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Types and Harms of Pathogenic Microorganisms: A Review

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Abstract

Diseases occur due to a malfunction in one part of the body. There are many microbes that cause diseases in humans, as these microbes attack one part of the body and cause health problems. These microbes mainly enter through the digestive or respiratory system via wounds, after which they begin to multiply and infect the host cell, causing a malfunction. These microbes include bacteria, fungi, viruses, and parasites. They cause many types of damage, some related to the respiratory system and others related to the digestive system, causing diarrhea and high temperatures. These microbes can be eliminated by taking antibiotics.

Keywords: Pathogenic, Microorganisms, bacteria, fungi, viruses, parasites

1. Introduction

Pathogenic microorganisms cause many infectious diseases worldwide (1). The four main groups are bacteria, viruses, fungi, and parasites. Unified by their ability to cause disease, they nevertheless adopt highly varied lifestyles—free-living and parasitic, opportunistic and obligate, intracellular and extracellular, with or without toxin production, highly transmissible or unable to spread from host to host (2). Bacteria are unicellular prokaryotes typified by classic pathogens such as Streptococcus spp. and Staphylococcus spp. (3). Viruses lack independent metabolism and do not multiply outside host cells; significant pathogens include rhinoviruses, coronaviruses, and HIV. Fungi are eukaryotic organisms ranging in form from single-celled yeasts to multicellular molds; examples of pathogens are Candida spp. and Trichophyton spp. (4). Parasites are a heterogeneous group of eukaryotes from simple protozoans to complex metazoans; pathogens include Plasmodium spp., Trichinella spp., and the liver fluke, Fasciola gigantica (5).

2. Classification of Pathogenic Microorganisms

Pathogenic microorganisms span diverse groups including bacteria, viruses, fungi, and parasites; these phylogenetically heterogeneous classes share a trait harmful to humans and other hosts, underscoring their medical and ecological significance (6). The first three groups—bacteria, viruses, and fungi—comprise the prokaryotes, viruses, and eukaryotes, respectively; notably, bacteria encompass the most versatile groups in terms of pathogenicity profiles. The final group, the parasites, contains a wide variety of unicellular and multicellular eukaryotes that temporarily or permanently infest other cells or tissues (7). The diversity among these classes is such that a comparative analysis of their pathogenicity requires focusing on specific pathogenic mechanisms (8). These pathogenic traits have been broadly grouped into adhesion, penetration, invasion, and evasion

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mechanisms. Another classification has distinguished passive and active penetration of host cells, multiplication within host cells, and toxicity (9).

Pathogenic microorganisms can also be classified according to their routes of infection. In this context, four main routes are distinguished: direct contact with the infectious agent; contamination by respiratory droplets; transmission by vectors such as mosquitoes, which transfer pathogens from an infected organism into another; and transmission via the fecal–oral route, which can occur through excreta contamination of water or food, often associated with inadequate infection control and sanitation (10). The damage inflicted on human health by pathogenic microorganisms correlates strongly with the modes of infection (11). The relationship between infectious diseases caused by different pathogens and their transmission routes continues to shape human pathogenicity profiles; H. sapiens has established numerous ways of minimizing the consequences of infection, however. These countermeasures act on different aspects of the chain of events necessary for pathogens to cause disease (12). For example, water chlorination and sanitation block the transmission of many pathogens, particularly those that depend on vector-borne or fecal–oral routes (13). The intensive use of antibiotics, antivirals, and vaccines introduces enormous resistance for some groups of microorganisms, particularly bacteria and viruses. Despite these efforts, pathogenic infection remains a leading cause of mortality worldwide (14).

2.1. Bacteria

Bacteria are the most prevalent microorganisms both externally and internally. Intracellular bacteria invade and survive within host cells, whereas extracellular bacteria reside outside cells. Beneficial bacteria constitute an important component of the human microbiome; others are pathogenic and cause infections (15). These organisms are well-equipped to evade host immune responses through diverse mechanisms to establish infections (16). The susceptibility of a host depends on the effectiveness of the immune system, overall health, and genetic factors. Conditions such as malnutrition or comorbid chronic diseases may influence severity, as may age-related factors (17). Pathogenic bacteria transmit efficiently in healthcare settings, placing additional burdens on hospital infrastructures through increased levels of morbidity and mortality (18). Bacterial pathogens comprise a diverse variety of species, and quantifying their global burden has critical implications for immunization strategies and targeted vaccine development, as well as for addressing the rise in antimicrobial resistance (19). Many bacterial species have developed resistance mechanisms, which affect the management of infectious diseases and the survival of antimicrobial-resistant microorganisms (20).

