Chapter 4
Cryptosporidiosis in Farmed Animals

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Abstract Cryptosporidiosis was first identified as a disease of veterinary, rather than human medical, importance, and infection of farmed animals with different species of *Cryptosporidium* continues to be of veterinary clinical concern. This chapter provides insights into *Cryptosporidium* infection in a range of farmed animals – cattle, sheep, goats, pigs, cervids, camelids, rabbits, water buffalo and poultry – presenting not only an updated overview of the infection in these animals, but also information on clinical disease, infection dynamics and zoonotic potential. Although extensive data have been accrued on, for example, *Cryptosporidium parvum* infection in calves, and calf cryptosporidiosis continues to be a major veterinary concern especially in temperate regions, there remains a paucity of data for other farmed animals, despite *Cryptosporidium* infection causing

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significant clinical disease and also, for some species, with the potential for transmission of infection to people, either directly or indirectly.

4.1 Introduction

4.1.1 *Species of* Cryptosporidium *Relevant to Different Farmed Animals: Overview*

Farmed animals, also commonly referred to as livestock or domesticated animals, are those animals that are reared in an agricultural setting in order to produce various commodities – usually food (meat, organs, eggs, dairy products), and/or hair or wool. In some settings farmed animals are also used to supply labour, and the manure of domesticated animals is often used as fertilizer. Animals were probably first farmed, that is their breeding and living conditions controlled by their human owners, around 7000–8000 BC during the first transitions from hunter-gatherer lifestyles to more settled agricultural living. The physiologies, behaviours, lifecycles of farmed animals generally differ quite substantially from those characteristics of the equivalent wild animals, and this difference impacts the interactions of these farmed animals with their parasites. Farmed animals are exposed to different stresses than wild animals, are kept at different densities, and their lifecycles regulated to such an extent that a parasite-host interaction in a farmed animal may differ significantly from that in a wild animal. Additionally, for infections that are of significant clinical importance, farmers may implement control measures (including treatment or prophylaxis) that alter the infection dynamics. With respect to *Cryptosporidium* infection, for which a satisfactory chemotherapeutic cure or prophylaxis is not yet available, different species infect different species of farmed animal, and may or may not be of clinical relevance.

Table 4.1 provides an overview of the farmed animals included in this chapter, the species of *Cryptosporidium* to which they are susceptible and brief notes on the clinical relevance. Greater details are provided in the appropriate chapter sections. Various categories of animals that are 'farmed', including mink, foxes, guinea pigs etc. are not included, largely because of a lack of information on *Cryptosporidium* in these animals in the domesticated setting. Additionally, farmed fish are not included in this chapter.

4.1.2 *Relevance of Cryptosporidiosis in Farmed Animals to Human Infections*

While cryptosporidiosis in farmed animals is of veterinary relevance, resulting in clinical morbidity, mortality, and associated production losses, the zoonotic nature

Table 4.1 Overview of farmed animals and major relevant species of *Cryptosporidium*

Farmed animal	Species of *Cryptosporidium*	Clinical notes
Bovines, including cattle (*Bos taurus* and *B. indicus*), banteng (*Bos javanicus*), gayal (*Bos frontalis*), water buffalo (*Bubalus bubalis*), and yaks (*Bos grunniens*)	*C. parvum*[a]	Common in pre-weaned calves – acute onset diarrhoea. Intestinal location
	C. bovis	Common in post-weaned calves – less pathogenic than *C. parvum*
	C. andersoni	Older post-weaned calves, yearlings and adults- some failure to thrive. Infects the gastric glands of the abomasum
	C. ryanae	Common in post-weaned calves
	A range of other species has been reported from cattle and other bovines. These seem to be unusual and are apparently of minor clinical significance	
Small ruminants, including sheep (*Ovis aries*) and goats (*Capra aegagrus hircus*)	*C. parvum*[a]	Relatively common in pre-weaned lambs, associated with diarrhoea
	C. xiaoi	Common in older lambs and sheep, often apparently asymptomatic
	C. ubiquitum[a]	Common in older lambs and sheep, often apparently asymptomatic
Pigs (*Sus scrofa domesticus*)	*C. parvum*[a]	Less common than in bovines and small ruminants; diarrhoea and vomiting
	C. suis	Relatively common, mild symptoms
	C. scrofarum	Relatively common, mild symptoms
Deer (cervids), including red deer (*Cervus elaphus*) fallow deer (*Dama dama*), elk/wapiti (*Cervus canadensis*), white-tailed deer (*Odocoileus virginianus*), and reindeer (*Rangifer tarandus*)	*C. parvum*[a] *C. ubiquitum*[a]	Information on species detected amongst farmed deer is lacking; diarrhoea in young calves, possibly severe, but can also be asymptomatic
Camelids, including dromedaries (*Camelus dromedarius*), llama (*Lama glama*), and alpaca (*Lama pacos*)	*C. parvum*[a]	Relatively little information on species that are infectious to camelids; diarrhoeal disease, particularly in young alpaca (crias)
Rabbits (*Oryctolagus cuniculus*)	*C. cuniculus*[a]	Clinical symptoms in rabbits are apparently mild or lacking
Poultry, including chickens (*Gallus gallus domesticus*), ducks (*Anas platyrhynchos*), turkeys (*Meleagris gallopavo*), geese (*Anser anser domesticus*), ostriches (*Struthio camelus*), pigeons (*Columba livia domestica*) etc.	*C. meleagridis*[a]	Appears to have a wide host range, including farmed poultry (and mammals). Mostly infects the intestines and has been associated with generally mild clinical symptoms
	C. baileyi[a]	A wide avian host range reported, including various farmed poultry species. Detected in many different anatomical sites including digestive tract, respiratory tract, and urinary tract. Has been associated with high morbidity and mortality

(continued)

Table 4.1 (continued)

Farmed animal	Species of *Cryptosporidium*	Clinical notes
	C. galli	May affect a wide range of avian species, including farmed poultry. Infects the proventriculus, and has been associated with acute diarrhoeal disease
	Various host species-associated genotypes have been described from different poultry, including ducks and geese. Their clinical importance and host specificity is generally not known	

[a]Noted for zoonotic potential

of various *Cryptosporidium* species (see Table 4.1) means that public health may also be affected by infections in farmed animals. Infection may be direct, from animal to human, or indirect, via a transmission vehicle. A large number of small outbreaks associated with *C. parvum* in calves and in veterinarians or veterinary students that have been exposed to calf faeces are documented in the literature (e.g. Grinberg et al. 2011; Gait et al. 2008; Robertson et al. 2006). In addition, a number of outbreaks have been documented associated with children visiting 'petting farms' or similar venues, where interaction with young animals such as lambs or calves is encouraged. Less commonly, transmission between animals such as camels or alpacas and their carers has also been reported. Drinking water, and less often food, has been associated with transmission of *Cryptosporidium* infection from animals to human populations, with *C. parvum* from grazing cattle contaminating water supplies particularly implicated. The high densities of farmed animals in water catchment areas mean that implementation of catchment control measures, including preventing defecation into water courses, may have a significant effect on minimising the risk from this potential transmission pathway.

4.2 *Cryptosporidium* Infection in Bovines

There are various species of farmed bovines, with cattle and zebu (*Bos taurus* and *Bos indicus*, respectively) amongst the most important livestock worldwide. Both provide meat, milk and other dairy products, and are also used as draught animals, with an estimated 1.5 billion head globally (3 cattle for every 14 people). A comprehensive overview of *Cryptosporidium* infection in cattle has been published by Santín and Trout (2008) and the information presented here is largely an update built on this solid basis. Domesticated water buffalo (*Bubalus bubalis*) is also important domestic livestock in the bovinae sub-family. Domesticated buffalo consist of swamp buffalo and river buffalo. In 2011, the world population of buffalo was estimated to approximately 195 million animals, of which 97 % were in Asia (FAOSTAT 2012).

