oocysts present in contaminated soil, water, or inadequately washed fresh produce. In addition, transplacental transmission can occur following a primary maternal infection during pregnancy, potentially leading to severe congenital disease (Dubey *et al*., 2020).

Most immunocompetent individuals are asymptomatic or experience mild, self-limited lymphadenopathy. However, congenital toxoplasmosis can cause severe neurological and ocular sequelae, and reactivation in immunocompromised patients (e.g., people with AIDS, transplant recipients) can cause life-threatening encephalitis (Dubey *et al*., 2020). Serological surveys show substantial variation in *T. gondii* exposure worldwide, reflecting differences in dietary habits, climate, animal husbandry and cat populations (Briciu *et al*., 2024). Beyond its often asymptomatic course in immunocompetent adults, *T. gondii* infection poses particular concern for pregnant women, in whom primary infection may result in congenital toxoplasmosis with severe neurological and ocular sequelae in the fetus, and for occupationally exposed groups such as veterinarians, livestock farmers and meat industry workers.

Food safety interventions include freezing meat, adequate cooking, avoiding cross-contamination in kitchens, and improving farm biosecurity and cat management. For pregnant women and immunocompromised individuals, advice often includes avoiding consumption of undercooked meat and unwashed produce and careful handling of cat litter. Table 2 summarises selected foodborne parasites highlighted in FAO/WHO global rankings, together with their main reservoirs, food vehicles and clinical consequences.

Table 2. Selected foodborne parasites of global importance, main reservoirs, key food vehicles and principal clinical manifestations.

Parasite	Main reservoir(s)	Key food vehicles	Principal clinical manifestations
Taenia solium	Humans (definitive host), pigs (intermediate host)	Undercooked pork, foods contaminated due to poor sanitation	Intestinal taeniasis, cysticercosis and neurocysticercosis (seizures, epilepsy)
Toxoplasma gondii	Cats (definitive host), many warm-blooded animals	Undercooked pork, lamb and goat meat, contaminated produce and water	Congenital toxoplasmosis (neurological and ocular disease), encephalitis in immunocompromised individuals
Echinococcus granulosus / E. multilocularis	Dogs, foxes	Meat and offal from livestock and small mammals, contaminated vegetables and water	Cystic or alveolar echinococcosis (hepatic and extra-hepatic cysts, organ failure)
Trichinella spp.	Pigs, wild boar and other wildlife	Raw or undercooked pork and game meat	Myalgia, fever, facial oedema, myocarditis and encephalitis
Fish-borne liver flukes (*Clonorchis sinensis*, *Opisthorchis viverrini*)	Dogs, cats, humans	Raw or undercooked freshwater fish	Chronic cholangitis and cholangiocarcinoma
Cryptosporidium spp.	Humans, ruminants and other animals	Contaminated drinking and recreational water, fresh produce, RTE foods	Watery diarrhoea and weight loss, severe disease in children and immunocompromised individuals
Cyclospora cayetanensis	Humans	Fresh herbs (basil, cilantro), berries and salad mixes	Prolonged, relapsing watery diarrhoea and weight loss

6.3. Fish- and seafood-borne parasites

6.3.1. Fish-borne trematodes and cestodes

Fish-borne trematodes (e.g., *Clonorchis sinensis*, *Opisthorchis viverrini*, *Metagonimus yokogawai*) and cestodes (e.g., *Diphyllobothrium* spp.) are transmitted through consumption of raw, undercooked or inadequately processed freshwater or marine fish (Keiser & Utzinger, 2005). Chronic infection with *Opisthorchis viverrini* and *Clonorchis sinensis* is strongly associated with cholangiocarcinoma, making these parasites major public health concerns in parts of Southeast Asia (Keiser and Utzinger, 2005; Havelaar *et al*., 2015).

Traditional dishes involving raw or lightly marinated fish (e.g., certain forms of ceviche, sushi, sashimi, carpaccio) can also transmit fish-borne cestodes such as *Diphyllobothrium nihonkaiense* unless fish are frozen or adequately heated. Risk mitigation includes freezing regimes (e.g., −20°C for ≥7 days) or cooking to ≥63°C, as well as public health campaigns targeting traditional high-risk dishes.

6.3.2. *Anisakis* spp.

Anisakis spp. are marine nematodes whose larvae may be present in a wide range of marine fish and cephalopods. Human infection (anisakiasis) is acquired by consumption of raw or undercooked fish (e.g., anchovies, herring, mackerel, salmon) and can cause acute abdominal pain, nausea and vomiting, sometimes mimicking surgical emergencies. *Anisakis* allergens can also induce IgE-mediated allergic reactions even when larvae are dead, which has implications for fish processing and labeling.

Effective control relies on visual inspection, evisceration soon after catch, and freezing or cooking. In some jurisdictions, regulations require that fish intended for raw consumption be frozen under defined conditions to kill parasites.

6.4. Water- and produce-borne protozoa

6.4.1. *Cryptosporidium* spp. and *Giardia duodenalis*

Cryptosporidium spp. and *Giardia duodenalis* are protozoan parasites transmitted via ingestion of oocysts/cysts in contaminated water and food (Checkley *et al*., 2015). Their oocysts and cysts are environmentally robust and resistant to standard chlorination, making them important causes of waterborne outbreaks.

