Toxoplasma and *Cryptosporidium* in the food supply

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Abbreviations and definitions

Anthroponosis – is a disease spread from human to human with no animal vector. It can also mean a disease spread from humans to animals. The adjective is anthroponotic.

Burden of Disease – is a measure[1] used to assess and compare the relative impact of different diseases and injuries on populations. It quantifies health loss due to disease and injury that remains after treatment, rehabilitation or prevention efforts of the health system and society generally. One measure of burden of disease is disability-adjusted life years or DALYs (see below).

Cryptosporidiosis – is a diarrhoeal disease caused by protozoa of the genus *Cryptosporidium*. Human infection is predominantly caused by the species *C. hominis* and *C. parvum*. The incubation period ranges from 2 to 12 days (usually 7–10 days), and symptoms typically last for 1–2 weeks in otherwise healthy individuals. More severe and prolonged illness is seen in patients with HIV/AIDS, in otherwise immunocompromised patients and in young children in developing countries (Ethelberg et al., 2009).

DALY – Disability Adjusted Life Years: One DALY[2] can be thought of as one lost year of "healthy" life. The sum of these DALYs across the population, or the burden of disease, can be thought of as a measurement of the gap between current health status and an ideal health situation where the entire population lives to an advanced age, free of disease and disability. DALYs for a disease or health condition are calculated as the sum of the Years of Life Lost (YLL) due to premature mortality in the population and the Years Lost due to Disability (YLD) for people living with the health condition or its consequences:

COI – Cost of Illness is defined[3] as the value of the resources that are expended or foregone as a result of a health problem. The COI includes health sector costs, the value of lost productivity by the patient (indirect cost), and the cost of pain and suffering (intangible costs). To make informed choices concerning which health problems to address and what interventions to use to alleviate them, decision makers need to know the economic burden imposed by the various health problems. The COI provides a monetary estimate for the economic burden of diseases.

Oocysts – are the environmentally robust infectious stages of *Cryptosporidium* and *Toxoplasma*. They have pronounced resistance to most chemical disinfectants, including chlorine[4].

Parasite – is an organism that lives on or in a host organism and gets its food from or at the expense of its host[5].

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[1] http://www.aihw.gov.au/burden-of-disease/
[2] http://www.who.int/healthinfo/global_burden_disease/metrics_daly/en/
[3] http://www.cdc.gov/owcd/eet/Cost/fixed/3.html
[4] http://www.fda.gov/downloads/Food/FoodSafety/FoodborneIllness/FoodborneIllnessFoodbornePathogensNaturalToxins/BadBugBook/UCM297627.pdf
[5] http://www.cdc.gov/parasites/about.html

Protozoa – are microscopic, one-celled organisms that can be free-living or parasitic in nature[6]. They are able to multiply in humans, which contributes to their survival and also permits serious infections to develop from just a single organism. Transmission of protozoa that live in a human intestine to another human typically occurs through a faecal-oral route (for example, contaminated food or water or person-to-person contact). Protozoa that live in the blood or tissue of humans are transmitted to other humans by an arthropod vector (for example, through the bite of a mosquito or sand fly).

QALY – Quality Adjusted Life Years: is a measure of the state of health of a person or group in which the benefits, in terms of length of life, are adjusted to reflect the quality of life. One QALY[7] is equal to 1 year of life in perfect health.

Toxoplasma gondii[8] – is a protozoan parasite that lives inside animal cells. It occasionally causes serious illness in immunocompromised individuals and unborn babies. It is estimated that one third of all humans have been exposed to *T. gondii*, but most instances of toxoplasmosis are mild or asymptomatic. Humans can contract foodborne toxoplasmosis from eating raw/undercooked meat containing parasitic cysts or from consuming food contaminated by infected cat faeces. T. gondii has 3 infective life stages:

> **Tachyzoites** disseminate within the animal. They are able to invade and multiply rapidly in virtually all vertebrate cell types including: brain, heart, lung, eye, muscles, placenta etc.

> **Bradyzoites** result from the conversion of tachyzoites into a slow-dividing stage that forms tissue cysts. Bradyzoites have a latent metabolism, well adapted to long-term survival. The resistance of bradyzoites to digestion (1 to 2 hour survival in acid pepsin) allows their transmission through ingestion.

> **Sporozoites** are an environmental stage and are protected within an oocyst. The oocyst wall is an extremely robust multilayer structure protecting the parasite from mechanical and chemical damage. It enables the parasite to survive for long periods, more than a year, in a moist environment. Oocysts are excreted in faeces by felids. They mature and become infectious after sporulation which occurs a few days after excretion.

Zoonosis – a disease which is communicable to humans from another animal species. The adjective is zoonotic.

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[6] http://www.cdc.gov/parasites/about.html
[7] http://www.nice.org.uk/website/glossary/glossary.jsp?alpha=Q
[8] http://www.foodsafety.govt.nz/elibrary/industry/Toxoplasma_Gondii-Science_Research.pdf

Executive summary

This survey was conducted to determine whether current interventions and advisory materials in place regarding Toxoplasmosis and Cryptosporidium are appropriate in light of current international knowledge.

Toxoplasmosis is caused by infection with the protozoan parasite *Toxoplasma gondii*. Infections are usually either asymptomatic or self-limited but infection in immunosuppressed persons can be severe. Infections in pregnant women can cause serious health problems in the child such as mental retardation, blindness, and epilepsy. Infection with *T. gondii* in adults with healthy immune systems can lead to impaired eye sight.

Toxoplasmosis has ranked very highly in two studies of death and disability attributable to foodborne pathogens. Significant amounts of illness are due to consumption of raw or undercooked meat containing *T. gondii* tissue cysts and consumption of raw vegetables or water with *T. gondii* oocysts from cat faeces. The risk of acquiring a *Toxoplasma* infection via food varies with cultural and eating habits in different human populations.

Australian data for a range of foodborne pathogens was extracted by Kirk et al. (In press). They calculated there were 30 hospitalisations and 1 death due to toxoplasmosis annually (circa 2010). These figures are based on hospitalisation data from state and territory health departments and deaths data from the Australian Bureau of Statistics.

A multi-centre European study identified these risks: raw sausage eaten at least once a week; dry cured meat eaten more than once a week; salami eaten more than once a week; raw/undercooked beef; raw/undercooked lamb; other raw/undercooked meats (e.g. venison, horse); tasting meat when cooking; unpasteurised milk; untreated water; contact with soil; working with animals and travel outside of Europe/US or Canada.

An American study confirmed the significance of a number of factors previously linked with *T. gondii* infection, such as eating undercooked meat and drinking unpasteurised goats milk. It also recognised eating raw oysters, clams or mussels as new risk factors.

Expert groups have drafted guidance that will reduce illness. The focus has been on women of childbearing age to prevent congenital toxoplasmosis however, the threat of ocular disease means that the guidance has universal importance.

Cryptosporidium species have been found to infect mammals, birds, reptiles, amphibians and fish. The two species that most commonly infect humans are *Cryptosporidium hominis* and *C. parvum*, and while the former species seems to be primarily limited to humans, the latter has a wide range of hosts, including most major domestic livestock animal species. In humans, cryptosporidiosis mainly involves infection of the gut, resulting in watery diarrhoea lasting up to 2 weeks with the potential for recurrence.

In immune-deficient and immunosuppressed individuals the infection may not resolve and may involve the bile and pancreatic ducts, stomach and lungs. The persistent diarrhoea and malabsorption can become life-threatening, particularly in acquired immunodeficiency syndrome patients.

The disease is primarily related to exposure to water that contains *Cryptosporidium* oocysts or with direct contact with faecal material from infectious young animals. Food contamination is a less significant exposure but can occur from: contaminated water, direct contact with manure, contaminated shellfish or from contamination during food preparation. Kirk et al. (in press) estimate there are 17,900 case of cryptosporidiosis Australia per year (circa 2010), with about 10% or 1700 cases being foodborne.

Again experts have designed a comprehensive set in interventions for use on farm, in recreational water situations and for drinking water protection. High levels of personal hygiene are also required to overcome the highly infectious nature of the parasite.

Introduction

A key results area for the NSW Food Authority (the Food Authority) is that safe food is produced and sold in NSW. The Food Authority has a strategy to identify and investigate contributors to foodborne illness with the aim of reducing foodborne illness in the community. This report is about two parasitic diseases that are in-part foodborne and cause considerable amounts of illness worldwide.

Toxoplasma gondii was recently ranked very highly in burden of disease estimates for foodborne pathogens in the Netherlands and the USA (Havelaar et al., 2012) (Hoffmann, Batz, & Morris Jr, 2012). The high burden of disease estimate results from very serious illnesses that are reported in a small number of exposed people. Congenital illness, resulting from infection of the unborn baby, can result in life-long disability. Infections in immune-compromised people can be severe. Eye problems can result from toxoplasmosis acquired later in life.

Several members of the *Cryptosporidium* genus, but particularly *C. hominis* and *C. parvum* cause diarrhoeal illness in humans. They can also result in very serious illness in immune-compromised people. In the general community, young children and their carers are more prone to infection.

This report is a brief overview of what is known about the parasites and the diseases that they cause. It was used to check that the interventions and advisory materials we have in place are appropriate in light of current knowledge.

Toxoplasma gondii

Toxoplasmosis is caused by infection with the protozoan parasite *Toxoplasma gondii*. In the United States, an estimated 23% of adolescents and adults have laboratory evidence of infection with *T. gondii*. Although these infections are usually either asymptomatic (i.e. shows no visible symptoms) or associated with self-limited symptoms (e.g., fever, malaise, and lymphadenopathy), infection in immunosuppressed persons (e.g., persons with acquired immunodeficiency syndrome [AIDS]) can be severe. In addition, infections in pregnant women can cause serious health problems in the foetus if the parasites are transmitted (i.e., congenital toxoplasmosis) and cause severe sequelae in the infant (e.g., mental retardation, blindness, and epilepsy) (Lopez, Dietz, Wilson, Navin, & Jones, 2000).