4.2.1 Occurrence (Prevalence)

4.2.1.1 Cattle

The first report on bovine cryptosporidiosis was published in 1971, when parasites were identified in an 8-month-old heifer with chronic diarrhoea (Panciera et al. 1971). Since then, *Cryptosporidium* infection in cattle has been documented in most countries worldwide. Four major *Cryptosporidium* species infect cattle: *C. parvum*, *C. bovis*, *C. ryanae* and *C. andersoni* (Table 4.2; Fayer et al. 2007; Feng et al. 2007; Santín et al. 2004; Langkjaer et al. 2007). *Cryptosporidium parvum* has a broad host range and apparently has the ability to infect most mammals, including humans and cattle. In contrast, the other three species have almost exclusively been found in cattle. In addition to these four common species, sporadic natural infections with *C. felis*, *C. hominis*, *C. scrofarum*, *C. serpentis*, *C. suis* and *C. suis*-like genotype have been detected in cattle (Bornay-Llinares et al. 1999; Geurden et al. 2006; Langkjaer et al. 2007; Santín et al. 2004; Smith et al. 2005; Chen and Huang 2012). The extent to which these findings reflect true infections or accidental carriage, i.e. ingested oocysts that pass intact through the gastrointestinal tract, remains to be clarified. Cattle have also been experimentally infected with *C. canis*, but natural infection has not been reported (Fayer et al. 2001).

Many reports on *Cryptosporidium* in cattle in different countries and settings have been published over the years, showing that *Cryptosporidium* spp. infections are common worldwide. Both dairy and beef cattle are infected and the prevalence estimates vary considerably among studies. Reported herd level prevalences range from 0 to 100 % (Olson et al. 1997; Chang'a et al. 2011; Maddox-Hyttel et al. 2006; Santín et al. 2004). Infected animals have been reported from all age groups but infection is most common in preweaned calves. When calves up to 2 months of age have been investigated in point prevalence surveys, 5–93 % of the calves shed oocysts (Table 4.2; Maddox-Hyttel et al. 2006; Santín et al. 2004; Uga et al. 2000). Longitudinal studies performed in infected dairy herds showed that all calves in such herds shed oocysts at some time during their first months of life (O'Handley et al. 1999; Santín et al. 2008).

The overall picture is that there is an age-related pattern in the species distribution. *C. parvum* is mostly found in preweaned, monogastric calves up to 2 months of age where it is often the most prevalent species, responsible for more than 80 % of *Cryptosporidium* infections (Brook et al. 2009; Fayer et al. 2007; Plutzer and Karanis 2007; Santín et al. 2004, 2008; Trotz-Williams et al. 2006). In some areas, however, *C. bovis* is the dominating species found in preweaned calves (Budu-Amoako et al. 2012a; Wang et al. 2011a; Silverlås et al. 2010b). The prevalence of *C. parvum* is considerably lower in older calves and young stock, and there are few reports of *C. parvum* infection in adult cows (Fayer et al. 2007; Langkjaer et al. 2007; Silverlås et al. 2010b; Castro-Hermida et al. 2011a; Khan et al. 2010; Ondrácková et al. 2009; Muhid et al. 2011; Budu-Amoako et al. 2012a, b).

Table 4.2 Location, age, and prevalence of *Cryptosporidium* spp. in cattle in some recent studies[a]

Location	Age	No. of animals/farms or locations	Prevalence	Microscopy (M), ELISA (E), Molecular (Mo) or Other (O)	Molecular identification/ species or genotype (% investigated)	Reference
Australia, NSW	Calves	196/20 herds	74 %	Mo	18S rRNA and GP60/ *C. parvum* 59 % *C. bovis* 20 % *C. ryanae* 10 % Mixed infections 10 % Not identified 1 %	Ng et al. 2012
Brazil	≤30 days	196/dairy herds	11 %	Mo	18S rRNA and GP60/ *C. parvum* 33 % *C. bovis* 5 % *C. ryanae* 10 % *C. andersoni* 10 % Not identified 42 %	Meireles et al. 2011
Canada	<2 months 2–6 months >6 months	752/20 dairy herds	17 % 14 % 15 %	M	18S rRNA/ *C. parvum* 5 % *C. bovis* 51 % *C. ryanae* 17 % *C. andersoni* 27 %	Budu-Amoako et al. 2012a
Canada	≤6 months >6 months	739/20 beef herds	18 % 15 %	M	18S rRNA and HSP70/ *C. parvum* 24 % *C. bovis* 20 % *C. ryanae* 7 % *C. andersoni* 49 %	Budu-Amoako et al. 2012b
Czech Republic	20–60 days	750/24 dairy herds	21 %	M	18S rRNA RFLP/ *C. parvum* 86 % *C. bovis* 2 % *C. andersoni* 13 %	Kváč et al. 2011

China	0–>48 months	2,056/14 dairy herds	19 %	M	18s rRNA/ *C. parvum* 48 % *C. bovis* 16 % *C. andersoni* 29 % *C. hominis* 6 % *C. serpentis* 1 %	Chen and Huang 2012
China	0–8 weeks	801/8 herds	21 %	M	18s rRNA and GP60/ *C. parvum* 31 % *C. bovis* 38 % *C. ryanae* 11 % *C. andersoni* 7 % Mixed infections 12 %	Wang et al. 2011b
England and Wales	≤3 months	229 dairy or beef calves/diagnostic lab	45 %	M	18s rRNA/ *C. parvum* 91 % *C. bovis* 2 % Not identified 7 %	Featherstone et al. 2010a
England and Wales	Preweaned Immature Adult	116/11 herds connected with human cryptosporidiosis	81 % 58 % 19 %	M	18s rRNA/ *C. parvum* 77 % *C. bovis* 5 % *C. andersoni* 16 % Not identified 2 %	Smith et al. 2010
Egypt	<6 weeks	96/2 dairy herds	30 %	M	18S rRNA and COWP *C. parvum* 93 % *C. andersoni* 7 %	Amer et al. 2010
Egypt	1 day–3 months >3 months–1 year >1–2 years >2 years	593	30 % 13 % 13 % 5 %	O	18S rRNA and GP60/ *C. parvum* 65 % *C. bovis* 4 % *C. ryanae* 14 % Mixed infections17%	Helmy et al. 2013

(continued)

Table 4.2 (continued)

Location	Age	No. of animals/farms or locations	Prevalence	Microscopy (M), ELISA (E), Molecular (Mo) or Other (O)	Molecular identification/ species or genotype (% investigated)	Reference
Hungary	Preweaned calves	79 diarrhoeic/52 herds	49 %	M	18S rRNA and GP60/ *C. parvum* 95 % *C. ryanae* 5 %	Plutzer and Karanis 2007
India	<3 months	461/various	16 %	M	18s sRNA/ *C. parvum* 100 %	Maurya et al. 2013
India	0–2 months 3–12 months >12 months	180/2 dairy herds	20 % 14 % 4 %	M	18S rRNA/ *C. parvum* 29 % *C. bovis* 38 % *C. ryanae* 14 % C. *andersoni* 14 % *C. suis*-like 5 %	Khan et al. 2010
Iran	1–20 weeks	272/15 dairy herds	19 %	M	18S rRNA/ *C. parvum* 73 % *C. bovis* 8 % C. *andersoni* 18 % Atypical isolates 2 %	Keshavarz et al. 2009
Italy	0 day–<12 months	2,024/248 dairy and beef herds	8 %	ELISA, M	COWP and GP60/ *C. parvum* 100 %	Duranti et al. 2009
Japan	3–48 days	80 diarrhoeic/different herds	75 %	Mo	18S rRNA/ *C. parvum* 53 % *C. bovis* 2 % Not identified 45 %	Karanis et al. 2010
Malaysia	1 day–≤4.5 months >4.5–12 months	250/16 herds	31 % 23 %	Mo	18S rRNA and GP60/ *C. parvum* 17 % *C. bovis* 25 % *C. ryanae* 15 %	Muhid et al. 2011