Foodborne transmission occurs when fresh produce, juices or RTE foods are contaminated with oocysts/cysts through irrigation with contaminated water, use of untreated manure, or handling by infected persons (Dubey *et al*., 2020). Cryptosporidiosis and giardiasis cause watery diarrhea, abdominal cramps and weight loss; disease can be chronic and severe in immunocompromised individuals and malnourished children (Checkley *et al*., 2015; Havelaar *et al*., 2015). *Cryptosporidium* oocysts and *Giardia* cysts are highly resistant to conventional chlorination and can persist for extended periods in surface waters and on fresh produce, so effective control requires adequate filtration and disinfection of drinking- and irrigation-water together with strict hygiene among food handlers. Improved water treatment, sanitation, hygiene, and application of good agricultural and manufacturing practices are crucial to reduce risk. In endemic settings, these parasites contribute significantly to the environmental enteric dysfunction and growth faltering in children.

6.4.2. *Cyclospora cayetanensis*

Cyclospora cayetanensis has emerged as a notable foodborne protozoan pathogen in North America and Europe, causing recurrent outbreaks linked to imported fresh produce (e.g., raspberries, basil, cilantro, pre-packaged salads) (Hall *et al*., 2012; Dubey *et al*., 2020). Unlike *Cryptosporidium*, *Cyclospora* oocysts require days to weeks in the environment to become infective, so direct person-to-person transmission is unlikely; contamination usually reflects poor sanitation at production sites (Hall *et al*., 2012).

Clinical illness is characterized by prolonged, relapsing watery diarrhea, fatigue and weight loss, especially in immunocompromised hosts. Control focuses on improving sanitation, worker hygiene, water quality and traceability in global produce supply chains, as well as seasonal surveillance in importing countries.

6.4.3. *Fasciola* spp.

Fasciola hepatica and *Fasciola gigantica* are liver flukes primarily affecting livestock but also infecting humans, who become accidental hosts through ingestion of aquatic plants (e.g., watercress) or water contaminated with metacercariae (Mas-Coma *et al*., 2018). Human fascioliasis can cause chronic biliary disease with pain, fever and eosinophilia. While often classified as a water/plant-borne zoonosis rather than a classical foodborne parasite, fascioliasis illustrates how contaminated freshwater produce can act as an important transmission route (Mas-Coma *et al*., 2018).

6.5. Emerging issues, diagnostics and control

Growing awareness of the public health burden associated with foodborne parasites has been driven by advances in diagnostic technologies, including multiplex PCR, serological assays, and next-generation sequencing, as well as by improved recognition of complex farm-to-fork transmission pathways in which contamination can occur at multiple stages (Bouwknegt *et al*., 2018; Dubey *et al*., 2020). Despite this progress, several challenges continue to hinder effective control. These include limited surveillance and substantial underreporting, particularly in settings with constrained diagnostic capacity; persistent difficulties in quantifying exposure and attributing illness to specific food sources; complex parasite life cycles that necessitate integrated veterinary and public health approaches; and the effects of climate change and globalization, which may alter parasite distributions and introduce emerging risks (Briciu *et al*., 2024). Addressing these challenges requires a set of coordinated priority actions, including strengthening surveillance through harmonized diagnostic protocols, developing risk-based standards for parasites in high-risk commodities such as fish, fresh produce, and ready-to-eat salads, implementing targeted control programmes in livestock for pathogens such as *Trichinella* and *Toxoplasma*, enhancing consumer education on the safe preparation of raw or lightly cooked animal products, and fully integrating foodborne parasites into national and international food safety agendas alongside bacterial and viral hazards.

7. Prevention and control along the farm-to-fork continuum

7.1. Principles of prevention

Prevention of foodborne disease is grounded in classic epidemiological principles that emphasize interruption of transmission at the level of the agent, host and environment. While case management and outbreak investigation remain essential (Heymann, 2015), the greatest and most sustainable health gains are usually achieved through primary prevention—that is, preventing contamination and exposure along the food chain before illness occurs (Havelaar *et al*., 2015).

In modern food systems, this preventive philosophy translates into integrated control measures from farm to fork. These measures include Good Agricultural Practices (GAPs), Good Veterinary Practice (GVP), Good Hygienic Practices (GHP), Good Manufacturing Practices (GMP), Hazard Analysis and Critical Control Points (HACCP)–based food safety management systems and effective risk communication targeting both professionals and consumers.

