Animals to Human Transmission of Intestinal Diseases: A Review of the Mechanism and Factors Involved

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Bilal Khan[1*], Abdullah Channo[2,3], Raza Rehman[1], Azhar Hyder Qazi[4], Haris Akbar[1], Namrah Zaynub[1], Hamad Ali[1], Khushboo Soomro[1] and Muhammad Hassan[1]

ABSTRACT

Many intestinal diseases in animals either in pets or livestock animals have the potential to transmit to humans and cause disease. These diseases include bacterial (campylobacteriosis, salmonellosis, yersiniosis, plesiomonas and Aeromonas, clostridial disease, shigellosis, colibacillosis), protozoal (coccidiosis, giardiasis, amoebiasis, balantidiasis, trichomonas), helminthic (strongyloidiasis, echinococcosis, echinococcus multilocularis, taeniasis, coenurosis, dipylidium caninum, cutaneous larva migrans) and even viral (parvovirus infection). The mechanism of transmission, clinical features and intermediate vectors vary with disease to disease. The most common route for transmission of these diseases is the fecal-oral route. Many of the causative agents for these diseases are the normal inhabitants of the intestinal tract of the animals. The widespread presence of these reservoir hosts determines the prevalence of these diseases. Other factors such as the persistence of causative agent in the environment, effective fecal shedding and efficient use of transmission vectors also determine the prevalence of a zoonotic disease. Transmission of campylobacter to humans principally occurs through contaminated animal-origin food, water and direct contact with infected animals specially pets. Salmonella transmission primarily occurs through close contact with the infected animals, contaminated food, contaminated raw poultry and meat, aerosols and oropharyngeal secretions. The primary source of Yersinia serotypes 0.3 and 0.9 are swine. Cryptosporidial transmission happen in two ways: direct or indirect transmission. Direct transmission occurs through oral exposure to oocytes and indirect transmission occurs through cross-contamination. Transmission of giardia occurs through cysts that are very resistant to harsh environmental conditions. Helminthic diseases are transmitted by ingestion of either larvae (Strongyloidiasis), eggs (Echinococcosis) or meat of infected animal (Taeniasis).

Keywords: Zoonosis, Intestinal diseases, Bacterial zoonosis, Protozoal zoonosis, Helminthic zoonosis, Viral zoonosis

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[1]Shaheed Benazir Bhutto University of Veterinary and Animal Sciences, Sakrand
[2]Pakistan Agricultural Research Council-Arid Zone Research Centre (PARC-AZRC), Umerkot, 69100

[3]Department of Animal Reproduction, Faculty of Animal Husbandry and Veterinary Sciences, Sindh Agriculture University, Tandojam, 70060
[4]Sindh Agriculture University, Tandojam
***Corresponding author:** dr.bilalkhan.vet@gmail.com

1. INTRODUCTION

Humans have long been prone to intestinal diseases transmitted from animals. These intestinal diseases include bacterial, protozoal, helminthic, or viral infections. In recent times, certain factors, such as the use of immunosuppressive drugs (Bulbuloglu et al. 2022) and the prevalence of acquired immunodeficiency syndrome (AIDS) (Kelly et al. 2009) have heightened the importance of these conditions in the human population. The presence of many pathogens in the stool has made it complicated for researchers to discover the disease's actual cause. Therefore, many of these diseases lack comprehensive understanding. Pathogens can often exist in many species, indicating a potential absence of host specificity. This phenomenon then raises concerns about the possibility of transmission between different species. Despite knowledge about the host specificity of a pathogen, understanding its zoonotic risk is often impeded by challenges posed by its transmission mode. This raises concerns about humans health, which can only be prevented when we have accurate information about the host specificity and mode of transmission of the pathogen from one species to another.

Both pet animals and livestock species are essential for their zoonotic risks. In a pet-centric society, the involvement of dogs and cats in transmitting these diseases gains importance, while in rural areas, livestock animals are mainly involved in the incidence of zoonotic diseases.

2. BACTERIAL DISEASES

2.1. CAMPYLOBACTERIOSIS

Campylobacter is widely recognized as the primary cause behind bacterial foodborne diarrheal disease globally. Symptoms vary from mild to severe infections, particularly in children and the elderly, with potential permanent neurological effects. Lastovica et al. (2014) described 30 species and sub-species of Campylobacter genera, 17 of *Arcobacter*, and 7 of *Sulfurospirillum.* This microorganism, characterized by its cytochrome oxidase positivity, curved Gram-negative, non-spore-forming (Garénaux et al. 2008), rod shape, and unique corkscrew motility, thrive in low oxygen environments and is commonly found in the intestinal tracts of numerous wild and domestic animals, avian species notably poultry, are among the primary carriers of this organism (Silva et al. 2011; Davis and DiRita 2008; Ragimbeau et al. 2014). In 1906, two British veterinarians documented *Campylobacter* for the first time. They reported the discovery of a peculiar organism in large quantities in the uterine mucus of a pregnant sheep (Skirrow 2006). Subsequently, *Campylobacter* species were also isolated from the fecal samples of cattle and pigs experiencing diarrhea (Klein-Jöbstl et al. 2016; Debruyne et al. 2008; Epps et al. 2013). The studies raised increased interest in *Campylobacter* as the researchers observed a high occurrence of these bacteria in human diarrhea cases (On 2001; Huang et al. 2009).

Amongst the various *Campylobacter* spp., *Campylobacter jejuni* and *Campylobacter coli* are widely acknowledged as the most important enteropathogens. These two species are particularly noteworthy for their impact on causing gastrointestinal infections in humans. During the early 20th century, *Campylobacter* was identified as a pathogen primarily associated with animals. Extensive research during this period shed light on its prevalence and impact as a pathogen affecting various animal species (Acheson and Allos 2001). In the late 1970s, *Campylobacter* was recognized as an essential human

pathogen (Rautelin and Hanninen 2000). Before that, its role in causing human infections and its impact on human health was not fully understood.

2.1.1. TRANSMISSION TO HUMANS

Campylobacter is a part of the normal intestinal flora in a wide range of domesticated and wild animals (Horrocks et al. 2009). Moreover, *Campylobacter* can also be carried by pet animals, including dogs, cats, and birds (Pintar et al. 2015). Given the widespread presence of its reservoirs, it has the potential to contaminate surface water and soil. Transmission to humans principally occurs through ingesting contaminated animal-origin food, consuming contaminated water, and direct contact with infected animals, particularly pets (Luangtongkum et al. 2009; Fernandes et al. 2015). Nichols and Shane have described the transmission of *Campylobacter* by flies. Their findings have highlighted the role of flies as potential vectors for *Campylobacter* transmission (Shane et al. 1985; Nichols 2005).

2.1.2. CLINICAL FEATURES

Campylobacter infection usually manifests as an acute gastrointestinal illness that presents symptoms resembling those caused by *salmonella* or s*higella*. It is necessary to detect *Campylobacter* in the patient's stool because of its similarity with other diarrheal disorders. This ensures the definitive diagnosis of the specific pathogen responsible for the disease. In the outbreak investigations, the usual incubation period after the ingestion of *Campylobacter* has been observed to be four days or less. However, incubation periods of one week or longer have also been observed in other instances, and these may be more common when low levels of pathogens are ingested (Horn and Lake 2013). In addition to affecting the incubation period, the infective dose, the virulence of the strain, and the host's susceptibility to infection also affect clinical symptoms. Symptoms usually start with abdominal cramps, watery diarrhea, and fever. More than eight bowel movements are in the severe form of illness, followed by bloody diarrhea (Acheson and Allos 2001) in almost 1/3 of the patients, indicating that infection has spread to the large intestine and rectum. Other non-specific symptoms include headache, vomiting, rigor, and myalgia (Lastovica et al. 2014).
However, the most critical aspect of *campylobacter* infection is the cramping of the abdomen, which may be so severe that it imitates appendicitis. The disease may last around four days. However, the patient continues to shed the pathogen in stool for up to 69 days (Black et al. 1988). Shedding may last up to 54 days in poultry (van Gerwe et al. 2009), so poultry litter plays a significant role in transmitting *Campylobacter* infection to humans. Nosocomial infections in neonatal intensive care units have also been reported (Wagenaar et al. 2014).