Infection with *T. gondii* is increasingly being recognised as a problem in non-pregnant, adults with healthy immune systems, where acute infection may lead to impaired eye sight (Petersen, Vesco, Villari, & Buffolano, 2010).

Toxoplasmosis can be transmitted to humans by three main routes. First, humans can eat raw or inadequately cooked infected meat or eat uncooked foods that have come in contact with contaminated meat. Second, humans can inadvertently ingest oocysts that cats have passed in their faeces, either in a cat litter box or outdoors in soil (e.g. soil from gardening or unwashed fruits or vegetables). Third, a woman can transmit the infection to her unborn foetus (Lopez, et al., 2000).

Water is increasingly being investigated as a risk factor and has been demonstrated to be an important source of infection in tropical and subtropical countries, where surface water may be used for human consumption without any purification, but has also been found to be a risk factor in parts of Europe. The largest and best documented outbreak of acute toxoplasmosis in humans occurred in 110 individuals on Vancouver Island, Canada, in 1995. Comprehensive, retrospective epidemiological studies provided strong evidence that this outbreak was caused by contamination of municipal drinking water with oocysts (Petersen, et al., 2010).

Toxoplasmosis has assumed particular interest since it ranked very highly in two studies of death and disability attributable to foodborne pathogens. (Havelaar et al. (2012) found that *T. gondii* (congenital and acquired cases) caused the highest disease burden as measured by Disability Adjusted Life Years (DALY per year 2009). This fell to second place when an economic discount factor was applied because the disability was very long term. The DALY per patient for congenital disease was second to perinatal *Listeria monocytogenes*. Approximately 45% of the total burden (for all the pathogens studied) was attributed to food.

Hoffman et al. (2012) ranked *T. gondii* second when measured by estimated annual cost of illness and third in terms of losses of Quality Adjusted Life Years (QALY loss). The rankings used figures from a previous paper in which toxoplasmosis was estimated to be 50% foodborne (Scallan et al., 2011).

Despite the similarities rankings there are some large differences in the estimates for congenital and acquired/adult toxoplasmosis.

Table 1: *T. gondii* burden of illness estimates: Netherlands and USA

	DALY Netherlands 2009 not discounted	Estimated cost of illness US $mil 2009	Mean QALY loss USA
Campylobacter spp	3250	1747	13,256
Salmonella spp – non-typhoidal	1270	3309	16,782
Norovirus	1480	2002	5027
T. gondii (congenital)	2270	122	1642
T. gondii (acquired / adult)	1350	2852	9323
Reference	(Havelaar, et al., 2012)	(Hoffmann, et al., 2012)	

While the papers agree on the significance of toxoplasmosis in total they differ on the contribution of congenital and acquired. Deaths in congenital cases are estimated to be 13 in the Netherlands and 15 in the USA. Deaths in acquired or adult cases are estimated to be nil in the Netherlands and 327 in the USA. Severe cases, requiring hospitalisation, of adult toxoplasmosis were estimated to be 4,428 in the USA.

Australian data for a range of foodborne pathogens was extracted by Kirk et al. (In press). They calculated there were 30 hospitalisations and 1 death due to toxoplasmosis annually (circa 2010). These figures are based on hospitalisation data from state and territory health departments and deaths data from the Australian Bureau of Statistics. A multiplier of 2 was used to account for under reporting, just as it was in the US estimates. The differences between US, Dutch and Australian hospitalisation and death figures are hard to reconcile.

Petersen et al. (2010) note that the risk of acquiring a *Toxoplasma* infection via food varies with cultural and eating habits in different human populations. Even so the differences between three western countries are pronounced. One possible problem with the US estimates is the assumption that 15% of those that sero-convert will become symptomatic. Jones and Holland (2010), reporting on ocular toxoplasmosis, estimate that 2% of infected people develop ocular lesions but only 0.2–0.7% develop symptomatic retinitis.

How do humans become infected with *T. gondii*?

Petersen et al. (2010) note that even though toxoplasmosis is the most prevalent parasitic zoonosis in humans, the system for routine monitoring and reporting is inadequate and there are still many unanswered questions about modes of transmission to humans. Risk factor studies are needed that more accurately identify common sources of infections, including the environment.

Figure 1 shows sources of *T. gondii* infection in humans. The role of waterborne contamination is not as evident in the graphic as it could be. Unfiltered surface waters have caused large outbreaks of toxoplasmosis.

Figure 1: Sources of *T. gondii* infection in humans (Robert-Gangneux & Dardé, 2012)
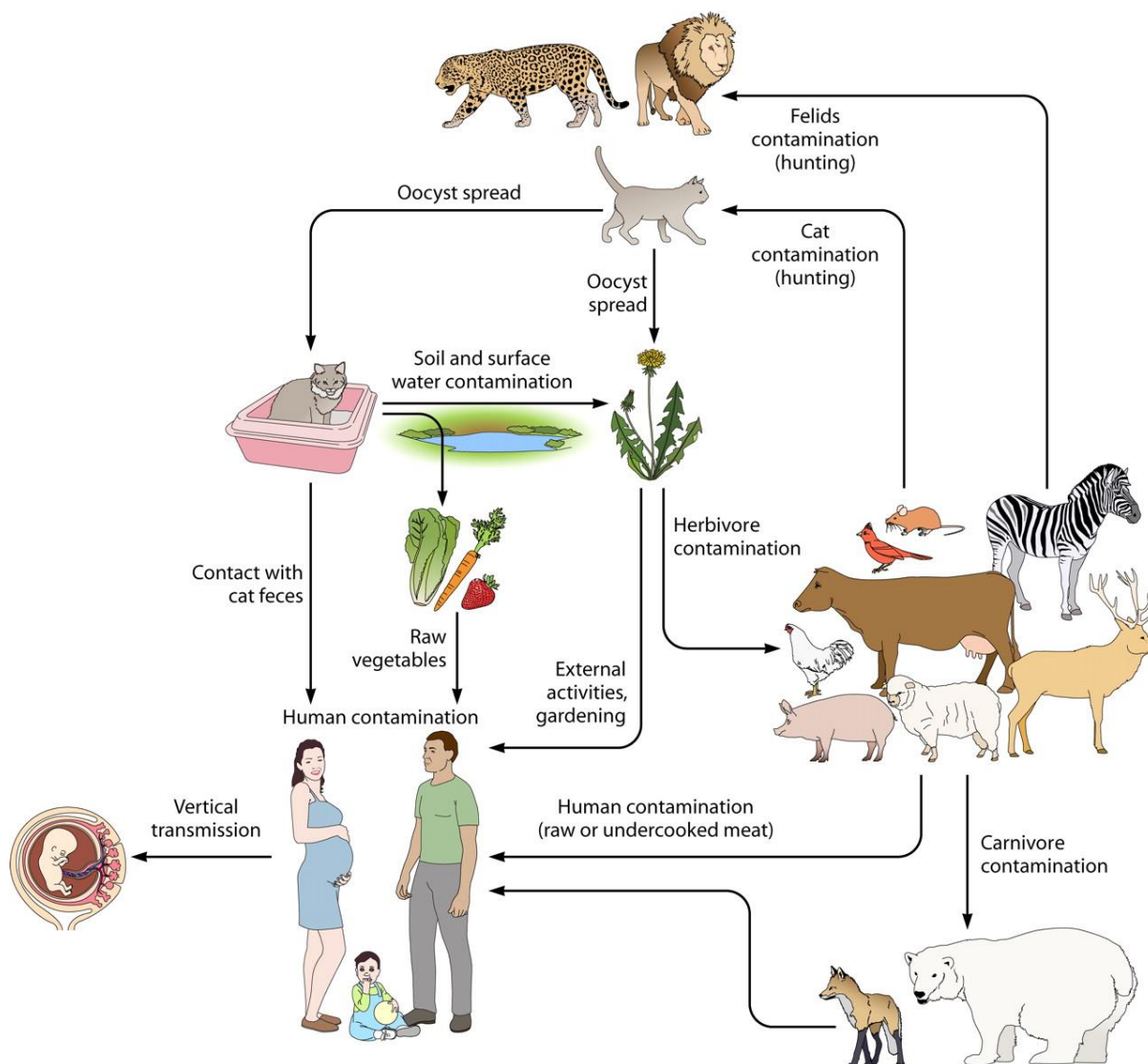
A number of studies have tried to determine which paths of infection of humans are most important. A number of authors (Petersen, et al., 2010) (Robert-Gangneux & Dardé, 2012) note difficulties with these studies. They tend to be small, focussed on women of childbearing age and risk factors for infection remain unexplained in a significant number of cases. The results are only applicable to the community where the studies were completed. Even given the weaknesses, European and US studies are likely to be relevant in Australia.

Cook et al (2000) reported a multi-centre European study of 252 infected women and 708 controls. They identified the following risk factors: raw sausage eaten at least once a week; dry cured meat eaten more than once a week; salami eaten more than once a week; raw/undercooked beef; raw/undercooked lamb; other raw/undercooked meats (venison,

horse, rabbit, whale and game birds); taste meat when cooking; unpasteurised milk; untreated water; contact with soil; working with animals and travel outside of Europe/US or Canada. In this and most other studies, contact with cats in the home was not identified as a risk. Raw/undercooked pork was not identified as a statistically significant risk, but several authors note this might change as animal friendly (organic, free range) approaches to animal husbandry become more common. Between 30% and 63% of infections in the different centres could be attributed to meat consumption, but the type of meat differed. Eating lamb and 'other meat' was more important in northern and central European centres than in Italy. The proportion of infections attributed to eating salami was 10% to 14% in Milan, Naples, and Brussels and 3-5% elsewhere. In Lausanne 14% of infections were attributed to consumption of unpasteurised milk or milk products, whereas elsewhere it was 5% or less. In all centres a large proportion of infection (14% to 49%) remained unexplained by the exposures studied.