7.2. Primary production: animals, crops and the environment

At the level of primary production, food safety prevention focuses on minimizing the introduction, persistence, and circulation of pathogens within animal reservoirs, crop production systems, and the surrounding environment. This upstream control relies on several interrelated measures. Robust biosecurity practices in livestock and poultry production, including controlled animal movements, all-in/all-out management, and effective sanitation of water and feed, are essential to limit colonization by major zoonotic pathogens such as *Salmonella*, *Campylobacter*, and pathogenic *Escherichia coli* (Abebe *et al*., 2020; EFSA and ECDC, 2021, 2024). In parallel, the rational use of antimicrobials and the application of vaccination programmes within a One Health framework contribute to disease prevention while reducing selective pressure for antimicrobial resistance (Aslam *et al*., 2021; Velazquez-Meza *et al*., 2022; Robles Ramirez *et al*., 2024). Effective manure management and wastewater treatment further reduce the environmental dissemination of bacterial pathogens, protozoa, and antimicrobial-resistant organisms, particularly when animal wastes are applied to land or enter surface waters, thereby protecting crops, shellfish-growing areas, and recreational environments (Fleming *et al*., 2006; Bouwknegt *et al*., 2018; Velazquez-Meza *et al*., 2022). In plant production, adherence to Good Agricultural Practices (GAPs), including the use of microbiologically safe irrigation water, avoidance of untreated human or animal waste, and good hygiene among farm workers, is critical to prevent pre-harvest contamination of fruits, leafy greens, and herbs with pathogens such as norovirus, *Salmonella*, STEC, and *Cyclospora* (Hall *et al*., 2012; Okafor, 2024). Together, these measures constitute the first and most upstream line of defense in an integrated farm-to-fork food safety system.

These measures also contribute to control of food- and waterborne diseases traditionally associated with drinking water—for example, cholera—highlighting the continuum between water safety, sanitation and food hygiene (Ali *et al*., 2015; Checkley *et al*., 2015).

7.3. Slaughter, processing and manufacturing

The slaughter, processing, and manufacturing stages constitute critical points along the food chain at which microbiological hazards may be substantially reduced or, if inadequately controlled, amplified. At these stages,

the application of general principles of food hygiene and Hazard Analysis and Critical Control Point (HACCP) systems, as codified in Codex standards, is central to effective risk control. Hygienic slaughter and carcass dressing practices are essential to minimize fecal contamination and carcass-to-carcass transmission of pathogens such as *Campylobacter* and *Salmonella* in poultry and red meat production chains (Abebe *et al*., 2020; EFSA and ECDC, 2024). In addition, robust process controls—particularly those related to temperature management, including rapid carcass chilling, validated cooking and pasteurization steps, and the prevention of temperature abuse during cooling and storage—are critical for controlling vegetative pathogens and spore-forming bacteria such as *Clostridium perfringens* and *Bacillus cereus* (Riemann and Cliver, 2006; Grass *et al*., 2013). Traditional preservation practices, including salting techniques used in cheese production, can also play an important role in enhancing microbiological safety and product stability (Atasever *et al*., 2003). For ready-to-eat (RTE) foods, environmental monitoring programmes and hygienic facility design are particularly important to prevent post-process contamination with persistent hazards such as *Listeria monocytogenes* and *Staphylococcus aureus* (Farber and Peterkin, 1991; Allerberger and Wagner, 2010). Finally, the use of validated decontamination and preservation technologies, including high-pressure processing, modified-atmosphere packaging, and fermentation, offers effective means of inhibiting or inactivating pathogens while responding to consumer demand for minimally processed foods (Havelaar *et al*., 2015; Hassan *et al*., 2023; Mazlum and Atasever, 2023).

Microbiological risk assessment (MRA) provides a quantitative basis for determining which control measures at these stages are most effective, for example in reducing *L. monocytogenes* in RTE meats or *Salmonella* in poultry meat.

7.4. Distribution, retail and food service

During distribution, retail, and food service operations, foodborne hazards may be introduced or amplified through cross-contamination, inadequate temperature control, and suboptimal personal hygiene practices. Numerous outbreaks involving pathogens such as *Salmonella*, *Campylobacter*, Shiga toxin–producing *Escherichia coli* (STEC), *Clostridium perfringens*, *Bacillus cereus*, *Staphylococcus aureus*, and norovirus have been linked to failures at these stages of the food chain (Koo *et al*., 2010; Grass *et al*., 2013; Kadariya *et al*., 2014; Silverberg, 2018). Effective prevention therefore depends on a combination of core control measures. Strict cold chain management during transport and storage is essential to limit the growth of psychrotrophic pathogens such as *Listeria monocytogenes* and *Yersinia enterocolitica*, as well as mesophilic organisms in chilled or temperature-abused foods (Farber and Peterkin, 1991; Naik *et al*., 2023). Equally important is the physical and operational separation of raw and ready-to-eat (RTE) foods, including the use of dedicated equipment, utensils, and handling areas, to reduce cross-contamination by zoonotic pathogens such as *Salmonella*, *Campylobacter*, and STEC (Abebe *et al*., 2020; EFSA and ECDC, 2021). Control of food handler–related risks is achieved through the exclusion of ill workers, rigorous hand hygiene, and the appropriate use of gloves or utensils when handling RTE foods, thereby limiting contamination with norovirus and *S. aureus* (Koo *et al*., 2010; Kadariya *et al*., 2014; CDC, 2024). Finally, robust cleaning and disinfection programmes, employing agents effective against non-enveloped viruses and verified through routine microbiological and visual checks, are critical to interrupt transmission along these downstream stages of the food chain (Carlson *et al*., 2024).

Increasing globalization and complex supply chains mean that failures in one food business operator can trigger multi-country outbreaks, as seen with STEC in sprouts, Salmonella in low-moisture foods and norovirus in frozen berries (Baker-Austin *et al*., 2018; EFSA and ECDC, 2021; Davydova *et al*., 2025; FDA, 2025). This underscores the need for strong traceability systems and timely international information sharing.