					C. andersoni 20 % Mixed infections 6 % Not identified: 17 %	
Nigeria	2–365 days	194/20 herds	16 %	Mo	18S rRNA/ *C. bovis* 45 % *C. ryanae* 26 % *C. andersoni* 16 % Mixed infections 13 %	Maikai et al. 2011
Northern Ireland	<1 month	779 diarrhoeic/ diagnostic lab	37 %	M	18S rRNA/ *C. parvum* 95 % *C. bovis* 4 % *C. ryanae* 1 %	Thompson et al. 2007
Romania	1–30 days	258 diarrhoeic/9 dairy herds	25 %	M	18S rRNA and GP60/ *C. parvum* 100 %	Imre et al. 2011
Spain	Neonatal Heifers Cows	649/not specified	61 % 15 % 8 %	M	18S rRNA/ *C. parvum* 56 % *C. andersoni* 23 % Not identified 21 %	Castro-Hermida et al. 2011a
Spain	≤21 days	61 diarrhoeic/27 herds	49 %	M	18S rRNA and GP60 *C. parvum* 100 %	Díaz et al. 2010a
Sweden	≤2 months 4–12 months Cows	1,202/50 dairy herds	52 % 29 % 6 %	M	18S rRNA and GP60/ *C. parvum* 14 % *C. bovis* 75 % *C. ryanae* 9 % *C. andersoni* 2 %	Silverlås et al. 2009b; Silverlås et al. 2010b
USA 7 states	5 days–2 months 3–11 months	971/15 dairy herds	50 % 20 %	M	18S rRNA/ *C. parvum* 50 % *C. bovis* 28 % *C. ryanae* 16 % *C. andersoni* 6 %	Santín et al. 2004

(continued)

Table 4.2 (continued)

Location	Age	No. of animals/farms or locations	Prevalence	Microscopy (M), ELISA (E), Molecular (Mo) or Other (O)	Molecular identification/ species or genotype (% investigated)	Reference
USA 7 states	12–24 months	571/14 dairy herds	12 %	Mo	18S rRNA/ *C. parvum* 6 % *C. bovis* 35 % *C. ryanae* 15 % *C. andersoni* 43 % *C. suis* 1 %	Fayer et al. 2006
USA 20 states	6–18 months	819/49 beef herds	20 %	Mo	18S rRNA/ *C. bovis* 23 % *C. ryanae* 9 % *C. andersoni* 68 %	Fayer et al. 2010

[a]See e.g. Santín and Trout (2008) for a review of older studies

In older calves and young stock, *C. bovis* and *C. ryanae* are the most commonly found species (Santín et al. 2008; Fayer et al. 2007; Muhid et al. 2011; Langkjaer et al. 2007; Silverlås et al. 2010b). *C. andersoni* is mainly found in young stock and adult cattle (Enemark et al. 2002; Wade et al. 2000; Fayer et al. 2007; Ralston et al. 2003). Older studies, based on microscopy alone, overestimated the *C. parvum* prevalence in weaned animals because the similarity in oocyst size makes it impossible to differentiate between *C. parvum* (~5.0 × 4.5 μm), *C. bovis* (~4.9 × 4.6 μm) and *C. ryanae* (~3.7 × 3.2 μm).

Molecular studies have revealed different genetic subtypes within the *C. parvum* and *C. hominis* species and DNA sequence analysis of the 60 kDa glycoprotein gene is commonly used to further characterize the isolates. A number of *C. parvum* GP60 subtype families, designated IIa-IIo, have been described. Of these, IIc and IIe are considered anthroponotic, whereas IIa and IId are commonly found in both humans and animals. The other subtype families are uncommon and their zoonotic potential has not been determined. In cattle, *C. parvum* of the IIa subtype family is especially common. In addition to IIa, IId and occasionally IIl (sometimes named IIj) subtypes are found (Table 4.3; Xiao 2010). Whether there is a difference in pathogenicity between subtypes is currently unknown, as most genotyping studies to date have focused on herds with a history of calf diarrhoea. It has been suggested that herd management strategies affect subtype distribution. Studies from areas with closed herd management (limited animal movements between herds) have shown a high number of subtypes in the calf population, but only one subtype in each herd (Brook et al. 2009; Mišic and Abe 2007; Soba and Logar 2008; Silverlås et al. 2013). It has also been shown that a unique GP60 subtype can persist over time in a closed dairy herd (Björkman and Mattsson 2006). In contrast, only a few subtypes have been identified in areas with more animal movements between herds, but several subtypes could be present in a herd (Brook et al. 2009; Peng et al. 2003; Trotz-Williams et al. 2006).

4.2.1.2 Water Buffalo

The information on the distribution of *Cryptosporidium* infection in water buffalo is rather fragmentary. Reported prevalences vary between 3 % and 38 % (Table 4.4). An association between prevalence of infection and age of the animals, with the highest prevalence in young calves, has been found (Helmy et al. 2013; Maurya et al. 2013; Nasir et al. 2009; Bhat et al. 2012; Díaz de Ramírez et al. 2012).

The first report on *Cryptosporidium* species identification in water buffalo was published in 2005, in which Gómez-Couso et al. (2005) used molecular tools to characterize *Cryptosporidium* oocysts from an asymptomatic neonatal calf in a dairy buffalo farm in Spain. Sequence analysis of a fragment of the oocyst wall protein (COWP) gene revealed that the isolate was closely related to the *Cryptosporidium* 'pig' genotype. A few years later, *C. parvum* was identified in water buffalo calves from Italy (Cacciò et al. 2007) and today *C. ryanae*, *C. bovis* and

Table 4.3 *Cryptosporidium parvum* GP60 subtypes in cattle in some recent studies[a]

Country	Number subtyped	IIa subtypes[b]	IId subtypes[b]	Reference
Australia	36	IIaA16G3R1 (1), IIaA17G4R1 (4), IIaA18G3R1 (29), IIaA20G3R1 (2)		Waldron et al. 2011
Australia	84	IIaA18G3R1 (57), IIaA19G3R1 (11), IIaA17G2R1 (7), IIaA19G2R1 (6), IIaA16G3R1 (2), IIaA20G3R1 (1)		Ng et al. 2012
Brazil	7	IIaA15G2R1 (7)		Meireles et al. 2011
China	67		IIdA19G1 (54)	Wang et al. 2011b
Czech Republic	1	IIaA16G1R1 (1)		Ondráčková et al. 2009
Czech Republic	131	IIaA16G1R1 (56), IIaA15G2R1 (48), IIaA22G2R1 (12), IIaA15G1R1 (12), IIA18G1R1 (3)		Kváč et al. 2011
England and Wales	13	IIaA17G1R1(10), IIaA15G2R1 (3)		Smith et al. 2010
Egypt	24	IIaA15G2R1 (1)	IIdA20G1 (23)	Amer et al. 2010
Egypt	71	IIaA15G1R1 (16)	IIdA20G1 (54), IIdA19G1 (1)	Helmy et al. 2013
Hungary	21	IIaA16G1R1 (15), IIaA17G1R1 (3), IIaA18G1R1 (1)	IIdA19G1(1), IIdA22G1 (1)	Plutzer and Karanis 2007
Iran	25	IIaA15G2R1 (22), IIaA16G3R1 (1)	IIdA15G1 (2)	Nazemalhosseini-Mojarad et al. 2011
Italy	62	IIaA15G2R1 (34), IIaA18G2R1 (10), IIaA17G2R1 (9), IIa A14 (5), IIaA13 (4)		Duranti et al. 2009
Malaysia	8	IIaA17G2R1 (2), IIaA18G3R1 (1)	IIdA15G1 (5)	Muhid et al. 2011
Romania	13	IIaA15G2R1 (8), IIaA16G1R1 (5)		Imre et al. 2011
Spain	27	IIaA15G2R1 (26), IIaA13G1R1 (1)		Díaz et al. 2010a
Sweden	13	IIaA15G1R1 (2), IIaA18G1R1 (2), IIaA21G1R1 (2), IIaA16G1R1 (1)	IIdA20G1 (2), IIdA23G1 (2) IIdA16G1 (1), IIdA22G1 (1)	Silverlås et al. 2010b

