

# CRYPTOSPORIDIUM PARVUM AND CRYPTOSPORIDIUM HOMINIS

## THE ORGANISM/TOXIN

*Cryptosporidium parvum* and *Cryptosporidium hominis* are intracellular protozoan parasites that may produce gastrointestinal symptoms when ingested by humans. Up until 2002 *C. parvum* was named *C. parvum* genotype 2 (cattle genotype) and *C. hominis* was named *C. parvum* genotype 1 (the human genotype) (Morgan-Ryan *et al.*, 2002). They are now recognised as different species based on genetic distinctions, but care should be taken when reviewing pre-2002 literature for this reason.

There are a further thirteen recognised species of the genus *Cryptosporidium*. *C. parvum* and *C. hominis* are the most widespread and most frequently associated with human infections.

*C. hominis* and *C. parvum* are transmitted person-to-person. *C. parvum* is also transmitted animal-to-animal or animal-to-human (zoonotically) (Awad-El Kariem 1999). Both organisms move between hosts in an environmentally-resistant oocyst form that is excreted with faeces. An oocyst is the resting stage of the protozoan, similar to a bacterial spore, and may facilitate transmission of the organism via contaminated water or food.

## GROWTH AND CONTROL

N.B. Viability refers to whether the organism is still alive, infectivity relates to the organism's ability to infect a host.

### Growth

The organism does not replicate outside the intestines of hosts.

### Survival

#### Temperature

Oocysts remain infective in water at:

- 67.5°C for 1 minute
- -5°C for up to 8 weeks
- -10°C for up to 7 days
- -15°C for up to 24 hours
- -20°C for up to 5 hours.

(Fayer 1994; Fayer and Nerad 1996; Fayer *et al.*, 1998)

### Inactivation

#### Temperature

Oocysts become non-infective when:

- Pasteurised (71.7°C, 15 seconds) (Harp *et al.*, 1996)
- Held in water at 64.2°C for 2 minutes (Fayer 1994)
- Frozen in de-ionised water (-20°C for 24 hours and -70°C for 1 hour) (Fayer and Nerad, 1996)
- Frozen in liquid nitrogen (-196°C) (Robertson *et al.*, 1992)
- Frozen in ice-cream (-20°C for 24 hours) (Deng and Cliver, 1999).

#### pH

- pH <4 or >11 results in minor viability loss (Jenkins *et al.*, 1998; Kniel *et al.*, 2003).
- Ammonia can enhance inactivation at low and high pH values (Jenkins *et al.*, 1998).
- Fruit juice organic acids inhibit oocyst infectivity (Kniel *et al.*, 2003).

#### pH

Viable *C. parvum* oocysts were detected after 14 days, in media based on citric acid (pH 4.6, 3.6 and 2.6), acetic acid (pH 3.6) or lactic acid (pH 4.6) held at 4°C or 22°C (Dawson *et al.*, 2004).

#### Water Activity

Oocysts survive on wet stainless steel (93.1% viable after 30 minutes) (Deng and Cliver, 1999).

#### Water activity

Oocysts are sensitive to air drying (e.g. at ambient temperatures). Viability on stainless steel after 10 minutes = 81%, 1 hour = 69%, 2 hours = 45%, 4 hours = 5% (Deng and Cliver, 1999).

At  $a_w$  0.85, 99.9% are non-infective after 24 hours at 28°C, or 1 week at 7°C. At  $a_w$  0.95, 99.99% are non-infective after 1 week at 28°C, or 2 weeks at 7°C (Rose and Slifko, 1999).

#### Radiation

Sensitive to UV from sunlight and UV lamps. Under strong sunlight for 8 and 12 hours, oocyst viability reduced from 98% to 11.7% and 0.3%, respectively. A synergistic effect occurs at temperatures greater than 45°C (Méndez-Hermida *et al.*, 2007).

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## Disinfectants / Sanitisers

Oocysts are resistant to chlorine and monochloramine (Carey *et al.*, 2004). Swimming pool transmission has been problematic for this reason.

Only 5/35 disinfectants tested were effective with short exposure (O'Donoghue, 1995). These were:

- Ammonia 5% for 120 minutes or 50% for 30 minutes
- Formalin 10% for 120 minutes

- Hydrogen peroxide 10 vol; 3% for 30 minutes
- "Exspor" working dilution 30 minutes
- Oo-cide (ammonia) 5% for 5 minutes.

Ozone (25°C, 1ppm) renders oocysts non-infective after 3 minutes (90% decrease), 5 minutes (99%) and 10 minutes (99.9%) (Korich *et al.*, 1990).

## CLINICAL PICTURE

**Incubation:** Time from ingestion to illness usually 3-5 days, up to 2 weeks (Fayer, 2004).

**Symptoms:** Copious watery non-bloody diarrhoea, vomiting, anorexia, fever, malaise, abdominal cramping and weight loss. Usually self-limiting. Symptoms typically last 2-4 days but can last up to two weeks. Infectious oocysts usually excreted for 6-9 days, but can be excreted by asymptomatic carriers for up to 2 months or longer following infection. The respiratory system may also be adversely affected by the illness.

**Condition:** Cryptosporidiosis.

**Dose:** Healthy human volunteer studies have shown that the infectious dose varies depending on the isolate used. The dose needed to infect 50% of the population ranged from 9-1042 oocysts (Chappell *et al.*, 2006; Okhuysen *et al.*, 1999).

