

Updates on the Environmental Risks and Control of Cryptosporidiosis

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Abstract

Cryptosporidiosis is an emerging infectious disease that remains to be fully understood. The role of environmental issues such as water quality, livestock management, animal waste disposal and insect control are beginning to be recognized in the transmission of cryptosporidiosis. Prevention is highly emphasized as there is no consistently effective pharmacologic treatment for human cryptosporidiosis until now. Based on literature review, there are four environmental strategies in controlling cryptosporidiosis: (1) promotion of hygienic practices in the livestock industry (2) sanitary disposal of animal manure and human waste (3) constant surveillance of water supply and (4) protection of the immunocompromised population. More studies are needed to further understand the ecological factors that may contribute to the effective measures in controlling cryptosporidial transmission and infection.

INTRODUCTION

Cryptosporidiosis is an emerging infectious disease which involves several environmental health issues such as water quality, control of zoonotic transmission and environmental health policy. It is a self-limiting diarrheal disease which can develop into a life threatening infection for the immunocompromised. Primarily recognized as a zoonotic infection, cryptosporidiosis has been reported all over the world to cause significant burden of disease. The greatest toll occurs among those with HIV/AIDS, post-transplant immunosuppression, malignancies, the frail elderly and the very young. These issues are essential in fully understanding the zoonotic potential of *Cryptosporidium*. It forms the basis for evidence-based recommendations that could be transformed into rational and effective environmental policies.

REVIEW OF LITERATURE

It was in 1907 when Ernest Tyzzer first described cryptosporidiosis in laboratory mice [1]. In the early 1970s, cryptosporidiosis was considered as a veterinary disease affecting turkeys and cows [2]. Only in 1976 was cryptosporidiosis identified to cause disease in humans [3, 4]. It is increasingly being recognized as a significant cause of diarrhea, intestinal malabsorption and mortality among malnourished children worldwide [1].

The taxonomy of this organism has been traditionally based on morphology but the advent of advanced molecular techniques has led to the identification of other species and reorganization of the taxonomy [5]. As of 2004, there are 22 identified species [6]. There is no strict host specificity as other species once thought to infect poultry, domestic animals and mice only have been found in humans as well [2]. *Cryptosporidium parvum* was the first and most well known species to cause human cryptosporidiosis [7].

C. parvum predominates in human infection. It is classified into two genotypes. The natural cycle of genotype 1 is mostly human-to-human transmission while genotype 2 has a broad zoonotic coverage [5]. The other non-*parvum* species also appear in a small percentage of individuals. The epidemiology of non-*parvum* cryptosporidiosis is still being identified as new diagnostic techniques are being developed. We could probably expect increased disease accountability for non-*parvum* species in the coming years as we begin to understand more of the biology of the *Cryptosporidium* spp.

Cryptosporidium oocysts are only 4-6 µm size which can evade traditional water filtration methods [1]. The oocysts are also resistant to chlorination in drinking water and swimming pool treatment [8]. A study by Goldstein et al. suggest that these characteristics might be the reason for the 1994 outbreak in Las Vegas, Nevada, in spite of full chlorination and advanced water treatment procedures

implemented at the local water district [9].

The clinical course and severity of infection varies individually based on immune status. Immunocompetent individuals may have asymptomatic illness, abdominal discomfort, anorexia, nausea or watery diarrhea that normally persists for two to three days. In contrast, patients with immune deficiencies can have chronic, life threatening, and disseminated cryptosporidiosis. They may have profuse watery diarrhea that can persist for more than two weeks. An unusual complication is the development of gas-filled cysts in the intestinal walls which could rupture and cause life-threatening peritonitis [10]. The infection could also disseminate into the biliary tract, the pancreas and even in the lungs and sinuses [11].

Among HIV patients complaining with diarrhea, the worldwide prevalence of cryptosporidiosis is estimated at 32% [11]. Only 29% of immunocompromised patients with cryptosporidiosis experience remission. It is also being implicated as a cause of chronic diarrhea among cancer patients. *Cryptosporidium* oocysts have been detected in fecal samples of post-transplant and renal dialysis patients with debilitating diarrhea [12].

There is no consistently effective treatment for cryptosporidiosis. Some experimental drugs need to reach toxic human doses in order to reduce the parasite. Paromomycin, previously used in bovine cryptosporidial diarrhea [13], has inconsistent results on humans [2]. Nitazoxanide, a new thiazolide antiparasitic has just been approved by the U.S. Food & Drug Administration for cryptosporidiosis in children. Its use for the immunocompromised is still being evaluated [14]. For most HIV/AIDS patients, enhancement of immune response by increasing CD4+ T-lymphocyte count remains as the most effective strategy in controlling disseminated cryptosporidiosis [15]. Since no effective treatment is available, the emphasis of public health programs regarding cryptosporidiosis relies heavily on preventive strategies such as assuring water safety and strengthening individuals' immune defenses.