Sweden	171	IIaA16G1R1 (58), IIaA17G1R1c (17), IIaA21G1R1 (12), IIaA17G1R1 (6), IIaA18G1R1c (7), IIaA22G1R1 (7), IIaA20G1R1 (5), IIaA15G2R1 (4), IIaA13G1R1 (3), IIaA18G1R1 (3), IIaA14R1 (2), IIaA16G1R1b (2), IIaA18G1R1d (2), IIaA13G1R2 (1), IIaA14G1R1b (1), IIaA17R1 (1), IIaA21G1R1b (1), IIaA23G1R1 (1)	IIdA20G1e (14), IIdA16G1b (4), IIdA22G1 (4), IIdA24G1c (4), IIdA22G1c (3), IIdA23G1 (3), IIdA17G1d (2), IIdA19G1 (2), IIdA26G1b (2)	Silverlås et al. 2013

[a]See Xiao et al. (2010) for a summary of results of older studies
[b]Numbers in parentheses are number of samples with the subtype

Table 4.4 Location, age, and prevalence of *Cryptosporidium* spp. and *C. parvum* subtypes in water buffalo[a]

Location	Symptoms	Host age	Study design	Prevalence % positive (no. positive/no. examined)	Diagnostic technique	Molecular analyses gene/species or subtype[b]	Reference
Egypt	Diarrhoeic and non-diarrhoeic	<3 months	Cross-sectional	Individual 14 % (65/458) Herd 55	Modified Ziehl-Neelsen (mZN)		El-Khodery and Osman 2008
Egypt	Diarrhoeic and non-diarrhoeic	Not stated	Cross-sectional	22 % (16/71)	mZN and Sheather's flotation followed by mZN		Shoukry et al. 2009
Egypt	Not stated	1 week–4 months and adults	Cross-sectional	Calves: 10 % (17/179) Adults: 0 Herds: 71 % (5/7)	Sheather's flotation followed by mZN	18S rRNA/ *C. parvum* (7) *C. ryanae* (10) GP60/ IIdA20G1 (5) IIaA15G1R1 (2)	Amer et al. 2013
Egypt	Not stated for calves, diarrhoeic adults	Calves and adults	Cross-sectional	Overall: 20 %. (43/211) 1 day–3 months: 40 % (34/85) <3 months–1 year: 11 % (6/56) >1 year: 4 % (3/70)	Antibody based copro-antigen test	18S rRNA/ *C. parvum* (30) *C. bovis* (2) *C. ryanae* (3) Mixed infections (10) GP60/ IIdA20G1 (28) IIaA15G1R1 (4)	Helmy et al. 2013
India	Not stated	Not stated	Cross-sectional	17 % (10/60)	Kinyon acid fast stain		Dubey et al. 1992

India	Diarrhoeic and non-diarrhoeic	Not stated	Cross-sectional	25 % (76/305)	mZN and formol-ether sedimentation followed by mZN		Mohanty and Panda 2012
India	Diarrhoeic and non-diarrhoeic	<5 months	Cross-sectional	Overall prevalence: 38 % (62/162) 1-2 months: 49 % (18/37) 2–3 months: 34 % (13/38) 3–4 months: 20 % (76/305) 4–5 months: 6 % (1/17)	mZN		Bhat et al. 2012
India	Diarrhoeic and non-diarrhoeic	<3 months	Cross-sectional	24 % (64/264)	mZN or Sheather's flotation followed by mZN	18S rRNA/ *C. parvum* (16)	Maurya et al. 2013
Nepal	Not stated	2–7 months	Cross-sectional	37 % (30/81)	Water-ethyl sedimentation followed by mZN	18S rRNA/ *C. ryanae* (16) Unique variant	Feng et al. 2012
Pakistan	Diarrhoeic and non-diarrhoeic	1 day–>1 year	Cross sectional	Overall prevalence: 24 % (60/250) 1–30 days: 42 % (29/69) 1–3 months: 26 % (17/66) 4–8 months: 15 % (9/60) 9 months->1 year: 9 % (5/55)	Sheather's flotation followed by mZN		Nasir et al. 2009

(continued)

Table 4.4 (continued)

Location	Symptoms	Host age	Study design	Prevalence % positive (no. positive/no. examined)	Diagnostic technique	Molecular analyses gene/species or subtype[b]	Reference
Philippines	Not stated	1–12 days	Longitudinal	3 % (1/38)	Formalin ether sedimentation followed by Kinyon acid fast stain		Villanueva et al. 2010
Venezuela	Not stated	0–12 weeks	Longitudinal	88 % (22/25)	Formalin-ethylacetate sedimentation and sodium chloride flotation followed by carbol-fuchsin staining		Díaz de Ramírez et al. 2012
Italy	Asymptomatic	Calves	Cross-sectional	14 % (8/57)	ELISA, followed by formol-ether extraction and IFAT	18S rRNA/ *C. parvum*	Cacciò et al. 2007
Italy	Not stated	2–60 days/ 43 farms	Cross-sectional	20 % (35/177) Herds 35 % (15/43)	ELISA		Rinaldi et al. 2007a
Italy	Not stated	1–9 weeks/ 90 farms	Cross-sectional	15 % (51/347) Herd 24 % (22/90)	ELISA		Rinaldi et al. 2007b

[a]A few studies are not included in the table due to inaccessibility of the publications

C. ubiquitum have also been reported (Table 4.4). Most molecular investigations have been done in calves and thus it is not known if the species distribution differs between animals of different age. One of the few studies that include faecal samples from both calves and older animals was done in Egyptian smallholder herds (Helmy et al. 2013). *C. parvum* was most common in calves younger than 3 months but was detected in animals up to 2 years of age. In another study, also from Egypt but from another part of the country, *C. parvum* was only found in calves, whereas none of the sampled cows shed any *Cryptosporidium* oocysts (Amer et al. 2013). When the *C. parvum* isolates were subtyped by sequence analysis of the GP60 gene, subtype families IId and IIa were found, with a majority of IId in both studies. Subtype IId is the dominating subtype family also in cattle in Egypt (Amer et al. 2010; Helmy et al. 2013).

4.2.2 Association of Infection with Clinical Disease

4.2.2.1 Cattle

Cryptosporidium parvum and *C. andersoni* are the two species that have been associated with clinical disease in cattle. *C. parvum* infection is considered a major cause of diarrhoea in young calves (Blanchard 2012; Radostits et al. 2007). Calves are often already infected during the first week of life (Uga et al. 2000) and clinical cryptosporidiosis is mostly seen in calves up to 6 weeks of age. The most prominent finding is pasty to watery diarrhoea, sometimes accompanied by lethargy, inappetence, fever, dehydration and/or poor condition. The calf most often recovers spontaneously within 1–2 weeks, but there is a large variation between individuals in how they respond to, and recover from, infection. In some cases the infection may be fatal (Tzipori et al. 1983; Fayer et al. 1998). A decrease in growth rate may be seen in the weeks after the calves have recovered from the acute phase of the disease (Klein et al. 2008), but no long-term effects on growth and performance have been reported. The pathogenesis of bovine cryptosporidiosis is not fully understood but the clinical signs are attributed to both malabsorption and an increase in fluid secretion in the ileum and proximal portions of the large intestine. For references and a brief overview see O'Handley and Olson (2006). Cryptosporidiosis may be seen in individual calves, but frequently it soon develops among the calves into a herd problem. Concomitant infection with other pathogens, e.g. rotavirus, coronavirus and enteropathogenic *Escherichia coli* (*E. coli* F5+) can worsen the clinical signs and prolong the duration of illness (Blanchard 2012).

