Cryptosporidium infection in Australian wildlife

Fact sheet

January 2023

Key points

- *Cryptosporium* spp. are protozoal parasites that can cause gastrointestinal disease in host species.
- *Cryptosporium* infections have been reported in a wide range of Australian native mammal, bird and reptile species.
- In the majority of cases, infection with *Cryptosporidium* in native wildlife species does not result in disease.
- *Cryptosporidium* spp. are extremely environmentally resistant and highly transmissible, and treatment options are limited.

Introductory statement

Cryptosporidium spp. are protozoal parasites that infect a range of vertebrate hosts including mammals, birds, reptiles, fish and amphibians, causing predominantly gastrointestinal disease. Infections are often subclinical, but juvenile and immunocompromised hosts may experience debilitating or even fatal disease (cryptosporidiosis). While there have been numerous reports of *Cryptosporidium* spp. infection in Australian wildlife, the extent of the parasite's prevalence, host range and disease burden is not known. Surveillance for *Cryptosporidium* should be prioritised in both free-living and captive Australian wildlife, as should prevention practices when working with wildlife species.

Aetiology

Cryptosporidium spp. (Apicomplexa: Cryptosporidiidae) are protozoa that infect the gastrointestinal and occasionally, respiratory and biliary tract of a wide variety of vertebrates, including humans (Vanathy 2022). At least 44 *Cryptosporidium* species and over 120 genotypes are recognised, with differing host ranges and virulence (Pane and Putignani 2022). Molecular analysis is necessary to characterise *Cryptosporidium* to species or genotype level (Ryan et al. 2021b).

One Health implications

Humans: Cryptosporidiosis is a major diarrhoeal disease of humans, with children and the immunocompromised most at risk (Pane and Putignani 2022). Most of the *Cryptosporidium* species

detected in Australian wildlife can infect humans, with the most common species found in human cases in Australia being *C. parvum*, *C. hominis* and *C. meleagridis* (Zahedi et al. 2018). Australians living in tropical and remote communities were found to be nearly eight times more likely to have cryptosporidiosis than those in urban centres (Forbes et al. 2021). The disease burden in Australia disproportionally affects the Aboriginal population, with notification rates up to 50 times higher in Aboriginal communities than in non-Aboriginal communities in WA (Ng-Hublin et al. 2017). There are two reports of humans infected with marsupial-derived *C. fayeri*, in 2010 (Waldron et al. 2011) and in 2020 (Braima et al. 2021). Both humans were immunocompromised and symptomatic and were infected with subtypes that were identified in the faeces of kangaroos inhabiting their relevant drinking water catchments. Additionally, the presence of *C. hominis* in Australian bats and kangaroos highlights the potential for reverse-zoonotic transmission (Schiller et al. 2016).

Domestic animals: Transmission can occur between species and disease may occur in ruminant species.

Wildlife and environment: Disease reports in wildlife species are rare.

Natural hosts

The *Cryptosporidium* genus has an extensive host range, that will likely continue to expand with further genetic investigation (Ryan et al. 2021b) and especially studies of wildlife species. A list of the major hosts of *Cryptosporidium* species and genotypes is provided in Ryan et al. (2021a).

Cryptosporidium has been detected in a broad range of **mammalian** hosts (Zahedi et al. 2016). Over 30 species of **bird** have been found infected with *Cryptosporidium* spp. (Ryan 2010). Infection has been recorded in over 57 **reptile** species including snakes, lizards, crocodilians and chelonians (Jacobson 2007). Several species of wild, cultured and ornamental marine and freshwater **fish** have been infected (Golomazou and Karanis 2020). A small number of cases have been reported in **amphibians**, all in frogs and toads (Zahedi et al. 2016).

World distribution

Cryptosporidium is globally distributed, with infections in humans and animals reported in Asia (El-Alfy and Nishikawa 2020), Africa (Bodager et al. 2015), North America (Feng et al. 2007), South America (Nakashima et al. 2022), Europe (Sangster et al. 2016), Oceania (Braima et al. 2021) and Antarctica (Rengifo-Herrera et al. 2013). The prevalence and distribution of *Cryptosporidium* species and genotypes varies with region.