2.2. Viruses

Viruses constitute a significant part of the microbial world and are considered one of the important origins of infectious diseases, causing great harm to human health and even outbreaks 21. Viruses consist of only one type of nucleic acid—either DNA or RNA—that can change from a single strand to a double strand and contain only 8–10 genes at most (22). The basic structure of a complete virus includes a nucleocapsid and nucleic acid, and may also contain an envelope comprised of lipids and proteins (23). During infection, viruses are unable to generate energy or synthesize proteins independently because of the lack of cell structures such as enzymes, ribosomes, and mitochondria (24). Therefore, they must rely entirely on host cells for energy and protein synthesis, using the genetic information of the host cells to achieve self-replication (25).

The route through which a pathogen enters the human body is an important factor determining the affected organs and the clinical manifestations of the resulting disease. Viral entry pathways often mirror the natural reservoir or source of infection (26). The principal routes include respiratory droplet transmission (influenza) and H5N1 avian influenza via infective droplets or contaminated objects; fecal–oral transmission (polio) through the ingestion of water or food polluted by the excrement of infected persons or animals; contact transmission (HIV, herpes simplex virus, subtypes of human papillomavirus) through sexual activity, cuts in the skin, or saliva and tears of an infected person; and mosquito vector transmission (Congo hemorrhagic fever virus) via mosquito bites (27). Consequently, pathogenicity mechanisms may involve the release of toxins and invasiveness; however, no virus is known to undergo invasion comparable with bacteria or other pathogens (28).

2.3. Fungi

The fungal kingdom encompasses millions of species, ranging from the largest organisms on earth to microscopic forms, some pathogenic for plants and animals (29). Many fungi perceive environmental cues and switch between forms, influencing their capacity to reproduce, invade tissues, and evade immune responses (30). Fungi play a vital role as degraders of organic matter, releasing nutrients from dead organisms, with enzymatic suites such as proteases and cellulases that also contribute to virulence as pathogens. They produce secondary metabolites, including antibiotics, immunosuppressants, and mycotoxins detrimental to health (31). Although once thought to be related to plants owing to their morphology, molecular analyses in the 1990s revealed a closer affinity to animals, complicating drug development due to fewer metabolic differences. Fungi form symbiotic relationships such as mycorrhizae and lichens, and commonly exist in complex communities and biofilms (32). As key members of microbiota, they promote ecosystem stability through interactions with bacteria in various environments. Some fungi harvest electromagnetic radiation for growth, displaying a degree of autotrophy (33). Their resilience enables habitation of extreme environments, including radioactive sites like Chernobyl and conditions simulating Mars.

Diseases caused by filamentous fungal human pathogens have increased considerably, especially among immunosuppressed individuals such as those with AIDS or undergoing chemotherapy, although immunocompetent persons can also be affected. Filamentous fungal human pathogens cause localized infections in lungs, sinuses, and other sites. Inhalation of conidia represents the principal infection route. Aspergillus fumigatus stands out as the predominant pathogen involved in 85–90% of invasive aspergillosis cases, mainly affecting patients with hematological malignancies or undergoing stem cell transplantation. Aspergillus flavus and other species also contribute significantly. Mucor species infect immunocompromised patients, with recent cases linked to contaminated yogurts; mucormycosis exhibits high mortality rates, particularly in gastrointestinal tract infections. Fusarium species are emerging as noteworthy opportunistic pathogens, with infection prevalence varying geographically (34, 35).

2.4. Parasites

Parasitic diseases are among the most neglected, and they primarily affect poor populations. The symptoms caused by parasites can vary from mild to life-threatening, determined largely by the parasite group and the number of parasites present. The main groups that appear in humans are protozoa, nematodes, flatworms, and external parasites such as lice, fleas, bedbugs, ticks, mites, and certain flies (36).

Parasitic infections are more common in low-income countries, partly because they are vector-borne and the eggs are found in the soil, remaining viable for many years until they are ingested. Infections caused by external parasites are generally transmitted during overcrowding or by poor hygiene conditions affecting the entire population relatively rapidly (37).