A number of studies have reported an association between *C. parvum* infection and diarrhoea in young calves. Many of these were published at the time when all *Cryptosporidium* oocysts of around 3–5 μm in diameter were considered to be *C. parvum*. More recent investigations, applying molecular methods to analyse faecal samples from diarrhoeic calves, corroborate these earlier findings. When samples from young calves with diarrhoea were analysed, *C. parvum* is found to be

the dominant species (Quílez et al. 2008b; Imre et al. 2011; Karanis et al. 2010; Soba and Logar 2008; Plutzer and Karanis 2007). Interestingly, this dominance of *C. parvum* in diarrheic calves was also seen in a recent Swedish investigation of diarrheic calves (Silverlås et al. 2013), although *C. bovis* is the predominant species in randomly selected calves in Sweden.

Only one experimental trial has been performed with *C. bovis* (Fayer et al. 2005). Three calves under 1–8 weeks of age were orally inoculated with oocysts. This resulted in subclinical infection in 2 of 3 calves. Both animals had, however, previously been infected with *C. parvum* and cross-protective immunity could not be excluded. Calves with diarrhoea are significantly more likely to be infected with *C. parvum* than with *C. bovis* (Silverlås et al. 2013; Starkey et al. 2006; Kváč et al. 2011). Based on these findings, and based on the fact that *C. bovis* is not common in calves, but is a widespread subclinical infection in older animals in most countries, *C. bovis* is commonly considered to be apathogenic to cattle. However, the pathogenic potential deserves further attention as high numbers of *C. bovis* oocysts in samples from diarrhoeic calves have been reported, even in the absence of *C. parvum* or other diarrhoeal agents (Silverlås et al. 2010a, b, 2013).

Cryptosporidium ryanae was first described as a separate species by Fayer et al. (2008), and until then it was known as *Cryptosporidium* deer-like genotype. An experimental trial was performed in two colostrum-deprived calves 17–18 days old. Both calves started excreting oocysts 11 days after inoculation, but neither of them showed any clinical signs (Fayer et al. 2008). There are several reports of the distribution of *Cryptosporidium* deer-like genotype and *C. ryanae*. Most studies found a predominance of the parasite in older calves and young stock. So far no association with clinical disease has been reported.

In contrast to the other species, *C. andersoni* infects the abomasum. It does not cause diarrhoea, but *C. andersoni* infections have been associated with maldigestion. The infection may cause moderate to severe weight gain impairment in young stock and reduced milk production in cows (Anderson 1998; Esteban and Anderson 1995; Lindsay et al. 2000).

A major obstacle from a disease control perspective is the lack of effective means to control *Cryptosporidium* infection and decrease the level of contamination of the environment with oocysts. Preventive hygiene measures and good management are currently the most important tools to control cryptosporidiosis. Reducing the number of oocysts ingested by neonatal calves may reduce the severity of infection and allow immunity to develop. A common recommendation is to ensure good hygiene in calf facilities and ascertain that all newborn calves ingest an adequate amount of colostrum during their first 24 h of life. Sick calves should be housed in a clean, warm, and dry environment and isolated to prevent spreading of the infection to other calves. Acutely infected animals may need supportive care with fluid and electrolytes, and milk should be given in small quantities several times daily to optimise digestion and minimise weight loss.

Over the years, several substances have been tested for potential anti-cryptosporidial effects with limited success (Santín and Trout 2008). Halofuginone lactate has shown some beneficial effects such as milder clinical signs and reduced

oocyst output when used as prophylactic treatment (De Waele et al. 2010; Silverlås et al. 2009a). This drug is approved in Europe to treat calf cryptosporidiosis. However, the safety margin is narrow and the substance is toxic at only twice the therapeutic dose, so careful dosage is necessary. Halofuginone lactate treatment should only be considered in herds with severe diarrhoeal problems strongly associated with *C. parvum*. When treatment is used, it should always be in conjunction with applying measures to reduce environmental contamination and risk of infection.

A recent study investigated if an antibody-biocide fusion consisting of a monoclonal antibody "armed" with membrane-disruptive peptides (biocides) could be used for treatment of cryptosporidiosis in calves (Imboden et al. 2012). Calves 36–48 h of age were challenged once with *C. parvum* oocysts and were simultaneously administered the antibody-biocide fusion mixed with milk replacer. The antibody-biocide fusion treatment was repeated 5–8 times. Control calves were given milk replacer with placebo. Calves receiving the antibody-biocide fusion had a significantly higher health score and shed fewer oocysts than control calves. These results suggest that this concept might be effective in cattle, but further testing is necessary (Imboden et al. 2012).

Vaccination is successfully used to control many infectious diseases in livestock. However, it takes weeks for a protective immune response to develop after a vaccine has been administered, and as calves may be exposed to *Cryptosporidium* oocysts immediately after birth, vaccination of newborn calves is unlikely to be successful in preventing cryptosporidiosis. Thus it has been suggested that the most feasible approach is likely to involve passive immunisation (Innes et al. 2011). Dams are immunised in late gestation and their colostrum is fed to the calves. A recent study investigated antibody responses in calves fed colostrum from heifers vaccinated with a recombinant *C. parvum* oocyst surface protein (rCP15/60). The calves had measurable quantities of the specific antibody in their serum. However, as the calves were not subsequently challenged with oocysts it remains to be seen whether this immunisation scheme can also prevent symptomatic infection and eliminate oocyst shedding (Burton et al. 2011).

4.2.2.2 Water Buffalo

An association between *Cryptosporidium* oocyst shedding and diarrhoea in buffalo calves has been reported from investigations performed in Egypt, India and Venezuela (Mohanty and Panda 2012; Bhat et al. 2012; Maurya et al. 2013; El-Khodery and Osman 2008; Díaz de Ramírez et al. 2012) suggesting the *Cryptosporidium* infection is part of the calf diarrhoea syndrome in water buffalo, as it is in cattle. Species identification was only performed in one of these studies, and *C. parvum* was the only species that was found (Maurya et al. 2013).

4.2.3 *Infection Dynamics: Oocyst Excretion and Transmission*

4.2.3.1 Cattle

Calves begin shedding *C. parvum* oocysts 2–6 days after infection and shedding continues for 1–13 days (Fayer et al. 1998; Tzipori et al. 1983). During the first 2 weeks an infected calf can shed millions of oocysts (Fayer et al. 1998; Uga et al. 2000) resulting in heavy environmental contamination, and efficient dissemination of the parasite within the herd and to the environment. In faecal samples obtained from symptomatic calves naturally infected with *C. parvum* 10^6–10^8 oocysts per gram faeces (OPG) are often seen (Silverlås et al. 2013). In herds with established *C. parvum* infection, most calves are excreting oocysts between 2 and 4 weeks of age (O'Handley et al. 1999; Santín et al. 2008; Uga et al. 2000). When Santín et al. (2008) repeatedly sampled the same 30 calves in a dairy herd from birth to 2 years of age they found *C. parvum* oocysts in faeces of all individuals before they were 3 weeks old, i.e. a cumulative prevalence of 100 %. *C. parvum* oocysts were also found in samples collected from a calf at 16 weeks of age and from another at 6 months of age, indicating that oocysts can be shed intermittently over a long period after the initial infection. Alternatively, these late-shedding individuals might not have developed a fully protective immunity after the first infection, and rather than this being a sign of prolonged infection, they had acquired new infections. In this study, molecular analyses indicated the same sub-genotype at the GP60 locus. However, this does not necessarily indicate prolonged infection, as re-infection with the same genotype in the environment may occur if the immunity is not protective. It has been suggested that an increase in *C. parvum* oocyst shedding may occur in adult cows around calving (so called periparturient rise), but to date there have been few reports to support this. In a recent study, however, dams in a suckler beef herd were found to shed low levels of *C. parvum* oocysts around the time of calving (De Waele et al. 2012).

Only one experimental infection for each of *C. bovis* and *C. ryanae* has been reported so far. Regarding *C. bovis*, one calf shed oocysts from 10 to 28 days after infection and the other only for 1 day (day 12) (Fayer et al. 2005). For *C. ryanae*, oocyst shedding started 11 days after inoculation. Both infected calves excreted oocysts during 15 and 17 consecutive days, respectively (Fayer et al. 2008). Shorter prepatent periods have been seen for both *C. bovis* and *C. ryanae* in natural infections (Silverlås et al. 2010b; Silverlås and Blanco-Penedo 2013). No oocyst excretion rate values were determined from the experimental infections, but in naturally infected calves 300 to 8×10^6 OPG and 100–835,000 OPG have been reported for *C. bovis* and *C. ryanae*, respectively (Silverlås and Blanco-Penedo 2013).