Occurrences in Australia

Cryptosporidium has been reported to occur across much of Australia, primarily in marsupials (Zahedi et al. 2016). Prevalence in Australian wildlife has typically been determined from the study of animal faecal samples in water catchments. Prevalence in faecal samples collected has been reported as 5–25.8% in NSW, 1.6–2.8% in Vic, 6.7-17.6% in WA and 25.9% in Qld (see Zahedi et al. 2018). At least 22 *Cryptosporidium* species and several novel genotypes have been detected in Australian wildlife (Koehler et al. 2020).

Of the host species studied, the highest prevalence reported is in marsupials (particularly macropods) and rodents. 'Marsupial-specific' *C. fayeri* and *C. macropodum* have been detected in Australia for decades (Ryan and Power 2012). While *C. hominis* is traditionally considered to be human-specific, recent detection in marsupials suggests reverse-zoonotic capabilities (Zahedi et al. 2018). Both native and invasive rodent species provide a natural reservoir for *C. viatorum* in Australia (Ryan et al. 2021b). *Cryptosporidium* infection occurs in captive reptiles (snakes, and also lizards and turtles) in Australia (Ladds 2009).

Epidemiology

Cryptosporidium spp. are primarily **transmitted** via the faecal-oral route, either by direct contact between animals, or through contact with a contaminated environment, usually a shared **food** or **water** source (Zahedi and Ryan 2020). Respiratory transmission has been reported in humans (Sponseller et al. 2014). Transmission can occur within species, between different animal species and between animals and humans (Pumipuntu and Piratae 2018). When the infective stage (sporulated oocyst) is ingested, the parasites are released and invade target host cells where they undergo asexual reproduction and finally sexual reproduction to produce the infective oocyst that is excreted in faeces. The pre-patent period is 2-14 days (Ikiroma and Pollock 2021). The oocysts are capable of surviving in waterbodies and in moist, temperate terrestrial environments for several months (Santos et al. 2019). It is likely that the warmer, wetter conditions associated with climate change will impact the seasonal patterns of outbreaks (summer and autumn peaks in Australia) and favour transmission (Ikiroma and Pollock 2021).

Clinical signs

Reports of clinical disease in animal hosts are rare. In mammals, birds and fish (Santín 2013; Palermo 2016; Hatam-Nahavandi et al. 2019), juveniles may be more severely affected than adults, whereas reptiles may become ill at any life stage (Cranfield et al. 1999).

If disease occurs, the primary clinical sign is watery diarrhoea, which is often accompanied by dehydration, weight loss, lethargy, and reduced growth rate and body condition. The disease is usually self-limiting but death occasionally occurs (Doneley et al. 2017). Other clinical signs vary with parasite species and genotype as well as host species, age and immunocompetency (Santin 2020).

Cryptosporidiosis in **birds** may be respiratory, intestinal or renal. Ocular disease has been observed in owls (Molina-Lopez et al. 2010). Some birds experience chronic vomiting (Makino et al. 2010), and *C. baileyi* may cause dyspnoea, coughing and sneezing (Santín 2013).

If clinical disease occurs in **reptiles**, it may be protracted and fatal (Terrell et al. 2003). Infected individuals may show regurgitation one to three days after feeding, firm mid-body swelling and recurrent cloacal prolapse (McCracken et al. 2017).

In **fish**, *C. molnari* infection has been associated with whitish faeces, abdominal swelling and ascites (Alvarez-Pellitero and Sitjà-Bobadilla 2002). Regurgitation, swollen coelomic cavities, atrophied skeletal muscle and tucked abdomens may also occur in larval or juvenile fish.

Infection is largely asymptomatic in native **mammals** and **amphibians** (Ladds 2009).

Diagnosis

Diagnosis can be confirmed by microscopy or polymerase chain reaction (PCR) analysis of faecal material (Santos et al. 2019). Antigen detection techniques such as immunofluorescent antibody (IFA) tests and enzyme-linked immunosorbent assay (ELISAs) are commercially available and have been used to detect *Cryptosporidium* spp., however, they have reduced specificity and sensitivity for species other than *C. hominis* and *C. parvum* (Zahedi 2018).

DNA sequencing is required to identify the species and genotype (Zahedi and Ryan 2020).

Histological examination may be conducted to investigate gross pathology but is not required for diagnosis.

