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Invited review

The zoonotic transmission of *Giardia* and *Cryptosporidium*

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Abstract

The molecular characterisation of *Giardia* and *Cryptosporidium* has given rise to a more epidemiological meaningful and robust taxonomy. Importantly, molecular tools are now available for 'typing' isolates of the parasites directly from clinical and environmental samples. As a consequence, information on zoonotic potential has been obtained although the frequency of zoonotic transmission is still poorly understood. Analysis of outbreaks and case–control studies, especially when coupled with genotyping data, is slowly providing information on the public health significance of zoonotic transmission. Such studies support the hypothesis that *Cryptosporidium hominis* is spread only between humans but that the major reservoir for *Cryptosporidium parvum* is domestic livestock, predominantly cattle, and that direct contact with infected cattle is a major transmission pathway along with indirect transmission through drinking water. The situation is less clearcut for *Giardia duodenalis* but the evidence does not, in general, support zoonotic transmission as a major risk for human infections. However, for both parasites there is a need for molecular epidemiological studies to be undertaken in well-defined foci of transmission in order to fully determine the frequency and importance of zoonotic transmission.

Keywords: Giardia; Cryptosporidium; Epidemiology; Zoonoses; Zoonotic transmission

1. Introduction

Giardia and *Cryptosporidium* are the most common enteric parasites of humans and domestic animals, and are being increasingly recognised as parasites of a diverse range of wildlife species (Fayer, 2004; Thompson, 2004; Thompson and Monis, 2004; Appelbee et al., 2005). Their clinical significance is largely restricted to humans and young livestock (Olson et al., 2004). *Giardia* is a common cause of diarrhoeal disease in humans, particularly among disadvantaged groups where chronic infections contribute to poor growth and other nutritional disorders particularly in children (Thompson and Monis, 2004). In young livestock, *Giardia* infections may adversely impact on production (Olson et al., 2004). The significance of *Cryptosporidium* was initially recognised to be one of an opportunistic pathogen in AIDS patients but the impact of such infections is now lessening, at least in developed countries, with the advent of retroviral therapies (Nannini and Okhuysen, 2002). Although *Cryptosporidium* infections are usually of short duration and self-limiting in individuals with an intact immune system the lack of effective anticryptosporidial drugs means the very young and elderly may be at risk of severe disease as a results of *Cryptosporidium* infection.

The life cycles of each parasite include asexual phases of proliferation on the mucosal surface, in addition to a sexual phase of reproduction in *Cryptosporidium* that also exhibits an unusual intracellular phase of development in its life cycle (reviewed in Thompson et al., in press). The infective stages of both parasites are encysted when released in the faeces and capable of prolonged survival in the environment. Re-infection is achieved when the cysts/oocysts are ingested which may be through direct host to host contact or via contaminated materials, water, food or arthropods.

The link between human and animal infections has been a question that has dominated much of the research effort on

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Giardia and *Cryptosporidium*. Since both organisms can be transmitted in water, the source of water contamination remains a critical issue for water authorities throughout the world (Fayer, 2004; Thompson, 2004). The role that animal infections may play in this regard remains controversial, particularly that of livestock and wildlife because of their potential role as zoonotic reservoirs of infection.

In order to better understand the zoonotic potential of *Giardia* and *Cryptosporidium* infections in wild and domestic animals, it has been important to determine whether humans and other animals are susceptible to infection with genetically identical forms of each parasite. The taxonomy of *Giardia* and *Cryptosporidium* has been extensively reviewed and will not be reviewed in detail here (Monis and Thompson, 2003; Thompson and Monis, 2004; Xiao et al., 2004; Caccio et al., 2005). However, elucidating a correct taxonomy for both *Giardia* and *Cryptosporidium* has provided the basis for better understanding the links between infections in humans and other animals. The issue has been difficult to resolve because of a paucity of morphological characters on which to discriminate species. In this respect, *Giardia* and *Cryptosporidium* share this problem with many other protozoa, and it is only recently with the advent of molecular typing tools that both the taxonomy and epidemiology of many protozoal infections are now being resolved.