Young stock and adults may also be infected by the larger *C. andersoni* (oocyst size ~7.4 × 5.5 μm) and may shed oocysts intermittently for many years (Olson et al. 2004; Ralston et al. 2003). A periparturient rise in *C. andersoni* oocyst shedding, seen both as increase in prevalence and in number of oocysts in faeces, has been reported (Ralston et al. 2003).

Several studies have shown that age is associated with *Cryptosporidium* infection and that young calves have the highest risk of being infected (Maddox-Hyttel et al. 2006; Santín et al. 2004; Sturdee et al. 2003; Fayer et al. 2007). This is also the age group that is most often infected with *C. parvum* and suffers from clinical cryptosporidiosis. Thus, from clinical and zoonotic perspective, knowledge on the epidemiology of cryptosporidiosis in young calves is highly valuable. When potential risk factors for *Cryptosporidium* infection in pre-weaned calves have been explored, the results differ between studies. One factor that recurs in several studies is the type of flooring in the calf housing area. In Spain, Castro-Hermida et al. (2002) found that straw on the floor or earth floors in the calf pens increased the risk for infection compared with cement flooring, and in Malaysia calves kept in pens with slatted floors and sand floors had an increased risk compared with those in pens with cement floors (Muhid et al. 2011). A protective effect of cement floors was also reported from the USA (Trotz-Williams et al. 2008). It was suggested that the reason for this protective effect of cement floors is that they facilitate thorough cleaning. This assumption corroborates the finding that a low frequency of cleaning of the calf pens increased the risk for infection (Castro-Hermida et al. 2002). It is also consistent with the finding that the use of an empty period in the calf pen between introductions of calves was associated with a lower risk for infection in Danish dairy herds (Maddox-Hyttel et al. 2006). When cows as a cause of infection were investigated, a higher risk of infection was identified in calves that were housed separately from their dams (Duranti et al. 2009), and a lower risk of infection in dairy calves kept with the cow for more than 6 h after birth (Silverlås et al. 2009b).

In one of the few reports to investigate risk factors for infection with different *Cryptosporidium* species in pre-weaned dairy calves to date (Szonyi et al. 2012), risk of infection with *C. parvum* differed to some extent from that of *C. bovis*. Both *C. parvum* and *C. bovis* were more common in the younger calves, but herd size and hay bedding were associated with an increased risk for *C. parvum* infection, whereas Jersey breed was a risk factor for *C. bovis* infection.

4.2.3.2 Water Buffalo

Experimental *Cryptosporidium* infections in water buffalo have not been reported. However, oocyst shedding dynamics were investigated in naturally infected buffalo calves in a farm located in a tropical dry forest area in Venezuela. Twenty-five calves were sampled from birth to 12 weeks of age. Oocysts were detected from day 5 and 72 % of the calves shed oocysts before they were 30 days (Díaz de Ramírez et al. 2012).

Regarding risk factors for infection, there are some reports of seasonal variations in prevalence (Bhat et al. 2012; El-Khodery and Osman 2008; Maurya et al. 2013; Mohanty and Panda 2012), and (El-Khodery and Osman 2008) identified type of flooring, frequency of cleaning and water source as risk factors for infection in small-scale herds in Egypt.

4.2.4 Zoonotic Transmission

4.2.4.1 Cattle

There are numerous reports of cryptosporidiosis outbreaks in humans after contact with infected calves. These have often involved veterinary students and students at farm schools (see, e.g., Gait et al. 2008; Grinberg et al. 2011; Pohjola et al. 1986; Robertson et al. 2006; Kiang et al. 2006), but also young children have fallen ill after visiting petting zoos or open farms (Gormley et al. 2011; Smith et al. 2004). Contact with cattle has been identified as a risk factor for disease also in case-control studies of sporadic human cryptosporidiosis (Hunter et al. 2004; Robertson et al. 2002; Roy et al. 2004). Altogether, there is plenty of evidence to conclude that *Cryptosporidium* can be transmitted from calves to humans by direct contact or by contaminated equipment. The risk for zoonotic transmission is likely to be highest in herds with *Cryptosporidium* associated calf diarrhoeal problems, where oocyst contamination in the barn can reach high levels and where contact with naïve individuals is most likely to occur. Key measures to prevent visitors becoming infected are to ensure good hygiene in the visitor area, providing suitable hand-washing facilities and ensure that they are used when workers and visitors leave the premises. *C. bovis* infections have recently been detected in a few persons living or working on cattle farms (Khan et al. 2010; Ng et al. 2012). It is not known if these were active infections and the implication of these findings is thus unclear.

As molecular typing methods become more accessible, epidemiological studies can investigate *C. parvum* GP60 subtype distribution in cattle and human populations in different regions. The reports so far indicate that in many areas the subtypes that are most common in cattle are those most often found in humans. For example, *C. parvum* IIaA15G2R1 was the predominant subtype in both bovine and human infections in Slovenia and Portugal (Soba and Logar 2008; Alves et al. 2006). In New South Wales, Australia *C. parvum* IIaA18G3R1 dominated in both calves and people living on cattle farms (Ng et al. 2012), whereas IIaA16G2R1 was the predominant genotype identified in beef cattle and humans in Prince Edward Island, Canada (Budu-Amoako et al. 2012c). Further information is provided in the review by Xiao (2010). That the same subtypes are found in cattle and humans might be taken as an indication of zoonotic transmission. However, it is important to note that even when zoonotic *C. parvum* subtypes are identified in humans, cattle are not necessarily the source of the infection. These zoonotic subtypes can circulate and propagate in the human population in addition to the anthroponotic subtypes. The occasional finding of *C. hominis* (Smith et al. 2005; Chen and Huang 2012) in cattle highlights the fact that cryptosporidiosis may be transmitted not only from cattle to humans, but also from humans to cattle.

Food-related cryptosporidiosis outbreaks have sometimes been associated with cattle. Foodborne transmission was implicated in cases of children who had drunk unpasteurized milk (Harper et al. 2002) or cider, made from apples collected in an orchard where calves from an infected herd had grazed (Millard et al. 1994).

Other outbreaks in which cattle were suspected as the source involved vegetables that had been sprayed with water that could have been contaminated with cattle faeces. Often there was only circumstantial evidence that cattle were the source of contamination, and it was not possible to exclude other potential sources (see e.g. CDC 2011; Robertson and Chalmers 2013).

Outbreaks of cryptosporidiosis associated with drinking water have often been attributed to contamination of water catchments by cattle manure. The evidence implicating cattle has sometimes been substantial (Bridgman et al. 1995; Smith et al. 1989), but for others the evidence was not conclusive. Grazing cattle or slaughterhouse effluent contaminating Lake Michigan were mentioned as two possible sources of *Cryptosporidium* oocysts in the large outbreak in Milwaukee, Wisconsin in 1993 (Mac Kenzie et al 1994), but retrospective analysis of clinical isolates revealed that it was caused by the anthroponotic species *C. hominis* (Sulaiman et al. 1998). This was also the case in the most recent outbreaks in UK (compiled by Chalmers 2012) and a large drinking waterborne outbreak in Sweden in 2010 (Anonymous 2011). Given that pre-weaned calves are the most likely age group to shed *C. parvum* oocysts, any measure to prevent waterborne zoonotic transmission should be directed towards this age group. Protective measures could be to prevent young ruminants from accessing water catchments, and compost or spread calf manure on fields where runoff cannot occur. The manure of older ruminants is generally not a zoonotic concern with respect to *Cryptosporidium*.

