Etiology and Transmission Routes for Cryptosporidiosis

Ghunwa Javed[1,*], Mirvasif Seyidov[2], Jiayi Tian[3], Shahid Hussain Farooqi[4] and Qamar un Nisa[5]

[1]Institute of Biochemistry and Biotechnology, University of Veterinary and Animal Sciences, Lahore, Pakistan
[2]Nakhchivan State University, Faculty of Natural Sciences and Agriculture, Department of Veterinary Medicine, Nakhchivan, Azerbaijan
[3]College of Veterinary Medicine, Nanjing Agricultural University, Nanjing 210095, China
[4]Department of Clinical Sciences, KBCMA College of Veterinary and Animal Sciences, Narowal, Sub-campus UVAS-Lahore, Pakistan
[5]Department of Pathology, University of Veterinary & Animal Sciences, Lahore, Pakistan
*Corresponding author: ghunwa.javed5@gmail.com

Abstract

The gastrointestinal tract cells of vertebrate animals become infected by Cryptosporidium species which exist as protozoan parasites. Immunocompetent humans experience self-healing infections from Cryptosporidium yet the high-risk groups including elderly people and patients with suppressed immune systems and especially those with HIV infection suffer from extreme diarrhea combined with tissue spread. The infectious oocysts found in host faeces become ready to transmit immediately following excretion which supports direct transmission. The ability of Cryptosporidium oocysts to stay dormant in environmental settings and survive typical water treatment procedures allows the specific species *Cryptosporidium parvum* to spread across multiple host populations because these resilient parasites can enter humans through the combination of animal contact and exposure to oocyst-contaminated water and food sources. Cryptosporidiosis shows an intricate epidemiology because the disease spreads through various transmission pathways. Scientific studies on single cryptosporidiosis occurrences together with outbreak investigations have enhanced the identification of infection origins and risk elements for this disease. We will examine the origin of cryptosporidiosis in animals in this chapter together with multiple spread methods alongside prevention approaches.

Keywords: Cryptosporidium, Aetiology, Transmission Routes, Pathogenesis, Species

Cite this Article as: Javed G, Seyidov MS, Tian J, Farooqi SH and Nisa QU, 2025. Etiology and transmission routes for cryptosporidiosis. In: Kun Li (ed), Protozoan Zoonoses: Advances in the Diagnosis, Prevention, and Treatment of Cryptosporidiosis and Toxoplasmosis. Unique Scientific Publishers, Faisalabad, Pakistan, pp: 1-8. https://doi.org/10.47278/book.HH/2025.335

A Publication of Unique Scientific Publishers

Chapter No: 25-01

Received: 20-Feb-2025
Revised: 25-Apr-2025
Accepted: 12-May-2025

Introduction

The genus Cryptosporidium exists within the Apicomplexa phylum as eukaryotic organisms of its own taxonomic group. When Cryptosporidium parasites release their obligatory feces the products of their endogenous developmental cycles inside host cells cause encystment (Current & Garcia, 1991; Chen et al., 2002).

Most phylum species depend on the oocysts for dispersed parasite survival and transmission potential. The oocyst serves multiple critical purposes for both detection and identification methods. Specialists identify species by evaluating biological features of Caryospora, Cyclospora, Eimeria, Isospora, Sarcocystis and Toxoplasma at the oocyst level through examining both oocyst shape and sporocyst composition and sporozoite inner structure (Chalmers & Davies, 2010). The microscopic analysis using high resolution inner-view reveals shape distinctions between oocysts that mostly range from 10 to 40 mm. Successful species identification through morphometrics becomes challenging because oocysts from different species share indistinguishable size arrangements and morphological features. The small oocysts of Cryptosporidium represent a challenging case for morphological identification. Scientists encounter two main obstacles when properly identifying Cryptosporidium spp. in faecal and environmental samples through differentiation of oocysts from other small objects including yeasts, molds, algae and plant matter. Differentiation of Apicomplexa species proves difficult during evaluation of oocyte dimensions alongside shape characteristics and unclear inner morphological features (Kosek et al., 2001; Chalmers & Davies, 2010).

The traditional biological analysis approach struggles with long research times and expensive costs to identify Apicomplexa members as species even though host specificity works well. Their investigations demand large numbers of oocysts from multiple host species together with exacting preservation techniques in accurate timing. This chapter aims to identify Cryptosporidium species with their aetiology, evaluate the hosts, human outbreaks, transmission patterns, prevention and control of the disease.

2. Aetiology and Pathophysiology

Intestinal apicomplexan protozoa (coccidian parasites) trigger human cryptosporidiosis outbreaks. The protozoa which produce cryptosporidiosis fall within Sporozoasida as their class then Coccidiasina as their subclass after which Eucoccidiorida serves as their order and Eimeriarina acts as their suborder before Cryptosporiidae identifies as their family and Cryptosporidium (from Greek –occult spore) stands as their genus. *C. parvum* (referred to as *C. parvum* Type 1) and *Cryptosporidium hominis* (initially named *C. parvum* Type 2) act as leading infectious pathogens causing diseases in humans (Dragomirova, 2022). Recent scientific evidence indicates that human infections exist from

five Cryptosporidium species including *C. felis*, *C. meleagridis*, *C. canis*, *C. muris* and *C. cuniculus*. According to expert's livestock including cattle, functions as main carriers for zoonotic infections. Studies demonstrated Cryptosporidium species along with genotypes can spread between wild and domestic animals even though public health impacts of these infections generally remain negligible. Medical science confirms that a human infection can be triggered by consuming just 101 to 103 oocysts (which comprise a 50% infectious dose of 102 oocysts) (Mosier & Oberst, 2000). The infectivity of Cryptosporidium parasites starts after expulsion from the host body yet ongoing parasite regeneration occurs throughout intestinal tissues. Gut cells host Cryptosporidium parasites yet the pathogens exist both inside and outside of cellular membranes creating a cellular structure which makes these parasites harder to treat. Standard chlorine-based water treatments used in water infrastructure operations fail to remove the high densities of resilient oocysts produced by the parasite (Cacciò & Pozio, 2006).