7.5. Household-level and community interventions

At the consumer end of the food chain, household-level practices represent a critical final line of defense against foodborne illness. The adoption of simple, evidence-based measures can substantially reduce individual and domestic risk. Central to these efforts is access to safe water, sanitation, and hygiene (WASH), which provides protection against a wide range of enteric pathogens transmitted via both food and water (Checkley *et al*., 2015; Havelaar *et al*., 2015). Widely disseminated public health guidance, commonly summarized by the “clean, separate, cook, chill” framework, offers practical and actionable advice, including effective hand hygiene, thorough cooking, avoidance of high-risk foods such as raw shellfish and undercooked minced meats, and prompt refrigeration of leftovers (CDC, 2024). In addition, targeted risk communication is essential for vulnerable populations. Pregnant women, for example, are advised to avoid certain soft cheeses, deli meats, and raw animal products to reduce the risk of listeriosis and toxoplasmosis, with similar precautionary measures

recommended for older adults and immunocompromised individuals (Farber and Peterkin, 1991; Dubey *et al*., 2020). Finally, the success of consumer-focused interventions depends on culturally appropriate education strategies that take into account local food practices, perceptions of risk, and relevant social or religious norms related to food preparation and hygiene (Ali *et al*., 2015; Okafor, 2024).

Household-level measures are particularly important in low- and middle-income settings, where informal markets, limited refrigeration and inadequate water and sanitation infrastructure constrain the feasibility of industrial-level controls (Havelaar *et al*., 2015; Okafor, 2024).

7.6. Integrating climate change and environmental change into prevention

Climate change and environmental degradation are increasingly recognized as modifiers of foodborne disease risk. Rising sea surface temperatures and extreme weather events influence the distribution and seasonality of *Vibrio* spp., harmful algal blooms and marine biotoxins, while floods and droughts can compromise water and sanitation systems, affecting contamination of crops and livestock (Fleming *et al*., 2006; Havelaar *et al*., 2015; Baker-Austin *et al*., 2018).

Integrating climate-informed risk assessments into food safety planning—such as early warning systems for *Vibrio* risk in shellfish-growing areas or for *Cyclospora* contamination of produce—will be essential to maintain and improve protection in a warming world (Hall *et al*., 2012; Baker-Austin *et al*., 2018; Zhang *et al*., 2024). These efforts closely align with One Health approaches discussed in Section 8, where animal, human and environmental health are considered jointly (Velazquez-Meza *et al*., 2022).

8. Emerging challenges and future directions

8.1. Antimicrobial resistance (AMR) and the One Health framework

8.1.1. AMR as a global food safety and public health threat

Antimicrobial resistance (AMR) is often cited as the global health problem that most clearly illustrates the One Health concept, because resistant bacteria and resistance genes circulate continuously between humans, animals and the environment (Velazquez-Meza *et al*., 2022). Resistant foodborne pathogens—particularly *Salmonella*, *Campylobacter*, *Escherichia coli* and *Listeria*—complicate clinical management of infections, increase the risk of severe disease and death, and generate substantial economic losses (EFSA and ECDC, 2021; Aslam *et al*., 2021). Fluoroquinolone-resistant *Campylobacter* and extended-spectrum cephalosporin-resistant *Salmonella* have been associated with longer illness duration, higher hospitalisation rates and, in some settings, higher case-fatality ratios (EFSA and ECDC, 2021). The detection of ESBL-producing *E. coli* and carbapenemase-producing Enterobacterales in food animals, retail meat and the wider environment further raises concerns about food-mediated dissemination of critical resistance determinants (Havelaar *et al*., 2015; Aslam *et al*., 2021).

8.1.2. Drivers of AMR across human–animal–environment interfaces

The emergence and spread of antimicrobial resistance (AMR) among foodborne pathogens are driven by a set of interconnected pressures operating across human, animal, and environmental interfaces. In human medicine, inappropriate antimicrobial prescribing, including use for viral infections, as well as suboptimal dosing and poor adherence, contributes to the selection of resistant organisms. In parallel, antimicrobial use in animal production for therapeutic purposes, metaphylaxis, and, in some settings, growth promotion continues to exert substantial selective pressure, particularly within intensive livestock and aquaculture systems (Robles Ramirez *et al*., 2024). Environmental pathways further amplify these dynamics, as antimicrobial residues, resistant bacteria, and resistance genes are disseminated through manure application, aquaculture effluents, wastewater discharges, and sludge applied to agricultural land (Velazquez-Meza *et al*., 2022). These drivers are compounded by globalization, with international trade and travel facilitating the rapid spread of resistant strains and mobile genetic elements along food supply chains and across borders (Havelaar *et al*., 2015; EFSA and ECDC, 2021). Importantly, these processes operate within a broader socio-economic context shaped by increasing food demand, agricultural and veterinary policies, regulatory frameworks, and consumer expectations related to both low food prices and “antibiotic-free” production, underscoring the complexity of implementing effective, integrated AMR mitigation strategies.