An American study (J. L. Jones et al., 2009) looked at 148 infected cases and 413 controls. This study confirmed the significance of a number of factors that had previously been linked with *T. gondii* infection, such as eating undercooked meat and drinking unpasteurised goats milk. It also recognised eating raw oysters, clams or mussels as new risk factors. They also noted that exposure to more than 3 kittens increased the risk and the authors noted that this might reflect exposure to a litter of kittens. They were not able to explain the risk for 48% of the infections in their study.

Commenting on 16 case-control studies Petersen et al. (2010) note the studies so far fail to explain the high frequency of seropositivity (24–47%) in some populations of vegetarians. The role of water that may contain infective *Toxoplasma* oocysts and thereby contaminate fruit or vegetables during growth and production of animals such as swine and sheep is important. Data on the frequency, severity and duration of symptoms of human toxoplasmosis are crucial to improve the determination of the burden of the disease which could lead to more adequate prevention strategies.

Toxoplasma interventions

Participants in the National Workshop on Toxoplasmosis: Preventing Congenital Toxoplasmosis (Lopez, et al., 2000) developed the following recommendations:

- To prevent toxoplasmosis and other foodborne illnesses, food should be cooked to safe temperatures. A food thermometer should be used to measure the internal temperature of cooked meat to ensure that meat is cooked all the way through. Beef, lamb, and veal roasts and steaks should be cooked to at least 145 F (63°C), and pork, ground meat, and wild game should be cooked to 160 F (71°C) before eating. Whole poultry should be cooked to 180 F (82°C) in the thigh to ensure doneness.
- Fruits and vegetables should be peeled or thoroughly washed before eating.
- Cutting boards, dishes, counters, utensils, and hands should always be washed with hot soapy water after they have contacted raw meat, poultry, seafood, or unwashed fruits or vegetables.
- Pregnant women should wear gloves when gardening and during any contact with soil or sand because cat waste might be in soil or sand. After gardening or contact with soil or sand, wash hands thoroughly.

- Pregnant women should avoid changing cat litter if possible. If no one else is available to change the cat litter, use gloves, then wash hands thoroughly. Change the litter box daily because *Toxoplasma* oocysts require several days to become infectious. Pregnant women should be encouraged to keep their cats inside and not adopt or handle stray cats. Cats should be fed only canned or dried commercial food or well-cooked table food, not raw or undercooked meats.
- Health education for women of childbearing age should include information about meat-related and soil borne toxoplasmosis prevention. Health-care providers should educate pregnant women at their first prenatal visit about food hygiene and prevention of exposure to cat faeces.
- Health-care providers who care for pregnant women should be educated about two potential problems associated with *Toxoplasma* serology tests. First, no assay exists that can determine precisely when initial *Toxoplasma* infection occurred. Second, in populations with a low incidence of *Toxoplasma* infection, such as in the United States, a substantial proportion of the positive IgM test results will probably be false positive.
- The government and the meat industry should continue efforts to reduce *Toxoplasma* in meat.

Robert-Gangneux et al. (2012) compiled a more recent set of hygiene measures.

Table 2: Basis for hygienic measures for prevention of toxoplasmosis

Source of infection	Type of risk	Prevention measure(s)
Oocysts and cat faeces	Direct contact with cat faeces; oocysts become infectious only 2–3 days after shedding, shedding usually occurs only once during a cat's life, duration of shedding is about 2 weeks, oocysts are killed within 1–2 min by heating to 55°C–60°C, and oocysts are resistant to chemical disinfectants such as sodium hypochlorite	Wash hands carefully after stroking a cat, wear gloves when changing cat litter, change the litter frequently and wash tray with hot water (>60°C), avoid putting cat litter in the kitchen, and feed cats dried or canned food
Oocysts in the environment	Soil contact for gardening, playing, or other outdoor activities; oocysts can survive more than 1 year in a moist environment at 4°C, 106 days at −10°C, 32 days at 35°C, and 9 days at 40°C	Wash hands thoroughly and brush nails after any outdoor activities in contact with soil, and wear gloves for gardening
	Consumption of unfiltered water (raw surface water, reservoir, wells, recreation areas)	Prefer mineral water to tap water (in countries where the water network is supplied mainly by surface water)

Source of infection	Type of risk	Prevention measure(s)
	Oocysts can survive for long periods of time in water and resist freezing and moderately high water temperatures, chlorination and ozone treatment, in seawater, and in various species of shellfish	Avoid raw oysters, clams, and mussels, and avoid occasional ingestion of water (lakes, rivers) during recreation activities
	Raw vegetables or fruit consumption	Thoroughly wash vegetables, fruits, and herbs eaten raw, especially if they grow close to the ground, and avoid raw vegetables at restaurants
Tissue cysts in meat	Meat consumption or manipulation; any type of meat may be infected, with sheep, goats, and pigs from organic outdoor production systems and wild game being at the most risk; cysts are killed immediately at 67°C and after at least 3 days at <−12°C, depending on the thickness of the piece of meat; cysts can survive in refrigerated meat for up to 3 weeks, for >11 days at −6.7°C, and for about 4 min at 60°C and 10 min at 50°C	Cook the meat well done (oven, pan) or stew; avoid microwave cooking; eat meat frozen for at least −20°C for at least 15 days; and wash hands, knives, any containers, and table thoroughly after meat manipulation or cutting

The *Food Safety During Pregnancy* brochure on the NSW Food Authority Website[9] addresses many of these issues. However, infection with *T. gondii* is increasingly being recognised as a problem in non-pregnant adults with healthy immune systems, where acute infection may lead to impaired eye sight. Therefore, the focus of research and education must be expanded from infection during pregnancy to human infections in general (Petersen, et al., 2010).

Recommendations developed by the National Workshop on Toxoplasmosis: Preventing Congenital Toxoplasmosis (Lopez, et al., 2000) include 'the government and the meat industry should continue efforts to reduce *Toxoplasma* in meat'. The observed decline in *Toxoplasma* seroprevalence as noted in many developed countries over past decades has been attributed to the introduction of modern farming systems resulting in a lower prevalence of *Toxoplasma* cysts in meat in combination with an increased use of frozen meat by consumers. Tactics to reduce prevalence in farm animals include cat control, rodent control, indoor production systems, decontamination of animal feed and bedding and

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[9] http://www.foodauthority.nsw.gov.au/_Documents/consumer_pdf/pregnancy-brochure.pdf

vaccination against cyst formation. Public demand for animal friendly production systems may lead to a re-emergence of *T. gondii* in pork and poultry (Kijlstra & Jongert, 2008).

Cryptosporidium

Members of the genus *Cryptosporidium* are parasitic protozoa capable of infecting the digestive or respiratory tracts of humans and most other animals. These parasites were not thought to cause disease, and received little attention from researchers until the 1950s when the first case of cryptosporidiosis, a disease leading to mortality in poultry, was reported. In the early 197Os, the interest among veterinarians in this organism increased, as the first case of *C. parvum* associated with bovine diarrhoea was reported. Between 1961 and 1986, cases of cryptosporidiosis were also reported in fish, reptiles, birds and mammals. *Cryptosporidium parvum* is now recognised as an important cause of diarrhoea in calves and other neonatal mammals, and *Cryptosporidium baileyi*, as an important cause of respiratory disease in poultry (Laberge, Griffiths, & Griffiths, 1996).

The first cases of human cryptosporidiosis were reported in 1976. Since the beginning of the 198Os, this disease has been diagnosed increasingly often in immune-competent persons with short-term diarrhoeal illness and in immunocompromised patients, especially those with the acquired immune deficiency syndrome (AIDS), with prolonged, life-threatening, cholera-like illness. The increased identification of cryptosporidiosis in immunocompromised patients stimulated the awareness of the medical community toward this protozoon, leading to more research. *Cryptosporidium parvum* is considered a major gastroenteric pathogen world-wide (Laberge, et al., 1996).

Cryptosporidium species have been found to infect mammals, birds, reptiles, amphibians and fish. The two species that most commonly infect humans are *Cryptosporidium hominis* and *C. parvum*, and while the former species seems to be primarily limited to humans, the latter has a wide range of hosts, including most major domestic livestock animal species. In humans, cryptosporidiosis mainly involves infection of the gut, resulting in a watery diarrhoea lasting up to 2 weeks with the potential for recurrence (Leitch & He, 2012).

In immunodeficient and immunosuppressed individuals the infection may not resolve and may involve the bile and pancreatic ducts, stomach and lungs. The persistent diarrhoea and malabsorption can become life-threatening, particularly in acquired immunodeficiency syndrome (AIDS) patients.

The major features of the life cycle of *C. parvum* or *C. hominis* are that it begins with the ingestion of fully sporulated, environmentally resistant oocysts. This fully sporulated thick-walled form is resistant to prolonged environmental exposure in various water sources, and is also resistant to many commonly used disinfecting agents, including dilute bleach. *Cryptosporidium* oocysts, unlike *Toxoplasma* oocysts, are fully infectious when they are excreted in faeces.

Cryptosporidium infections may be zoonotic (see Figure 2) or anthroponotic (see Figure 3). Most of the major outbreaks of cryptosporidiosis have been attributed to contaminated drinking water, but oocysts have been recovered from food, such as fresh vegetables and seafood, and person-to-person transmission may also be possible (Leitch & He, 2012).