Initially, species of *Giardia* and *Cryptosporidium* were described on the basis of host occurrence (Thompson et al., 1990; O'Donoghue, 1995). Subsequently, such an approach was criticised and the numbers of species was rationalised. Now, the picture is changing once again and many of the 'host based' species have been resurrected (Thompson and Monis, 1994; Xiao et al., 2004).

Table 1 lists currently recognised and recently proposed species of *Giardia* and *Cryptosporidium* and their host ranges, as well as a number of intraspecific variants, or genotypes, that have been characterised on the basis of their genetic distinctness, as well as other phenotypic characteristics including host origin. The taxonomic status of these latter forms remains to be resolved, and requires further studies in which both their geographic and host ranges are further investigated.

With both *Giardia* and *Cryptosporidium*, a large number of species and genotypes are now recognised that differ principally in their host range. Some species and genotypes appear to be restricted to particular species of hosts

Table 1
Species and genotypes of *Cryptosporidium* and *Giardia*

Cryptosporidium		Giardia	
Species	Major hosts	Species	Major hosts
C. muris	Rodents	G. duodenalis (=Assemblage A)	Humans and other primates, dogs, cats, livestock, rodents and other wild mammals
C. parvum	Cattle & other livestock, humans	Assemblage B[a]	Humans and other primates, dogs
C. melegridis	Birds	G. agilis	Amphibians
C. wrairi	Guinea pigs	G. muris	Rodents
C. felis	Cats	G. psittaci	Birds
C. serpentis	Reptiles	G. ardeae	Birds
C. baileyi	Poultry	Assemblage C[a]	Dogs
C. saurophilum	Lizards	Assemblage F[a]	Cats
C. galli		Assemblage E[a]	Cattle and other hoofed livestock
C. andersoni	Cattle	G. simondi (=Assemblage G)	Rats
C. canis	Dogs		
C. molnari	Fish		
C. hominis	Humans		
C. suis	Pigs		
Genotypes			
Ferret	Deer mice		
Mouse	Squirrel (×2)		
Skunk	Bear		
Marsupial (×4)	Goose (×2)		
Horse	Duck		
Rabbit	Bovine		
Monkey	Snake		
Pig (×2)	Tortoise		
Cervid (×2)	Lizard		
Fox	Woodcock		
Muskrat (×2)			

Details in Thompson and Monis (2004); Xiao et al. (2004).

[a] Recently proposed to be separate species: *G. enterica*, *G. canis*, *G. cati* and *G. bovis*. See Thompson and Monis (2004).

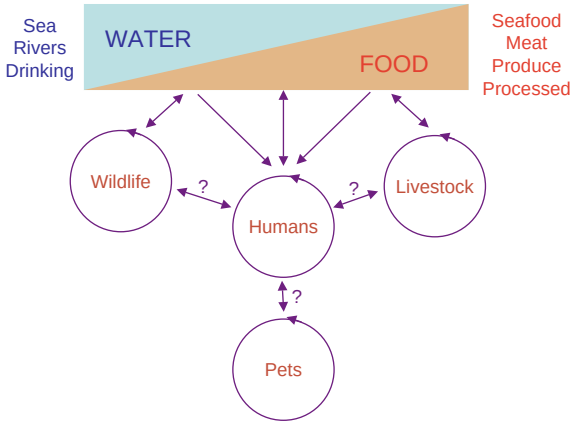

Fig. 1. Diagram showing the different, most important cycles of transmission for maintaining *Giardia* and *Cryptosporidium*. As well as direct transmission, water and food may also play a role in transmission. Question marks indicate uncertainty regarding the frequency of interaction between cycles.

(e.g. *Giardia psittaci*; *Cryptosporidium canis*; Table 1) or closely related host assemblages (e.g. *Giardia bovis*; *Cryptosporidium baileyi*; Table 1), whereas others have broad host ranges including humans (e.g. *Giardia duodenalis*; *Cryptosporidium parvum*; Table 1) and are therefore of zoonotic significance.

Giardia duodenalis and *C. parvum* are maintained in a variety of transmission cycles that can be maintained independently and do not require interaction between them (Fig. 1). Thus *G. duodenalis* can be maintained in independent cycles involving wildlife or domestic animals. Similarly, *C. parvum* can be maintained in cycles involving livestock, especially cattle. What is not understood are the circumstances under which such cycles may interact resulting in zoonotic transfer.