8.1.3. One Health surveillance and integrated data

A One Health approach to antimicrobial resistance (AMR) surveillance emphasizes coordinated and integrated data collection across human health, veterinary, food, and environmental sectors, using harmonized indicators and methodologies (Velazquez-Meza *et al*., 2022). This approach is implemented through global initiatives such as the WHO Global Antimicrobial Resistance and Use Surveillance System (GLASS), which primarily compiles

data on human AMR and is increasingly incorporating community and environmental isolates, as well as through regional frameworks such as the European Union's integrated AMR surveillance, which jointly analyzes resistance patterns in human clinical isolates, food-producing animals, and food products (EFSA and ECDC, 2021). At the national level, integrated programmes including DANMAP and NARMS monitor AMR in human pathogens, foodborne zoonoses, commensal bacteria, and retail meat. Together, these systems enable the early detection of emerging resistance phenotypes, such as mobile colistin resistance genes and plasmid-mediated extended-spectrum beta-lactamases (ESBLs), and support the identification of source–sink relationships and transmission pathways across human, animal, and food reservoirs (Aslam *et al*., 2021; Velazquez-Meza *et al*., 2022; Atasever, 2025b). Nevertheless, substantial disparities in surveillance capacity remain, as many low- and middle-income countries lack the infrastructure and resources required for comprehensive implementation, resulting in persistent geographic data gaps.

8.1.4. Control strategies in animal production and along the food chain

Control strategies implemented in animal production systems and along the food chain play a dual role in both food safety and antimicrobial resistance (AMR) mitigation. Farm-to-fork interventions such as improved biosecurity, vaccination, good husbandry and hygiene practices, and effective manure and waste management reduce the incidence of infectious diseases, thereby lowering the overall need for antimicrobial use in both animals and humans (Velazquez-Meza *et al*., 2022; Robles Ramirez *et al*., 2024). By preventing infections at their source, these measures address one of the fundamental drivers of antimicrobial selection pressure. In parallel, antimicrobial stewardship programmes in human and veterinary medicine remain a cornerstone of efforts to slow the emergence and spread of resistance. Such programmes, supported by evidence-based treatment guidelines and restrictions on the use of critically important antimicrobials, aim to optimize therapeutic outcomes while minimizing unnecessary exposure to antibiotics.

In parallel, a range of complementary or alternative approaches is being explored within a One Health framework. These include vaccines targeting specific foodborne pathogens in animals (e.g., *Salmonella* in poultry), bacteriophages and phage-derived enzymes for targeted decontamination, probiotics and competitive exclusion cultures in poultry, and antimicrobial peptides as substitutes or adjuncts to conventional antibiotics (Robles Ramirez *et al*., 2024; Singh *et al*., 2025). Optimising manure and wastewater treatment (for example through composting or anaerobic digestion) can further limit environmental dissemination of resistant bacteria and mobile genetic elements (Velazquez-Meza *et al*., 2022). While many of these options are promising, their wider adoption will depend on context-specific evidence for efficacy, safety, cost-effectiveness and acceptability to producers and consumers. Table 3 provides illustrative examples of One Health interventions along the farm-to-fork continuum that are relevant to mitigating antimicrobial resistance in foodborne pathogens.

Table 3. Examples of One Health interventions along the farm-to-fork continuum relevant to antimicrobial resistance (AMR) in foodborne pathogens.

Stage of the food chain	Example intervention	Target hazards / mechanisms
Primary production (farm)	Improved biosecurity, vaccination, herd/flock health programmes, reduced and targeted antimicrobial use	Prevents infections and lowers overall antimicrobial exposure and selection pressure
Environment	Manure treatment (composting, anaerobic digestion), improved wastewater treatment, controlled sludge application	Reduces dissemination of resistant bacteria and resistance genes to soil and water
Slaughter and processing	Hygienic slaughter, decontamination steps, HACCP-based process controls	Lowers carcass contamination with resistant zoonotic pathogens
Food manufacturing	Environmental monitoring, hygienic design, validation of control measures	Prevents persistence and spread of resistant strains in processing environments
Retail and food service	Temperature control, prevention of cross-contamination, staff training	Limits growth and spread of resistant pathogens on food and surfaces
Consumers and communities	Education on safe food handling, WASH interventions, awareness campaigns on prudent antibiotic use	Reduces exposure to resistant pathogens and supports stewardship

8.1.5. Policy, governance and global initiatives

International efforts to address antimicrobial resistance (AMR) are led by a coalition of global organizations, including the WHO, FAO, the World Organisation for Animal Health (WOAH), and the United Nations Environment Programme (UNEP), which have jointly advanced One Health–oriented Global Action Plans. These frameworks emphasize coordinated surveillance, antimicrobial stewardship, infection prevention, and research across human, animal, and environmental sectors. Within the food safety context, this has resulted in targeted initiatives, including Codex Alimentarius guidelines on integrated AMR surveillance and the prudent use of antimicrobials in food-producing animals, as well as FAO/WHO expert consultations addressing AMR risks along the food chain. At the national level, effective implementation typically relies on a combination of policy instruments, such as restrictions or bans on the use of antibiotics for growth promotion, prescription-only access with veterinary oversight for therapeutic use, incentives to support farmers in adopting alternatives such as vaccination and improved husbandry and biosecurity, transparent public reporting of antimicrobial use and resistance trends, and the integration of AMR considerations into food safety, animal welfare, and environmental regulatory frameworks (Aslam *et al*., 2021; Velazquez-Meza *et al*., 2022).