![NSW Government Food Authority logo]

Figure 2: *Cryptosporidium* - zoonotic routes of infection
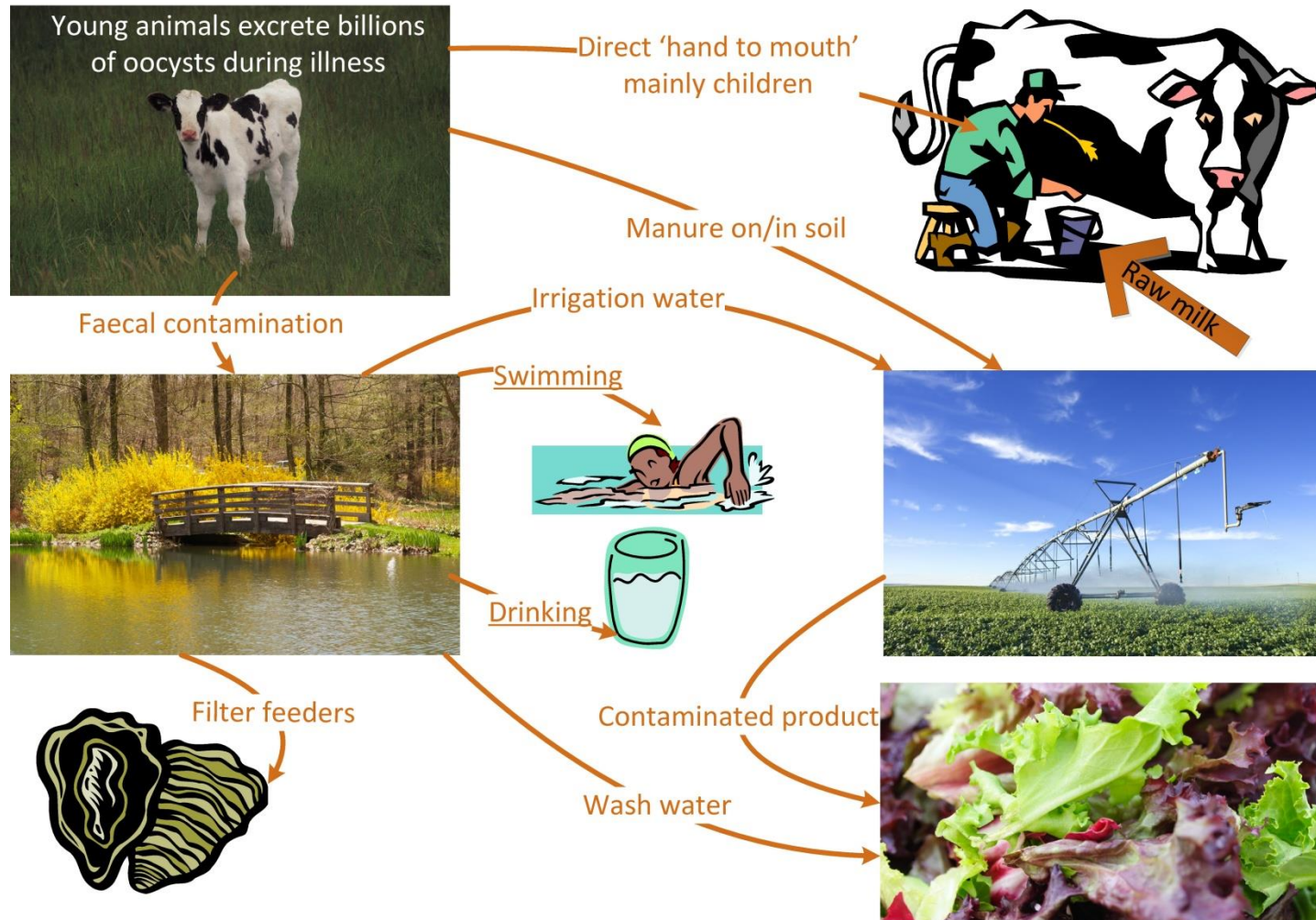
**Figure 3: *Cryptosporidium anthroponotic* routes of infection**

Swimming with untrained todlers

Child in nappies – worse with diarrhoea

Children in childcare

Water supply

Treatment plant

Infected food service worker

International travel

Person in the home with diarrhoea
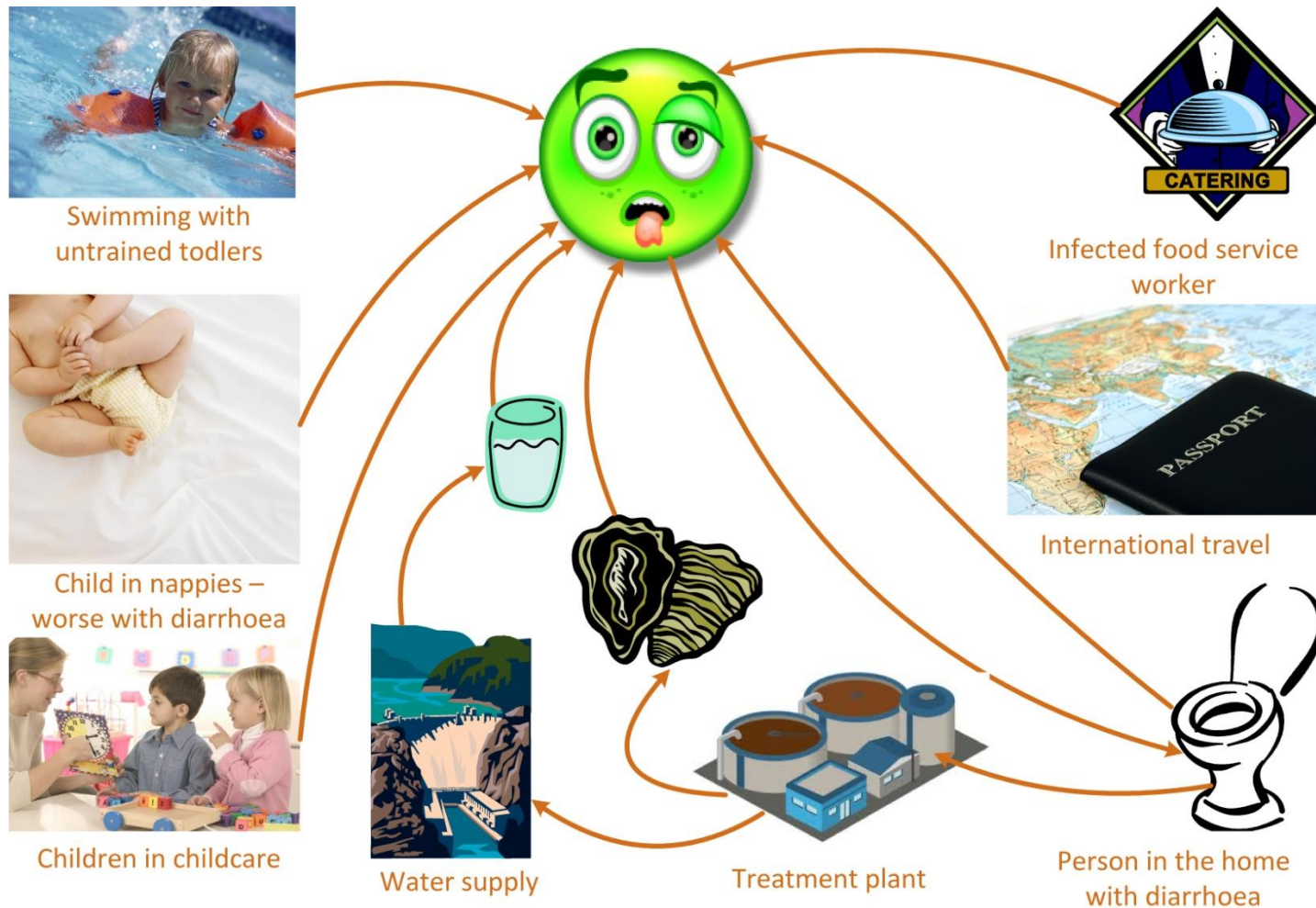
The largest cryptosporidiosis outbreak reported to date in the USA occurred in Milwaukee, Wisconsin in 1993 when over 403,000 individuals were sickened out of a potentially exposed population of 1.6 million. Long term, the number of deaths resulting from cryptosporidiosis approximated 54, mostly AIDS patients. Cost estimates of the economic impact of the Milwaukee outbreak were US$96 million in 1993 dollars for treatment and lost productivity costs.

Outbreaks of cryptosporidiosis have also been linked to contaminated recreational water, such as parks and swimming areas. In most human cryptosporidiosis studies, *C. hominis* is the major causative species. Individuals, particularly children, in rural areas may have a higher prevalence rate of *C. parvum* infections than children in urban areas where *C. hominis* predominates and there is a greater *Cryptosporidium* species diversity in rural areas, in keeping with greater exposure to livestock and other animals.

In spite of the advances that have been made in the study of cryptosporidiosis, there is not full agreement on the taxonomy of the various *Cryptosporidium* species. Nevertheless, it appears that *C. hominis* is primarily limited to humans and its transmission is therefore usually anthroponotic, while transmission of *C. parvum* found in many mammals, particularly livestock, is usually zoonotic (Leitch & He, 2012).

Joachim (2004) used Figure 4 to describe the understanding at the time. Full lines represent regular transmission while dotted lines represent rare transmission. Only genotypes and species diagnosed in humans to the date manuscript preparation are listed.

Figure 4: Postulated transmission cycles of Cryptosporidium involving humans.
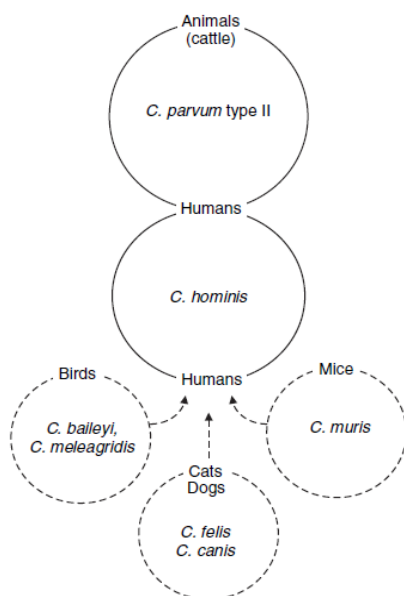
Leith and He (2012) added 3 species reported to cause isolated human cases to Joachim's list. *C. baileyi*, which Joachim reported as a rare human pathogen, was not classified as rare in Leith and He's list. The understanding of *Cryptosporidium* is still changing.