Numerous studies have characterised isolates of *Giardia* and *Cryptosporidium* collected from different hosts and have demonstrated the occurrence of the same species/genotype in humans and other animals (Monis and Thompson, 2003). Such data is indicative of zoonotic potential but gives no information on the frequency of zoonotic transmission. Such information can be obtained from molecular epidemiological studies that genotype isolates of the parasites from susceptible hosts in localised foci of transmission or as a result of longitudinal surveillance and genotyping of positive cases. In the former, recent research in a localised endemic focus of transmission has provided convincing data on the zoonotic transmission of *G. duodenalis* between dogs and humans (Traub et al., 2004). Although companion animals have long been considered potential sources of human *Cryptosporidium* infection, the only studies in which oocysts recovered from dogs and cats have been genotyped have shown that they are usually infected with host-adapted species; *C. canis* and *Cryptosporidium felis* (Abe et al., 2002). Thus dogs and cats and possibly other companion animals may not be important zoonotic reservoirs of *Cryptosporidium* infection. However, with *Cryptosporidium*, there is considerable epidemiological data demonstrating strong links between contact with infected livestock and human infections (Fayer et al., 2000; Stantic-Pavlinic et al., 2003). This is not the case with *Giardia*. However, with both *Giardia* and infected livestock have often been incriminated as sources of contamination for waterborne outbreaks of cryptosporidiosis and giardiasis (Fayer et al., 2000; Thompson, 2004). Interestingly, the application of genotyping procedures to the contaminating isolate(s) has often incriminated human effluent as the source. However, in a study undertaken of cryptosporidiosis patients in Scotland, C. parvum was shown to be the causative agent in 84% of 67 cases, supporting livestock faecal pollution of water sources as the leading cause of human sporadic cryptosporidiosis (Goh et al., 2004).

2. Epidemiological evidence

2.1. Cryptosporidiosis

Widespread interest in human cryptosporidiosis can be dated back to the early 1980 s. At that time it was generally assumed that cryptosporidiosis was primarily a zoonotic pathogen, though with the potential for person-to-person transmission (Casemore et al., 1985). This view came largely from the investigation of outbreaks of infection that were usually associated with visits to farms and zoos or through contamination of drinking water, believed to be largely due to contamination by livestock (Meinhardt et al., 1996; Hunter, 1997). Outbreaks due to person-to-person spread were described, especially associated with swimming pools or with pre-school children. Fortunately, for those people researching cryptosporidiosis this relatively simple model of its epidemiology began to unravel due in part to a series of important discoveries.

Perhaps, the most important of such discoveries was the finding that *C. parvum* could be separated into two genotypes (Peng et al., 1997). Although the initial study was done on only 39 isolates detected in human and bovine specimens, all type 1 genotypes were found only in humans whilst type 2 genotypes were found in both humans and cattle. Type 1 genotypes were subsequently called human (H type) and type 2 cattle (C type). This raised the point that *C. parvum* was not after all a single homogenous species with fairly identical epidemiology but rather two related types, one of which was a strictly human pathogen and the other primarily zoonotic. This observation was soon confirmed by several other research groups with access to larger and more varied sample collections (Morgan et al., 1998; McLauchlin et al., 1998, 1999; Homan et al., 1999) and also in experimental studies (Widmer et al., 1998).

However, the definitive paper was published by McLaughlin et al. (2000). They studied 1705 isolates from human faeces and 105 isolates from animal faeces. Of

the human isolates, 37.8% were genotype 1 (H) and 61.5% genotype 2 (C), the remainder were *Cryptosporidium meleagridis*. In contrast, all of the animal isolates were genotype 2 (C). The authors also reported geographical and seasonal variation in the distribution of genotypes with type 2 being more common in the spring, the North West of England and people reporting contact with farm animals. Type 2 isolates were more common in autumn and in people who travelled abroad. The proposed model for the epidemiology of cryptosporidiosis at the turn of the millennium was of two subtypes one of which (genotype 1) was exclusively a human pathogen and the drivers for maintenance of this genotype in circulation were, in part due to infection during travelling. For genotype 2 the primary driver was contact with livestock either directly or indirectly as a result of contamination of water supplies by animal faeces. These two genotypes have now been proposed as two separate species, genotype 1 as *C. hominis* and genotype 2 as *C. parvum* (Morgan-Ryan et al., 2002). For the rest of this discussion, we will use the proposed species names.