8.1.6. AMR, foodborne disease burden and future priorities

Quantifying the incremental public health burden attributable specifically to antimicrobial-resistant (AMR) foodborne infections, beyond that associated with susceptible strains, remains methodologically challenging. Persistent surveillance gaps, confounding clinical factors, and difficulties in attributing adverse outcomes directly to resistance complicate burden estimation (Havelaar *et al*., 2015). Nevertheless, the available evidence consistently indicates a substantial additional burden in terms of Disability-Adjusted Life Years (DALYs), particularly for resistant *Salmonella*, *Campylobacter*, and *Escherichia coli* infections in settings where access to effective treatment options is limited (EFSA and ECDC, 2021; Aslam *et al*., 2021). Addressing this multifaceted challenge requires a set of clearly defined priorities. These include strengthening integrated One Health AMR surveillance using harmonized methodologies; improving data integration and modeling approaches to better link antimicrobial use, resistance emergence along the food chain, and downstream human health outcomes; and investing in context-appropriate interventions for low- and middle-income countries. Continued research and innovation in vaccines, alternatives to antibiotics, rapid diagnostics, and environmental mitigation strategies will also be essential. Ultimately, effective AMR mitigation depends on embedding resistance considerations within broader policy agendas on food security, climate resilience, and sustainable agriculture (Velazquez-Meza *et al*., 2022; Robles Ramirez *et al*., 2024; Singh *et al*., 2025).

9. Conclusions

Despite substantial advances in water treatment, food processing, refrigeration, and hygiene over recent decades, foodborne microbial diseases continue to pose a major and uneven global public health burden. The synthesis presented in this review highlights that this burden remains disproportionately concentrated among young children and populations in low- and middle-income countries. Bacteria remain responsible for the majority of recognized foodborne illnesses, while viruses, particularly norovirus, account for an exceptionally large number of acute gastroenteritis episodes worldwide. In addition, foodborne parasites contribute substantially to chronic, long-term, and disabling disease outcomes. The reviewed evidence demonstrates how key pathogens, including nontyphoidal *Salmonella*, *Campylobacter*, pathogenic *Escherichia coli*, *Listeria monocytogenes*, *Staphylococcus aureus*, *Clostridium perfringens*, *Bacillus cereus*, norovirus, and a wide range of parasites transmitted via meat, fish, and fresh produce, interact with increasingly complex and interconnected food systems. Intensification of animal production, globalization of food trade, shifts in dietary patterns, and climate change collectively shape contemporary food safety risks, often in ways that are not yet fully understood.

From a methodological perspective, microbiological risk assessment has become a central component of modern food safety, providing a structured and quantitative basis for estimating risks and comparing the effectiveness of control measures. When integrated within broader risk analysis frameworks and linked to microbiological criteria, performance objectives, and HACCP-based management systems, this approach supports more transparent and science-based decision-making at both national and international levels. At the same time, antimicrobial resistance has emerged as a defining challenge for food safety and a clear illustration of the One Health nature of current public health threats. Infections caused by resistant *Salmonella*, *Campylobacter*, and *Escherichia coli* are associated with prolonged illness, increased hospitalization rates, and higher case fatality, particularly in settings with limited access to effective antimicrobial therapy. Integrated surveillance of antimicrobial resistance across humans, animals, food, and the environment, together with antimicrobial

stewardship, improved husbandry practices, vaccination, and the development of alternative control strategies, should therefore remain high policy priorities. Ultimately, technical interventions alone will be insufficient without sustained political commitment and societal engagement. Food safety must be embedded within broader agendas of food security, nutrition, environmental sustainability, animal welfare, and social equity, with the overarching goal of building resilient food systems that protect human health while supporting sustainable and equitable patterns of food production and consumption.

Data availability
Not applicable.

Conflict of interest
None to declare.

Author's contribution
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References
Abebe E, G Gugsa and M Ahmed, 2020. Review on major foodborne zoonotic bacterial pathogens. J. Trop. Med., 2020: 4674235.

Ali M, AR Nelson, AL Lopez and DA Sack, 2015. Updated global burden of cholera in endemic countries. PLoS Negl. Trop. Dis., 9: e0003832.

Allerberger F and M Wagner, 2010. Listeriosis: a resurgent foodborne infection. Clin. Microbiol. Infect., 16: 16-23.

Aslam B, M Khurshid, MI Arshad, S Muzammil, M Rasool, N Yasmeen, S Taif, TH Chaudhry, MH Rasool, A Shahid, X Xueshan and Z Baloch. 2021. Antibiotic resistance: One Health, one world outlook. Front. Cell. Infect. Microbiol., 11: 771510.

Atasever M, 2025a. *Listeria monocytogenes*: an overview. Asian J. Med. Biol. Res., 11: 5-12.

Atasever M, 2025b. Veterinarians' role in global food safety and public health. Asian Australas. J. Biosci. Biotechnol., 10: 1-5.

Atasever M, A Keles, G Uçar and A Güner, 2003. Use of different salting techniques in Halloumi cheese: effect on sensory, microbiological and chemical properties. Acta Aliment., 32: 7-14.

Atasever M, M Alişarlı and F Tombak, 2015. Gıdalarla taşınan virüsler [Foodborne viruses]. Turkiye Klin. J. Food Hyg. Technol.-Spec. Top., 1: 102-108.