3. Mechanisms of Pathogenicity

Pathogenic microorganisms employ diverse strategies to damage the host and facilitate infection. Initial colonization involves attachment to epithelial surfaces through pili, adhesion molecules, and other structures, enabling resistance to mechanical elimination. Certain pathogens then breach the epithelium by transcytotic or endocytic mechanisms; for example, Salmonella and Shigella escape from phagocytic vacuoles into the cytoplasm, spreading directly to neighboring cells (38). Micro-organisms that penetrate beyond the mucosa must withstand host defenses, particularly phagocytosis, to establish systemic infection (39). The relative importance of direct microbial damage versus host-mediated injury varies among pathogens; ubiquitous viruses and intracellular bacteria like mycobacteria, Brucella, Rickettsiae, Mycoplasma, and Chlamydia cause cellular disruption by interfering with essential functions, whereas few viruses or rickettsiae produce potent exotoxins (40). Nevertheless, toxin production enhances virulence in organisms that multiply extracellularly without entering the blood or lymph, as exemplified by Clostridium and Bacillus species. Intracellular microbes typically do not generate powerful toxins, since premature host cell death would be counterproductive; accordingly, exotoxins rarely contribute to the pathogenesis of intracellular infections (28). Endotoxin—a complex of lipopolysaccharide and protein in the outer membrane of many gram-negative bacteria—may be regarded as an integral structural component rather than a secreted toxin. Rare bacterial factors that act at a distance from the site of infection might better be classified as “secondary products” (41).

3.1. Adhesion

Adhesion represents the preliminary phase of bacterial colonization and/or infection on abiotic solid surfaces (42). A number of clinically relevant bacteria, including Escherichia, Staphylococcus, Klebsiella, Enterobacter, and Salmonella species, can adhere tenaciously to surfaces in food-processing plants. Following adhesion, they can proliferate and generate biofilms that remain insusceptible to cleaning and sanitation procedures (43).

3.2. Invasion

Invasion is a critical step in pathogenicity, involving the entry and spread of microorganisms within host tissues. Certain bacteria possess surface receptors for integrins (a, d, and e) that trigger a pinocytic-like response, facilitating uptake into host cells (38). The process of bacterial invasion is orchestrated by the polymerization of actin filaments, activation of Rho GTPases, and engagement of various cellular kinases. Surface proteins such as invasin, InlA, and InlB mediate entry, while bacterial effectors like SopE, SipA, and IpgD manipulate the host cytoskeleton to promote internalization (44). Post-entry, species exhibit distinct intracellular behaviors: Shigella spp. escape into the cytoplasm, whereas Salmonella spp. alter the host endocytic pathway. Pathogens can survive and disseminate within polymorphonuclear cells, lymphocytes, and macrophages, thereby evading humoral immune components such as complement (45,46). Through the bloodstream, they can reach distant organs, including lungs, liver, spleen, and kidneys, giving rise to metastatic infections. Cell-to-cell spread is achieved by dissolution of adjoining membranes, allowing bacterial propagation without exposure to extracellular immune defenses, though innate immune responses remain stimulated (47).

3.3. Evasion of Host Defenses

Fungi have developed various evasion strategies to counteract the host immune system (48). Pathogens use stealth tactics to avoid detection, control mechanisms to suppress or divert immune responses, and attack strategies that directly impair immune functions through the secretion of inhibitory molecules (49).

Bacteria exhibit diverse immune evasion capabilities to enhance survival and persistence within the host (19). Approaches include inhibiting autophagolysosome fusion, residing in specialized vacuoles, escaping into the cytosol, and disrupting immune components such as inflammatory responses, complement pathways, cytokines, and chemokines (50). Bacterial species have also evolved methods to evade phagocytic killing: Shigella and Listeria secrete lysins to breach vacuolar membranes; Salmonella, Legionella, Brucella, Mycobacteria, Tropheryma, Anaplasma, and Coxiella employ type III or IV secretion systems to manipulate vesicular trafficking and alter phagosomal pathways; Yersinia delivers factors that target immune signaling cascades, inducing immunosuppression (51).