How do humans become infected with *Cryptosporidium*

Menzies (2002) reported on cryptosporidiosis in NSW from 1990-2000. During this period the epidemiology of cryptosporidiosis in NSW was dominated by epidemics every 3–4 years, with little seasonal pattern in between. This pattern extended beyond NSW to other parts of Australia. There were three epidemics during that time: 1991, 1994–1995, and 1997–1998. The 1991 epidemic was simultaneously documented in Adelaide and Sydney. A study in Adelaide at the time found a protective effect from drinking only rain water. For the epidemic in 1994–95, the peak was recorded in Sydney, Melbourne and Brisbane. In Southern Sydney, infection was associated with swimming in one public swimming pool. During the 1997–98 epidemic, cases were documented in Sydney, Canberra, Melbourne, and Brisbane; and in NSW infection was associated with swimming in public pools, rivers or lakes, and with not drinking bottled water. In these epidemics, most cases were young children. Data since 1997 show that those living in non-metropolitan areas had a higher rate of infection. Cryptosporidiosis resulted in 239 recorded hospitalisations in NSW over a four-year period, and those hospitalised had a similar age and geographic distribution to the cases notified by laboratories.

In July–September 1998 a series of 'boil water' alerts were made to Sydney residents, following the detection of high levels of *Cryptosporidium parvum* and *Giardia lamblia* in samples of drinking water. These alerts occurred shortly after the end of the epidemic of 1997–98 and that there was no detectable rise in notifications. A household survey also found no measurable increase in illness attributable to drinking Sydney water at that time.

Majowicz et al (2001) reported on endemic illness (sporadic cases of illness with outbreaks excluded) in Ontario and reported probable sources of infection. Overall they identified 487 cases and excluded 36 cases outbreak related cases. Of the remaining endemic cases a probable source of infection was notified for 157 cases. Children under 5 have a very much higher rate of infection than older groups. People living in rural areas have much higher rates of infection than those living in urban areas.

Table 3. Probable sources of infection for endemic cryptosporidiosis reported in Ontario 1996-1997

Probable source	Number	Per cent
Water – lake stream river pond	41	26.1
Livestock	33	21.0
Person-to-person	23	14.7
Water – private well	15	9.6
Food	13	8.3
Pets	13	8.3
Water – municipal unfiltered	8	5.1

Probable source	Number	Per cent
Water – pool spa	6	3.8
Water – municipal filtered	4	2.5
Water - bottled	1	0.6
Total	157	

Robertson et al (2002) carried out case-control studies assessing risk factors for sporadic disease in Melbourne and Adelaide, which have water supplies from different ends of the raw water spectrum. In addition to examining drinking water, they assessed several other exposures. Two hundred and one cases and 795 controls were recruited for Melbourne and 134 cases and 536 controls were recruited for Adelaide. Risk factors were similar for the two cities, with swimming in public pools and contact with a person with diarrhoea being most important. The consumption of plain tap water was not found to be associated with disease.

For Melbourne, crude analysis identified statistically significant risk factors associated with sporadic cryptosporidiosis including swimming in public pools (both toddler's and adult's), immune system impairment, having children at home in nappies, having contact with children or adults with diarrhoea and having children at home attending childcare. Other risk factors included calf and lamb contact away from home, the consumption of unboiled water from a river, lake or dam within rural Australia, overseas travel and the consumption of unboiled water, ice-cubes or salad overseas.

For Adelaide, crude analysis identified statistically significant risk factors associated with sporadic cryptosporidiosis including the consumption of unpasteurised milk products, swimming in a public toddlers' pool, immune system impairment, having children at home in nappies, having contact with persons with diarrhoea and having children at home attending childcare. Other risk factors included the consumption of unboiled water from a river, lake or dam within rural Australia, overseas travel and the consumption of unboiled water, ice-cubes or salad overseas.

Roy et al (2004) studied risk factors for sporadic (not outbreak related) cryptosporidiosis among persons with healthy immune systems in the US from 1999 to 2001. The authors found significant risk for persons in contact with children >2 to 11 years of age with diarrhoea. In contrast, they found no significant risk of transmission from contact with older children and adults. This suggests that the risk of transmission is influenced by the age of the index case. Young age is probably a surrogate for inadequate hygiene, faecal incontinence, and the need for more assistance during illness.

Contact with cattle was a risk factor for cryptosporidiosis, whereas contacts with numerous other animals, including dogs, cats, sheep, goats, pigs, horses, reptiles, and birds, showed no significant associations. The transmission of cryptosporidia from calves to humans has been well established, with documented outbreaks among veterinary hospital staff and visitors to farms. An estimated 15 to 56% of dairy calves shed cryptosporidia and perhaps >90% of dairy farms in the United States may have *Cryptosporidium* spp. on their premises.

Travel is a recognized risk factor for cryptosporidiosis. Although domestic travel was not a risk factor in their study, international travel was highly associated with cryptosporidiosis.

Swimming in freshwater was a risk for sporadic disease in this study, unlike swimming in pools and water parks. This finding differs from surveillance data. From 1991 through 2000, 90% of recreational water cryptosporidiosis outbreaks reported to the CDC were associated with swimming pools and water parks, whereas only 10% were associated with freshwater venues. The authors speculate that the transmission patterns in chlorinated recreational water venues favour outbreaks over sporadic cases. High bather densities with routine use of recreational waters by incontinent persons, including children in nappies and toddlers, coupled with oocyst resistance to chlorine, a low infectious dose, and immediate release of potentially large numbers of oocysts from a single faecal accident into small volumes of water relative to lakes and oceans likely facilitate outbreaks in public pools and water parks.

Interestingly they found that raw vegetable consumption was a protective factor, seemingly contradicting much of the available data on food-borne transmission of this disease. CDC's Foodborne Disease Outbreak Surveillance System documented eight food-borne-related cryptosporidiosis outbreaks from 1990 through 2000. One explanation may be that regular consumption of oocyst-contaminated vegetables results in immunity to illness from *Cryptosporidium* infection. Serologic responses have been shown to develop after both symptomatic and asymptomatic infections. Although pre-existing antibody responses may not protect against subsequent infection, they may protect against subsequent illness.

Snel et al (2009) studied the epidemiology of cryptosporidiosis in New Zealand 1997-2006. Notification rates showed large geographic variations, with rates in rural areas 2.8 times higher than in urban areas, and with rural areas also experiencing the most pronounced spring peak.

Figure 5. Cryptosporidiosis notification rate per 100,000 comparing urban and rural areas of New Zealand 1997-2006 (Snel, et al., 2009)
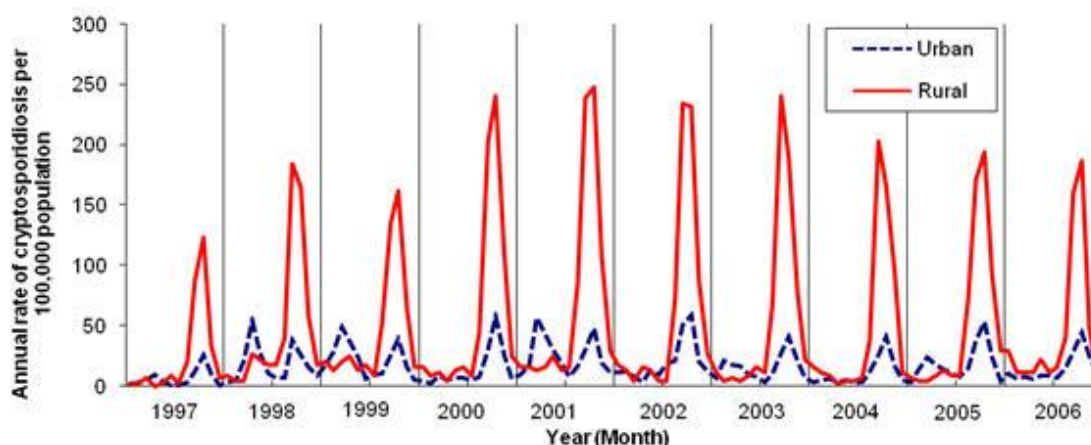
Possibly the most distinctive feature of cryptosporidiosis epidemiology in New Zealand is the remarkably high rate in rural areas, with a strong and consistent dose-response relationship with increasing levels of rurality. A positive correlation with farm animal density suggests that these animals may be reservoirs responsible for the high rates of disease in rural areas.

In the early part of the observation period (particularly 1998, 1999, and 2001), higher cryptosporidium rates were also seen during late summer/early autumn in urban areas. This spatio-temporal pattern would be consistent with anthroponotic transmission of *C. hominis* through contaminated swimming pools during the swimming season, as shown in previous studies. The publicity following a large cryptosporidiosis outbreak in 1998 may have contributed to improved regulations and filtration systems in public swimming pools resulting in the subsequent disappearance of this 'swimming pool peak' after 2001.

Table 4 shows the number and proportion of cases reporting specific exposures. Contact with farm animals was the most commonly reported risk factor (59.4%) followed by attending school or childcare (43.4%) and drinking or using untreated drinking water (38.7%).

Table 4. Cryptosporidiosis – self-reported risk factors (Snel, et al., 2009)

Exposure	Number reporting information	Positive exposure (%)
Farm animals	4479	59.4
School, childcare	6430	43.4
Untreated drinking water	3697	38.7
Recreational water	4471	32.7
Faecal matter, vomit	4064	30.2
Other symptomatic case	4103	26.9
Food premise	2793	26.0
Sick animals	3519	25.0
Confirmed case	4925	12.4
Overseas	6273	5.7

Yoder et al (2010) reported on cryptosporidiosis surveillance in the United States in the period 2006-2008. During 2006--2008, the number of reported cases of cryptosporidiosis increased dramatically (79.9%), from 6,479 for 2006 to 11,657 for 2007, and then decreased (9.9%) to 10,500 in 2008. Although cryptosporidiosis affects persons in all age groups, the number of reported cases was highest among children aged 1-9 years and adults aged 25-39 years (Figure 2). These data are consistent with reports of cryptosporidiosis incidence being higher among younger children and of transmission to their caregivers (e.g., child care staff, family members, and other household contacts).