2.2.3. TREATMENT

Infection is usually self-limiting for mild infections; however, erythromycin is attempted as a drug of choice in people with compromised immune systems or with severe symptoms like bloody diarrhea and fever. Alternatively, tetracyclines and fluoroquinolones may also be used as effective antibiotics. The increasing use of these antibiotics in humans and food animals has raised the frequency of resistant campylobacteriosis (Aarestrup and Engberg 2001; Wieczorek and Osek 2013).

2.2. SALMONELLOSIS

The genus Salmonella causes salmonellosis. Two species, *S. enterica* and *S. bengari,* are responsible for the disease. They are motile, gram-negative, rod-shaped, and facultative anaerobic bacteria. There are

more than 2400 serotypes (serovars) of *salmonella* (Brenner et al. 2000). They are present in the environment and produce illness only in people with compromised immune systems. They have a broad host range, from humans to domestic animals and avian species. Salmonella can be isolated and identified in almost all vertebrate species, but many salmonella serotypes have immensely evolved to be associated with a single host; examples include *S. Typhi* (primarily in humans), *S. Dublin* (in bovines), *S. Gallinarum* (specific to poultry), *S. Choleraesuis* (in swine) (Griffith et al. 2019). In the case of horses, salmonellosis is non-specific and can be caused by many of the serotypes.

In most cases, salmonellosis in humans is caused by four serotypes of *S. enterica* (*S. enteritidis, S. Typhimurium, S. Newport, S. Heidelberg*). They can survive for long periods in the environment because of their resilient nature, up to 54 days in water reservoirs and more than a year in cattle feces (McGuirk and Peek 2003; Moore et al. 2003). They can proliferate in a temperature range of 7°C to 45°C and a broad pH range of 6.5 to 7.5. They are sensitive to sunlight exposure and many commonly employed disinfectants, including phenolic compounds, iodophors, and sodium hypochlorite (Joseph et al. 2001). The source of infection for animals may vary but typically is from contaminated feed, water, and contaminated environment or by excreta from infected animals. Outbreaks may be a result of fertilizers and feed supplements that contain meat meal, bone meal, fish meal, and other by-products or plant products that may be contaminated. Water can also serve as a source of infection only when surface water is used for consumption. Tap water is unlikely a source of infection.

Centers for Disease Control and Prevention (CDC) reported that four serotypes of *salmonella enterica* were responsible for most of the *salmonella* cases (almost 60%), *S. enteritidis* (24.7%), *S. typhimurium* (23.5%), *S. newport* (6.2%), *S. heidelberg* (5.1%). S. typhi, which is restricted to humans only, other host-specific salmonella serotypes such as *S. Dublin* in bovines, *S. gallinarum* in poultry, and *S. choleraesuis* in swine can also be transmitted to humans. Moreover, non-host-specific serotypes can also be transmitted to humans and cause disease.

2.2.1. TRANSMISSION TO HUMANS

Salmonella has all the necessary characteristics that enable them to have a wide distribution and a wide range of reservoir hosts, their persistence in the environment for long periods, effective fecal shedding, and efficient use of transmission vectors. Its serotypes can be found and isolated from the feces of almost all cold-blooded and warm-blooded animals. Transmission to humans primarily occurs through the fecal-oral route. Transmission can occur by close contact with the infected animal or human by contaminated environment and food. Direct contact with the infected animal, clinically or sub-clinically, is an essential source of human infection. However, contaminated food remains the most significant mode of transmission for humans. Contaminated raw poultry and meat are the primary culprits of transmitting *salmonella* infection. In addition, aerosol transmission (for short distances) and transmission through oropharyngeal secretions because tonsils become contaminated following illness are also modes of transmission. *Salmonella* spp. are widespread in the environment and the reservoir hosts. When examined for salmonella in their feces, non-diarrhoeic dogs showed a 9.47% prevalence in Xuzhou and 3.6 percent in Trinidad and are carriers of a maximum of 28 salmonella serotypes. Multiple serotypes may be present simultaneously (Seepersadsingh et al. 2004; Wei et al. 2020). They shed them in their feces and may serve as a potential source of infection for humans. Dogs usually shed *salmonella* in their feces for 3 to 4 weeks and rarely as long as 100 days following infection (Bagcigil et al. 2007). In the case of cats, the isolation rate from feces was 1.77 percent (Wei et al. 2020). It suggests that pet lovers may have higher chances of acquiring the pathogen and developing the disease than others. Salmonellosis in humans is more common in fall and winter (Oloya et al. 2007).

2.2.2. CLINICAL FEATURES

Clinical features of the disease vary with the virulence of serotypes, host defenses, and the level of initial inocula. Non-typhoidal salmonellosis, along with other foodborne illnesses, is a worldwide health problem. Clinical symptoms for non-typhoidal salmonellosis begin to appear after 12 to 796 hours but sometimes may extend to a week or longer after entry of the bacteria into the body and vanish in 5-7 days (Eikmeier et al. 2018). Clinical manifestations of non-typhoidal salmonellosis are usually self-limiting diarrhea, fever, abdominal cramps, and vomiting (Onwuezobe et al. 2012). A non-typhoidal salmonellosis investigation in an outbreak found that all the cases had diarrhea that was followed by fever in 96.2%, headache in 84.9%, abdominal pain in 50.1%, nausea and vomiting in 49.1% and body ache in 39.6% (Singh et al. 2013). Septicemia may also occur in some instances (mainly in elderly >50) that may lead to pneumonia, osteomyelitis, or, in some cases, meningitis (Chen et al. 2012). Unlike other non-typhoidal pathogens, *S. Choleraesuis* produces severe septicemia (Griffith et al. 2019). The mortality rate is more in immuno-compromised people and infants.

2.2.3. TREATMENT

The disease is usually self-limiting. In severe cases, supportive therapy should be recommended. Antibiotic therapy should be avoided as it may prolong the carrier state of the pathogen. *In-vitro* resistance pattern for non-typhoidal salmonella infections was 79% for ampicillin, 72% for co-trimoxazole, 55% for gentamicin, and far less for chloramphenicol (0.3%). Thus, chloramphenicol may be considered the drug of choice for non-typhoidal salmonellosis (Graham et al. 2000). However, samples should be tested for antimicrobial sensitivity before antibiotic therapy because *salmonella spp.* show variable patterns of resistance. In septicemic patients, fluid therapy, intravenous steroids, and sometimes plasma transfusion may also be recommended.