The key sign people experience when infected with Cryptosporidiosis is diarrhea that mostly appears as water-like stool. The parasite's specific mechanism behind symptom generation includes elevated intestinal permeability with associated chloride flux and impaired absorption resulting in contractive diarrhea. Expert medical insights show that host reactions to infection infections result in these symptoms. Most cases experience that infection solely impacts their small intestinal area. Studies show Cryptosporidium can lead to infections in people whose immune system functions poorly such as those living with HIV (Clark, 1999).

3. Distribution and Prevalence

Medical experts tracked the very first Cryptosporidium human infection cases that occurred across two scenarios between 1976 and 1982 and after that identified eleven subsequent instances spanning a six-year time frame. Scientists have tracked Cryptosporidium infections existing across 90 countries throughout six continents (Ungar, 2018). The latest understandings of Cryptosporidium infection come primarily from the reporting of isolated cases and outbreak investigations. Research into Cryptosporidium parasites exists throughout scientific literature emanating from medical periodicals through executed surveys and individual case reports. Students from developed nations most commonly provide samples for testing after developing gastrointestinal illness yet outbreak samples remain hard to obtain. Medical facilities report positive Cryptosporidium findings among 2% of patients whose stool tests physicians examine. Each year there are 15 million health visits directly related to diarrhea. Stool testing reveals Cryptosporidium every year in 300000 Americans. Statistical analytics from Food Net surveillance show 4500 annual reports while laboratory Commissioned reports demonstrate 102000 annual cases accounting for 45 times higher incidence (Mead et al., 1999).

CDC surveillance data recorded only a single cryptosporidiosis outbreak during five years. The authors state that their research data needs to be interpreted with precision. The data needs cautious interpretation because its reporting scope remains limited. Official reports of cases fall far below the actual total number of infected persons (Beach, 2007). Several developing nations found elevated infection rates via their survey responses. Latest research shows developing countries experience more infections than industrialized countries do. Rates of incidence between industrialized nations show the greatest impact as the dominant contributor. Populated regions include multiple specific groups who face improved chances of infections (Caccio & Putignani, 2013). Our survey showed elevated infection rates especially among children who are malnourished combined with immunocompromised patients and diverse population types. Patients diagnosed with AIDS along with transplant recipients and chemotherapy patients and institutionalized patients form the high-risk infection group (Bean et al., 1990; Wang et al., 2018).