DISCUSSION

More than half of human cryptosporidiosis is attributed to *C. parvum* genotype 2 which is transmitted zoonotically [16]. There is strong evidence to indicate that a significant percentage of human cryptosporidiosis is attributable to exposure to cattle manure and contamination of drinking

water. There are also molecular epidemiologic studies documenting transmission of *C. parvum* genotype 2 between humans and livestock by direct contact in animal handling and not only through water-borne or food contamination [17, 18]. *Cryptosporidium* oocysts are readily infective once ingested from any contaminated source [1].

Bodies of water that receive sewage from cattle grazing areas have increased concentrations of oocysts [19]. As we concentrate more livestock in a geographic locality due to increasing food demand, we are also increasing the load of oocysts in the environment. In the U.S. alone, the estimated annual cattle manure production is 1.2 Billion tons [2].

Recent discoveries in the transmission of cryptosporidiosis also suggest involvement of flies. Synanthropic insects such as houseflies and other common filth flies have been documented in several studies to carry *Cryptosporidium* oocysts in their exoskeleton and digestive tracts [20,21,22]. Graczyk et al. suggest that flies are effective carriers of oocysts since they are strongly attracted to cattle manure [23]. This problem could be aggravated in areas where there is improper disposal of animal and human waste. Flies can spread *Cryptosporidium* oocysts beyond the usual route of water contamination. However, there has been no study that has successfully quantified the effect of flies in the overall transmission of cryptosporidiosis in humans. Nevertheless, we can not neglect the contribution of flies in the transmission of this disease, noting that the infective dose of *Cryptosporidium* has been documented to be as little as nine oocysts [24].

Drastic weather changes have also been implicated in the propagation of oocysts. Increased concentration of oocysts have been observed to coincide with heavy rainfall [25] such as the outbreaks in Milwaukee, Wisconsin and in other foreign countries. Flood water can flow from contaminated lands towards surface water for domestic use. In the Milwaukee outbreak, contamination of Lake Michigan with human and animal waste that may have overwhelmed the local water treatment process [26]. Similarly, Las Vegas is being supplied by Lake Mead and there have been sporadic reports identifying oocysts from the lake [9].

Recognizing that animal manure is a major contributor of *Cryptosporidium* oocysts in the environment, strategies for prevention should focus on safe disposal of animal and human waste as well as protection of water supplies.

First, livestock industries should be constantly reminded of

the threat of zoonoses from farm animals. Ramirez et al. suggest limiting the number of animals in enclosed facilities to limit animal transmission and oocysts load [6]. Animal facilities should be zoned away from waterways that are sources for the local community's drinking water supply. The goal is to reduce run-offs from animal farms. Grass or strips of vegetation on the edges of surface water may serve as natural buffer zones. They could trap sediments and reduce the organic matter that could contaminate the water [27].

Second, cattle manure should be promptly and safely disposed. Flies can carry oocysts to human water and food sources. Physical barriers such as insect screens and other environmental control strategies should be placed in areas where animal manure is densely concentrated. The same strategies also apply in managing human wastes. After all, the safe disposal of human and animal wastes has been a standard tenet in public sanitation.

Third, constant surveillance of water supply is advised as more studies are needed to understand the waterborne properties of *Cryptosporidium*. Analysis of the Las Vegas outbreak suggests that the municipal water was responsible for the transmission of cryptosporidiosis [9]. And yet, the quality of Las Vegas water source and water treatment process is beyond the prescribed U.S. national standards. There were speculations of possible cross contamination between unprocessed source and finished water within the treatment facility but these have not been proven [9]. Other possibilities entertained would be the contamination of the water distribution system. All of these concerns have not been resolved and more studies are needed

Lastly, immunocompromised individuals should avoid contact with animals that are host or carriers of *Cryptosporidium* spp. [28]. Providing safe water to immunocompromised individuals may be the most cost-effective means to prevent incidence of disseminated cryptosporidiosis. There is evidence in the literature advising HIV patients with CD4 counts less than 200/mm³ to boil their drinking water [11]. Also, controlling the environmental load of *Cryptosporidium* and other opportunistic pathogens can be a significant contribution in the protection of the immunocompromised.

CONCLUSION

Cryptosporidiosis is an emerging infectious disease. The organism has physical characteristics that enable it to survive in standard water processing. Various environmental agents

play a role in its transmission including flies and livestock. More studies are needed to understand the nature of the organism in terms of its detection and viability in water treatment processes. Prevention is still the primary and most effective strategy. The role of the environment can not be disregarded as climatic changes, increased demand for livestock production and pollution of our water reservoirs increasingly threatens human health.