2.3. YERSINIOSIS

Yersiniosis is generally a self-limiting gastrointestinal disease of worldwide concern. It is caused by 3 of the 11 species of the genus *Yersinia* of the family *Enterobacteriaceae*. *Y. pestis, Y. pseudotuberculosis,* and *Y. enterocolitica* are the three most important species because of their disease association with animals and humans (Duan et al. 2014). *Y. kristensenii, Y. intermedia, Y. aldovae, Y. fredericksenii, Y. bercovieri, Y. mollaretii, Y. ruckeri, and Y. rohdei* are widespread in the environment, but they are not associated with the disease (Sulakvelidze 2000). These are gram-negative bacilli with a facultative anaerobic nature. *Yersinia* colonies are lactose-negative. These bacteria can be isolated by 'cold enrichment' (the capability of bacteria to grow at 4°C) (Jiang et al. 2000). Virulence of the *Yersinia* species depends on the presence of 70-75 kb plasmid. *Y. enterocolitica* (YE) is the primary causative agent behind human yersiniosis. The related *Y. pseudotuberculosis* (YPT) can also cause the disease, but human infections are less common than YE. *Y. pestis* is associated with the respiratory system. There are 70 serovars of *Y. enterocolitica* and 21 of *Y. pseudotuberculosis* (Kenyon et al. 2017; Nieckarz et al. 2020).

2.3.1. TRANSMISSION TO HUMANS

YE infections are typically transmitted by fecal-oral route. Yersiniosis is found worldwide but is most commonly observed in Europe (Galindo et al. 2011). *Yersinia* is prevalent in food animals, particularly

pigs (Fredriksson-Ahomaa et al. 2006). Serovars 0:3 and 0:9 of YE are primarily found in swine and are the chronic carriers of these strains. However, these are very rarely isolated from the environment. They carry 0:3 and 0:9 serovars of YE in their feces and throat. *Yersinia* species were also isolated from domestic dogs in China (Wang et al. 2010) and bats in Germany (Mühldorfer et al. 2010). In a recent European study, wild rodents were found to carry YE, which suggested that they might facilitate reservoir transmission (Backhans et al. 2011). YE has also been isolated from flies (Rahuma et al. 2005), further complicating human disease transmission. Swine represents the primary source of yersiniosis. However, recent reports have also indicated the presence of *Yersinia* in contaminated chicken, milk, tofu, and water (Lynch et al. 2006; Bonardi et al. 2010). Serovar 0:8 of YE is much more prevalent in the environment than the 0:3 and 0:9 serovars. Sources of contamination of 0:8 serovar mainly include drinking water from wells or streams (Terech-Majewska et al. 2016), food washed from water (tofu, bean sprouts), and milk products (Longenberger et al. 2014). It was found in Germany that most infection cases due to pathogenic serotypes (0:3, 0:9) in humans were due to the ingestion of raw pork in the country (Bucher et al. 2008). Infection can also be transmitted from person to person involving the fecal-oral route. Healthy individuals may be asymptomatic carriers of yersiniosis and may be a source of infection for others. This problem becomes particularly concerning in the case of blood transfusion. *Y. enterocolitica*, present in blood products stored at 4°C, may proliferate and produce a septic shock upon transfusion. This condition is fatal in up to 54.5% of the cases (Guinet et al. 2011). Person-to-person transmission of serovar 0:8 has not been evidenced. Also, humans are not chronic carriers of this serovar. Transmission of *Y. pestis* mainly occurs through vectors; the most common is flea bites. Fleas ingest the bacteria along with blood and transfer it to the next host when they bite the infected host. The source of *Y. pestis* is primarily wild rodents. Outbreaks occur when bacteria pass from wild rodents to domestic ones and then to humans via flea bites. Person-to-person transmission can also occur via aerosols. Unlike *Y. pestis*, YPT transmission does not involve any vector and occurs through ingesting contaminated food and water. Direct contact with infected animals or humans can also transmit the bacteria to healthy people.

2.3.2. CLINICAL FEATURES

Y. enterocolitica infection is characterized by self-limiting diarrhea, abdominal pain, and low-grade fever. The infection starts with the ingestion of contaminated food or water. After ingestion, bacteria adhere to the small intestinal inner wall, cross the intestinal barrier, multiply in Peyer's patches, and eventually cause lymphadenitis. However, in individuals with compromised immune systems, chronic conditions such as arthritis can also develop (Galindo et al. 2011). Infection sometimes spreads to mesenteric lymph nodes and then disseminates into the spleen and liver. Subsequently, extracellular replication leads to monoclonal abscesses (Trülzsch et al. 2007). Acute gastroenteritis is observed mainly in children owing to their immature immune system. However, in older children and adults, yersiniosis can lead to a range of other complications such as pseudo appendicular syndrome, mycotic aneurysm (Maykel and Steele 2011; Robins-Browne and Hartland 2003), and sepsis from blood transfusions or as a secondary complication. Moreover, yersiniosis can also result in chronic conditions such as erythema nodosum, glomerulonephritis, uveitis, reactive arthritis, and myocarditis (Galindo et al. 2011). Nonetheless, enteropathogenic yersiniosis is commonly self-limiting unless the individual is not immunocompromized. As a result of bacteremia, the mortality rate can reach up to 60% in people with compromised immune systems (Robins-Browne 2012). Y. pseudotuberculosis infection is usually self-limiting. Common Symptoms include low-grade fever, mild diarrhea, and abdominal pain on the lower right side, mimicking appendicitis. But on opening the abdomen, the appendix is found normal

with inflamed mesenteric lymph nodes surrounding it. Infection can rarely lead to sepsis with a high rate of mortality (>70%) (Deacon et al. 2003). There are two forms of Y. pestis infection (plague): bubonic and pneumonic. In bubonic form, bacteria colonize the proximal lymph nodes, causing 'bubon.' Sometimes, bacteria enter the bloodstream and reach the lungs. Lungs are the primary site for Y. pestis multiplication. Bacteria multiply rapidly and spread to the bloodstream, causing septicemia and death within a few hours. Thus, this form of disease is characterized by the absence of clinical symptoms.

2.3.3. TREATMENT

In the case of mild disease, antimicrobial therapy is not needed. However, systemic infection should be treated. Antimicrobial-susceptibility tests should be recommended because YE, YPT, and Y. pestis are resistant to many antibiotics. Before the susceptibility test results, a combination of aminoglycosides and doxycycline can be started. Resistance to penicillin and 1st generation cephalosporins is widespread (Fàbrega and Vila 2012).

3. MISCELLANEOUS BACTERIA

3.1. PLESIOMONAS AND AEROMONAS

These two genera of bacteria are vibrios, gram-negative, and facultatively anaerobic. These are not the ordinary inhabitants of the human GI tract. These have been suggested as causative agents for diarrhea because of their prevalence in patients with diarrhea (Von Graevenitz 2007). The bacteria have not been isolated from poultry, cattle, and pigs (Arai et al. 1980). However, samples from dogs, cats, fish, and river water were found to harbor these organisms. Dogs and cats are not known in detail for their role in transmitting these organisms. A major source of human infection may be contaminated water sources. Clinical symptoms are commonly presented as self-limiting diarrhea and dysentery in infants. *Aeromonas spp*. cause septicemia in debilitated individuals (Janda 2002). These are of low prevalence. However, diarrhea due to an unexplained cause may be suggested to be due to *Plesiomonas* or *Aeromonas*. Antimicrobial sensitivity tests should be adopted for the treatment of these bacteria.

