

Calderon 1996, National Research Council 1997). This under reporting is due to a number of factors. Many people experiencing gastrointestinal illness do not seek medical attention. Where medical attention is provided, the pathogenic agent may not be identified through routine testing. Physicians often lack sufficient information to attribute gastrointestinal illness to any specific origin, such as drinking water, and few States have an active outbreak surveillance program. Consequently, outbreaks are often not recognized in a community or, if recognized, are not traced to a drinking water source.

In addition, an unknown but probably significant portion of waterborne disease is endemic (*i.e.* isolated cases not associated with an outbreak) and, thus, is even more difficult to recognize. The Economic Analysis for the proposed LT2ESWTR (USEPA 2003a) uses data on *Cryptosporidium* occurrence, infectivity, and treatment to estimate the baseline endemic incidence of cryptosporidiosis attributable to drinking water, as well as the reductions projected as a result of this rule.

Most waterborne pathogens cause gastrointestinal illness with diarrhea, abdominal discomfort, nausea, vomiting, and other symptoms. The effects of waterborne disease are usually acute, resulting from a single or small number of exposures. Such illnesses are generally of short duration in healthy people. However, some pathogens, including *Giardia lamblia* and *Cryptosporidium*, may cause disease lasting weeks or longer in otherwise healthy individuals, though this is not typical for *Cryptosporidium*. Waterborne pathogens also cause more serious disorders such as hepatitis, peptic ulcers, myocarditis, paralysis, conjunctivitis, swollen lymph glands, meningitis, and reactive arthritis, and have been associated with diabetes, encephalitis, and other diseases (Lederberg 1992).

There are populations that are at greater risk from waterborne disease. These sensitive subpopulations include children (especially infants), the elderly, the malnourished, pregnant women, the disease impaired (*e.g.*, diabetes, cystic fibrosis), and a broad category of those with compromised immune systems, such as AIDS patients, those with autoimmune disorders (*e.g.*, rheumatoid arthritis, lupus erythematosus, multiple sclerosis), transplant recipients, and those on chemotherapy (Rose 1997). This sensitive segment represents almost 20% of the population in the United States (Gerba *et al.* 1996). The severity and duration of illness is often greater in sensitive subpopulations than

in healthy individuals, and in a small percentage of such cases, death may result.

2. *Cryptosporidium* Health Effects and Outbreaks

Cryptosporidium is a protozoan parasite that exists in warm-blooded hosts and, upon excretion, may survive for months in the environment (Kato *et al.*, 2001). Ingestion of *Cryptosporidium* can lead to cryptosporidiosis, a gastrointestinal illness. Transmission of cryptosporidiosis often occurs through consumption of feces contaminated food or water, but may also result from direct or indirect contact with infected persons or animals (Casemore 1990). Surveys (described in Section III) indicate that *Cryptosporidium* is common in surface waters used as drinking water supplies. Sources of *Cryptosporidium* contamination include animal agriculture, wastewater treatment plant discharges, slaughterhouses, birds, wild animals, and other sources of fecal matter.

EPA is particularly concerned about *Cryptosporidium* because, unlike pathogens such as bacteria and most viruses, *Cryptosporidium* oocysts are highly resistant to standard disinfectants like chlorine and chloramines. Consequently, control of *Cryptosporidium* in most treatment plants is dependent on physical removal processes. Finished water monitoring data indicate that *Cryptosporidium* is sometimes present in filtered, treated drinking water (LeChevallier *et al.* 1991; Aboytes *et al.* 2002). Moreover, as noted later, many of the individuals sickened by waterborne outbreaks of cryptosporidiosis were served by filtered surface water supplies (Solo-Gabriele and Neumeister, 1996). In some cases, these outbreaks were attributed to treatment deficiencies, while in other cases the cause was unidentified (see Table II-1).

These data suggest that surface water systems that filter and disinfect may still be vulnerable to *Cryptosporidium*, depending on the source water quality and treatment effectiveness. Today's proposed rule addresses concern with passage of *Cryptosporidium* through physical removal processes during water treatment, as well as in systems lacking filtration.

a. *Health effects.* *Cryptosporidium* infection is characterized by mild to severe diarrhea, dehydration, stomach cramps, and/or a slight fever. Symptoms typically last from several days to two weeks, though in a small percentage of cases, the symptoms may persist for months or longer in otherwise healthy individuals. Human feeding studies

have demonstrated that a low dose of *Cryptosporidium parvum* (*C. parvum*) is sufficient to cause infection in healthy adults (DuPont *et al.* 1995, Chappell *et al.* 1999, Messner *et al.* 2001). Studies of immunosuppressed adult mice have demonstrated that a single viable oocyst can induce patent *C. parvum* infections (Yang *et al.* 2000).

There is evidence that an immune response to *Cryptosporidium* exists, but the degree and duration of this immunity is not well characterized. In a study by Chappell *et al.* (1999), individuals with a blood serum antibody (IgG), which can develop from exposure to *C. parvum*, demonstrated immunity to low doses of oocysts. The investigators found the ID50 dose (*i.e.*, dose that infects 50% of the challenged population) of one *C. parvum* isolate for adult volunteers who had pre-existing serum IgG to be 1,880 oocysts in comparison to 132 oocysts for individuals reported as serologically negative. However, the implications of these data for studies of *Cryptosporidium* infectivity are unclear. Earlier work did not observe a correlation between the development of antibodies after *Cryptosporidium* exposure and subsequent protection from illness (Okhuysen *et al.* 1998). A subsequent investigation by Muller *et al.* (2001) observed serological responses to *Cryptosporidium* antigens in samples from individuals reported by Chappell *et al.* as serologically negative.

Cryptosporidium parvum was first recognized as a human pathogen in 1976 (Juranek 1995). Cases of illness from *Cryptosporidium* were rarely reported until 1982 when documented disease incidence increased due to the AIDS epidemic (Current 1983). As laboratory diagnostic techniques improved during subsequent years, outbreaks among immunocompetent persons were recognized as well. Human, cattle, dog and deer types of *C. parvum* have been found in healthy individuals (Ong *et al.* 2002, Morgan-Ryan *et al.* 2002). Other *Cryptosporidium* species (*C. felis*, *C. meleagridis*, and possibly *C. muris*) have infected healthy individuals, primarily children (Xiao *et al.* 2001, Chalmers *et al.* 2002, Katsumata *et al.* 2000). Cross-species infection occurs. The human type of *C. parvum* (now named *C. hominis* (Morgan-Ryan *et al.* 2002)) has infected a dugong and monkeys (Spano *et al.* 1998). The cattle type of *C. parvum* infects humans, wild animals, and other livestock, such as sheep, goats and deer (Ong *et al.* 2002).

As noted earlier, there are sensitive populations that are at greater risk from pathogenic microorganisms.