Aydemir Atasever M, H Özlü and M Atasever, 2025. Occurrence and dietary risk assessment of AFB1 in tomato and pepper pastes in Türkiye. Ital. J. Food Sci., 37: 410-420.

Baker-Austin C, J Trinanes, N Gonzalez-Escalona and J Martinez-Urtaza, 2018. Non-cholera vibrios: the microbial barometer of climate change. Trends Microbiol., 25: 76-84.

Bottone EJ, 2010. *Bacillus cereus*, a volatile human pathogen. Clin. Microbiol. Rev., 23: 382-398.

Bouwknegt M, B Devleesschauwer, H Graham, LJ Robertson and JW van der Giessen, 2018. Prioritization of foodborne parasites in Europe, 2016. Eurosurveillance, 23: 17-00161.

Briciu V, M Opsteegh, SD Giurgiu, IA Colosi, V Mircean, V Cozma and R Blaga, 2024. Serological evidence of human exposure to foodborne parasites in Romania. Food Waterborne Parasitol., 38: e00163.

Carlson KB, L. Hu, N. Collins and J Vinjé, 2024. A narrative review of norovirus epidemiology, biology, and vaccine development. NPJ Vaccines, 9: 45.

Centers for Disease Control and Prevention (CDC), 2023. Foodborne Diseases Active Surveillance Network (FoodNet). Available at: https://www.cdc.gov/foodnet/index.html

Centers for Disease Control and Prevention (CDC), 2024. Norovirus – about norovirus. Available at: https://www.cdc.gov/norovirus/about/index.html

Checkley W, AC White Jr, D Jaganath, MJ Arrowood, RM Chalmers, XM Chen, R Fayer, JK Griffiths, RL Guerrant, L Hedstrom, CD Huston, KL Kotloff, G Kang, JR Mead, M Miller, WA Petri Jr, JW Priest, DS Roos, B Striepen, RCA Thompson, HD Ward, WAV Voorhis, L Xiao, G Zhu and ER Houpt, 2015. A review of the global burden, novel diagnostics, therapeutics, and vaccine targets for *Cryptosporidium*. Lancet Infect. Dis., 15: 85-94.

Dasti JI, AM Tareen, R Lugert, AE Zautner and U Groß, 2010. Campylobacter jejuni: a brief overview on pathogenicity-associated factors and disease-mediating mechanisms. Int. J. Med. Microbiol., 300: 205-211.

Davydova A, BA Smith, GK Jones and DL Patterson, 2025. Source attribution studies of foodborne pathogens, 2010–2023: a systematic review. Foodborne Pathog. Dis., 22: 125-142.

Dubey JP, DE Hill and SK Verma, 2024. Over a century of progress on *Trichinella* research in pigs at the USDA Agricultural Research Service. Parasitol. Res., 123: 1001-1020.

Dubey JP, KD Murrell and JH Cross, 2020. Foodborne parasites. In: Rose JB and B Jiménez-Cisneros (eds.), Global Water Pathogens Project. Michigan State Univ., East Lansing, USA.

EFSA and ECDC, 2021. The European Union One Health 2020 zoonoses report. EFSA J., 19: e06971.

EFSA and ECDC, 2024. The European Union One Health 2023 zoonoses report. EFSA J., 22: e09106.

Fabrega A and J Vila, 2013. Salmonella enterica serovar Typhimurium skills to succeed in the host: virulence and regulation. Clin. Microbiol. Rev., 26: 308-341.

Farber JM and PI Peterkin, 1991. Listeria monocytogenes, a foodborne pathogen. Microbiol. Rev., 55: 476-511.

Fleming LE, K Broad, A Clement, E Dewailly, S Elmir, A Knap, SA Pomponi, S Smith, HS Gabriele and PJ Walsh, 2006. Oceans and human health: emerging public health risks in the marine environment. Mar. Pollut. Bull., 53: 545-560.

Franco MP, M Mulder, RH Gilman and HL Smits, 2007. Human brucellosis. Lancet Infect. Dis., 7: 775-786.

Gao J, Z Zhang, Y Jiao, Y Xu, Y Chen and L Xue, 2025. Global burden of human norovirus contamination in drinking water sources and drinking water: a systematic review and meta-analysis identifying GII.4 and GII.17 as dominant genotypes. Sci. Total Environ., 989: 179854.

Garcia HH, AE Gonzalez, CA Evans and RH Gilman, 2014. *Taenia solium* cysticercosis. Lancet, 362: 547-556.

Giannella RA, 1996. *Salmonella*. In: Baron S (ed.), Medical Microbiology (4th ed.). Univ. Texas Med. Branch, Galveston, USA.

Grass JE, LH Gould and BE Mahon, 2013. Epidemiology of foodborne disease outbreaks caused by *Clostridium perfringens*, United States, 1998–2010. Foodborne Pathog. Dis., 10: 131-136.

Hall RL, JL Jones, BL Herwaldt, Centers for Disease Control and Prevention (CDC), 2012. Cyclosporiasis—U.S. outbreaks of diarrhea associated with imported raspberries and other fresh produce. Clin. Infect. Dis., 54(Suppl. 4): S218-S224.

Hassan A, M Khan and S Ali, 2023. Challenges and strategies for ensuring food safety. Emerg. Trends Life Sci., 3: 32.