However, at this point, most of our knowledge on the exact transmission pathways came from the analysis of outbreaks of infection. It was far from clear that sporadic cases of infection were acquired from the same transmission routes associated with outbreaks. Cases associated with detected outbreaks represent only a small proportion ($<$ 10%) of all diagnosed cases (Nichols, 2003). Some proportion of apparently sporadic cases is likely to be associated with undetected outbreaks but this is still likely to represent only a small proportion of all cases (Hunter et al., 2001). When trying to determine transmission pathways, the problem with existing national surveillance systems, where they even exist, is that they rarely have adequate risk factor data associated with them.

The next set of advances came from several large case–control studies conducted in Australia, the UK and US. Although case control studies have been done earlier they have generally enrolled small numbers of cases or to have been performed during investigation of outbreaks. One group of humans that are particularly at risk from *Cryptosporidium* infection is the immune compromised, especially those living with AIDS. The epidemiology of infection in this group has been reviewed recently in detail and we will not repeat much of this review (Hunter and Nichols, 2002). In this group most of the case control studies have highlighted sexual transmission as being one of the most important transmission pathways with having multiple partners, 'insertive anal sex' and visiting sex venues being particular risks (Sorvillo et al., 1994; Pedersen et al., 1996; Caputo et al., 1999; Hellard et al., 2003). However, one study reported an increased risk in HIV positive patients who owned dogs (Odds ratio = 2.19; 95% Confidence Intervals 0.9–5.3; $P = 0.05$) (Glaser et al., 1998). There was no association with other pets.

In developing countries we have been able to identify only one good case–control study, this from Guinea-Bissau (Molbak et al., 1994). Technically, this study was a case–control study nested within a longer cohort study. Some 125 cases and an identical number of matched controls were interviewed for possible risk factors. Significant risk factors were keeping pigs (OR = 2.5; 95% CI 1.4–4.7) and dogs (OR = 2.1; 95% CI 1.0–4.2), storage of cooked food for later consumption (OR = 1.8; 95% CI 1.0–3.3), and male sex (OR for boys = 1.9; 95% CI 1.0–3.4). Breast feeding was negatively associated with risk (OR = 0.3; 95% CI 0.1–1.1).

The first high quality study from a developed country was from South Australia and included a relatively small 51 laboratory confirmed cases and the same number of matched controls (Weinstein et al., 1993). The only significant association was a negative association with the consumption of rainwater suggesting that the consumption of spring water or mains water may have been the transmission pathway.

The first adequately powered case–control study, also from Australia, was reported by Robertson and colleagues (2002). The authors actually conducted two case control studies, one in Melbourne with 201 cases and 795 controls and one in Adelaide with 134 cases and 536 controls. Significant associations are presented in Table 2. Of particular note for this paper is the negative association with contact with domestic pets but the positive association with contact with calves. Also of interest was the positive associations with contact with other cases of diarrhoea at home, swimming in Melbourne, but not Adelaide and drinking unboiled river water from Adelaide but not Melbourne. There was also a negative association with eating uncooked carrots.

The next adequately powered study was reported by Roy et al. (2004) from the United States. They recruited 282 persons with laboratory confirmed cases and 490 matched controls. Risk factors are shown in Table 2. The key findings were strong association with travel abroad, contact with cases of diarrhoea and contact with calves or cows. Eating raw vegetables was also negatively associated with illness.

The third well-powered study was done in North Cumbria, two local government districts in the North of England (Goh et al., 2004). This study was done over a 4-year period, a very long time for most such studies. Overall, 152 cases and 466 unmatched controls were recruited. As far as we can tell the study focussed predominantly on water-related risk factors. Of 67 isolates that were typed, 46 (84%) were *C. parvum* (genotype 2). The only significant risk factors were the usual daily volume of cold unboiled tap water drunk (OR 1.40; 95% CI 1.14–1.71 per pint consumed per day) and also short visits to farms (OR 2.02; 95% CI 1.04–3.90; $P = 0.04$). Contact with farm animals was not independently associated, though this was likely to have been due to the inclusion of a farm visit in the model. It should be noted that the study was done at a time when the mains water supply was not adequately filtered to remove *Cryptosporidium*. After the installation of