3.4. Toxin Production

Toxin production is a key mechanism of pathogenicity in some pathogenic microorganisms and constitutes an evolutionary strategy exploitable by parasites (52). A toxin is a poisonous substance produced by an organism that can cause disease or death in another. Toxins are produced by prokaryotes such as bacteria and cyanobacteria and by eukaryotes like dinoflagellates, diatoms, fungi, and animals (53). They show a wide variety of modes of action and chemical composition, including small molecules, peptides, alkaloids, and proteins. A single toxin can have a single effect (e.g., membrane permeabilisation) or can affect various cellular processes (e.g., interference with membrane traffic, signal transduction disruption, inhibition of protein synthesis, cytoskeleton alterations, and blocks in voltage-gated ion channels) (54). Because of their prevalence in natural aquatic environments worldwide, some microorganisms and their associated toxins are of concern for waters and food safety, public health, and the economy. Bacterial protein exotoxins and lipopolysaccharide endotoxins are two major classes that are known to cause disease and death, and no other known non-protein compounds are unique to either pathogenic organisms or pathogens (55). The chemical and biochemical action of toxins derives from their nonstoichiometric basis, which clearly differentiates the mode of action of exotoxins and endotoxins from other virulence factors. Bacterial and fungal toxins alter host-cellular metabolism to favor the spread of the pathogen or survival within the host (56). Bacterial exotoxins are usually proteins of A–B structure, in which the A-subunit is enzymatically active and responsible for the cytotoxicity. These can be classified as enterotoxins, neurotoxins, or leukotoxins (57). Tissue-specific toxins (tetanus and botulinum neurotoxins) act on specific target tissues, while broad-spectrum toxins such as hemolysins and leukocidins do not discriminate between different cell types (58). The B-subunit is responsible for binding to the host-cell receptor and delivery of the A-subunit (59). Four main strategies are used sequentially by pathogenic microorganisms to cause disease: adhesion to host cells, colonisation and invasion of host tissues, evasion of host defence systems, and toxin production. Each of the main pathogenic microbial groups (bacteria, viruses, fungi, and parasites) comprises taxonomic groups that also exhibit one or more of these mechanisms (60,61).

4. Transmission Routes

Direct contact is the simplest and one of the most common routes of transmitting infections. This can be from contact with an infected animal or person, or from contact with a contaminated surface (fomite). Droplet transmission involves contact with a large aerosol (> 5 μm diameter) through sneezing, coughing, or talking. This differs from airborne transmission, which refers to exposures to microorganisms carried by droplet nuclei (residue of evaporated droplets) usually of 5 μm or smaller (62). Airborne transmission also involves either direct deposition of the pathogen near the site of infection (usually the respiratory mucosa) or inhalation of the pathogen into the lungs. Airborne transmission can involve both short and long distances (63). Vector transmission includes an insect or animal that carries the infectious pathogen on its body or in its gut and transfers it to the next host either through biting or deposition of feces or other body fluids (64). A classic example of this is malaria, where mosquitoes carry the parasite that then grows within a person. The fecal–oral route of transmission occurs when pathogens found in an infected person's stool are ingested by another person, commonly through contaminated hands or environmental media such as food, soil, or water (65). This route is common with pathogens that infect the intestinal tract but can also affect other parts of the body. A particularly important group of pathogens that are transmitted via this route is the enteric pathogens, those that inhabit the gastrointestinal tract and are shed in large quantities in feces. Enteric pathogens include many viruses (e.g., norovirus, rotavirus), most bacteria (e.g., *Campylobacter jejuni*, enterohemorrhagic *Escherichia coli*, Salmonella), and some parasitic protozoa (e.g., Cryptosporidium, Giardia) (66,67).

4.1. Direct Contact

The incidence of disease associated with direct contact involves pathogens transmitted by physical contact between an infected individual and a susceptible host. Transmission can be by person-to-person contact, skin-to-skin contact, sexual contact, or direct contact with soil containing infectious microorganisms (62). Pathogens transmitted by direct contact include prions, viruses, bacteria, fungi, and parasites. Diseases transmitted by direct contact include Creutzfeldt-Jakob disease, rabies, herpes, chickenpox, influenza, smallpox, syphilis, gonorrhoea, Lyme disease, ringworm, hookworm, and leishmaniasis (68).

Infection by causative agents can occur by direct penetration of the skin, selective consumption of the skin, or crossing the mucous membranes lining the mouth, nose, and eyes. Crossing of the mucous membranes can occur following removal of the Protective layer of mucous (e.g., by inflammation) or by specialised mechanisms that allow pathogens to get through these protective layers (69).