Havelaar AH, MD Kirk, PR Torgerson, HJ Gibb, T Hald, RJ Lake, N Praet, DC Bellinger, NR de Silva, N Gargouri, N Speybroeck, A Cawthorne, C Mathers, C Stein, FJ Angulo, B Devleesschauwer and World Health Organization Foodborne Disease Burden Epidemiology Reference Group, 2015. World Health Organization global estimates and regional comparisons of the burden of foodborne disease in 2010. PLoS Med., 12: e1001923.

Heymann DL (ed.), 2015. Control of communicable diseases manual (20th ed.). American Public Health Association, Washington, DC, USA.

Jones MK and JD Oliver, 2009. Vibrio vulnificus: disease and pathogenesis. Infect. Immun., 77: 1723-1733.

Kaakoush NO, N Castaño-Rodríguez, HM Mitchell and SM Man, 2015. Global epidemiology of *Campylobacter* infection. Clin. Microbiol. Rev., 28: 687-720.

Kadariya J, TC Smith and D Thapaliya, 2014. *Staphylococcus aureus* and staphylococcal foodborne disease: an ongoing challenge in public health. Biomed. Res. Int., 2014: 827965.

Kaper JB, JP Nataro and HL Mobley, 2004. Pathogenic *Escherichia coli*. Nat. Rev. Microbiol., 2: 123-140.

Keiser J and J Utzinger, 2005. Emerging foodborne trematodiasis. Emerg. Infect. Dis., 11: 1507-1514.

Koepke R, J Sobel and SS Arnon, 2008. Global occurrence of infant botulism, 1976–2006. Pediatrics, 122: e73-e82.

Koo HL, N Ajami, RL Atmar and HL DuPont, 2010. Noroviruses: the principal cause of foodborne disease. Curr. Opin. Gastroenterol., 26: 21-27.

Kotloff KL, MS Riddle, JA Platts-Mills, P Pavlinac and AK Zaidi, 2018. Shigellosis. Lancet, 391: 801-812.

Mas-Coma S, MD Bargues, MA Valero, 2018. Human fascioliasis infection sources, their diversity, incidence factors, analytical methods and prevention measures. Parasitol., 145: 1665-1699.

Mazlum H and M Atasever, 2023. Probiotic cheese as a functional food. Bangladesh Acad. Sci., 7.

Naik BPK, A Singh and R Kumar, 2023. A foodborne pathogen: *Listeria monocytogenes*. Int. J. Curr. Microbiol. Appl. Sci., 12: 101-118.

Okafor CA, 2024. Impact and interventions of waterborne and foodborne diseases: a review. Food Health, 10: 316-322.

Özlü H and M Atasever, 2018. Foodborne bacterial zoonoses. N. Duran (Ed.), *Current Academic Studies in Health Sciences – 2018*, 262–269. IVPE.

Radoshevich L and P Cossart, 2018. *Listeria monocytogenes*: towards a complete picture of its physiology and pathogenesis. Nat. Rev. Microbiol., 16: 32-46.

Riemann HP and DO Cliver (eds.), 2006. Foodborne infections and intoxications (3rd ed.). Academic Press, London, UK.

Robles Ramirez O, S García and M López, 2024. Antimicrobial peptides in livestock: a review with a One Health perspective. Front. Cell. Infect. Microbiol., 14: 1339285.

Scallan E, RM Hoekstra, FJ Angulo, RV Tauxe, MA Widdowson, SL Roy, JL Jones and PM Griffin, 2011. Foodborne illness acquired in the United States—major pathogens. Emerg. Infect. Dis., 17: 7-15.

Silverberg R, 2018. Preventing foodborne illness: norovirus. USDA National Institute of Food and Agriculture. Available at: https://nifa.usda.gov

Singh S V. Kumar and A Patel, 2025. A One Health approach addressing poultry-associated antimicrobial resistance. One Health, 18: 100562.

Tan M and X Jiang, 2024. Norovirus vaccines: where are we now? Curr. Opin. Virol., 66: 102337.

Tan M, 2024. The impact of norovirus on children and adolescents. Curr. Pediatr. Rep., 12: 101-115.

Velazquez-Meza ME, M Galarde-López, B Carrillo-Quiróz and CM Alpuche-Aranda, 2022. Antimicrobial resistance: One Health approach. Vet. World, 15: 743-753.

Verhoef L, J Hewitt, L Barclay, S Ahmed, R Lake, AJ Hall, B Lopman, A Kroneman, H Vennema, J VinjA(c) and M Koopmans, 2015. Norovirus genotype profiles associated with foodborne transmission, 1999–2012. Emerg. Infect. Dis., 21: 592-599.

WHO Regional Office for Europe, 2017. The burden of foodborne diseases in the WHO European Region. WHO Regional Office for Europe, Copenhagen, Denmark.

Yu SM, ZQ Wang and RD Liu, 2025. Molecular diagnosis of *Trichinella* spp.: current status and future perspectives. Parasitol. Res., 124: 203-219.

Zhang P, Y Li and J Wang, 2024. Global prevalence of norovirus gastroenteritis after the COVID-19 pandemic: a systematic review and meta-analysis. Front. Public Health, 12: 1373322.

Zhu ML, Y Chen and X Liu, 2025. Global burden and trends of norovirus-associated diseases. Front. Public Health, 12: 1483149.