3.2. CLOSTRIDIAL DISEASE

Clostridium difficile causes chronic diarrhea and pseudomembranous colitis in infected persons (Kuipers and Surawicz 2008). It is a gram-positive, obligate, anaerobic, rod-shaped bacterium. This disease cannot be associated with the bacteria in the stool. One study found that 21 percent of people have this bacterium in their stool. The presence of toxigenic strains establishes the disease. Sufficient toxins are necessary to associate this organism as the causative agent for diarrhea in a patient (Sambol et al. 2002). Dogs and pigs harbor *C. difficile* and shed in their feces (Viegas et al. 2020). There is not much evidence that pets may transmit the pathogen to humans. The disease may be nosocomial in humans in some instances. *C. difficile* can also cause disease in dogs. Metronidazole has been effective in dogs. Metronidazole and vancomycin have been curative in people.

3.3. SHIGELLOSIS

Shigellosis is caused by *Shigella* spp., which are gram-negative, aerobic bacteria. The bacterium has been isolated from dogs with low prevalence rates of 0.3 to 0.5 percent. There is no such data available for the cats. This suggests that dogs may be an unlikely but possible source for humans. However, they are immune

to the disease. The bacteria enter the GIT through the oral route and invade the intestines' epithelial cells. The disease in people is characterized by several clinical presentations, of which fever, dysentery, and abdominal pain are best known. However, asymptomatic infection may also occur. In suspected individuals, sulphonamides, tetracyclines, or beta-lactams may be tried (Christopher et al. 2010).

3.4. COLIBACILLOSIS

*Through various mechanisms, Escherichia coli causes diarrhea in humans, cattle, pigs, and many other animal*s. These mechanisms include the action of both heat-stable and heat-labile enterotoxins, invasion of enterocytes, and mechanical obstructions and disruptions to the brush border. Human infection is clinically manifested as severe abdominal pain, diarrhea, which is often bloody, and vomiting. Farm animals are a significant source of infection for humans. In contrast, there is little evidence that companion animals carry non-invasive, enterotoxigenic *E.coli.*

4. PROTOZOAL DISEASES

4.1. COCCIDIOSIS

Coccidiosis, primarily cryptosporidiosis, is a widespread intestinal disorder in animals and humans. It can affect the GIT, respiratory tract, kidneys, and biliary tract (Hunter and Nichols 2002). It can be either a contributing factor or a critical cause of acute enteritis in humans and animals. *Cryptosporidia* spp. are coccidial protozoa with a small size of 4-5µm. It is not confirmed whether it has multiple species; however, it can infect numerous host ranges (Pumipuntu and Piratae 2018).

4.1.1. TRANSMISSION TO HUMANS

Farm animals are considered the most important source of cryptosporidiosis for humans. Researchers in the United States, United Kingdom, Ireland, and Australia have implicated contact with cattle as a significant risk factor for humans to get the infection (Robertson et al. 2002; Roy et al. 2004; Hunter et al. 2004; Goh et al. 2004). However, sheep have also been considered a source of human cryptosporidiosis, with only a few studies pointing to their involvement. Companion animals are less frequently implicated a source of human cryptosporidiosis. However, in the United States, a weak association was observed between cryptosporidiosis in HIV+ persons and dog contact (Glaser et al. 1998).

Transmission to humans can happen in two ways: direct or indirect transmission. Direct transmission occurs through oral exposure to oocytes excreted in feces. Transmission can happen from animal to human and from human to human, usually in hospitals, daycare centers, water parks, swimming pools, and direct contact with human feces during anal sexual contact. This becomes particularly important during sexual intercourse between men (Hellard et al. 2003). Direct transmission can also happen through direct exposure to infected animals. Veterinarians and animal researchers are at high risk of getting the disease through direct contact with the infected animal.

Indirect transmission can occur through cross-contamination, including contaminated food materials, drinking water, and fomites such as contaminated footwear and clothes used in farms and wildlife facilities. Oocytes resist environmental odds and many disinfectants; direct contact is often unnecessary. Each oocyte contains four sporozoites inside it. Once in the intestine, the enterocyte brush border is the primary attachment site for sporozoites. Sporozoites are then converted to merozoites. Merozoites infect more enterocytes and increase in number. The gametogony stage follows, and two types of oocytes are formed: thin-walled and thick-walled oocytes. The former ruptures inside the intestine

and causes hyper infection (Abou-Bakr et al. 2019), while the thick-walled oocytes are shed in the feces. The protozoa can be found throughout the small and large intestines; however, the ileum is most affected (Del Coco et al. 2012). Oocytes are excreted in the feces and contaminate the environment, such as soil and water bodies, sewage, or slurry, mainly insufficiently treated domestic water supplies. High rainfall and flooding can distribute the contamination to longer distances (Jiang et al. 2005).

4.1.2. CLINICAL FEATURES

Cryptosporidiosis symptoms in humans depend on the immunity status of the individual. In immunocompetent people, infection is usually asymptomatic or self-limiting diarrhea for 5 to 10 days, sometimes accompanied by fever, nausea, abdominal pain, constipation, or weight loss (Siciliano et al. 2020). These symptoms usually subside with the development of immunity in the affected people. In immuno-deficient people, symptoms typically comprise severe watery diarrhea, fever, abdominal pain, and weight loss and can persist for more than a year (Tzipori 1983; Current 1985).

4.1.3. TREATMENT

In immune-competent persons, infection usually subsides without treatment; however, supportive therapy with fluids and electrolytes should be considered. In immuno-deficient individuals, anticryptosporidial drugs may be attempted, but the protozoa are resistant to many of the medicines (Al-Matha and Alsalem, 2012). Spiromycin, clarithromycin, paromomycin, and nitazoxanide are recommended treatment regimens (Acikgoz et al. 2012). Immune-suppressive and cytotoxic drugs should be avoided (Angus 1983; Pitlik et al. 1983a, b).

4.2. GIARDIASIS

Giardiasis is a common enteric illness in the human population and domestic animals, including livestock, dogs, cats (Thompson and Monis 2004; Thompson 2004), and wildlife (Appelbee et al. 2005). The species *Giardia duodenalis* is responsible for most of the cases of giardiasis in humans and most mammals. Thus, it is considered a zoonotic disease. In Asia, Africa, and Latin America, about 200 million people are affected annually (Yason and Rivera 2007). Asymptomatic giardiasis is prevalent in the developing world (Hellard et al. 2000; Thompson 2000). Symptomatic giardiasis typically causes self-limiting diarrhea, abdominal pain, bloating, and weight loss.

4.2.1. TRANSMISSION TO HUMANS

The infective stage of *giardia* is a cyst. Once inside the host, the cyst is excysted in the first part of the small intestine and releases trophozoites, a self-replicating stage of the parasite. The trophozoites divide and increase in number. In response to bile salts and other intra-intestinal conditions, the trophozoites are again converted to cysts and excreted in the feces of the affected host. Cysts are then spread in the environment and reach the intestinal tract of the following host by contaminated food, water, fomites, and through direct contact with the infected host. A minimum of 10 cysts can cause disease in a person. Since 1954, 132 waterborne outbreaks have been reported; 104 were associated with drinking water, 18 were linked to recreational water, and 10 were connected to foreign travel (Karanis et al. 2006). Beavers are a significant cause of water contamination with *giardia* (Tsui et al. 2018).

Furthermore, foodborne outbreaks were found to be associated with food handlers infected with *giardia* and the food handlers who had been changing the diapers of infected children before handling food (Hoffmann et al. 2007). Vegetables, ice, and chicken salad have also been responsible for foodborne outbreaks. Outbreaks due to person-to-person transmission of giardiasis in childcare centers are common.

The disease is common in cattle, pigs, sheep, goats, deer, and elk. Dogs are also a common source of *giardia* for humans. 1 to 12 percent of dogs have been reported to have asymptomatic giardiasis, while 25 to 36 percent prevalence of giardiasis has been reported in dogs with diarrhea. Cats usually have a 1.4 to 5 percent prevalence.