Table 2
Case–control studies of sporadic cryptosporidiosis in developed nations reported since the early 1990s

Reference	Location	No. of cases	No. of controls	Significant risk factors	Odds ratios (95% CI)
Robertson et al. (2002)	Melbourne	201	795	Eating uncooked carrots	0.6 (0.4–0.9)
				Swimming in public pool	2.7 (1.9–3.8)
				Children < 6 at home with diarrhoea	7.4 (4.0–13.8)
				Persons > 5 at home with diarrhoea	1.8 (1.1–2.9)
				Animal contact in home	0.6 (0.4–0.8)
				Calf contact away from home	2.9 (1.5–5.7)
				Drink unboiled water from river, lake or dam	1.5 (0.8–2.7)
Robertson et al. (2002)	Adelaide	134	536	Eating uncooked carrots	0.6 (0.4–0.9)
				Swimming in public pool	1.2 (0.8–1.9)
				Children < 6 at home with diarrhoea	8.6 (4.8–15.6)
				Persons > 5 at home with diarrhoea	3.7 (2.2–6.2)
				Animal contact in home	0.6 (0.4–0.9)
				Calf contact away from home	5.1 (1.5–17.3)
				Drink unboiled water from river, lake or dam	3.1 (1.5–6.5)
Roy et al. (2004)	United States	282	490	Contact with persons (> 2 to 11 yr old) with diarrhoea	3.0 (1.5–6.2)
				Contact with calves or cows	3.5 (1.8–6.8)
				International travel	7.7 (2.7–22.0)
				Freshwater swimming	1.9 (1.0–3.5)
				Eating raw vegetables	0.5 (0.3–0.7)
				Chronic medical condition	2.2 (1.2–4.0)
Hunter et al. (2004)	Wales and NW England	427	427	Travel outside of UK	5.7 (2.9–11.2)
				Case contact	4.6 (2.4–8.7)
				Touch cattle	3.9 (1.4–10.0)
				Toileting child < 5 years	1.9 (1.1–3.2)
				No. of glasses unboiled water	1.1 per glass (1.0–1.3)
				Eat ice cream	0.5 (0.3–0.7)
				Eat raw vegetables	0.5 (0.3–0.8)
				Eat tomatoes	0.6 (0.4–1.0)

an adequate membrane filter the incidence of cryptosporidiosis in the area fell by about 79% (Goh et al., 2005).

Finally, Hunter et al. (2004) reported a large study conducted in Wales and the North West Region of England. These authors were able to recruit 427 cases and 427 controls. Risk factors are listed in Table 2. The clearest associations were with travel outside of the UK, contact with another case, touching cattle, and toileting children under 5 years of age. The number of glasses of unboiled water drunk at home was borderline significant. Eating ice cream, raw vegetables and tomatoes were all negatively associated.

From these studies, it is becoming obvious that the main risk factors for human cryptosporidiosis at least in developed countries are contact with other cases, travel abroad and contact with cattle. Contact with family pets is either not significant or negatively associated with risk. There is also an interesting negative association across most of the studies with the consumption of raw vegetables. However, one of the problems with the studies presented so far has been the general lack of genotyping data. Clearly if *C. hominis* and *C. parvum* have very different host ranges and transmission pathways then combining them together for analysis would emphasise risk factors common to both species and downplay risk factors unique to one or other species.

Only one study so far has done independent analyses of these two species (Hunter et al., 2004). In their larger study the authors were able to type 191 isolates; 115 were *C. hominis* and 76 *C. parvum*. The final models for these two species are shown in Table 3. The key findings for *C. hominis* are the association with foreign travel and changing children's nappies. There was a weak negative association with eating fresh fruit. For *C. parvum* the only positive association was with contact with cattle but there were strong negative associations with eating tomatoes and raw vegetables. The association of contact with cattle is all the more interesting as much of the study was done at the time of a foot and mouth epidemic during which access to farmland was severely restricted (Hunter et al., 2003).

Taking the evidence from the case–control studies and genotyping studies, we can now suggest models for the transmission of both *C. hominis* and *C. parvum*. *Cryptosporidium hominis* appears to be a strictly human pathogen. As such its reservoir is the gut of other humans. New cases become infected directly or indirectly from other humans.