Note that *Cryptosporidium* spp. in ingested prey animals of the target host may be detected which does not constitute true infection (Doneley et al. 2017).

Laboratory diagnostic specimens and procedures

Multiple, temporally distinct faecal samples per individual are desirable, since clinically affected animals tend to shed oocysts intermittently (Santos et al. 2019). Collect fresh faecal specimens into sterile containers and store in 2.5% potassium dichromate at 4°C until analysis (Baz-González et al. 2022). Collect post-mortem tissue samples (≤ 1cm) from the intestines or stomach depending on parasite species (both, if species unconfirmed) (Hyndman and Marschang 2017), fixed in 10% buffered formalin (Shilton 2017). Samples taken from the respiratory tract, lungs, gills, bursa of Fabricius, liver and kidneys may also demonstrate pathology (Koehler et al. 2020).

Direct faecal examination: direct and concentrated faecal smears are used to identify *Cryptosporidium* oocysts by microscopy. Prior to examination, samples should be modified using oocyst concentration and purification methods followed by staining, to improve sensitivity of detection (Santos et al. 2019).

Gross necropsy: investigation of multiple organs, particularly intestines, stomach and gastrointestinal tract, to determine the presence and nature of any gross lesions (Golomazou and Karanis 2020).

Pathology

Pathological changes range from mild to severe.

Gross lesions

Mammals: pathological description of *Cryptosporidium* infection in Australian native mammals is lacking, but lesions are presumably comparable to other mammalian hosts (Ladds 2009). Subcutaneous oedema and visceral congestion may occur, and the large intestine may contain light yellow pasty material. Lungs may be diffusely to moderately oedematous and have pale pink to dark red, multifocal to coalescing discolouration, particularly in the caudal lobes. Fibrin deposits may be observed in the lumen of the distal trachea and bronchi (Gonzalez-Astudillo et al. 2021).

Birds: in several avian species superficial mucous throughout the respiratory and oculo-nasal tracts, distended mucous glands and suppurative inflammation has been noted (Ladds 2009). Birds with renal cryptosporidiosis may have enlarged, pale kidneys, occasionally with urate crystals in surface tubules (Santín 2013). Airsacculitis was observed as a result of experimental infection (Lindsay et al. 1987).

Reptiles: Oedematous gastric mucosa, increased stomach diameter, mucosal petechiae, brush haemorrhages and focal necrosis can occur in snakes (Kuroki et al. 2008). Pathogenic gastroenteritis is more common in lizards, involving ecchymoses, acute inflammation and red discolouration of gastrointestinal tract mucosa (Ladds 2009). Additional pathological changes in lizards can include renal oedema and congestion, cholecystitis, cystitis and an absence of fat bodies (Gałęcki and Sokół 2018). Intestinal lesions also occur in infected tortoises (Traversa 2010).

Fish: clinical infection typically results in mucous contents, gas and whitish faeces in the intestines causing distension. Intestines may also have a thickened mucosa, haemorrhages and be pale in colour. The liver may be congested, with granulomatous inflammatory lesions and pale brown discoloration. Hyperaemia of the gills and splenomegaly may also occur (Golomazou and Karanis 2020).

Histology

Parasites typically enter the mucosal epithelium or are free within the lumen and can result in the following changes:

Mammals: diffuse intestinal villous atrophy, crypt dilation and presence of sloughed epithelial cells and cellular debris in the lumen. Reduced alveolar space caused by multifocal infiltration by sloughed cells may be observed, particularly in animals suffering from upper respiratory disease (Zahedi and Ryan 2020).

Birds: epithelial hypertrophy and hyperplasia, loss of cilia, distended mucous glands, infiltration of stroma by sloughed cells and focal suppurative inflammation. Villous atrophy is also seen, as well as bursal follicular atrophy and detachment of enterocytes (Ladds 2009).

Reptiles: reduction of brush border, flattening of epithelial cells and proliferation of gastric mucous cells (Ladds 2009). Intestinal lesions in lizards, chelonians and (rarely) snakes consist of heterophil, lymphocyte and macrophage infiltration (Oros et al. 1998). More than 80% of the intestinal cells may harbour parasites (Jacobson 2007).