4.2.2. CLINICAL FEATURES

Human giardiasis has severe acute symptoms characterized by explosive (severe), watery diarrhea, abdominal cramps, nausea, flatulence, and anorexia. There may be fecal blood and mucous, but it is uncommon (Newman et al. 2001). Acute symptoms are usually eliminated after 1 to 3 weeks without treatment. Chronic disease characterized by weight loss, irritable bowel syndrome (IBS), food allergies, arthritis or chronic fatigue syndrome (Einarsson et al. 2016). Immune-deficient people may present severe symptoms of giardiasis. Less common, long-term consequences of giardiasis include cholecystitis, fever, urticaria, and ocular inflammation (Khalifa et al. 2007; Lamps and Lamps 2010).

Giardiasis in dogs is usually asymptomatic. Symptomatic infection develops after 1 to 3 weeks post-exposure and is characterized by soft stool with mucous, hematochezia, and symptoms related to chronic ulcerative colitis have been reported. Giardiasis symptoms in cats are usually the same as in dogs, except that these are generally eliminated within 4 to 5 weeks.

4.2.3. TREATMENT

Nitroimidazoles (for example, metronidazole, ornidazole, tinidazole, ipronidazole), quinacrine or furazolidone are the recommended medications used against giardiasis. Most drugs have efficacy more than 80 percent (Ordóñez-Mena et al. 2018). Metronidazole and tinidazole usually have efficacy of more than 90 percent (Upcroft and Upcroft 2001). In one Malaysian trial, ornidazole showed 100 percent efficacy (Wright et al. 2003). Metronidazole has been reported to be 100 percent efficacious at high doses in people (Brandborg et al. 1980), while it has been documented to be 67 percent effective in dogs. There is one report of quinacrine and metronidazole having a synergistic effect when a combination of both treated giardiasis in an immuno-deficient person who did not respond to several treatments with quinacrine and metronidazole separately (Smith et al. 1982).

5. MISCELLANEOUS PROTOZOAL DISEASES

5.1. AMOEBIASIS

Unlike other parasitic protozoa, *Entamoeba histolytica* has a simple life cycle. It lives as the motile trophozoite in the host's intestine or the infective cyst. Human beings or primates are the only natural hosts of this protozoa. Cysts are ingested through food and water that has been contaminated. Once inside, cysts are excysted in the intestine and trophozoites invade the intestinal epithelium causing ulcerations. Sometimes, they can spread further and cause abscesses, particularly in the liver. Metronidazole is the recommended drug of choice (Kumanan et al. 2021). However, furazolidone and tetracycline may also be used.

5.2. BALANTIDIASIS

Balantidia coli is a protozoal parasite of humans, non-human primates, rodents, and swine. It primarily causes infection in the large intestine. Transmission occurs through the fecal-oral route, and cysts are ingested through contaminated food and water. Cysts can remain infective in the environment for up to 10 days. One study in captive African great apes found that trophozoites of B. coli can also be infective (Pomajbíková et al. 2010). No vector transmits this parasite (Schuster and Ramirez-Avila 2008). Once ingested, cysts, surviving the stomach's acidic environment, move to the small intestine where they excyst, and trophozoites are released. These motile trophozoites are then passed to the large intestine. Infection can be asymptomatic to severe bloody diarrhea. Watery diarrhea, dehydration, anorexia, and reduced growth are typical clinical findings of Balantidiasis. Metronidazole, furazolidone, and secnidazole can be used against *Balantidia coli.*

5.3. TRICHOMONAS

Two species of *trichomonas* are of zoonotic importance*, Dientamoeba fragilis* and *Pentatrichomonas hominis*. These two species have a broad host range and have been isolated from domestic and farm animals. Little is known about its pathogenesis and transmission. However, it is suggested that the species mentioned above of trichomonas are transmitted by the fecal-oral route and cause mild diarrhea even with a large parasite load. Metronidazole can cure the disease, and there is little chance of an outbreak.

6. HELMINTHIC DISEASES

6.1. STRONGYLOIDIASIS

Strongyloidiasis is a helminthic disease of zoonotic importance, easily transmissible from one host to another. It is caused by a nematode, *Strongyloides stercoralis* that can live as a parasite and a saprophytic organism in the environment. Females produce eggs in the intestine of the host. These eggs hatch, producing filariform larvae in the host's intestine. These filariform larvae cause hyperinfection. Thus, a large load of the parasite builds up in the intestine despite a small number of initial infective exposures. Hyperinfection caused by *Strongyloides stercoralis* has a high mortality rate (15 to 87 percent) (Marcos et al. 2008). Infection begins when the infective form, filariform larvae, penetrates the skin or mucous membrane of the host. The larvae penetrate until they reach a blood vessel and eventually into the lungs. Adults burrow into the small intestine, producing eggs and damaging the intestinal mucosa. Reservoirs for *Strongyloides stercoralis* are people, dogs, cats, foxes, and the environment, especially in hot and humid climates.

Infection is usually asymptomatic, but some people show symptoms like watery and mucoid diarrhea and abdominal cramps (Vadlamudi et al. 2006). Coughing, fever, and creeping eruptions around the anus and buttocks have been documented in some cases. Hyperinfected people present severe, fatal symptoms. These patients can have a hemorrhage, hemoptysis, secondary sepsis, toxic intestinal dilation, and death. Antihelminthic drugs are used to prevent and treat the infection.

6.2. ECHINOCOCCOSIS

Echinococcosis, known as hydatid disease, is a rare but potentially lethal disease in people. The definitive hosts of this disease are members of the dog family (dogs, wolves, jackals, and dingos). The disease is caused by tapeworm *Echinococcus granulosus.*

Once eggs are ingested by an intermediate host (man, cattle, sheep), they hatch in the intestine and penetrate the intestinal mucosa and into the bloodstream. Subsequently, parasites travel throughout the host's body and live in body organs, mainly the liver and lungs, forming hydatid cysts. The cysts enlarge in size and may cause organ dysfunction. If the cysts are not removed, the daughter cysts are included, and the condition worsens. The parasite reaches the definitive host by ingesting meat contaminated with hydatid cysts. The parasite does not cause clinical disease in the definitive host but may be life-threatening in the intermediate host. Praziquantel, bunamidine, and arecoline are used to kill adult parasites in the definitive host. Hydatid cysts in the intermediate host need surgical removal. Care should be taken to avoid rupturing the cyst, as it can be fatal. Non-operable hydatid cysts can be destroyed in humans by mebendazole or praziquantel (El-On 2003).

7. MISCELLANEOUS HELMINTHS

7.1. ECHINOCOCCUS MULTILOCULARIS

Echinococcus multilocularis is similar to *E. granulosus* and is found in northern Asia, Canada, and Alaska. Dogs and foxes act as definitive hosts in the life cycle of this tapeworm. Unlike *E. granulosus*, scoleces of *E. multilocularis* grow uncontrollably, resulting in widespread metastasis, primarily affecting the liver (Hildreth et al. 2000). Hence, surgical removal is generally not feasible. Other considerations apply to *E. multilocularis* as to *E. granulosus.*

7.2. TAENIASIS

Taenia saginata and *Taenia solium* are significant public health considerations. However, human infection principally occurs through ingesting cysticerci-contaminated beef and pork. After ingestion, the cysts undergo evagination and attach themselves to the walls of the small intestine by their scolex. Over about two months, they mature into adult worms (Nyangi et al. 2022).