4.2. Airborne Transmission

Airborne transmission involves the spread of microorganisms through aerosols and causes human disease by invasion of the mucosa of the mouth, nose, throat, and/or lungs (70). The spread of infection depends on the presence of infectious particles suspended in the air at concentrations sufficient to initiate an infection; the particles are transmissible by air currents and inhalable by a susceptible host (71). Airborne transmissions account for a significant fraction of all infections worldwide, and the incidence of many airborne diseases remains uncomfortably high. Following inhalation by a susceptible host, infectious viruses may establish residence in host cells of the respiratory tract and potentially infect other tissues (72). Influenza, caused by viruses A, B, and C, is a contagious respiratory illness with subtypes delineated based on surface proteins. Type A viruses infect animals, while Type B circulates only among humans. Influenza causes annual epidemics, and in the US, over 200,000 hospitalizations and 36,000 deaths occur annually. All age groups are susceptible to influenza infection, but the most at-risk groups are those older than 65 years, individuals with chronic medical conditions, such as heart or lung disease, and very young children. Transmission occurs from person to person, mainly through respiratory droplets. (73 74)

4.3. Vector-Borne Transmission

A subset of pathogenic microorganisms is transmitted by vectors. Potential vectors can include blood-feeding animals, notably the three main mosquito genera Anopheles, Aedes, and Culex (75). The causative agents of numerous important diseases are transmitted to humans and animals by these vectors (64). Malaria remains the most important parasitic disease of humans and is endemic in more than 80 countries, mainly in sub-Saharan Africa, where over 85% of cases and 90%

of deaths occur (76). Worldwide, malaria still inflicts enormous public-health damage, with 228 million cases chiefly in Africa; the disease has recently caused severe outbreaks in many areas. The parasites Wuchereria bancrofti and Brugia spp. can be transmitted by numerous mosquito species and cause clinical manifestations such as hydrocele and lymphoedema, affecting at least 36 million people (77). Control of lymphatic filariasis, therefore, depends on controlling the vectors. Dengue virus comprises four serotypes in the family Flaviviridae and is transmitted primarily by Aedes aegypti, with Ae. Aedes aegypti acting as a secondary vector; the pathogen currently constitutes one of the most serious public-health threats. Chikungunya virus, a Togaviridae, causes chikungunya fever, characterised by severe articular pain, and recent outbreaks have been reported globally (78). The yellow fever virus (Flaviviridae) produces haemorrhagic disease that can lead to potentially lethal outbreaks recurring every 7–10 years, especially in unvaccinated populations. Research on insect-associated microbial species indicates that the vector's microbiome can alter its competence for arboviruses by modulating host immune responses, competing with arboviruses for resources, and secreting antiviral factors (79). Developing alternatives to the traditional approach of direct control of the vector, therefore, requires an understanding of the tripartite relationships between the insect, its microbiome, and the arboviral pathogens that it harbours (80).

4.4. Fecal-Oral Transmission

Fecal–oral transmission occurs when pathogens shed in feces infect a new host by direct contact or through contaminated fluids such as surface water and food (81). Most pathogens transmitted in this way are stable under a variety of conditions, allowing them to survive passage through the gastrointestinal tract during infection. In addition, zoonotic pathogens must adapt successfully to gut-specific selective pressures such as low oxygen levels and acidity (82). For example, in hepatitis E virus infection, fecal shedding predominantly follows liver tropism rather than intestinal replication. The availability and distribution of receptors in the intestine of susceptible hosts also influence pathogen tropism and efficacy of human-to-human transmission. A number of zoonotic respiratory viruses are detected in stool, but there is no evidence for fecal–oral transmission (83).

Microorganisms transmitted by the fecal–oral route are usually described as enteric pathogens because they infect the gastrointestinal tract (62). They tend to be stable in water and food and, in the case of enteric bacteria, are capable of growth outside the host under appropriate environmental conditions, although it is important to distinguish between persistence of a carrier state and replication in the nonhost environment (84). Pathogens shed in feces, urine, saliva, or tears contaminate air, water, food, or inanimate objects. Transmission to a susceptible individual occurs via ingestion of contaminated water or food or through contact with a contaminated surface. Incubation periods vary considerably between enteric infections (85). For example, illness caused by Escherichia coli generally lasts 3–5 days, whereas Cryptosporidium may produce clinical disease that persists for weeks to months. Certain infectious agents, such as adenovirus and norovirus, may be transmitted through contaminated food or water or by aerosol, and ingestion of a single dose of these viruses may induce infection. Pathogens readily transmitted in feces (e.g. Salmonella and Shigella) occur in high concentrations and promote transmission. Protection of water sources and treatment of contaminated drinking water supplies are central strategies for controlling fecal–oral transmission in most developed countries, and reductions in exposure to enteric pathogens have been observed following the application of effluent standards that lessen impacts on receiving waters. In addition, contamination of water sources after flooding or via irrigation or food handlers may increase the likelihood of widespread outbreaks (86,87).