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References

1. Dillingham RA, Lima AA, Guerrant RL. Cryptosporidiosis: epidemiology and impact. *Microbes Infect* 2002; 4(10):1059-66.
2. Fayer R. *Cryptosporidium*: a water-borne zoonotic parasite. *Vet Parasitol* 2004;126(1- 2):37-56.
3. Nime FA, Burek JD, Page DL, et al. Acute enterocolitis in a human being infected with the protozoan *Cryptosporidium*. *Gastroenterology* 1976;70(4):592-8.
4. Meisel JL, Perera DR, Meligro C, et al. Overwhelming watery diarrhea associated with a *cryptosporidium* in an immunosuppressed patient. *Gastroenterology* 1976;70(6):1156-60.
5. Chappell CL, Okhuysen PC. Cryptosporidiosis. *Curr Opin Infect Dis* 2002;15(5):523-7.
6. Ramirez NE, Ward LA, Sreevatsan S. A review of the biology and epidemiology of cryptosporidiosis in humans and animals. *Microbes Infect* 2004;6(8):773-85.
7. Monis PT, Thompson RC. Cryptosporidium and Giardia-zoonoses: fact or fiction? *Infect Genet Evol* 2003;3(4):233-44.
8. McNulty JM, Fleming DW, Gonzalez AH. A community-wide outbreak of cryptosporidiosis associated with swimming at a wave pool. *Jama* 1994;272(20):1597-600.
9. Goldstein ST, Juranek DD, Ravenholt O, et al. Cryptosporidiosis: an outbreak associated with drinking water despite state-of-the-art water treatment. *Ann Intern Med* 1996;124(5):459-68.
10. Sidhu S, Flamm S, Chopra S. Pneumatosis cystoides intestinalis: an incidental finding in a patient with AIDS and cryptosporidial diarrhea. *Am J Gastroenterol* 1994;89(9):1578-9.
11. Hunter PR, Nichols G. Epidemiology and clinical features of *Cryptosporidium* infection in immunocompromised patients. *Clin Microbiol Rev* 2002;15(1):145-54.
12. Clifford CP, Crook DW, Conlon CP, et al. Impact of waterborne outbreak of cryptosporidiosis on AIDS and renal transplant patients. *Lancet* 1990;335(8703):1455-6.
13. Fayer R, Ellis W. Paromomycin is effective as prophylaxis for cryptosporidiosis in dairy calves. *J Parasitol* 1993;79(5):771-4.
14. Fox LM, Saravolatz LD. Nitazoxanide: a new thiazolide antiparasitic agent. *Clin Infect Dis* 2005;40(8):1173-80.
15. Maggi P, Larocca AM, Quarto M, et al. Effect of antiretroviral therapy on cryptosporidiosis and

microsporidiosis in patients infected with human immunodeficiency virus type 1. *Eur J Clin Microbiol Infect Dis* 2000;19(3):213-7.

16. Guyot K, Follet-Dumoulin A, Lelievre E, et al. Molecular characterization of *Cryptosporidium* isolates obtained from humans in France. *J Clin Microbiol* 2001;39(10):3472-80.
17. Mallon M, MacLeod A, Wastling J, et al. Population structures and the role of genetic exchange in the zoonotic pathogen *Cryptosporidium parvum*. *J Mol Evol* 2003;56(4):407-17.
18. McLauchlin J, Amar C, Pedraza-Diaz S, et al. Molecular epidemiological analysis of *Cryptosporidium* spp. in the United Kingdom: results of genotyping *Cryptosporidium* spp. in 1,705 fecal samples from humans and 105 fecal samples from livestock animals. *J Clin Microbiol* 2000;38(11):3984-90.
19. Bagley ST, Auer MT, Stern DA, et al. Sources and fate of *Giardia* cysts and *Cryptosporidium* oocysts in surface waters. *J. Lake Res. Manage.* 1998;14:379-372.
20. Follet-Dumoulin A, Guyot K, Duchatelle S, et al. Involvement of insects in the dissemination of *Cryptosporidium* in the environment. *J Eukaryot Microbiol* 2001;Suppl:36S.
21. Graczyk TK, Grimes BH, Knight R, et al. Detection of *Cryptosporidium parvum* and *Giardia lamblia* carried by synanthropic flies by combined fluorescent in situ

hybridization and a monoclonal antibody. *Am J Trop Med Hyg* 2003;68(2):228-32.

22. Szostakowska B, Kruminis-Lozowska W, Racewicz M, et al. *Cryptosporidium parvum* and *Giardia lamblia* recovered from flies on a cattle farm and in a landfill. *Appl Environ Microbiol* 2004;70(6):3742-4.
23. Graczyk TK, Fayer R, Knight R, et al. Mechanical transport and transmission of *Cryptosporidium parvum* oocysts by wild filth flies. *Am J Trop Med Hyg* 2000;63(3-4):178-83.
24. Okhuysen PC, Chappell CL, Crabb JH, et al. Virulence of three distinct *Cryptosporidium parvum* isolates for healthy adults. *J Infect Dis* 1999;180(4):1275-81.
25. Atherholt T, LeChevalier M, Norton W. Effect of rainfall on *Cryptosporidium* and *Giardia*. *J. AWWA* 1998;90:66-80.
26. Mac Kenzie WR, Hoxie NJ, Proctor ME, et al. A massive outbreak in Milwaukee of cryptosporidium infection transmitted through the public water supply. *N Engl J Med* 1994;331(3):161-7.
27. Atwill ER, Hou L, Karle BM, et al. Transport of *Cryptosporidium parvum* oocysts through vegetated buffer strips and estimated filtration efficiency. *Appl Environ Microbiol* 2002;68(11):5517-27.
28. Juranek DD. Cryptosporidiosis: sources of infection and guidelines for prevention. *Clin Infect Dis* 1995;21 Suppl 1:S57-61.

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