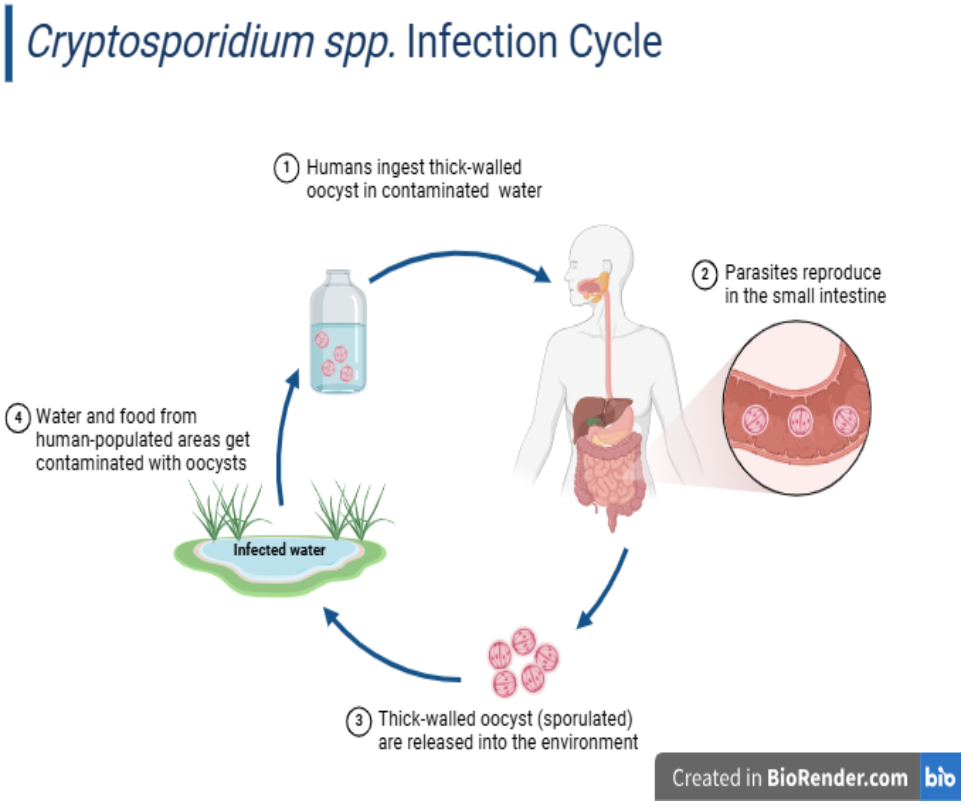

Fig. 1: Cryptosporidium Infection Cycle in Humans

4. Pathogenesis

Scientific knowledge about Cryptosporidium development needs further examination. Both rotaviruses and enteric viruses and intestinal protozoan diseases like amebiasis and giardiasis exhibit equivalent disease progression patterns. Severe tissue conditions occur within the gut while Cryptosporidium parasites persist in lungs and digestive tract and conjunctiva (Clark & Sears, 1996). Medical tests reveal the existence of enlarged mesenteric lymph nodes as one of the disease's diagnostic features. Intestinal parasite damage results in two distinct tissue abnormalities which cause villi atrophy and epithelial tissue irregularities while damaging both cryptal tissue and lamina propria layers. Numerous neutrophil cells reside in this tissue alongside macrophages and eosinophils and lymphocytes (Warren & Guerrant, 2007). A patient's bodies experience excessive diarrhea from enzyme shortages paired with lactose intolerance that results in malabsorption syndrome which triggers protein metabolic problems. A severe disturbance simultaneously affects water balance and electrolyte homeostasis and protein metabolic function. Infections harm the urogenital tract whereas other infections affect the urogenital system (Cacciò & Widmer, 2013). The medical research demonstrates that *C. parvum* and *C. hominis* continue releasing oocysts from the time diarrhea ends until it comes back. *C. muris* infecting immune-competent subjects maintains oocyst production capability for seven months following its entry into the body. Genome sequencing of *C. parvum* along with *C. hominis* revealed more than twenty-five potential virulence elements according to multiple molecular testing methods. Human immune systems regulate merozoite type 1 reproduction rates and oocyst wall thickness as shown in figure 1. Individuals with functioning immune systems who survived a previous cryptosporidium infection gain some protection against reinfection but also reduce disease complications by reducing oocyst counts after infection (Cacciò & Widmer, 2013; Gonzalez-Astudillo et al., 2021).

5. Transmission

During oocyst transmission from infected to unexposed hosts the route of transmission remains fecal-oral. Sustained transmission of *C. parvum* occurs via two exchange pathways: direct or indirect person-to-person interactions with sexual activities and through animal contact, exposures or consumption of contaminated food and water and airborne transmission routes (DuPont et al., 1995). An analysis of 29 healthy volunteers showed the minimum number of calf-stage oocysts needed to establish *C. parvum* infection from a single ingestion event. Research results confirms that one of five volunteers developed an infection after ingesting thirty oocysts. Eight out of seven participants developed *C. parvum* infection after consuming 1000 oocysts and more (Okhuysen et al., 1999). The study team established 132 oocysts as the median number required to cause infection. ID50 analysis indicated 87 oocysts as the minimum infective dose although the scientists noted inconsistent ID50 measurements across distinct *C. parvum* isolates (Meinhardt et al., 1996). Human subjects required between 9 to 1042 oocysts from the TAMU and UCP strains before the infection could take hold, according to the research team (Fayer et al., 1998)

5.1. The Impact of Desiccation and Temperature on Oocyst Survival

Studies confirm that *C. parvum* oocysts can thrive throughout multiple months. Exposure of oocysts to 208C temperature over six-month period did not degrade their capacity to infect suckling mice. During six months at storage temperature oocysts maintained their potential to transmit the parasite to suckling mice (Fayer et al., 1998). Oocyst survival rate diminishes more rapidly when maintained at superior temperature levels. When assayed at 25 or 308C the oocysts exhibited continued infectivity across 3 months of testing. endive warming *C. parvum* oocysts from 9 to 558C over twenty minutes prevented parasite transmission to suckling mice. Research revealed that *C. parvum* oocysts endured limited survival and capacity to infect after both five minutes of exposure to 59.78C and five seconds of exposure to 71.78C heat (Anderson, 1985). Cold treatment at 2708C immediately destroyed Cryptosporidium oocysts although they demonstrated tenacious survival at 2208C for eight hours without surviving beyond this duration (Peng et al., 2008). Storing oocysts at 2108C maintained their infectivity to mice during an entire week's time yet samples kept at 258C remained infectious for two months. Hereditary fluid material inside the oocysts does not confer sufficient protection against freezing damage for sporozoites. Two hours of desiccation exposure diminished oocyst viability to 3% followed by complete mortality in all oocysts after four hours under similar desiccation conditions (Walker et al., 2001; King & Monis, 2007).

5.2. Mechanisms of Transmission

When faecal waste touches the ground oocysts can get infected through wind and water-based soil transportation paths. Many elements including people or animals alongside environmental factors help move oocysts. A subject's infection begins after ingesting oocysts (Certad et al., 2017).

5.2.1. Mechanical Movement through and Over Soil

Combined complete fecal pollution of water and soil structures simultaneously leads to the pollution of drinking water sources as well as fresh agricultural products and recreational aquatic environments as shown in figure 2. Research on soil oocyst detection remains important yet scientists need to investigate the path that oocysts take through terrestrial surfaces onto water supplies (Anderson, 1986; Boyer et al., 2009). The scientific examination of oocyst migration through soils towards groundwater storage systems remains insufficiently studied. Through a greenhouse soil tilting platform researchers identified alterations occurring in period-to-period movement between observation intervals. Researchers conducted the experiment by spraying livestock faecal oocysts on soil samples for testing. The investigation showed Cryptosporidium oocysts could travel through soil surfaces for 70 days producing their maximum population (Dumètre et al., 2012). Lab findings discovered that *C. parvum* oocysts occupied mostly the soil surface at depths less than 2 cm and their numbers decreased more deeply downward (Anguish & Ghiorse, 1997). The moisture content did not exceed thirty centimeters at which point scientists discovered the presence of oocysts in a small sample set prior to the disappearance of all detected oocysts in seventy-centimeter-deep samples. The British investigation team could not confirm whether gulls carried *C. parvum* oocysts or avian species oocysts because the origin remained unknown yet they suggested gulls could spread oocysts over large distances (Kaucner, 2007). Canada geese (Branta canadensis) together with Peking ducks (Anas platyrhynchos) ingested *C. parvum* oocysts which completed gastrointestinal transit and appeared in fecal droppings before becoming infectious

to mice. The research team discovered living *C. parvum* oocysts in the excretions of Canadian geese during their stopovers in fields as they migrated (Ramirez et al., 2009).