5. Impact on Human Health

Pathogenic microorganisms encompass bacteria, viruses, fungi, and parasites that cause harm within and outside a host body (7). Infections can have significant consequences, leading to acute infectious diseases or precipitating chronic conditions such as cancer or asthma. Viral infections are major causes of morbidity and mortality globally, and antimicrobial resistance is an increasingly serious public health threat (62).

5.1. Infectious Diseases

Pathogenic micro-organisms fall into four broad classes: bacteria, viruses, fungi, and parasites (38). These organisms have developed diverse strategies for propagating infection within their hosts. To establish infection, micro-organisms must overcome the host's defences, colonize surface tissues, invade local tissues, and then move to other sites, or avoid or compromise host immunity (88). Pathogens can also harm their hosts by producing toxic substances. Humans encounter these alien, disease-producing entities everywhere in the environment (89). Transported through air, water, or food, they instantly colonize living hosts. Moreover, the number of new infectious diseases that have emerged in the last two decades highlights the failure of vaccination campaigns and resistance to antibiotics to contain an ever-increasing threat. Each class of microorganisms can be considered separately (12).

The first of these broad groups includes Bacteria, whose ubiquitous nature allows them to colonize virtually every habitat on the planet. While most species that contribute to the ''biogeochemical cycles'' are metabolically benign to humans, the introduction of pathogens such as Vibrio, Salmonella, and Campylobacter into environmental compartments, especially in conjunction with a human host, can result in substantial and widespread intoxication and illness (90). Bacterial pathogens can be classified as either extracellular or intracellular (91). The former do not enter host cells and must avoid host defences to replicate and disseminate, whereas intracellular pathogens are phagocytosed by host cells and must avoid destruction by host cell lysosomes. Elaborate mechanisms have been developed by both groups to interfere with immune system recognition and nutrient acquisition (92).

Viruses are obligate intracellular parasites and require a host cell for replication and dissemination. As a result, they do not possess any type of metabolism and are entirely dependent on the host for these functions. In addition to the metabolic constraints of an obligate intracellular lifestyle, viruses must also be transmitted between hosts, thus increasing their requirements for propagation and transmission. Several different classes of viruses exist, with differing modes of overall replication and transmission schemes (93,94).

Fungi are ubiquitous throughout terrestrial and aquatic ecosystems and fulfil important roles in nutrient cycling, medicine, food spoilage, and the production of foodstuffs such as breads, beer, and wine. Most fungal organisms are not considered to be serious pathogens, although serious fungal infections exist, particularly in individuals with compromised immune systems. Rather, it seems that fungi are primarily related to human disease through the production of allergenic compounds through the colonization of wounds and, most commonly, through fungal toxins produced during infection (95).

Parasitic organisms encompass a wide variety of lifestyles and tropisms. Ectoparasitic species primarily feed on host blood and therefore cannot act as vectors for other infections; these are commonly associated with mites, fleas, and lice, amongst others. Endoparasitic organisms can be divided into helminths (multicellular worms) and protozoa (unicellular eukaryotes), which differ substantially in their disease pathology (96).

5.2. Chronic Conditions

Following infection, three prolonged disease courses—complications, sequelae, and chronic infections—may ensue. Complications emerge during the acute illness due to excessive inflammation, the introduction of new pathogens, or inadequate immune responses (97). Examples include varicella zoster following upper respiratory tract infections and bacterial pneumonia after influenza (98). Sequelae persist after the acute infection resolves and may constitute chronic diseases, frequently involving autoimmune pathogenesis as observed with Epstein–Barr virus infections. Chronic diseases are often associated with five microorganisms: HIV, hepatitis B virus, hepatitis C virus, Helicobacter pylori, and Chlamydia pneumoniae. Prevention and treatment of these pathogens constitute a promising public-health strategy for reducing the chronic disease burden (99). Chronic hepatitis, caused by hepatitis B and C viruses, exemplifies this group. A chronic infection persists despite an apparently successful immune response that controls but does not clear the invader. In many cases, a classic acute stage is absent, although for a few pathogens, an acute stage precedes the chronic stage. In persistent Helicobacter pylori infections, the host fails to eradicate the bacterium for long time periods. Elevated levels of inflammatory mediators and neutrophils characterize chronic infections (100,101).