The data (Figure 6) shows the number of cryptosporidiosis case reports by month. The tenfold increase in onset of cryptosporidiosis occurring during summer through early fall is not unexpected. This increase coincides with increased swimming activities during the summer recreational water season and likely reflects the contribution of recreational water venues in the transmission of *Cryptosporidium*, particularly among younger children. *Cryptosporidium* is the leading cause of reported recreational water-associated outbreaks of gastroenteritis. Transmission through recreational water is facilitated by the substantial number of *Cryptosporidium* oocysts that can be shed by a single person; the extended periods of time that oocysts can be shed; the low infectious dose; the tolerance of *Cryptosporidium* oocysts to chlorine; and the prevalence of improper pool maintenance (i.e., insufficient disinfection, filtration, and recirculation of water), particularly of children's

wading pools. This seasonal variation also has been noted in state, Canadian provincial and previous U.S. national surveillance data for cryptosporidiosis and giardiasis

Figure 6. Number of cryptosporidiosis case reports, by date of illness onset --- National Notifiable Disease Surveillance System, United States, 2006—2008 (Yoder, et al., 2010)
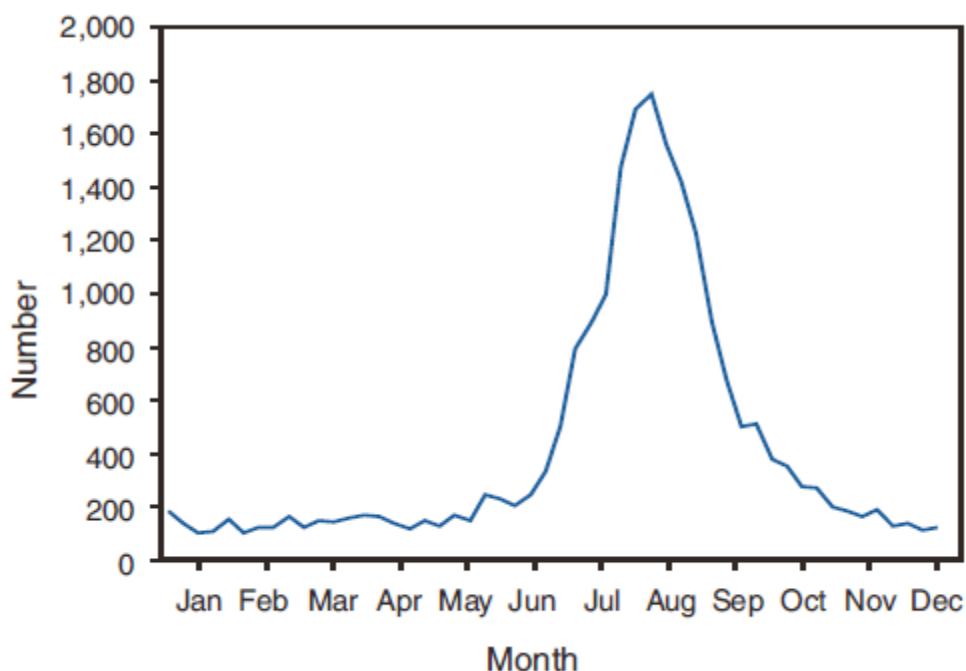
Waldron et al (2011) studied where and when sporadic cryptosporidiosis is found in NSW. Of the human faecal samples that were positive for Cryptosporidium, 66% were *C. hominis* and 33% were *C. parvum*. The dominant *C. hominis* subtype is a globally distributed subtype and is the most common cause of waterborne outbreaks and sporadic human cryptosporidiosis. The dominant *C. parvum* subtype was also the most common cause of cryptosporidiosis in cattle.

Waldron et al identified cryptosporidiosis hot spots in Sydney, Newcastle, Port Macquarie, Lismore, Wagga Wagga, Dubbo and Bathurst. Cryptosporidiosis infections in Dubbo, Wagga Wagga and the Sydney suburb of Richmond were dominated in spring by *C. parvum* which can probably be attributed to the calving season. They conclude that Cryptosporidium is widespread in NSW and has complex transmission pathways involving humans, cattle and Australian native wildlife.

National notifiable disease surveillance system (NNDSS) data

Cryptosporidiosis is a notifiable disease in all states and territories of Australia. The NNDSS data provides some useful insights into the nature of the disease. Figure 7 shows Australian cryptosporidiosis notifications by age for 2009 which was an epidemic year in the eastern states. Figure 8 shows data for 2010 which was not an epidemic year. The patterns are similar but the number of cases is markedly different. The two age-related peaks have been mentioned above. Infants experience most cases of illness followed by those in age groups

between 20 and 40. Infants are believed to pick up the disease in swimming pools and child care and their carers are exposed when changing nappies and assisting sick children.

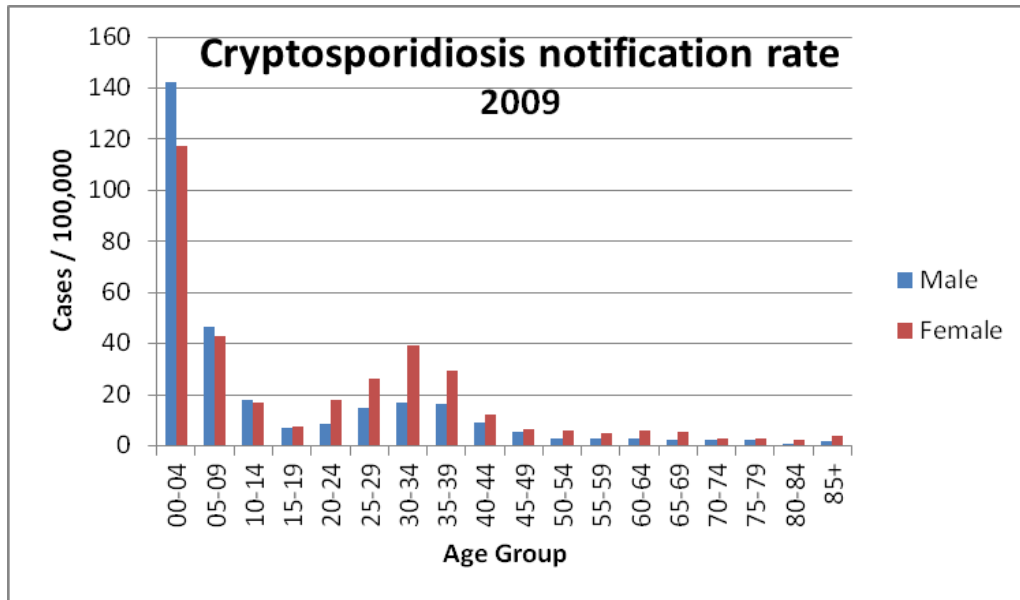**Figure 7. Age-based cryptosporidiosis notification rate 2009 (NNDSS) Australia**