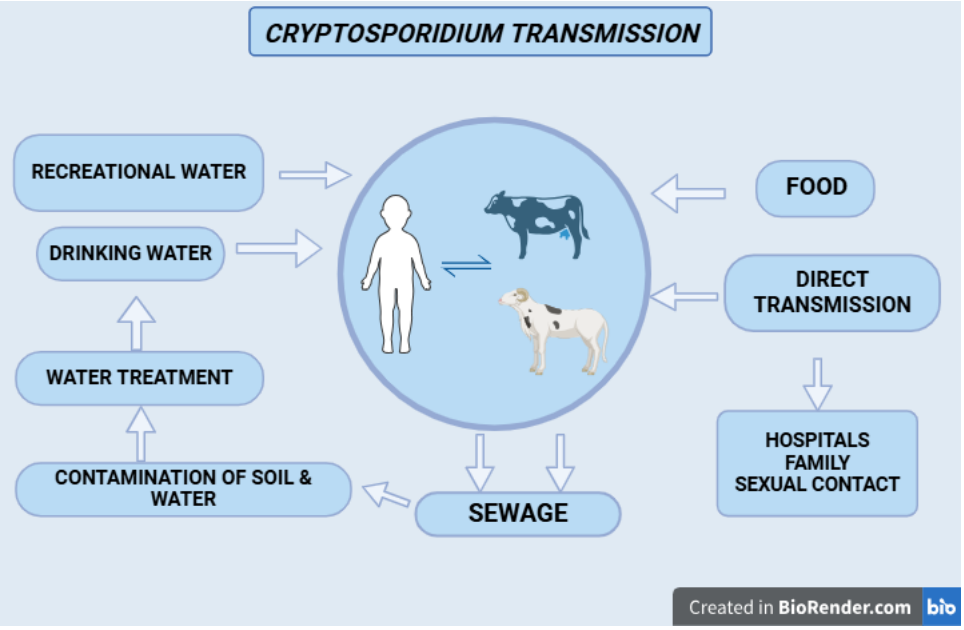

Fig. 2: This depicts the Transmission Routes for Cryptosporidium.

The examination under a microscope found that Periplaneta americana cockroach intestines contained possible *C. parvum* oocysts. Separate study found *C. parvum* oocysts in roaches discovered within a household after the child developed cryptosporidiosis thereby identifying roaches as potential disease carriers (King & Monis, 2007). Observational laboratory experiments confirmed that oocysts from *C. parvum* can persist after house files encounter bovine faeces containing *C. parvum* oocysts. Prior to encapsulation researchers observed that a calf living in the barn developed cryptosporidiosis leaving them to study the barn's wild flies and house flies in captivity. Both *C. parvum* oocysts were detected inside and on the surface of the examined flies. Study showed that dung beetles consumed most *C. parvum* oocysts but their ingested oocysts retained normal shape when they passed through beetle excrement. Oocysts were found to accumulate in regions of beetle anatomy that remain outside the body framework thus suggesting these oocysts could be dispersed across habitats. In lakes ponds puddles mosses and damp soil environments Rotifers distribute across the world within six unique genera among all microscopic invertebrates. Experimental research has shown that six rotifer species are capable of consuming *C. parvum* oocysts while feeding across mosses and soil that gets waterlogged and standing water surfaces. There was no evidence provided showing if the subjected oocysts survived digestive processing or died (Headd & Bradford, 2016).

5.2.3. Transmission via Drinking Water

Widespread fecal contamination is indicated by positive results of oocysts in untreated wastewater, filtered secondary treated wastewater, activated sludge effluent, combined sewer overflows, groundwater, surface water, and treated drinking water. Strong circumstantial evidence that tainted water is a significant risk factor for cryptosporidiosis is provided by several reports from throughout the world (Goldstein et al., 1996). Numerous studies have documented surface source water contamination in North America. Studies that reexamined the same locations after a 4-year break and discovered that 89 and 45%, respectively, of all samples tested positive for *C. parvum* oocysts are representative of these. Oocysts were detected in finished water 3.8±33.3% of the time at drinking water treatment facilities that used conventional filtration, with quantities ranging from 0.1 to 48 oocysts per 100. These amounts reflect the daily exposure of people in the USA who use filter-purified tap water (Meinhardt et al., 1996). In 1988, approximately 155 million people in the USA drank surface water from 6000 community water systems, of which 23% supplied 21 million people with unfiltered water. Disinfection was the only method of protecting against pathogenic pathogens. Households supplied by systems that provide unfiltered water may have increased exposure levels unless source water is safeguarded (Smith & Rose, 1990). The viability, species, and origin of the oocysts in tap water are aspects of Cryptosporidium that are not well described. The public health significance of oocysts detected in water is uncertain since species identification is not a normal process. While it might be challenging to record individual water-borne diseases, epidemics of cryptosporidiosis linked to drinking water Clearly confirm that live *C. parvum* oocysts infiltrate and make their way through the purification procedures of drinking water as shown in figure 2. Source water and finished water may also include nonviable oocysts of *C. parvum* and other species (Carmena, 2010).