5.3. Antimicrobial Resistance

Numerous infectious diseases caused by pathogens can be treated by a variety of antimicrobial agents. However, the overuse and abuse of antimicrobials have stimulated the emergence and spread of antimicrobial resistance (AMR). AMR in both the healthcare and agricultural sectors has imposed a substantial burden on the global economy and public health (102). Antibiotic resistance development is a gradual process wherein the microbes in question develop resilience against agents targeted towards their destruction (103). Addressing this threat requires aggressive action towards preventing infections and controlling transmission. Additionally, improving antibiotic use to slow the development of resistance through high-quality surveillance and usage guidelines is critical. Stopping the spread of resistant microbes when they develop can be managed through antimicrobial stewardship programs (104). Observed MDR profiles and increasing rates of drug-resistant campylobacteriosis pose Campylobacter species as a serious global health threat. Epidemics driven by MDR Shigella have risen worldwide within the last decade and require significant intervention efforts to prevent further disease. Salmonella enterica continues to be a major source of enteric infection, causing severe human health burdens without serious interventions. V. cholerae is one of the highest priority enteric bacterial pathogens due to its diverse resistance mechanisms and potential for further spread of resistant infections. C. difficile, transmitted as a metabolically inactive spore, has virulence enhanced by a mobile genome, with resistance to some treatments being rare. Resistance testing for anaerobes like C. difficile is not standard, possibly underestimating AMR prevalence (105,106). Pathobionts are increasingly recognized as significant sources of infection and reservoirs of antimicrobial resistance, largely due to advances in modern medicine (107).

6. Environmental Factors Influencing Pathogenicity

Environmental factors play a significant role in shaping pathogenicity and influencing the distribution of diseases. Climate change, sanitation issues, and the loss of biodiversity all contribute to shifts in pathogen dynamics and epidemiology (108). Health outcomes are affected as well, since alterations to the environment can extend the range of established diseases and increase the frequency or severity of new ones (12).

Certain types of pathogens are particularly impacted by those changes. Human and animal waste harbor pathogens that enter the environment directly after being shed, and they can survive for extended periods outside a host (62). Temperature is a key influence, with lower values increasing persistence times considerably (109). Freezing temperatures often reduce bacterial and protozoan burdens but may have little effect on viruses. Likewise, moisture levels modulate decay rates, and exposure to ultraviolet light from sunlight strongly inhibits the survival of bacterial pathogens, while viruses show greater resistance (110).

Most environmentally transmitted diseases can also undergo vector-borne, direct contact, or fecal-oral spread from person to person. Global warming, for example, may create additional habitats for vector species and new opportunities for microbial replication inside invertebrate hosts. Such range expansions could alter transmission dynamics and increase both the incidence and prevalence of vector-borne diseases in some regions (111). Anthropogenic habitat destruction and growing human migrations are liable to bring individuals into closer proximity to wildlife communities and novel infectious agents. This form of disturbance may prove especially dangerous for naive human populations that lack the evolutionary experience to mount effective immune responses when exposed to those microorganisms (112).

6.1. Climate Change

Climate exerts an influence with a magnitude comparable to that of immigration, sanitation, and poverty on the distribution of many pathogenic microorganisms, yet the impact of climate change on the epidemiology of infectious diseases remains poorly understood (113). Early studies examined climate change in the context of vector-borne diseases and demonstrated how a warming climate exerts a strongly non-linear impact on transmission, depending on how close local temperatures were already to the optimal temperature for transmission (114). More recently, studies on the impact of climate change on infectious diseases began to take into account the entire food system. The complexity of the food system necessitates a better understanding of the relative importance of different transmission routes along which pathogens spread (115). A systematic approach to estimating the likely impact of climate change on the epidemiology of foodborne disease is outlined, and a systematic literature review is performed to identify the likely climate drivers for a subset of key foodborne pathogens (116).

Climate can affect many stages of the farm-to-fork chain; for example, warm weather, high moisture, increased rainfall, and flooding may provide pathogens with conditions that support growth and survival both on and off the farm. Pathogens can nevertheless enter the food chain at many points, including at harvest, during transportation and processing, during retail, and also in the kitchen (117). Each of these provides a distinct set of climatic conditions which also affect the behaviour of consumers; for example, hot weather encourages people to barbecue food outside, where sanitation standards may be lower. Climate is expected to have impacts at many of these points and throughout the food chain (118). In this context, a total of 19 foodborne pathogens are identified from a much larger list of those of global importance, which might be affected by climate change. These pathogens cover the majority of major groups of pathogens, including bacteria, viruses, as well as protozoa and helminths, and the relative importance of different climatic factors for these pathogens is investigated (119).