7.3. COENUROSIS

Another tapeworm, *Multiceps multiceps*, is found in dogs. After ingesting eggs of this tapeworm by humans, it forms cysts (coenuri). These cysts primarily occur in the brain and can cause internal hydrocephalus and posterior fossa syndrome (Haddad et al. 2008).

7.4. DIPYLIDIUM CANINUM

Humans are occasional hosts of this tapeworm (Narasimham et al. 2013). However, it is commonly present in dogs and cats. The flea or dog louse is its intermediate host. It causes few symptoms in dogs and cats, except for anal pruritus, unless an extensive infestation causes obstruction. Occasionally, children may ingest the intermediate host, which results in mild gastroenteritis, eosinophilia, and restlessness caused by the tapeworm. Diagnosis is based on detecting the tapeworm's proglottids in the stool. Praziquantel effectively eradicates adult tapeworms, but the dog can be reinfected unless the fleas and lice are virtually eliminated from the dog's environment.

7.5. CUTANEOUS LARVA MIGRANS

Ancylostoma spp., canine hookworm larvae, can cause a pruritic creeping eruption when they localize in the skin. However, since human beings are not their natural hosts, skin penetration by these larvae causes

this syndrome rather than the intestinal infection as in dogs (Brenner and Patel 2003). After hatching, these larvae are susceptible to drying. Hence, transmission to people must occur relatively shortly after the hatching. In human cases, pruritus is a prominent symptom, and the tract created by the migrating larvae may be seen on the skin.

8. VIRAL DISEASES

8.1. PARVOVIRUS INFECTION

There is an increasing concern about various human illnesses that appear to be associated with parvovirus infection (Anderson et al. 1985; Plummer et al. 1985). These illnesses include pancytopenias and exanthematous conditions in children. However, these diseases and their link to parvovirus are in the early stages of being understood in detail. There is currently no provided evidence that demonstrates their transmission from pets to humans. However, further research is required to confirm or deny the possibility of such transmission.

REFERENCES

Aarestrup F and Engberg J, 2001. Antimicrobial resistance of thermophilic Campylobacter. Veterinary Research 32(3-4): 311-321.

Abou-Bakr et al., 2019. opportunistic parasitic pulmonary infections in human immunodeficiency virus (HIV) infected patients: with references to Egyptian parasites. Journal of the Egyptian Society of Parasitology 49(2): 423–438.

Acheson D and Allos BM, 2001. *Campylobacter jejuni* infections: update on emerging issues and trends. Clinical Infectious Diseases 32(8): 1201-1206.

Acikgoz Y et al., 2012. Cryptosporidiosis: A rare and severe infection in a pediatric renal transplant recipient. Pediatric Transplantation 16(4): E115–E119.

Al-Matha EM and Alsalem AM, 2012. Pomegranate (Punica granatum) peel is effective in a murine model of experimental Cryptosporidium parvum. Experimental Parasitology 131(3): 350–357.

Anderson et al., 1985. Experimental parvoviral infection in humans. Journal of Infectious Diseases 152(2): 257–265.

Angus KW, 1983. Cryptosporidiosis in man, domestic animals and birds: a review. Journal of the Royal Society of Medicine 76(1): 62-70.

Appelbee AJ et al., 2005. Giardia and Cryptosporidium in mammalian wildlife–current status and future needs. Trends in Parasitology 21(8): 370-376.

Arai T et al., 1980. A survey of *Plesiomonas shigelloides* from aquatic environments, domestic animals, pets and humans. Epidemiology and Infection 84(2): 203-211.

Backhans A et al., 2011. Occurrence of pathogenic *Yersinia enterocolitica* and *Yersinia pseudotuberculosis* in small wild rodents. Epidemiology and Infection 139(8): 1230-1238.

Bagcigil AF et al., 2007. Fecal shedding of *Salmonella spp.* in dogs. Journal of Veterinary Medical Science 69(7): 775–777.

Black RE et al., 1988. Experimental *Campylobacter jejuni* infection in humans. Journal of Infectious Diseases 157(3): 472-479.

Bonardi S et al., 2010. Detection, semiquantitative enumeration, and antimicrobial susceptibility of *Yersinia enterocolitica* in pork and chicken meats in Italy. Journal of Food Protection 73(10): 1785-1792.

Brandborg LL et al., 1980. Giardiasis and traveler's diarrhea. Gastroenterology 78(6): 1602–1614.

Brenner MA and Patel MB, 2003. Cutaneous larva migrans: The creeping eruption. CUTIS-NEW YORK 72(2): 111–123.

Bucher M et al., 2008. Epidemiological data on pathogenic Yersinia enterocolitica in Southern Germany during 2000–2006. Foodborne Pathogens and Disease 5(3): 273–280.

Bulbuloglu S et al., 2022. The effect of long-term immunosuppressive therapy on gastrointestinal symptoms after kidney transplantation. Transplant Immunology 70: 101515.

Chen P et al., 2012. Epidemiology, disease spectrum and economic burden of non-typhoidal Salmonella infections in Taiwan, 2006–2008. Epidemiology and Infection 140(12): 2256-2263.

Christopher PR et al., 2010. Antibiotic therapy for Shigella dysentery. Cochrane Database of Systematic Reviews 8.

Current W, 1985. Cryptosporidiosis. Journal of the American Veterinary Medical Association 187(12): 1334-1338.

Davis L and DiRita V, 2008. Growth and Laboratory Maintenance of *Campylobacter jejuni*: Epsilon Proteobacteria. Current Protocols in Microbiology 10(1): 8A.1.1-8A. 1.7.

Deacon A et al., 2003. Septicemia due to *Yersinia pseudotuberculosis*—a case report. Clinical microbiology and Infection 9(11): 1118-1119.

Debruyne L et al., 2008. Taxonomy of the family Campylobacteraceae. Campylobacter 2008: 1-25.

Del Coco VF et al., 2012. Experimental infection with Cryptosporidium parvum IIaA21G1R1 subtype in immunosuppressed mice. Veterinary Parasitology 190(3–4): 411–417.

Duan R et al., 2014. Homology analysis of pathogenic Yersinia species *Yersinia enterocolitica, Yersinia pseudotuberculosis*, and *Yersinia pestis* based on multilocus sequence typing. Journal of Clinical Microbiology 52(1): 20–29.

Eikmeier D et al., 2018. Incubation period for outbreak-associated, non-typhoidal salmonellosis cases, Minnesota, 2000–2015. Epidemiology and Infection 146(4): 423–429.

Einarsson E et al., 2016. An up-date on Giardia and giardiasis. Current Opinion in Microbiology 34: 47–52.

El-On J, 2003. Benzimidazole treatment of cystic echinococcosis. Acta Tropica 85(2): 243–252.

Epps SV et al., 2013. Foodborne Campylobacter: infections, metabolism, pathogenesis and reservoirs. International Journal of Environmental Research and Public Health 10(12): 6292-6304.

Fàbrega A and Vila J, 2012. *Yersinia enterocolitica*: Pathogenesis, virulence and antimicrobial resistance. Enfermedades Infecciosas y Microbiologia Clinica 30(1): 24–32.

Fernandes AM et al., 2015. Partial failure of milk pasteurization as a risk for the transmission of Campylobacter from cattle to humans. Clinical Infectious Diseases 61(6): 903-909.

Fredriksson-Ahomaa M et al., 2006. Sporadic human Yersinia enterocolitica infections caused by bioserotype 4/O: 3 originate mainly from pigs. Journal of Medical Microbiology 55(6): 747-749.