Although they have been frequently linked to water-borne outbreaks outside of the US, cattle (and sheep) have not been definitively identified (by genotyping) as the cause of any water-borne epidemics within the US. The only water-borne epidemic in North America where bovine genotype oocysts have been identified is the one in Cranbrook, British Columbia. The majority of epidemiologic studies have identified a number of contributing factors, such as excessive turbidity, polluted source water, and treatment plant malfunctions (Zahedi & Ryan, 2020).

5.2.4. Food-borne Transmission

Food-related epidemics are seldom recorded, are hard to record, and are significantly underreported. Small-group epidemics and individuals are less likely to be identified (Duffy & Moriarty, 2003). Cryptosporidium oocysts were discovered in estuary waters in the Chesapeake Bay and in seawater close to a sewage outfall site in Honolulu, Hawaii (Fayer et al., 1999). Molluscan shells are effective biological markers of water-borne diseases because they filter huge amounts of water and remove minute particles that stay on their gills. Oocysts of *C. parvum* have been found in mussels from the Irish coast, oysters from Galicia, Spain, and clams, mussels, and oysters that were taken from the Chesapeake Bay (Graczyk et al., 1999). Several outbreaks of bacterial and viral illnesses linked to eating raw shellfish should serve as a reminder that boiling shellfish lowers the risk of contracting illnesses from all of these pathogens, even if none of these findings were linked to cryptosporidiosis outbreaks (Ryan et al., 2018). Raw veggies from the market have been found to have oocysts on their surface. Vegetables that are cool and damp offer the best conditions for survival. Oocysts were discovered on the leaves and roots of cilantro, lettuce, radishes, tomatoes, cucumbers, and carrots in Costa Rica, but not on cabbage (Bier, 1991; Rinder, 2004).

Milk from a small, local producer in the UK that used an on-farm pasteurizer was linked to a cryptosporidiosis epidemic that affected fifty schoolchildren. After receiving a complaint about dirt in the milk, environmental health professionals discovered that the pasteurizer was not operating as intended throughout the epidemic (Gelletlie et al., 1997). Drinking freshly squeezed apple juice (non-alcoholic cider) was linked to outbreaks. 160 people in Maine, USA, contracted cryptosporidiosis after drinking cider made from apples grown close to a cow pasture. The guests' oocysts had genetic traits that suggested a bovine origin. It's possible that feces-contaminated well water in New York was used to wash apples for cider. Cider was not pasteurized in each of the incidents (Smith et al., 2007).

An epidemic among 50 attendees of a social gathering in Minnesota was linked to chicken salad. After changing a baby's diaper at her home daycare center, the caterer made chicken salad for the gathering. 54 out of 62 guests at a catered luncheon in Spokane, Washington, fell ill 3±9 days later (Besser-Wiek et al., 1996). There were seven uncooked produce products among the 18 food and drink items at the buffet. Uncooked green onions were present in the food consumed by 51 afflicted individuals. Two out of fourteen food preparers tested positive for Cryptosporidium between three and four weeks after the dinner, and one was exhibiting symptoms at the time of the event. Similarly, at a university in Washington, DC, four cafeteria staff members and 88 students were found to have cryptosporidiosis (CDC, 1998). Three days before to the implicated lunch, a prep cook who chopped up fruit and vegetables for raw consumption became unwell for ten days and may have contracted infection from a child who has a family history of diarrhea. DNA sequencing and restriction fragment length polymorphism (RFLP) examination of PCR results revealed that all positive specimens were identical, human genotypes, and connected to the food handler (Siwila, 2023).

These epidemics draw attention to significant problems. Before handling food products and utensils, food handlers should wash their hands properly. It is not advisable to touch raw fruits and vegetables or already cooked foods with your bare hands. Produce that hasn't been cooked should be carefully cleaned before being put on kitchen surfaces. Surfaces used for food preparation should be cleaned in between tasks. Food workers who are unwell with their gastrointestinal tract should not work.

5.2.5. Transmission via Recreational Water

Around the world, swimming is a fairly common recreational activity. In the United States alone, there are more than 350 million person-events every year. During 10,000 persons have been impacted by documented epidemics of cryptosporidiosis linked to recreational waterways during the last 12 years. Oocyst resistance to chlorine-borne transmission combined with frequent fecal contamination. Public health officials, pool operators, and consumers must work together to devise strategies to lower the risk of water-borne transmission as cryptosporidiosis is recognized as a major source of recreational water-borne illness. Engineering modifications include better filtration and turnover rates, as well as the separation of plumbing and filtration for high-risk "kiddie" pools, should be part of the plans (Carpenter et al., 1999). Pool rules should provide specific procedures for responding to fecal mishaps, evaluate the efficacy of barrier clothing such as swim diapers, and instruct both employees and customers. Education should emphasize the spread of water-borne diseases and recommend easy preventative strategies like avoiding water-related recreational activities while experiencing or having just finished a diarrheal episode, avoiding ingesting recreational water, practicing good hand washing and diaper changing techniques, encouraging showers to get rid of feces before using a pool, and frequent toilet breaks for young children (Beach, 2007; Rochelle & Di Giovanni, 2013).