4.2.4.2 Water Buffalo

The seemingly common occurrence of *C. parvum* in buffalo calves highlights the potential role of water buffalo in zoonotic transmission. Thus the same precautions to prevent transmission of the parasite to humans, by direct contact or through food or water, are also applicable to water buffalo.

4.3 *Cryptosporidium* spp. in Small Ruminants

Sheep (*Ovis aries*) and goats (*Capra aegagrus hircus*) are important in the global agricultural economy – producing meat, milk and wool – both in developing countries such as India and Iran, and industrialised countries such as Australia and The United Kingdom (de Graaf et al. 1999; Noordeen et al. 2000; Robertson 2009). In 2010, the world stocks were approximately one billion sheep and 910 million goats (FAOSTAT 2013a). Asia has the largest populations of both species, with 42 % and 60 % of the total world populations, followed by Africa (FAOSTAT 2013a). According to a FAO report, over 90 % of the goat population can be found in developing countries (FAO 2012). Sheep and goats tend to be managed differently to cattle, with flocks grazing large enclosures rather than being kept indoors. There have been fewer studies on *Cryptosporidium* infection in sheep than in cattle, and

even fewer studies have been performed on goats. Nevertheless, it is known that these protozoans are economically important parasites in both ruminant species (Noordeen et al. 2000; Robertson 2009). Infection and disease was first described in 1974 for sheep (Barker and Carbonell 1974) and in 1981 for goats (Mason et al. 1981). Younger animals are more susceptible to infection than older ones, reflected in high shedding rates and diarrhoeal prevalences in lambs and kids up to 1 month of age, whereas infection in older animals is usually subclinical with lower shedding rates (Vieira et al. 1997).

4.3.1 Occurrence (Prevalence)

As for other animals, ovine and caprine *Cryptosporidium* infection can be found throughout the world. The prevalence varies widely between studies, from 0 % to 77 % in sheep and from 0 % to 100 % in goats. All age groups are susceptible, but infection is more common in lambs and kids than in older animals (Tables 4.5 and 4.6). Study design factors other than age of sampled animals, such as whether only diarrhoeal animals were sampled or not, if a point prevalence study or a longitudinal study was performed and the diagnostic method(s) used, also can affect prevalence data. The effect of using different diagnostic methods is evident in, for example, Giadinis et al. (2012; see also Tables 4.5 and 4.6) and Ryan et al. (2005), where microscopy resulted in lower prevalences than detected by ELISA and PCR, respectively. Prevalence and species distribution for studies conducted on sheep dating back to 2007 are summarised in Table 4.5. Specific data for studies on sheep published before 2007 can be found in "*Cryptosporidium* and Cryptosporidiosis" (Santín and Trout 2008; Tables 18.9 and 18.10). Prevalence rates and species distribution for all identified surveys of goats are summarised in Table 4.6.

Several species and genotypes have been identified in sheep, and the species distribution varies between studies and with age of the animals. *Cryptosporidium parvum*, *C. ubiquitum* (previously *Cryptosporidium* cervine/cervid genotype) and *C. xiaoi* (previously *C. bovis*-like genotype) are the most common species. Sporadic infection with *C. hominis*, *C. suis*, *C. andersoni*, *C. fayeri* (previously marsupial genotype I), *C. scrofarum* (previously pig genotype II), sheep genotype I, and unknown/novel genotypes have been identified (Chalmers et al. 2005; Giles et al. 2009; Karanis et al. 2007; Ryan et al. 2005, 2008; Sweeny et al. 2011b; Wang et al. 2010c). Species distribution differs between studies and between age groups within studies. For instance, *C. parvum* is commonly found in lambs in Italy, Romania, Spain and the UK (Díaz et al. 2010a; Imre et al. 2013; Mueller-Doblies et al. 2008; Paoletti et al. 2009). In other studies, *C. ubiquitum* or *C. xiaoi* is the most common species (Fiuza et al. 2011a; Geurden et al. 2008; Robertson et al. 2010; Sweeny et al. 2011b). For example, Wang et al. (2010c) identified *C. ubiquitum* in 90 % of all analysed samples, and the species dominated in all age groups, whereas Sweeny et al. (2011b) found *C. xiaoi* to be the most common

Table 4.5 Studies on prevalence *Cryptosporidium* infections in sheep published in 2007–2012

Country	Number of animals	Age	Animal prevalence %	Positive herds	*Cryptosporidium* spp. (% of all determined)	Reference
Australia	477	≤8 w	9.3–56.3 Overall 24.5	5/5	*C. bovis* 36.5 *C. parvum* 46.1 *C. ubiquitum* 8.7 Mixed infections 8.7	(Yang et al. 2009)
Australia[a]	235	2 w–8 m	Age group point prevalences: 18.5–42.6 Cumulative prevalences: Herd A: 81.3 Herd B: 71.4	2/2	*C. andersoni* 1.0[d] *C. parvum* 5.9 *C. ubiquitum* 9.8 *C. xiaoi* 73.7 Sheep genotype I 1.6 Mixed infections 8.0	(Sweeny et al. 2011b)
Australia[a]	96	Ewes, 4 m prp Ewes, 2 w prp	Herd A, B: 6.3 Herd A, B: 8.3	2/2	*C. xiaoi* 100[d]	(Sweeny et al. 2011b)
Australia[a]	200	2–3 m 4–5 m	Herd I: 33.6, II: 31.9 Herd I: 28.1, II: 23.6 Cumulative prevalences: Herd I: 40.6 Herd II: 31.9	2/2	*C. parvum* 21.0[d] *C. ubiquitum* 44.5 *C. xiaoi* 31.9 Mixed infections 2.5	(Sweeny et al. 2012)
Belgium	137	≤10 w	13.1	4/10	*C. parvum* 10.0 *C. ubiquitum* 90.0	(Geurden et al. 2008)
Brazil	90 35	2–6 m >12 m	2.2 0 Overall 1.6		*C. ubiquitum* 100	(Fiuza et al. 2011a)
Brazil	1 10	<3 m >3 m	0 0	0/9	–	(Sevá et al. 2010)
China	378 585 580 158	preweaned postweaned ewe, prp ewe, pp	10.8 4.3 2.1 2.5	5/5	*C. andersoni* 4.9 *C. ubiquitum* 90.2 *C. xiaoi* 4.9	(Wang et al. 2010c)
China	213	6–9 m	14.6	?/8	*C. ubiquitum* 100[e]	(Shen et al. 2011)

(continued)

Table 4.5 (continued)

Country	Number of animals	Age	Animal prevalence %	Positive herds	*Cryptosporidium* spp. (% of all determined)	Reference
Cyprus[c]	39	4–15 d	61.5 (mZN) 76.9 (E)	12/15	ND	(Giadinis et al. 2012)
India	55	<3 m	1.8	–	ND	(Maurya et al. 2013)
Italy[c]	21	–	100	–	*C. parvum* 100	(Drumo et al. 2012)
Italy	149	2 w–3 m	17.4	5/6	*C. parvum* 100	(Paoletti et al. 2009)
Norway[a]	567	5–6 w	14.6	6/6	*C. ubiquitum* 83.3	(Robertson et al. 2010)
	528	6–10 w	24.1		*C. xiaoi* 16.7	
Romania[c]	58	1–7 d	13.8	5/7	*C. parvum* 83.3	(Imre et al. 2013)
	68	8–14 d	16.2		*C. ubiquitum* 8.3	
	49	15–21 d	10.2		*C. xiaoi* 8.3	
Spain[c]	127	<21 d	30.7	17/28	*C. parvum* 63.6	(Díaz et al. 2010a)
					C. ubiquitum 36.4	
Spain	446	Adult	5.3	13/38	ND	(Castro-Hermida et al. 2007)
St Kilda[a, b]		4–≥40 m	9.0–28.6	–	ND	(Craig et al. 2007)
Tunisia	30	<3 m	16.7	3/3	*C. xiaoi*[f]	(Soltane et al. 2007)
	59	>1 y	8.5			
Turkey[c]	151	≤7 d	44.4	26/34	ND	(Sari et al. 2009)
	104	8–14 d	37.5			
	95	15–21 d	40.0			
	50	22 d≤1 m	22.0			
			Overall 38.8			
United Kingdom	260	Mixed	39.6	11/17	*C. parvum* 88.9	(Mueller-Doblies et al. 2008; Smith et al. 2010)
					C. xiaoi [g] 11.1	

d days, *w* weeks, *m* months, *ND* not done, *Prp* prepartus, *pp* post partum, *mZN* modified Ziehl-Neelsen, *E* ELISA