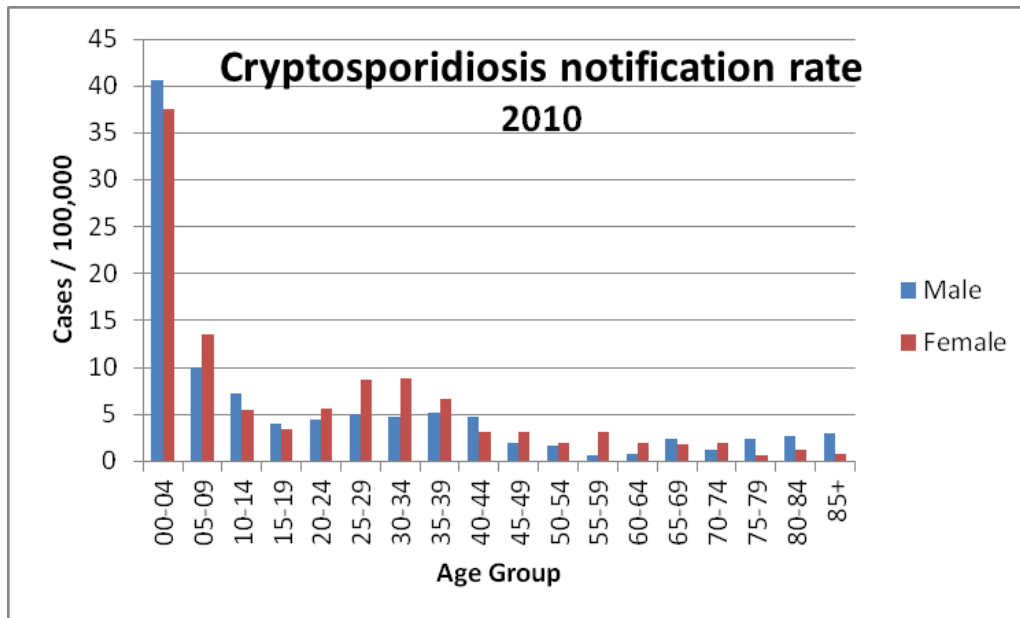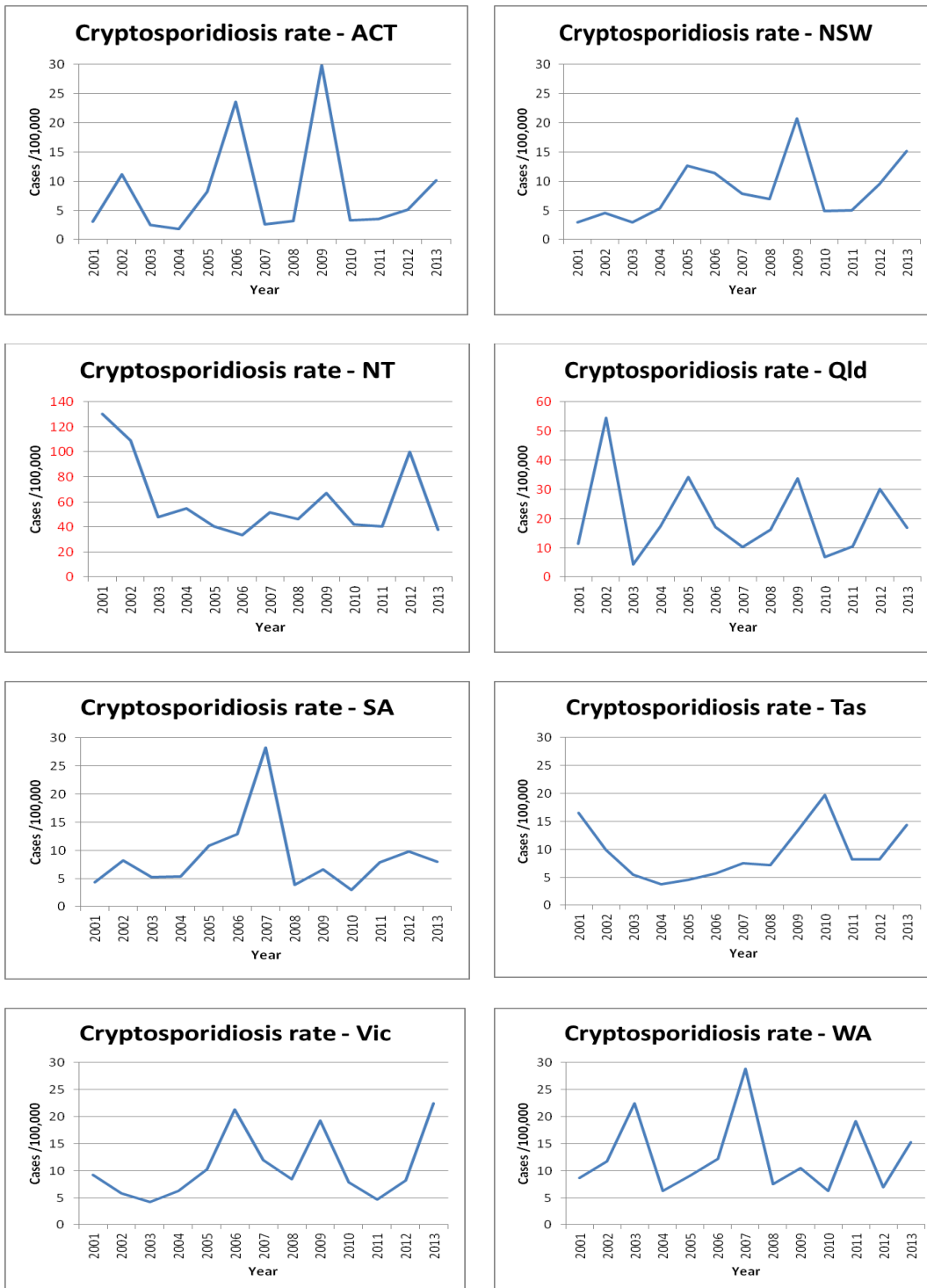**Figure 8. Age-based cryptosporidiosis notification rate 2010 (NNDSS) Australia**

Figure 9. Cryptosporidiosis rates by state or territory (NNDSS)

Note the different scales used for the NT and Qld.

Figure 10 shows the cryptosporidiosis rates across Australian states and territories. Epidemic years are obvious. Some high rates are still being reported in the Northern Territory. For comparison New Zealand averaged 22 cases/100,000 per year from 1997 to 2006 (Snel, et al., 2009). For the USA annual figures ranged from 2.2 to 3.9 over the period 2006 to 2010

(Yoder, et al., 2010; Yoder, Wallace, Collier, Beach, & Hlavsa, 2012). In the absence of a large recreational water outbreak most US states for most years had rates lower than shown in Figure 9. Australia-wide results are shown in Figure 10. Seasonality is displayed in Figure 11.

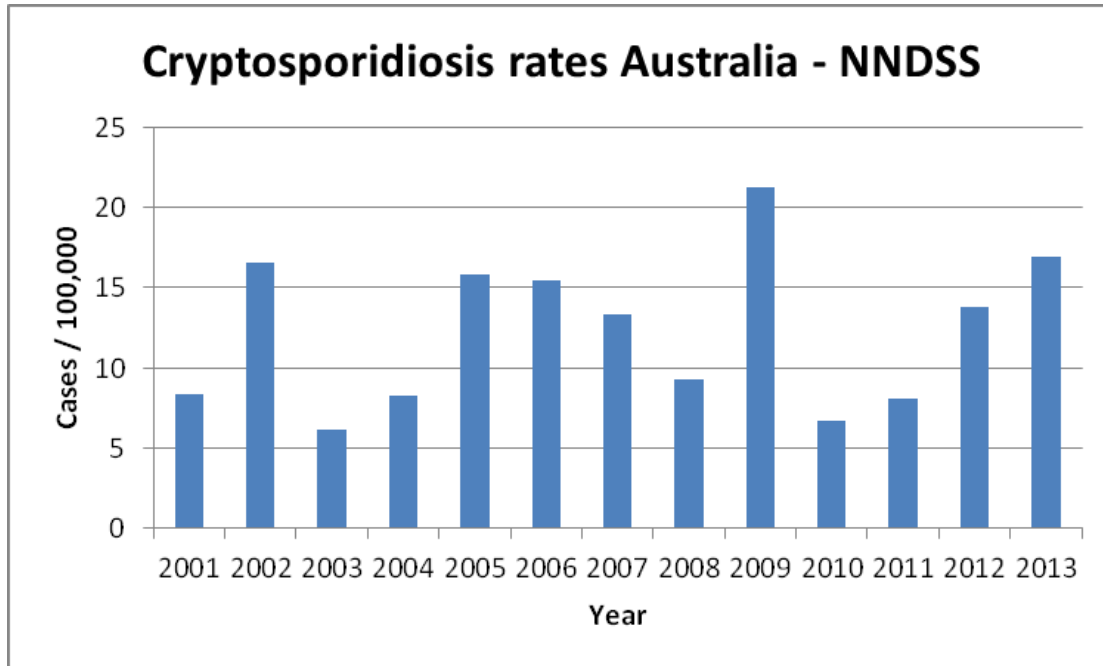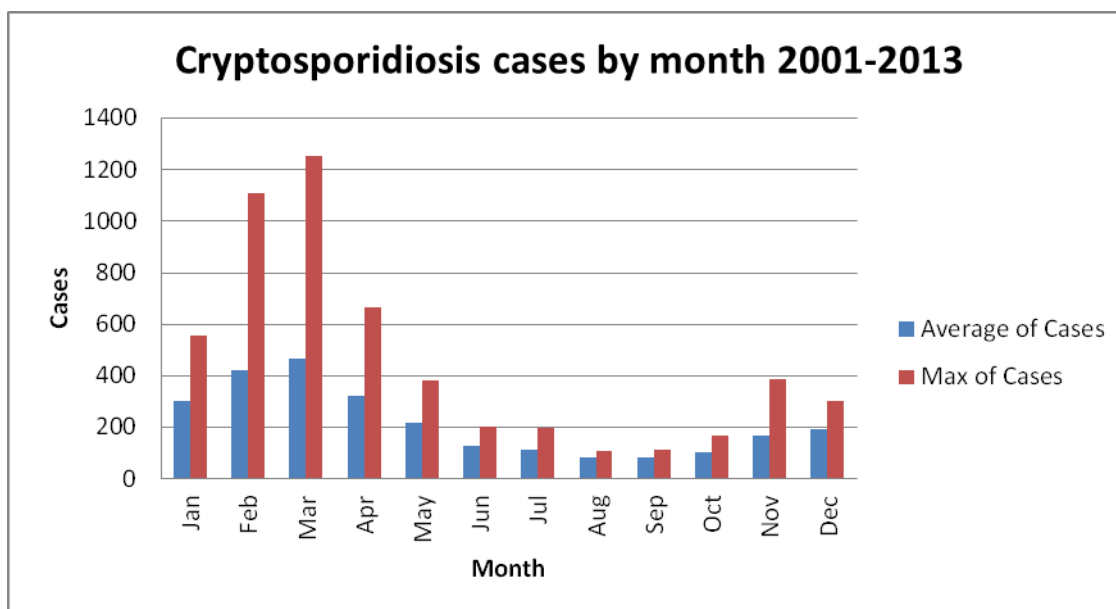**Figure 10. Cryptosporidiosis rates Australia**

Figure 11. Cryptosporidium seasonality Australia

The seasonal pattern of cryptosporidiosis in Australia is closer to the pattern seen in the USA than the one seen in New Zealand. This probably reflects the importance of infections associated with recreational water in Australia.

Foodborne cryptosporidiosis

Robertson and Chambers (2013) searched the literature and identified 19 outbreaks of cryptosporidiosis between 1983 and 2009 in which foodborne transmission was implicated or postulated. Thirteen reports (820 cases) were described as convincing foodborne outbreaks. Two of the outbreaks (one described as convincing) were from Australia. The convincing Australian outbreak involved the consumption of unpasteurised 'milk for pets' and affected 8 children (Harper, Cowell, Adams, Langley, & Wohlsen, 2002). An outbreak involving a mother and infant from 1983 had only scanty information that implicated consumption of raw goat's milk.

For the convincing outbreaks the known food vehicles were: ready to eat foods served cold – 6 (salad vegetables – 5, chicken salad –1); US style apple cider – 3; raw milk – 1; milk (following pasteurisation failure) – 1; and raw meat/liver – 1. Contamination originated from food handlers in four of those outbreaks.