5.2.6. Sexual Transmission

Though they were unable to confirm it, a number of stories strongly suggested that cryptosporidiosis may be contracted through sexual contact. Cryptosporidiosis was more common in males, according to data analyzing HIV/AIDS patients, homosexual men, and intravenous drug users. It was unable to completely rule out the likelihood of transmission linked to other behaviors, though (Griffiths, 1998; Chou & Fan, 2024).

5.2.7. Airborne Transmission

There have been several instances of youngsters and immunocompromised individuals with cryptosporidiosis experiencing an elevated level of cough or other respiratory symptoms (Rinder, 2004). Respiratory cryptosporidiosis is infrequently recorded, despite the fact that deadly cases have been documented in patients with AIDS, malignant lymphoma, and bone marrow transplantation (Højlyng et al., 1987). An overview of the anatomical geographic distribution of Cryptosporidium in naturally infected birds indicates that respiratory infections with avian Cryptosporidium species appear to occur more frequently in chickens, turkeys, quail, ducks, pheasants, peafowl, and budgerigars than in mammals (Lindsay & Blagburn, 2018; Ahmed et al., 2024).

5.3. Prevention and Control

All control strategies must target both therapy aspects as well as the reduction of oocysts in the environment because oocysts start the infection through ingestion. The strategy focuses on decreasing the presence of oocysts in environmental areas. Successful treatment methods

should decrease both the clinical manifestations of the disease and oocyst shedding. The effort to eliminate the pathogen from animal facilities stands as a difficult goal that usually leads to failure (Abubakar et al., 2007). The contamination management of those who have already suffered exposure pairs effectively with the medical treatments for patients and infected individuals. The infection load in the environment can be reduced through clean and disinfection procedures which represent essential hygienic measures (Shahiduzzaman & Daugschies, 2012). Cleaning stables thoroughly and swiftly disposing of contaminated waste while applying correct disposal methods successfully eliminates oocysts thus minimizing the chance of infection. A designated cleaning toolkit for each enclosure reduces the spread of pathogens which includes Cryptosporidium when proper sanitary measures are followed. Proper management methods decrease both the quantity of Cryptosporidium oocysts present in slurry together with water from calf houses which helps prevent surface water pollution and zoonotic infections (Collinet-Adler et al., 2010).

Conclusion

The parasitic organism Cryptosporidium spp. exists throughout the planet with its highest prevalence observed in underdeveloped countries and developing regions. The coccidial parasite leads to the generation of hardy oocysts following a multifaceted life-cycle that ends with faecal oocyst excretion. The microorganism *C. parvum* spreads throughout multiple host species that includes human beings and their domesticated livestock. Water surface acquisition of Cryptosporidium's oocyst occurs through fecal dissemination from humans and animals including wildlife and domestic animals and livestock. Hosts acquire Cryptosporidium's oocyst primarily through fecal-oral transmission from contaminated food and water or by directly and indirectly coming into contact with animal feces. Foodborne outbreaks of Cryptosporidium occur primarily because the infection spreads between animals and humans. Human and animal cryptosporidiosis prevention requires both monitoring systems and the One Health approach while knowing better how transmission works alongside stopping the disease life cycle. The prevention and reduction of future worldwide outbreaks depend on sanitary water management as well as food protection standards. The current diagnostic options present constraints that affect their ability to perform suitable isolation and detect dual infections with other pathogens while considering expenses. The limited diagnostic capabilities in developing nations leads to insufficient data reporting about cryptosporidiosis which results in substandard medical and public health practices for treating this disease. The detection and reporting dependence on current diagnostic tools is inadequate so it becomes essential to establish new fast and economical diagnostic techniques.

References

Abubakar, I. I., Aliyu, S. H., Arumugam, C., Hunter, P. R., & Usman, N. (2007). Prevention and treatment of cryptosporidiosis in immunocompromised patients. *Cochrane Database of Systematic Reviews, 1*(1), CD004932.

Ahmed, S. A. A., Quattrocchi, A., & Karanis, P. (2024). Cryptosporidium sp. infection in solid organ transplant recipients: A systematic review and meta-analysis. *Pathogens Global Health, 118*(4), 305-316.

Anderson, B. (1986). Effect of drying on the infectivity of cryptosporidia-laden calf feces for 3-to 7-day-old mice. *American Journal of Veterinary Research, 47*(10), 2272-2273.

Anderson, B. C. (1985). Moist heat inactivation of Cryptosporidium spp. *American Journal of Public Health, 75*(12), 1433-1434.

Anguish, L. J., & Ghiorse, W. C. (1997). Computer-assisted laser scanning and video microscopy for analysis of Cryptosporidium parvum oocysts in soil, sediment, and feces. *Applied and Environmental Microbiology*, *63*(2), 724-733.

Beach, M. J. (2007). Waterborne: recreational water. In R. Fayer, & L. Xiao (Eds.), *Cryptosporidium and Cryptosporidiosis, 2* (pp. 335-370). CRC Press.

Bean, N. H., Griffin, P. M., Goulding, J. S., & Ivey, C. B. (1990). Foodborne disease outbreaks, 5-year summary, 1983–1987. *Journal of Food Protection, 53*(8), 711-728.

Besser-Wiek, J., Forfang, J., Hedberg, C., Korlath, J., Osterholm, M., Sterling, C., & Garcia, L. (1996). Foodborne outbreak of diarrheal illness associated with Cryptosporidium parvum-Minnesota, 1995. *Journal of the American Medical Association, 276*(15), 1214.

Bier, J. W. (1991). Isolation of parasites on fruits and vegetables. *The Southeast Asian Journal of Tropical Medicine Public Health, 22*, 144-145.