Table 3
Key risk factors from case control study of cryptosporidiosis in Wales and the North West Region of England for identified species (Hunter et al., 2004)

Risk factor	Odds ratios	
	C. hominis	C. parvum
Travel out UK	6.8 (2.6–17.9)	
No. of times in toddler pool	1.3/time (1.0–1.7)	
Sleeping on ground	0.2 (0.1–1.0)	
Nappy changing	4.0 (1.8–8.6)	
Washing fruit and vegetables		
Always	1.0	
Usually	0.4 (0.2–0.8)	
Sometimes	0.9 (0.4–1.8)	
Never	1.6 (0.5–5.1)	
Number of children aged 5–15 in home	0.6/person (0.4–1.0)	
Eat fresh fruit	0.2 (0.1–0.9)	
Touch or handle any farm animals		2.7(1.1–6.3)
Eat tomatoes		0.3 (0.1–0.7)
Eat raw vegetables		0.2 (0.1–0.6)

The strong association with foreign travel for *C. hominis* suggests that exposure to lower hygiene standards may increase the probability of person-to-person transmission. The finding of an association with changing nappies in asymptomatic children tends to suggest that very young children may also act as a reservoir and source of infection.

For *C. parvum* the epidemiology is a more complex. Animals, especially cattle, are the main reservoir. Cattle to human transmission can be via direct contact with animals or indirect such as following the contamination of drinking water. Person-to-person transmission also undoubtedly occurs (as illustrated by certain swimming pool outbreaks), though the relative importance of this transmission pathway is unclear. The evidence does not support a strong role for domestic pets as sources of infection and if anything pets are negatively associated with infection. The negative association with pets and, even more strongly, the consumption of raw vegetables, tomatoes and carrots are perhaps the most intriguing aspect of the epidemiology.

It is our belief that these negative associations indicate the protective effect of repeated low dose exposure leading to enhanced immunity. It has been hypothesised that repeated low dose exposure could lead to reduced disease burdens compared to those communities that are exposed to infrequent and possibly higher dose exposures (Swift and Hunter, 2004). Recent evidence for this hypothesis came from a sero-epidemiological study that showed that people with high anti-cryptosporidial antibody levels were much less likely to self-report diarrhoeal disease than those with low levels of antibody (Frost et al., 2005). In this context regular contact with domestic pets or consumption of raw vegetable that may be contaminated with oocysts from domestic livestock would be expected to be protective.

2.2. Giardiasis

The epidemiology and transmission pathways of giardiasis are, in many ways, similar to cryptosporidiosis, at least as determined by the analysis of outbreaks (Flanagan, 1992; Hunter, 1997). Possibly reflecting its longer history as a recognised enteric pathogen and greater ease of diagnosis there are rather more adequate case–control studies. This review will concentrate on those studies reported since the early 1990s.

We have identified eight case control studies (Table 4). The earliest was reported by Isaac-Renton and Philion (1992) from British Columbia. There were two studies reported in the following year: one by Dennis et al. (1993) from New Hampshire and one by Mitchell et al. (1993) from Canterbury, New Zealand. This later study was relatively low powered with just 51 cases and 67 controls. In 1994 two relatively low powered studies were reported from England. One included just 33 cases from East Anglia (Warburton et al., 1994) and the other included 74 cases in Avon and Somerset (two counties in the South West of England) (Gray et al., 1994). More recently, two studies were reported from Auckland, New Zealand; one in people aged 15 to 64 years (Hoque et al., 2002) and one in children under 5 (Hoque et al., 2003). A final one was reported from the South West of England (Stuart et al., 2003). The analyses done in the Auckland studies in adults (Hoque et al., 2002), are somewhat confusing. The authors presented several models in their paper which, in our view, represent a degree of over-analysis and so require caution in their interpretation. The English study by Stuart et al. (2003) excluded people with a history of travel.

When looking at the studies in their totality the recurring themes are travel, swimming in surface waters, drinking water, especially when this does not come from a mains supply, and contact with young children, especially those still wearing nappies. Only one study has found a significant association with animal contact (both farm and companion animals). This study by Warburton et al. (1994) was one of the smaller studies to be reviewed in this paper. Other studies have not found such an association.