Galindo CL et al., 2011. Pathogenesis of *Y. enterocolitica* and *Y. pseudotuberculosis* in Human Yersiniosis. Journal of Pathogens 2011.

Garénaux A et al., 2008. Survival of *Campylobacter jejuni* strains from different origins under oxidative stress conditions: effect of temperature. Current Microbiology 56: 293-297.

Glaser CA et al., 1998. Association Between Cryptosporidium Infection and Animal Exposure in HIV-Infected Individuals. Journal of Acquired Immune Deficiency Syndromes 17(1): 79-82.

Goh S et al., 2004. Sporadic cryptosporidiosis, North Cumbria, England, 1996–2000. Emerging Infectious Diseases 10(6): 1007.

Graham SM et al., 2000. Clinical presentation of non-typhoidal Salmonella bacteraemia in Malawian children. Transactions of the Royal Society of Tropical Medicine and Hygiene 94(3): 310-314.

Griffith RW et al., 2019. Salmonellosis. Diseases of Swine 2019: 912-925.

Guinet F et al., 2011. Transfusion-transmitted Yersinia enterocolitica sepsis. Clinical Infectious Diseases 53(6): 583–591.

Haddad M et al., 2008. Imaging of parasitic diseases of the central nervous system. Imaging of Parasitic Diseases: 7–31.

Hellard ME et al., 2000. Prevalence of enteric pathogens among community based asymptomatic individuals. Journal of Gastroenterology and Hepatology 15(3): 290-293.

Hellard M et al., 2003. Risk factors leading to Cryptosporidium infection in men who have sex with men. Sexually Transmitted Infections 79(5): 412–414.

Hildreth MB et al., 2000. Failure to identify alveolar echinococcosis in trappers from South Dakota in spite of high prevalence of *Echinococcus multilocularis* in wild canids. Journal of Parasitology 86(1): 75–77.

Hoffmann S et al., 2007. Using expert elicitation to link foodborne illnesses in the United States to foods. Journal of Food Protection 70(5): 1220–1229.

Horn BJ and Lake RJ, 2013. Incubation period for campylobacteriosis and its importance in the estimation of incidence related to travel. Eurosurveillance 18(40): 20602.

Horrocks S et al., 2009. Incidence and ecology of *Campylobacter jejuni* and *coli* in animals. Anaerobe 15(1-2): 18-25.
Huang JL et al., 2009. Epidemiological surveillance of *Campylobacter jejuni* in chicken, dairy cattle and diarrhoea patients. Epidemiology and Infection 137(8): 1111–1120.
Hunter PR and Nichols G, 2002. Epidemiology and clinical features of Cryptosporidium infection in immunocompromised patients. Clinical Microbiology Reviews 15(1): 145–154.
Hunter PR et al., 2004. Sporadic cryptosporidiosis case-control study with genotyping. Emerging Infectious Diseases 10(7): 1241.
Janda JM, 2002. Aeromonas and Plesiomonas. In: Tang Y, Sussman M, Liu D, Poxton I, Schwartzman J, editors. Molecular medical microbiology: Elsevier; pp: 1237–1270.
Jiang GC et al., 2000. Enrichment procedures and plating media for isolation of Yersinia enterocolitica. Journal of Food Protection 63(11): 1483–1486.
Jiang J et al., 2005. Distribution of Cryptosporidium genotypes in storm event water samples from three watersheds in New York. Applied and Environmental Microbiology 71(8): 4446-4454.
Joseph B et al., 2001. Biofilm formation by Salmonella spp. On food contact surfaces and their sensitivity to sanitizers. International Journal of Food Microbiology 64(3): 367–372.
Karanis P et al., 2006. Waterborne transmission of protozoan parasites: A worldwide review of Outbreaks and lessons learnt. Journal of Water and Health 5(1): 1–38.
Kelly P et al., 2009. Susceptibility to intestinal infection and diarrhoea in Zambian adults in relation to HIV status and CD4 count. BMC Gastroenterology 9(1): 1-11.
Kenyon JJ et al., 2017. Genetics and evolution of Yersinia pseudotuberculosis O-specific polysaccharides: A novel pattern of O-antigen diversity. FEMS Microbiology Reviews 41(2): 200–217.
Khalifa EA et al., 2007. Ocular changes in giardiasis: Human and experimental studies. Tanta Medical Sciences Journal 2: 119–131.
Klein-Jöbstl D et al., 2016. Multilocus sequence typing and antimicrobial resistance of *Campylobacter jejuni* isolated from dairy calves in Austria. Frontiers in Microbiology 7: 72.
Kuipers EJ and Surawicz CM, 2008. *Clostridium difficile* infection. The Lancet 371(9623): 1486–1488.
Kumanan T et al., 2021. Metronidazole for Amoebiasis: A tale of more than half a century.
Kunwar R et al., 2013. Outbreak investigation: Salmonella food poisoning. Medical Journal Armed Forces India 69(4): 388-391.
Lamps LW and Lamps LW, 2010. Intestinal flagellates. Surgical Pathology of the Gastrointestinal System: Bacterial, Fungal, Viral, and Parasitic Infections 2010: 177–182.
Lastovica AJ et al., 2014. The family campylobacteraceae, Springer-Verlag Berlin Heidelberg.
Longenberger AH et al., 2014. *Yersinia enterocolitica* infections associated with improperly pasteurized milk products: Southwest Pennsylvania, March–August, 2011. Epidemiology and Infection 142(8): 1640–1650.
Luangtongkum T et al., 2009. Antibiotic resistance in Campylobacter: emergence, transmission and persistence.
Lynch M et al., 2006. Surveillance for foodborne-disease outbreaks: United States, 1998-2002.
Marcos LA et al., 2008. Strongyloides hyperinfection syndrome: An emerging global infectious disease. Transactions of the Royal Society of Tropical Medicine and Hygiene 102(4): 314–318.
Maykel JA and Steele SR, 2011. Other Benign Colorectal Disorders. The ASCRS Textbook of Colon and Rectal Surgery 2011: 565–596.
McGuirk SM and Peek S, 2003. Salmonellosis in cattle: A review. American Association of Bovine Practitioners 36th Annual Conference.
Moore BC et al., 2003. Survival of Salmonella enterica in freshwater and sediments and transmission by the aquatic midge Chironomus tentans (Chironomidae: Diptera). Applied and Environmental Microbiology 69(8): 4556–4560.
Mühldorfer K et al., 2010. Yersinia species isolated from bats, Germany. Emerging Infectious Diseases 16(3): 578.
Narasimham MV et al., 2013. Dipylidium caninum infection in a child: A rare case report. Indian Journal of Medical Microbiology 31(1): 82–84.
Newman RD et al., 2001. A longitudinal study of *Giardia lamblia* infection in north-east Brazilian children. Tropical Medicine and International Health 6(8): 624–634.
Nichols GL, 2005. Fly transmission of Campylobacter. Emerging Infectious Diseases 11(3): 361.

Nieckarz M et al., 2020. Urease expression in pathogenic *Yersinia enterocolitica* strains of bio-serotypes 2/O: 9 and 1B/O: 8 is differentially regulated by the OmpR regulator. Frontiers in Microbiology 11: 607.

Nyangi C et al., 2022. Knowledge, attitudes and practices related to *Taenia solium* cysticercosis and taeniasis in Tanzania. BMC Infectious Diseases 22(1): 534.