Boyer, D. G., Kuczynska, E., & Fayer, R. (2009). Transport, fate, and infectivity of Cryptosporidium parvum oocysts released from manure and leached through macroporous soil. *Environmental Geology, 58*, 1011-1019.

Cacciò, S. M., & Pozio, E. (2006). Advances in the epidemiology, diagnosis and treatment of cryptosporidiosis. *Expert Review of Anti-Infective Therapy, 4*(3), 429-443.

Cacciò, S.M., & Putignani, L. (2014). Epidemiology of Human Cryptosporidiosis. In S. M. Cacciò, & G. Widmer (Eds.), *Cryptosporidium: Parasite and Disease, 1* (pp. 43-79). Springer, Vienna.

Cacciò, S. M., & Widmer, G. (2013). *Cryptosporidium: parasite and disease*. Springer Science & Business Media.

Carmena, D. (2010). Waterborne transmission of Cryptosporidium and Giardia: detection, surveillance and implications for public health. *Current Research, Technology Education Topics in Applied Microbiology Microbial Biotechnology, 20*, 3-4.

Carpenter, C., Fayer, R., Trout, J., & Beach, M. J. (1999). Chlorine disinfection of recreational water for Cryptosporidium parvum. *Emerging Infectious Diseases, 5*(4), 579.

Certad, G., Viscogliosi, E., Chabé, M., & Cacciò, S. M. (2017). Pathogenic mechanisms of Cryptosporidium and Giardia. *Trends in Parasitology, 33*(7), 561-576.

Chalmers, R. M., & Davies, A. P. (2010). Minireview: clinical cryptosporidiosis. *Experimental Parasitology, 124*(1), 138-146.

Chen, X.-M., Keithly, J. S., Paya, C. V., & LaRusso, N. F. (2002). Cryptosporidiosis. *New England Journal of Medicine, 346*(22), 1723-1731.

Chou, Y. H., & Fan, H. J. (2024). Cryptosporidium-induced acute kidney injury in a patient with acquired immunodeficiency syndrome. *The American Journal of the Medical Sciences, 368*(3), 253-257.

Clark, D. P. (1999). New insights into human cryptosporidiosis. *Clinical Microbiology Reviews, 12*(4), 554-563.

Clark, D. P., & Sears, C. L. (1996). The pathogenesis of cryptosporidiosis. *Parasitology Today, 12*(6), 221-225.

Collinet-Adler, S., & Ward, H. D. (2010). Cryptosporidiosis: environmental, therapeutic, and preventive challenges. *European Journal of Clinical Microbiology & Infectious Diseases, 29*, 927-935.

Current, W. L., & Garcia, L. S. (1991). Cryptosporidiosis. *Clinical Microbiology Reviews, 4*(3), 325-358.

Dragomirova, P. V. (2022). Cryptosporidiosis: History, Etiology, Biology, Pathogenesis and Pathoanatomy-A Review. *Journal of Biomedical Clinical Research, 15*(1), 22-29.

Duffy, G., & Moriarty, E. M. (2003). Cryptosporidium and its potential as a food-borne pathogen. *Animal Health Research Reviews, 4*(2), 95-107.

Dumètre, A., Aubert, D., Puech, P.-H., Hohweyer, J., Azas, N., & Villena, I. (2012). Interaction forces drive the environmental transmission of pathogenic protozoa. *Applied Environmental Microbiology, 78*(4), 905-912.

DuPont, H. L., Chappell, C. L., Sterling, C. R., Okhuysen, P. C., Rose, J. B., & Jakubowski, W. (1995). The infectivity of Cryptosporidium parvum in healthy volunteers. *New England Journal of Medicine, 332*(13), 855-859.

Fayer, R., Lewis, E. J., Trout, J. M., Graczyk, T. K., Jenkins, M. C., Higgins, J., & Lal, A. A. (1999). Cryptosporidium parvum in oysters from commercial harvesting sites in the Chesapeake Bay. *Emerging Infectious Diseases, 5*(5), 706.

Fayer, R., Trout, J., & Jenkins, M. (1998). Infectivity of Cryptosporidium parvum oocysts stored in water at environmental temperatures. *The Journal of Parasitology,* 1165-1169.

Centers for Disease Control and Prevention (CDC) (1998). Foodborne outbreak of cryptosporidiosis--Spokane, Washington, 1997. *Morbidity and Mortality Weekly Report, 47*(27), 565-567.

Gelletlie, R., Stuart, J., Soltanpoor, N., Armstrong, R., & Nichols, G. (1997). Cryptosporidiosis associated with school milk. *The Lancet, 350*(9083), 1005-1006.

Goldstein, S. T., Juranek, D. D., Ravenholt, O., Hightower, A. W., Martin, D. G., Mesnik, J. L., & Herwaldt, B. L. (1996). Cryptosporidiosis: an outbreak associated with drinking water despite state-of-the-art water treatment. *Annals of Internal Medicine, 124*(5), 459-468.

Gonzalez-Astudillo, V., Sheley, M. F., Uzal, F. A., & Navarro, M. A. (2021). Pathology of cryptosporidiosis in raccoons: case series and retrospective analysis, 1990–2019. *Journal of Veterinary Diagnostic Investigation, 33*(4), 721-727.

Graczyk, T., Fayer, R., Lewis, E., Trout, J., & Farley, C. (1999). Cryptosporidium oocysts in Bent mussels (Ischadium recurvum) in the Chesapeake Bay. *Parasitology Research, 85*, 518-521.