In addition to these studies done in relatively affluent countries there have been several studies reported from low to mid income countries (Table 5). The first such study was a cohort study of a birth cohort in Egypt followed for a year (Mahmud et al., 1995). Two cross-sectional surveys from Mexico were done, one examined 6748 faecal samples (Cifuentes et al., 2000) and the other some 914 children during the dry and rainy seasons (Cifuentes et al., 2004). Finally a cross-sectional survey from Brazil has been reported (Prado et al., 2003). All of these studies have highlighted the key role of sanitation and personal hygiene as the major risk factor for giardiasis in low income countries suggesting that zoonotic transmission plays, at most, only a minor role.

Table 4
Case–control studies of sporadic giardiasis in developed nations reported since the early 1990s

Reference	Location	Patient age group	No. of cases	No. of controls	Significant risk factors	Odds ratios (95% CI)
Isaac-Renton and Philion (1992)	British Columbia	All ages	228	228	Use non-chlorinated surface water vs well water	12.0 (5.9–24.7)
					Travel to third world countries	6.3 (1.1–35.8)
Dennis et al. (1993)	New Hampshire	All ages	273	375	Shallow dug well as a residential water source	2.4 (1.3–47.0)
					Recent history of drinking untreated surface water	3.4 (2.1–5.5)
					History of swimming in a lake or pond	4.6 (2.4–86.0)
					Contact with a person thought to have giardiasis	2.3 (1.4–36.0)
					Recent contact with a child in day care	1.5 (1.0–2.1)
Mitchell et al. (1993)	Canterbury, New Zealand	All ages	51	67	Contact with sewage	10.6 (1.3–85.1)
					Travel overseas	∞ (1.8–∞)
Warburton et al. (1994)	East Anglia, UK	All ages	33	112	Contact with farm animals	4.8 (1.3–17.4)
					Contact with pets	14.6 (4.2–50.6)
Gray et al. (1994)	Avon and Somerset, UK	All ages	74	108	Swimming	2.4 (1.0–6.1)
					Travel to developing countries	7.6 (0.8–70.1)
					Camping, caravanning or use of holiday chalets	8.4 (0.8–70.1)
Hoque et al. (2002)	Aukland, New Zealand	15–64 yrs	183	336	Housewives and nursing mothers (especially those changing nappies)	2.1 (1.4–3.7)
					Occupational groups exposed to human wastes	4.1 (1.9–8.9)
					Consumption of drinking water from New Zealand supplies other than metropolitan mains supplies	2.1 (1.4–3.3)
					Consumption of drinking water from sources outside New Zealand	8.0 (4.2–15.1)
					Travelling (especially outside New Zealand)	7.6 (4.0–14.2)
					Swimming in pools or fresh water at least once a week	2.04 (1.3–3.1)
Hoque et al. (2003)	Aukland, New Zealand	<5 years	69	98	Wearing nappies	3.0 (1.0–8.9)
					Other children in household wearing nappy	6.5 (1.8–23.4)
					Drinking water consumed away from home	4.7 (2.2–10.1)
					Swimming at least once a week	2.4, (1.1–5.3)
					Travelling domestically	2.5 (1.03–6.0)
Stuart et al. (2003)	South West England	All	232	574	Swallowed water while swimming	6.2 (2.3–16.6)
					Recreational fresh water contact	5.5 (1.9–15.9)
					Each additional glass of tap water consumed per day	1.3 (1.1–1.5)
					Ate lettuce	2.2 (1.2–4.3)
					Ate ice cream	0.4 (0.2–0.7)

Taken together, the epidemiological evidence presented here, and older studies reviewed elsewhere (Hunter, 1997), do not support a major role for zoonotic transmission of *Giardia*, at least by direct contact. The association of illness with water can of course be due to contamination from animal sources. Waterborne outbreaks have been reported where the outbreak strain has been found in animals in the watershed, though most

Table 5
Studies of sporadic Giardiasis in low income nations reported since the early 1990s