Oloya J et al., 2007. Evaluation of Salmonella occurrence in domestic animals and humans in North Dakota (2000–2005). Foodborne Pathogens and Disease 4(4): 551–563.

On SL, 2001. Taxonomy of Campylobacter, Arcobacter, Helicobacter and related bacteria: current status, future prospects and immediate concerns. Journal of Applied Microbiology 90(6): 1S-15S.

Onwuezobe Ian et al., 2012. Antimicrobials for treating symptomatic non-typhoidal Salmonella infection. Cochrane Database of Systematic Reviews 11.

Ordóñez-Mena JM et al., 2018. Comparative efficacy of drugs for treating giardiasis: A systematic update of the literature and network meta-analysis of randomized clinical trials. Journal of Antimicrobial Chemotherapy 73(3): 596–606.

Pintar KD et al., 2015. A systematic review and meta-analysis of the Campylobacter spp. Prevalence and concentration in household pets and petting zoo animals for use in exposure assessments. PLoS One 10(12): e0144976.

Pitlik SD et al., 1983a. Cryptosporidial cholecystitis. New England Journal of Medicine 308(16).

Pitlik SD et al., 1983b. Human cryptosporidiosis: spectrum of disease: report of six cases and review of the literature. Archives of Internal Medicine 143(12): 2269-2275.

Plummer FA et al., 1985. An erythema infectiosum—like illness caused by human parvovirus infection. New England Journal of Medicine 313(2): 74–79.

Pomajbíková K et al., 2010. Discrepancies in the occurrence of *Balantidium coli* between wild and captive African great apes. Journal of Parasitology 96(6): 1139-1144.

Pumipuntu N and Piratae S, 2018. Cryptosporidiosis: A zoonotic disease concern. Veterinary World 11(5): 681.

Ragimbeau C et al., 2014. Investigating the host specificity of *Campylobacter jejuni* and *Campylobacter coli* by sequencing gyrase subunit A. BMC Microbiology 14: 1-12.

Rahuma N et al., 2005. Carriage by the housefly (Musca domestica) of multiple-antibiotic-resistant bacteria that are potentially pathogenic to humans, in hospital and other urban environments in Misurata, Libya. Annals of Tropical Medicine and Parasitology 99(8): 795–802.

Rautelin H and Hanninen ML, 2000. Campylobacters: the most common bacterial enteropathogens in the Nordic countries. Annals of Medicine 32(7): 440-445.

Robertson B et al., 2002. Case-control studies of sporadic cryptosporidiosis in Melbourne and Adelaide, Australia. Epidemiology and Infection 128(3): 419-431.

Robins-Browne RM and Hartland EL, 2003. International Handbook of Foodborne Pathogens.

Robins-Browne RM, 2012. Yersinia enterocolitica. Food Microbiology: Fundamentals and Frontiers 2012: 339–376.

Roy SL et al., 2004. Risk factors for sporadic cryptosporidiosis among immunocompetent persons in the United States from 1999 to 2001. Journal of Clinical Microbiology 42(7): 2944-2951.

Sambol SP et al., 2002. Colonization for the prevention of Clostridium difficile disease in hamsters. The Journal of Infectious Diseases 186(12): 1781–1789.

Schuster FL and Ramirez-Avila L, 2008. Current world status of *Balantidium coli*. Clinical Microbiology Reviews 21(4): 626-638.

Seepersadsingh N et al., 2004. Prevalence and antimicrobial resistance of *Salmonella spp.* In non-diarrhoeic dogs in Trinidad. Journal of Veterinary Medicine, Series B 51(7): 337–342.

Shane SM et al., 1985. Transmission of *Campylobacter jejuni* by the housefly (Musca domestica). Avian Diseases 1985: 384-391.

Siciliano V et al., 2020. Clinical management of infectious diarrhea. Reviews on Recent Clinical Trials 15(4): 298–308.

Silva J et al., 2011. Campylobacter spp. as a foodborne pathogen: A review. Frontiers in Microbiology 2: 200.

Skirrow MB, 2006. John McFadyean and the Centenary of the First Isolation of Campylobacter Species. Clinical Infectious Diseases 43(9): 1213-1217

Smith PD et al., 1982. Chronic giardiasis: Studies on drug sensitivity, toxin production, and host immune response. Gastroenterology 83(4): 797–803.

Sulakvelidze A, 2000. Yersiniae other than *Y. enterocolitica, Y. pseudotuberculosis*, and *Y. pestis*: The ignored species. Microbes and Infection 2(5): 497–513.

Terech-Majewska E et al., 2016. Characterization of *Yersinia enterocolitica* strains potentially virulent for humans and animals in river water. Journal of Applied Microbiology 121(2): 554–560.

Thompson RA, 2000. Giardiasis as a re-emerging infectious disease and its zoonotic potential. International Journal for Parasitology 30(12-13): 1259-1267.

Thompson RA, 2004. The zoonotic significance and molecular epidemiology of Giardia and giardiasis. Veterinary Parasitology 126(1-2): 15-35.

Thompson R and Monis P, 2004. Variation in Giardia: implications for taxonomy and epidemiology. Adv Parasitol 58(95): 69.

Trülzsch K et al., 2007. Invasion and dissemination of *Yersinia enterocolitica* in the mouse infection model. The Genus Yersinia: From Genomics to Function 2007: 279-285.

Tsui CKM et al., 2018. Beaver fever: Whole-genome characterization of waterborne outbreak and sporadic isolates to study the zoonotic transmission of giardiasis. Msphere 3(2): 10–1128.

Tzipori S, 1983. Cryptosporidiosis in animals and humans. Microbiological Reviews 47(1): 84-96.

Upcroft P and Upcroft JA, 2001. Drug targets and mechanisms of resistance in the anaerobic protozoa. Clinical Microbiology Reviews 14(1): 150–164.

Vadlamudi RS et al., 2006. Intestinal strongyloidiasis and hyperinfection syndrome. Clinical and Molecular Allergy 4(1): 1–13.

van Gerwe T et al., 2009. Quantifying transmission of *Campylobacter jejuni* in commercial broiler flocks. Applied and Environmental Microbiology 75(3): 625–628.

Viegas FM et al., 2020. Fecal shedding of *Salmonella spp*., *Clostridium perfringens*, and *Clostridioides difficile* in dogs fed raw meat-based diets in Brazil and their owners' motivation. PLoS One 15(4): e0231275.

Von Graevenitz A, 2007. The role of Aeromonas in diarrhea: A review. Infection 35(2): 59.

Wagenaar JA et al., 2014. Campylobacter fetus infections in humans: Exposure and disease. Clinical Infectious Diseases 58(11): 1579–1586.

Wang X et al., 2010. Pathogenic strains of *Yersinia enterocolitica* isolated from domestic dogs (Canis familiaris) belonging to farmers are of the same subtype as pathogenic *Y. enterocolitica* strains isolated from humans and may be a source of human infection in Jiangsu Province, China. Journal of Clinical Microbiology 48(5): 1604-1610.

Wei L et al., 2020. Prevalence and drug resistance of Salmonella in dogs and cats in Xuzhou, China. Journal of Veterinary Research 64(2): 263.

Wieczorek K and Osek J, 2013. Antimicrobial resistance mechanisms among Campylobacter. BioMed Research International 2013.

Wright JM et al., 2003. Efficacy of antigiardial drugs. Expert Opinion on Drug Safety 2(6): 529–541.

Yason JAD and Rivera WL, 2007. Genotyping of *Giardia duodenalis* isolates among residents of slum area in Manila, Philippines. Parasitology Research 101: 681-687.