Griffiths, J. K. (1998). Human cryptosporidiosis: epidemiology, transmission, clinical disease, treatment, and diagnosis. *Advances in Parasitology, 40*, 37-85.

Headd, B., & Bradford, S. A. (2016). Use of aerobic spores as a surrogate for Cryptosporidium oocysts in drinking water supplies. *Water Research, 90*, 185-202.

Højlyng, N., Holten-Andersen, W., & Jepsen, S. (1987). Cryptosporidiosis: a case of airborne transmission. *Lancet, 2*(8553), 271-272.

Kaucner, C. E. (2007). *Factors influencing overland mobility of Cryptosporidium Oocysts* (Masters Thesis, University of New South Wales Sydney).

King, B., & Monis, P. (2007). Critical processes affecting Cryptosporidium oocyst survival in the environment. *Parasitology, 134*(3), 309-323.

Kosek, M., Alcantara, C., Lima, A. A., & Guerrant, R. L. (2001). Cryptosporidiosis: an update. *The Lancet Infectious Diseases, 1*(4), 262-269.

Lindsay, D. S., & Blagburn, B. L. (2018). Cryptosporidiosis in birds. In J. P. Dubey, C. A. Speer, & R. Fayer (Eds.), *Cryptosporidiosis of Man and Animals, 1* (pp. 133-148). CRC press.

Mead, P. S., Slutsker, L., Dietz, V., McCaig, L. F., Bresee, J. S., Shapiro, C., & Tauxe, R. V. (1999). Food-related illness and death in the United States. *Emerging Infectious Diseases, 5*(5), 607.

Meinhardt, P. L., Casemore, D. P., & Miller, K. B. (1996). Epidemiologic aspects of human cryptosporidiosis and the role of waterborne transmission. *Epidemiologic Reviews, 18*(2), 118-136.

Mosier, D. A., & Oberst, R. D. (2000). Cryptosporidiosis: a global challenge. *Annals of the New York Academy of Sciences, 916*(1), 102-111.

Okhuysen, P. C., Chappell, C. L., Crabb, J. H., Sterling, C. R., & DuPont, H. L. (1999). Virulence of three distinct Cryptosporidium parvum isolates for healthy adults. *The Journal of Infectious Diseases, 180*(4), 1275-1281.

Peng, X., Murphy, T., & Holden, N. (2008). Evaluation of the effect of temperature on the die-off rate for Cryptosporidium parvum oocysts in water, soils, and feces. *Applied Environmental Microbiology, 74*(23), 7101-7107.

Ramirez, N. E., Wang, P., Lejeune, J., Shipitalo, M. J., Ward, L. A., Sreevatsan, S., & Dick, W. A. (2009). Effect of tillage and rainfall on transport of manure-applied Cryptosporidium parvum oocysts through soil. *Journal of Environmental Quality, 38*(6), 2394-2401.

Rinder, H. (2004). Transmission of microsporidia to humans: Water-borne, food-borne, air-borne, zoonotic, or anthroponotic. *The Southeast Asian Journal of Tropical Medicine and Public Health, 35*, 54-57.

Rochelle, P.A., Di Giovanni, G.D. (2014). Cryptosporidium Oocysts in Drinking Water and Recreational Water. In: S. Cacciò, & G. Widmer (Eds.), *Cryptosporidium: Parasite and Disease, 1* (pp. 489-513). Springer, Vienna.

Ryan, U., Hijjawi, N., & Xiao, L. (2018). Foodborne cryptosporidiosis. *International Journal for Parasitology, 48*(1), 1-12.

Shahiduzzaman, M., & Daugschies, A. (2012). Therapy and prevention of cryptosporidiosis in animals. *Veterinary Parasitology, 188*(3-4), 203-214.

Siwila, J. (2023). The Triple Food-borne Protozoan Parasites: Cryptosporidium spp., Giardia duodenalis, Cyclospora cayetanensis—Hope in Transmission Reduction. *Current Clinical Microbiology Reports, 10*(3), 99-107.

Smith, H., Caccio, S., Cook, N., Nichols, R., & Tait, A. (2007). Cryptosporidium and Giardia as foodborne zoonoses. *Veterinary Parasitology, 149*(1-2), 29-40.

Smith, H., & Rose, J. (1990). Waterborne cryptosporidiosis. *Parasitology Today, 6*(1), 8-12.

Ungar, B. L. (2018). Cryptosporidiosis in humans (Homo sapiens). In J. P. Dubey, C. A. Speer, & R. Fayer (Eds.), *Cryptosporidiosis of Man and Animals, 1* (pp. 59-82). CRC Press.

Walker, M., Leddy, K., & Hagar, E. (2001). Effects of combined water potential and temperature stresses on Cryptosporidium parvum oocysts. *Applied Environmental Microbiology, 67*(12), 5526-5529.

Wang, Z.-D., Liu, Q., Liu, H.-H., Li, S., Zhang, L., Zhao, Y.-K., & Zhu, X.-Q. (2018). Prevalence of Cryptosporidium, microsporidia and Isospora infection in HIV-infected people: a global systematic review and meta-analysis. *Parasites Vectors, 11*, 1-19.

Warren, C. A., & Guerrant, R. L. (2007). Clinical disease and pathology. In R. Fayer, & L. Xiao (Eds.), *Cryptosporidium and Cryptosporidiosis, 2* (pp. 235-254). CRC press.

Zahedi, A., & Ryan, U. (2020). Cryptosporidium–an update with an emphasis on foodborne and waterborne transmission. *Research in Veterinary Science, 132*, 500-512.