Reference	Location	Patient group	Significant risk factors	Odds ratios (95% CI)
Mahmud et al. (1995)	Egypt	<1 year old	Living in a household without a latrine	2.63 (1.4–4.9)
			Living in a household with a mud floor in the sleeping rooms	1.8 (1.0–3.0)
			Household exposure to more than 10 chickens	2.5 (1.1–5.6)
			Mothers education beyond the primary level	0.3 (0.1–0.9)
			Drinking water stored in metallic containers	0.3 (0.1–1.0)
			Male sex	0.5 (0.3–0.9)
Cifuentes et al. (2000)	Mexico		Storing drinking water in unprotected containers	1.76 (1.0–3.2)
			Lack of facilities for faeces disposal	1.2 (1.0–1.5)
			Purchasing vegetables at the city market vs the village shop	2.5 (1.0–6.2)
Prado et al. (2003)	Salvador, Brazil	2–45 months	Number of children in the household under five years	2.1 (1.3–3.3)
			Rubbish not collected from the house	2.0 (1.2–3.2)
			Presence of visible sewage nearby	1.9 (1.2–3.0)
			Absence of a toilet	2.5 (1.3–4.7)
Cifuentes et al. (2004)	Mexico		During the wet season, storing water in unprotected receptacles such as uncovered jars, cisterns or tanks, and buckets	Various
			Using a tap to bathing outside the dwelling	1.9 (1.1–3.4)
			Unsafe food hygiene practices (OR = and those with)	2.4 (1.1–5.3)
			Poor hand-washing	2.3 (1.0–5.2)

are likely to be due to human contamination (Hunter, 1997).

In general, the conclusions of the epidemiology that zoonotic transmission pathways are of primary importance for giardiasis are not supported by the evidence of genotyping studies (as discussed above). The finding that most types of *Giardia* are adapted to a single host species reduces the likelihood that zoonotic transmission pathways are going to play a major role. On the other hand, the report by Traub et al. (2004) shows that at least in some communities with inadequate sanitation some *Giardia* genotypes can circulate between humans and dogs, though it is not clear whether man or dog is the primary reservoir in this context.

3. Future perspectives

The next big advance in our understanding of the epidemiology of cryptosporidiosis and giardiasis is likely to come from more detailed characterisation of strains below the level of species and genotype (sub-genotype). Indeed, a complicating factor in our understanding of zoonotic transmission in *Cryptosporidium* and *Giardia* is the recent demonstration of the existence of sub-genotypes in both *C. parvum* and *G. duodenalis* (Hopkins et al., 1999; Alves et al., 2003; Leoni et al., 2003; Mallon et al., 2003; Peng et al., 2003; Xiao et al., 2004; Lalle et al., 2005). The epidemiological significance of such sub-genotypes is not understood and most research to date has been carried out on sub-genotypes of *C. parvum*. Although it is early days, one of the most interesting studies to date comes from work by Mallon et al. (2003) in Glasgow using microsatellite polymorphisms. These authors used three minisatellite and four microsatellite markers and were able to identify some 38 genotypes. Of particular interest was that within *C. parvum* (cattle genotype) there were three major groups, only one of which was found in cattle. The authors suggested that this indicates that there are sub-genotypes of *C. parvum* (GT2) that are adapted to purely human carriage. It is too early to say whether the authors are correct or not. The study looked at 180 isolates of which 44 were from cattle. All isolates were collected in Scotland. It is certainly possible that the authors have identified human only strains of *C. parvum* (GT2) though we will need to see similar studies from elsewhere before we can accept this hypothesis. It may be that the 'human' strains of *C. parvum* are zoonotic, but related to other animal species or cattle in other parts of the world. Alternatively, if some sub-genotypes of *C. parvum* are restricted to humans, then the occurrence of *C. parvum* in water is not necessarily indicative of a non-human source of contamination. Indeed, some sub-genotypes of *C. parvum* may only be capable of infecting humans and possibly others only cattle or other species of mammal. This obviously raises questions concerning their taxonomic status and emphasises the need for caution in describing species before sufficient information on host range has been obtained.

4. Conclusions

The results of epidemiological studies and strain characterisation certainly support the hypothesis that *C.*

hominis is spread only between humans but that the major reservoir for *C. parvum* is domestic livestock, predominantly cattle, and that direct contact with infected cattle is a major transmission pathway along with indirect transmission through drinking water. The situation is less clearcut for *Giardia* but the evidence does not, in general, support zoonotic transmission as a major risk for human infections.

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