**At Risk Groups:** Children aged under 1 year and immunocompromised people are more susceptible (Carey *et al.*, 2004).

**Long Term Effects:** Immunocompromised patients (e.g. AIDS patients or those receiving immunosuppressant drugs) develop severe symptoms that can last months or years and the condition can be fatal. The infection may also spread to other parts of the body in immunocompromised patients, such as the respiratory tract (Ortega, 2007).

**Treatment:** There is no specific treatment for cryptosporidiosis currently available, but some antibiotics (e.g. spiramycin) are effective (Ortega, 2007).

## SOURCES

**Human:** Person-to-person transmission occurs with *C. parvum* and *C. hominis*. More than  $10^8$ - $10^{10}$  oocysts can be excreted daily in the faeces of infected hosts (Fayer *et al.*, 2004).

**Animal:** *C. parvum* has been identified in 155 species of mammal including domestic animals. Preweaned ruminants, especially calves, are especially vulnerable to infection. Shedding of oocysts increases in calves to a peak at day 12 (excreting approximately  $3.89 \times 10^{10}$  oocysts) (Nydam *et al.*, 2001; Fayer 2004). Flies are potential vehicles of transmission as oocysts can pass unaltered through their gastro-intestinal tract and be deposited in fly faeces (Graczyk *et al.*, 2004; Graczyk *et al.*, 1999), but flies have never been linked to an outbreak. Around 80% of oocysts remain in an infected host's gut and can autoinfect the host. The remaining 20% develop thick (trilaminar) cell walls and are released in the faeces of the infected host into the environment. This can lead to subsequent infection of a new host (O'Donoghue, 1995).

**Food:** Oocysts have been detected in raw milk, raw meat, raw fruits and vegetables. Foods suspected as acting as vehicles of disease include raw milk, green onions, apple cider, sausage, fruit/vegetables and frozen tripe (Duffy and Moriarty, 2003).

**Environment:** Oocysts in the environment are particularly resilient, especially under cool, moist conditions. Infectivity can be retained for months, especially in low water temperatures (<5°C but above freezing) (Fayer, 2004). The long term viability of oocysts in animal faeces is unclear. A study of a variety of slurries, soil types, pH ranges and temperatures found that acid to neutral soils at 4°C were most conducive to oocyst survival (still viable after 162 days) (Warnes and Keevil, 2003).

**Transmission Routes:** Three main routes of transmission are person-to-person, zoonotic, and via faecally-contaminated water or food. Outbreak data for New Zealand suggests that foodborne transmission makes a very small contribution to the overall burden of cryptosporidiosis (Gilbert *et al.*, 2007).

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## OUTBREAKS AND INCIDENTS

**NZ Incidence:** 854 cases of cryptosporidiosis were notified in 2009 (19.8 per 100,000 population) and 34 cases were reported as being hospitalised (ESR, 2010b). The highest rates were reported in the West Coast DHB (55.2/100,000; 18 cases) and South Canterbury DHB (50.4/100,000; 28 cases). Of 851 cases, 54.9% were children aged less than 15 years.

### New Zealand epidemiological studies

Analysis of 1997-2006 data showed incidence was 2.84 times higher in rural areas (50.68/100,000) than urban areas (17.22/100,000). 55% of cases occurred in spring (Sept-Nov) and 11% occurred summer (Dec-Feb), and the spring peak was most pronounced in rural areas. A small late Summer/early Autumn peak was observed in urban areas in 1998, 1999 and 2001 (Snel *et al.*, 2009).

### New Zealand outbreaks

In 2009 there were 20 outbreaks of cryptosporidiosis, involving 68 cases (ESR, 2010a). No cases were hospitalised. Person-to-person transmission was implicated in 15 of these outbreaks (55 cases).

### Overseas outbreaks overseas associated with food/water:

**Chicken salad:** USA, 15 ill, 2 confirmed (26 exposed). Food handler related (CDC, 1996).

**Unpasteurised apple cider:** USA, 20 confirmed cases, 11 suspected. Apples washed in well-water (Peng *et al.*, 1997).

**Unpasteurised apple cider:** USA, 154 cases. Apples collected from ground grazed by livestock (Millard *et al.*, 1994).

**Green onions:** USA, 54 cases (62 exposed). Food handler related and onions unwashed (CDC, 1998).

**Fruits/vegetables:** USA, 148 cases. Food handler related. (Quiroz *et al.*, 2000).

**Salad vegetables:** Denmark, 2005, 99 cases (13 confirmed with *C. hominis* infection). Likely contaminated foods were peeled whole carrots served in water, grated carrots, red peppers. Food handler implicated (Ethelberg *et al.*, 2009).

**Salad vegetables:** Finland, 2008, 72 cases (4 confirmed, 1 with *C. parvum* infection). Salad implicated by case-control study but cause of contamination not identified (Pönkä *et al.*, 2009).

**Drinking water:** Milwaukee, USA, 403,000 cases (attack rate 55%). One of largest recorded waterborne outbreaks in USA. Hospitalisation rate estimated at 15%, case fatality rate 0.5%.

**Drinking water:** Ireland, > 476 cases. Three unrelated drinking water outbreaks. Related to septic tank and blocked drain water entering the drinking water system (Xiao 2002).

**Drinking water:** Australia, 1998. Two episodes of contaminated water supply in Sydney area. No measurable increase in disease (Anonymous, 1998).

**Drinking water:** Wales, 2005, 218 cases (*C. hominis*). Absence of effective treatment to remove *Cryptosporidium* oocysts at drinking water treatment plant (Mason *et al.*, 2010).

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