Fish: widespread pathology with multiple granulomas, vacuolisation, degradation, sloughing of epithelial cells and necrosis may be present in the tissue of infected organs. In severe cases, the mucosal architecture on the intestines is distorted resulting in large vacuoles full of oocyst clusters distending epithelial cells (Golomazou and Karanis 2020).

Pathology in **amphibians** has not been reported.

Differential diagnoses

Given the generic nature of clinical signs associated with *Cryptosporidium* spp. infection, and the lack of disease in most infections, other known causes of gastrointestinal disease in the target host species should be investigated. These include other enteric pathogens such as *Giardia* spp. or *Escherichia coli* (Santín 2013). McCracken et al. (2017) provide a comprehensive list of differential diagnoses for many of the clinical signs observed in reptiles with cryptosporidiosis.

Treatment

There is currently no standardised treatment for *Cryptosporidium* spp. infection. In farm animals, fluid therapy has been used to manage diarrhoea and dehydration (Santin 2020). Hyperimmune bovine colostrum has been used to treat reptiles with some success (Gałęcki and Sokół 2018). Paromomycin has been used in reptiles with mixed results (Gibbons 2014) as it does not eliminate the pathogen (Hyndman and Marschang 2017). Dimetridazole was apparently successful in eliminating infection in bilbies (*Macrotis lagotis*) after multiple doses (Warren et al. 2003).

Prevention and control

The best practice for preventing outbreaks in captive wildlife populations is a combination of *Cryptosporidium* screening, quarantining new individuals and regular maintenance and cleaning of habitats and enclosures (Koehler et al. 2020). There is no commercially available vaccine. Control of infection in the wild is not possible as, even with the culling of reservoir hosts, the soil column acts as an environmental reservoir for *Cryptosporidium* (King and Monis 2007).

Simple control procedures can limit the spread of cryptosporidiosis during an outbreak in captive species. Quarantining infected animals, scrubbing of contaminated surfaces and prompt removal and appropriate disposal of contaminated wastes will remove reservoirs of parasites thereby reducing the risk of spread of infection. Chronic shedding and mortality may occur in birds and reptiles, therefore, if clinical signs of cryptosporidiosis occur, euthanasia may be considered on humane grounds.

Hydrogen peroxide is lethal to oocysts following exposure at room temperature for 10 minutes and is not inhibited by the presence of organic matter (Carmel and Groves 1993). Infectivity of oocysts is neutralised by desiccation, ideally by exposure to high temperatures (>60°C) (Innes et al. 2020).

Research

Further research in the following areas would be particularly beneficial to expanding understanding of *Cryptosporidium* infection in Australian wildlife:

- The presence and nature of pathological changes in infected native mammals, especially marsupials.
- The prevalence of infection in free living Australian reptiles and amphibians.
- Geographic distribution of *Cryptosporidium* spp. in Australia, outside of major water catchments and captive animal populations.
- Improved treatment and prevention regimes for infected individuals.

- Measures to control environmental reservoirs of *Cryptosporidium*.
- The impact of climate change on the seasonal patterns of transmission.

Surveillance and management

Wildlife disease surveillance in Australia is coordinated by Wildlife Health Australia. The National Wildlife Health Information System (eWHIS) captures information from a variety of sources including Australian government agencies, zoo and wildlife parks, wildlife carers, universities and members of the public. Coordinators in each of Australia's States and Territories report monthly on significant wildlife cases identified in their jurisdictions. NOTE: access to information contained within the National Wildlife Health Information System dataset is by application. See the WHA website for more information https://wildlifehealthaustralia.com.au/ProgramsProjects/eWHIS-WildlifeHealthInformationSystem.aspx.

Cryptosporidium spp. infection in animals in Australia is not a notifiable disease.

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To provide feedback on this fact sheet

We are interested in hearing from anyone with information on this condition in Australia, including laboratory reports, historical datasets or survey results that could be added to the National Wildlife Health Information System. If you can help, please contact us at admin@wildlifehealthaustralia.com.au.

Wildlife Health Australia would be grateful for any feedback on this fact sheet. Please provide detailed comments or suggestions to admin@wildlifehealthaustralia.com.au. We would also like to hear from you if you have a particular area of expertise and would like to produce a fact sheet (or sheets) for WHA (or update current sheets). A small amount of funding is available to facilitate this.

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