6.2. Sanitation and Hygiene

Maintaining personal hygiene and environmental sanitation are effective measures to control the microbial diseases transmitted through airborne droplets (2). Vertical transmission by parents to newborn animals may take the form of congenital infections that occur mostly with viruses. For example, during the 1994-1995 respiratory syncytial virus (RSV) epidemic in Houston, Texas, the number of patients hospitalized to those placed on home care computed to a ratio of 1:7, but in the 2009 H1N1 influenza pandemic, the hospitalization-to-home-care ratio changed to 1:1 as a result of increased pathogenicity of the virus (120). However, the use of strict isolation and quarantine measures under these circumstances can easily limit transmission. Sanitation is all very well when water is available, but water may become scarce during times of social disruption when contagious diseases spread rapidly through direct or indirect contact with the fecal-oral route, being a primary mode of transmission for such diseases (121). The transmission of entero-epidemic diseases can be well prevented by effective sanitation and control of waste disposal, and can be further minimized by immunization with vaccines available for several enteric pathogens and some enteric protozoa. Hygiene and sanitation are closely related to the level of development of an area. Numerous parasitic worms commonly infect about 2 billion people worldwide, and they can be controlled by the maintenance of good hygiene and sanitation conditions (122). The four types of microorganisms that cause diseases are bacteria, viruses, fungi, and parasites. Of all these microbial pathogens, which also include protozoans, bacteria are the dominant cause of infection; bacteria, virions, and fungi are by far the most numerous among the infected population (88).

6.3. Biodiversity Loss

Biodiversity loss alters ecosystem structures and functioning, resulting in consequences for disease transmission in animals and humans. The dilution effect hypothesis—one of the leading theories for the underlying mechanisms for the relationship between biodiversity loss and human disease—suggests that in species-rich host communities, some host species can ‘‘dilute’’ infection by the amplification of hosts without amplifying the pathogen or reducing competent hosts. In contrast, the amplification effect implies the opposite, with greater species diversity increasing disease transmission. Both effects have empirical support, but the dilution effect is robust across a broader range of ecological settings and pathogen groups (123).

Biodiversity loss thus promotes disease transmission rates between humans and wildlife. Several studies have shown a positive association with declining species richness and levels of infection in zoonotic viruses originating in mammals: severe acute respiratory syndrome (SARS)-CoV in China during the 2002 and 2003 outbreaks; Middle East respiratory syndrome (MERS)-CoV in Saudi Arabia; Hendra Virus in Australia; and Nipah virus in Bangladesh. At a global scale, these associations have also been demonstrated for zoonotic diseases originating in mammal species, including mammal zoonotic viruses (123,124).

7. Conclusion

Despite the diversity of microorganisms in nature, only a limited number infect humans. Among these, bacteria, viruses, fungi, and protozoans are the principal types, with each class hosting pathogenic species. Consequently, it is unsurprising that the majority of emerging infectious diseases over the past century have been attributed to bacteria and viruses. The pathogens rarely cause disease unless introduced into a conducive environment—often due to lowered host immunity—and even then, they employ various strategies to circumvent host defenses before eliciting symptoms. Beyond acute infections, some pathogens establish long-term chronic or latent infections. These microbial traits directly influence transmission routes, which predominantly occur via fecal-oral or respiratory pathways in densely populated niches.

Importantly, pathogen presence is not synonymous with ongoing disease. Various physical, chemical, and immune clearing mechanisms can prevent or eliminate infections, underscoring the critical balance within natural ecosystems. The forms and modes of transmission elucidated here clarify how pathogens may spread between hosts and why particular transmission routes prevail. Nevertheless, shifts in environmental conditions can alter natural balances and favor the emergence or proliferation of new diseases. Climatic factors or habitat destruction, for instance, profoundly impact pathogenic microorganisms and their epidemiology.

At present, these issues constitute pressing concerns in public health. Understanding the relationship between pathogenic microorganisms and their environments requires extensive continued research. Such efforts are essential for advancing knowledge of microbial ecology, uncovering more effective control methods, and reducing the likelihood of the emergence and spread of infectious disease. Because bionts (living agents) substantially influence material flow, energy transfer, and information flow in natural ecosystems, further investigation is warranted to achieve these objectives.

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