Subsequent to preparation of that list there has been a press release[10] advising of 300 cases of cryptosporidiosis in the UK with strong evidence of an association with eating pre-cut bagged salad products. Twenty reports spanning a 30-year period is a short list. However there are believed to be many more cases of sporadic illness than outbreak cases. As diarrhoea is regarded by many as an inconvenience, rather than an illness, sporadic cases frequently go unreported. There is widespread agreement amongst authors quoted in this document that cases of foodborne cryptosporidiosis are under-reported.

Kirk et al. (in press) estimate there are 17,900 case of cryptosporidiosis Australia per year (circa 2010), with about 10% or 1700 cases being foodborne.

Shellfish

Robertson (2007) reviewed the potential for marine bivalve shellfish to act as transmission vehicles for outbreaks of protozoan infections in humans. Six of 9 surveys identified low levels of cryptosporidium oocysts in marine and estuarine waters. Sixteen of 19 surveys identified cryptosporidium oocysts in commonly eaten marine bivalve molluscs. Oocysts were determined to be viable or infectious in 6 of 7 studies of naturally infected shellfish and in 6 of 6 studies of experimentally contaminated shellfish. However, one of the studies suggested the numbers of viable, infectious oocysts in oysters may be insufficient to cause infection in healthy individuals, especially those with previous *Cryptosporidium* exposure.

Robertson's review identified two cases of parasitic illness linked to the consumption of shellfish. One of these was an epidemiological association between cryptosporidiosis in an immunocompromised individual and the consumption of raw oysters. She suggests that if cases of protozoan disease transmission occur via ingestion of contaminated shellfish, it is very probable that the association between shellfish contamination and the infection would not be recognised. Robertson recommends that the potential for transmission of parasites via shellfish consumption be recognised by foodborne illness investigators.

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[10]http://www.hpa.org.uk/NewsCentre/NationalPressReleases/2013PressReleases/130319Investigationintoanoutbreakofcryptosporidium/

Cryptosporidium interventions

Zoonotic spread

Although *Cryptosporidium* oocysts are widely dispersed in the environment, they are the only life cycle stage involved in disease transmission under normal circumstances, and their minimization or elimination is a key intervention in infection control (Chalmers & Giles, 2010). Good stockmanship and hygiene are central to the control of cryptosporidiosis on farms, with manure and slurry management providing feasible intervention points for farmers.

The UK Food Standards Agency[11] provides guidance on manure control to protect ready-to-eat to crops. Key points include:

- Do not graze fields or apply fresh solid manures and slurries within 12 months of harvest, including a minimum period of 6 months before drilling/planting.
- Ensure water sources used on the farm are not contaminated with manures or run-off.
- Keep livestock and pets out of cropped areas.
- Store solid manures and slurries well away from growing areas.

Visitors to farms on excursions or open days also require protection. Hand washing, preferably with liquid soap in warm running water and drying with disposable towels, after touching animals is central to this, and it should be noted that alcohol gels are not suitable (Chalmers & Giles, 2010).

Protection of waterways that might be used for drinking water supplies, recreation or aquaculture is also vital. Eliminating direct access by animals can be achieved by farm management and fencing. Young calves, in particular, should be kept well clear of waterways.

Since the implementation of improved risk assessment, catchment protection and water treatment following the revision of the water supply regulations in the UK in 2000, there has been a decline in reported cases of cryptosporidiosis, particularly in the spring (Lake et al., 2007).

Whether the source of infection is animal or human, high quality treatment of drinking water will complement catchment protection.

Anthroponotic spread

The infection and consequently the disease is most common in small children in the preschool age, which is probably both as a result of the susceptibility (first exposure) and the personal hygiene (or rather, the lack of it) in this age group which promotes the spread of the parasites via the faecal–oral route or indirectly in public areas such as day care centres, swimming pools, etc. (Joachim, 2004).

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[11] http://multimedia.food.gov.uk/multimedia/pdfs/manuresguidance.pdf

Comprehensive advice from the Centres for Disease Control is included in the Appendix. Key points include:

- Wash hands with soap and water for at least 20 ——seconds, rub hands together vigorously, and scrub all surfaces –
  - before preparing or eating food,
  - after using the toilet,
  - after changing nappies or cleaning up a child who has used the toilet,
  - before and after tending to someone who is ill with diarrhoea, and
  - after handling an animal or its faeces.
- At child care facilities –
  - to reduce risk for disease transmission, exclude children with diarrhoea from child care settings until the diarrhoea has stopped.
- At recreational water venues –
  - protect others by not swimming while experiencing diarrhoea (this is essential for children in nappies and aquatics staff). If cryptosporidiosis is diagnosed, do not swim for at least 2 weeks after diarrhoea stops.
  - shower before entering the water.
  - wash children thoroughly (especially their bottoms) with soap and water after they use the toilet or their nappies are changed and before they enter the water.
  - take children on frequent toilet breaks and check their nappies often.
  - change nappies in the toilet, not at the poolside.
  - do not swallow water while swimming in swimming pools, spas, interactive fountains, lakes, rivers, springs, ponds, streams, or the ocean.

Conclusions

Toxoplasmosis is a serious illness in a relatively small proportion of those exposed to the parasite. There is still much to be learned about the routes of human infection. This is in-part due to the complexity of the parasite's life cycle and in-part due the high proportion of illnesses that result from sporadic infection, which are difficult to investigate. However, it is clear that significant amounts of illness are due to consumption of raw or undercooked meat containing *T. gondii* tissue cysts, consumption of raw vegetables contaminated with oocysts from cat faeces and water also contaminated with *T. gondii* oocysts from cat faeces.

Expert groups have drafted guidance that will reduce illness. The focus has been on women of childbearing age to prevent congenital toxoplasmosis however, the threat of ocular disease means that the guidance has universal importance.

Cryptosporidiosis is a short-term illness for most people but can cause prolonged life-threatening disease in immune-compromised people. The routes of infection for cryptosporidiosis are better understood than they are for toxoplasmosis. The disease is primarily related to exposure to water which contains *Cryptosporidium* oocysts or with direct contact with faecal material from infectious young animals. Food contamination is a less significant exposure but can occur from contaminated water, direct contact with manure, from infectious food handlers, from contaminated shellfish or from cross-contamination during food preparation.

Again, experts have designed a comprehensive set of interventions for use on farm, in recreational water situations and for drinking water protection. High levels of personal hygiene are also required to overcome the highly infectious nature of the parasite.

Appendix

Table 5. CDC recommendations to prevent and control cryptosporidiosis

BOX 1. CDC recommendations to prevent and control cryptosporidiosis

Practice good hygiene.
- Everywhere
  - Wash hands with soap and water for at least 20 seconds, rub hands together vigorously, and scrub all surfaces
    - before preparing or eating food,
    - after using the toilet,
    - after changing diapers or cleaning up a child who has used the toilet,
    - before and after tending to someone who is ill with diarrhea, and
    - after handling an animal or its feces.

Additional information about hygiene is available at http://www.cdc.gov/healthywater/hygiene.

- At child care facilities
  - To reduce risk for disease transmission, exclude children with diarrhea from child care settings until the diarrhea has stopped.
- At recreational water venues
  - Protect others by not swimming while experiencing diarrhea (this is essential for children in diapers and aquatics staff). If cryptosporidiosis is diagnosed, do not swim for at least 2 weeks after diarrhea stops.
  - Shower before entering the water.
  - Wash children thoroughly (especially their bottoms) with soap and water after they use the toilet or their diapers are changed and before they enter the water.
  - Take children on frequent bathroom breaks and check their diapers often.
  - Change diapers in the bathroom, not at the poolside.

Additional information about recreational water illnesses and how to stop them from spreading is available at http://www.cdc.gov/healthywater/swimming.

- Around animals
  - Minimize contact with the feces of all animals, particularly young animals.
  - When cleaning up animal feces, wear disposable gloves, and always wash hands when finished.
  - Wash hands after any contact with animals or their living areas.
- Outside
  - Wash hands after gardening, even if wearing gloves.
- Immunocompromised persons
  - Avoid close contact with anyone who has cryptosporidiosis.
  - Do not handle animal feces because infection can be life threatening for immunocompromised persons.

Additional information about cryptosporidiosis prevention and control is available at http://www.cdc.gov/crypto/gen_info/prevent.html.

Avoid water that might be contaminated.
- Do not swallow water while swimming in swimming pools, spas, interactive fountains, lakes, rivers, springs, ponds, streams, or the ocean.
- Reduce contamination of treated recreational water venues by having pool operators install in-line secondary disinfection systems (e.g., ultraviolet light, ozone) to inactive this chlorine-tolerant parasite.
- Do not drink untreated water from lakes, rivers, springs, ponds, streams, or shallow wells.
- Do not drink inadequately treated water or ice made from water during communitywide outbreaks caused by contaminated drinking water.
- Do not use or drink inadequately treated water or use ice when traveling in countries where the water supply might be unsafe.
- If the safety of drinking water is in doubt (e.g., outbreak, poor sanitation, or lack of water treatment systems),
  - drink bottled water
  - disinfect water by heating it to a rolling boil for 1 minute, or
  - use a filter that has been tested and rated by National Safety Foundation (NSF) Standard 53 or NSF Standard 58 for cyst and oocyst reduction; filtered water will need additional treatment to kill or inactivate bacteria and viruses.

Additional information about water filters is available at http://www.cdc.gov/crypto/gen_info/filters.html.

Avoid eating food that might be contaminated.
- Use safe, uncontaminated water to wash all food that is to be eaten raw.
- After washing vegetables and fruit in safe, uncontaminated water, peel them before eating them raw.
- Avoid eating uncooked foods when traveling in countries with poor water treatment and food sanitation.

Information about how to prevent illnesses while traveling is available at http://wwwnc.cdc.gov/travel/content/safe-food-water.aspx.

Prevent contact and contamination with feces during sex.
- Use a barrier during oral-anal sex.
- Wash hands immediately after handling a condom used during anal sex and after touching the anus or rectal area.

(Yoder, et al., 2010)

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