[a]Longitudinal study

[b]Feral population

[c]Only diarrhoeal samples collected

[d]Species distribution for all positive samples independent of age

[e]Only four isolates successfully amplified and sequenced

[f]Reported as similar to *C. bovis*, 99.5–99.7 % identical to *C. xiaoi* isolates in GenBank

[g]Reported as *C. bovis*, 100 % identical to *C. xiaoi* isolates in GenBank

Table 4.6 Published studies on prevalence of *Cryptosporidium* infections in goats

Country	Number of animals	Age	Animal prevalence %	Positive herds	*Cryptosporidium* spp. (% of all determined)	Reference
Belgium	148	≤10 w	9.5	6/10	*C. parvum* (100)	(Geurden et al. 2008)
Brazil	49	<12 m	10.2	2/6	ND	(Bomfim et al. 2005)
	56	>12 m	0			
			Overall 4.8			
China	42	–	35.7	–	*C. xiaoi*[c]	(Karanis et al. 2007)
					Novel genotype[d]	
Cyprus[b]	75	4–15 d	64(mZN) 86.7 (E)	25/28	ND	(Giadinis et al. 2012)
France[a]	40	0-weaning	Age related point prevalences: 0–100	1/1	*C. xiaoi* (94.7)	(Rieux et al. 2013)
			Cumulative 77.5		*C. parvum* (5.3)	
France	200	5–12 m	2.5	–	ND	(Castro-Hermida et al. 2005)
France	879	5–30 d	16.2	32/60 (53.3)	ND	(Delafosse et al. 2006)
India	116	<3 m	3.5	–	*C. parvum* (100)	(Maurya et al. 2013)
Italy[b]	21	–	100	–	*C. parvum* (100)	(Drumo et al. 2012)
Spain[b]	5	<21 d	40	1/1	*C. xiaoi*	(Díaz et al. 2010b)
Spain	116	Adult	7.7	6/20 (30)	ND	(Castro-Hermida et al. 2007)
Spain	134	≤15 d	10.4	4/4	ND	(Sanz Ceballos et al. 2009)
	144	>15 d–2 m	13.4			
	304	>2 m	25.2			
Sri Lanka	558	≤6 m	33.0	23/24	ND	(Noordeen et al. 2000)
	133	6–12 m	30.8			
	329	>12 m	20.1			
			Overall 28.5			
Sri Lanka	72	1–12 m	0–75[b]	1/1	ND	(Noordeen et al. 2001)
Tunisia	184	1–7 y	0	0/5	–	(Soltane et al. 2007)
UK	15	Adult	20	1/4	Negative	(Smith et al. 2010)

d days, *w* weeks, *m* months, *ND* not done, *Prp* prepartus, *pp* post partum, *mZN* modified Ziehl-Neelsen, *E* ELISA

[a]Longitudinal study

[b]Only diarrhoeal samples collected

[c]Only two samples successfully amplified

[d]Clustered with *C. bovis*, *C. ryanae* (previously *Cryptosporidium* deerlike genotype) and *Cryptosporidium* deer genotype

species in lambs (73.1 %) and the only species in ewes (Table 4.5). *Cryptosporidium bovis* has also been reported in sheep (Mueller-Doblies et al. 2008; Soltane et al. 2007; Wang et al. 2010c). Whether *C. bovis* has actually been identified in sheep, or if it is the closely related species *C. xiaoi* is uncertain. For instance, Soltane et al. (2007) reported isolates similar to *C. bovis*, and Mueller-Doblies et al. (2008) reported *C. bovis*, but a BLAST search of the GenBank accession numbers identified these isolates as *C. xiaoi*. For the isolates reported as *C. bovis* by Yang et al. (2009), no GenBank accession numbers are available, so the true identity of those isolates is uncertain. Similarly, no GenBank records are available from the study of Ryan et al. (2005) reporting the "New bovine B genotype" in sheep. Since *C. bovis* was first identified as the bovine B genotype, this could actually be the "*C. bovis*-like genotype", i.e. *C. xiaoi*.

Cryptosporidium andersoni has been identified in a few naturally infected adult sheep (Wang et al. 2010c), but experimental infection in 4-month-old lambs failed (Kváč et al. 2004).

A couple of apparently related surveys from Mexico have been published, but because of lack of clarity in the data, they will not be reviewed in this text. Two studies from Brazil (Sevá et al. 2010) and Mongolia (Burenbaatar et al. 2008) failed to identify *Cryptosporidium* in any of the collected samples, but the number of sampled sheep was small – 11 and 5 animals, respectively.

Because of the small number of studies and isolates analysed, it is hard to draw any conclusion about the species distribution in goats. *Cryptosporidium parvum*, *C. xiaoi* and a novel genotype have been identified in naturally infected goats (Table 4.5). In addition, one report of natural infection with *C. hominis* is also available (Giles et al. 2009). The identification of *C. xiaoi* in a number of samples is in contrast with a failed attempt to infect 36-week-old goats to determine the host range of *C. xiaoi* (Fayer and Santín 2009). Because there is only scant information about cryptosporidiosis in goats, we do not know if an age-related resistance or immunity from a previous *Cryptosporidium* infection could have affected this experiment, as natural infections indicate that *C. xiaoi* is infectious to goats.

Two studies from Mongolia (Burenbaatar et al. 2008) and the United Kingdom (Smith et al. 2010) failed to identify *Cryptosporidium* in any of the collected samples, but the number of sampled goats was small – 16 and 15 animals, respectively.

4.3.2 Association of Infection with Clinical Disease

Cryptosporidiosis has been associated with high morbidity and mortality rates in both lambs and goat kids (Cacciò et al. 2013; Chartier et al. 1995; de Graaf et al. 1999; Giadinis et al. 2007, 2012; Johnson et al. 1999; Munoz et al. 1996; Paraud et al. 2011; Vieira et al. 1997). High mortality has been described both from natural infection and from experimental studies, where infection doses are generally high (Chartier et al. 1995; Giadinis et al. 2007; Paraud et al. 2010). In fact,

it has been stated to be one of the most important pathogens associated with diarrhoeal disease and mortality in neonatal lambs and kids (Quílez et al. 2008a).

Anorexia and apathy/depression are common symptoms, accompanied by abdominal pain and pasty to watery, yellow and foul-smelling diarrhoea (de Graaf et al. 1999; Snodgrass et al. 1984). Diarrhoea can last from a few days up to 2 weeks (de Graaf et al. 1999). Faecal consistency is correlated with oocyst excretion (de Graaf et al. 1999; Paraud et al. 2010, 2011), and a longer duration of diarrhoea is potentially associated with infection early in life (Paraud et al. 2010). Body condition score and growth are affected (de Graaf et al. 1999), probably due to both anorexia and the intestinal damage, that can reduce nutrient uptake for weeks (de Graaf et al. 1999; Klein et al. 2008).

Infection in animals older than 1 month is usually subclinical, and even younger animals can be subclinically infected. However, the infection can still affect production, with reduced body condition score (Sweeny et al. 2011a, 2012), reduced growth rate, and reduced carcass weight and dressing percentage at slaughter (Sweeny et al. 2011a).

As discussed above for cattle, before molecular methods were developed *C. parvum* was the only species considered to infect and cause disease in sheep and goats (Chartier et al. 1995; de Graaf et al. 1999; Munoz et al. 1996). *Cryptosporidium parvum* infection has since been associated with diarrhoea in studies using molecular methods (Caccio et al. 2013; Díaz et al. 2010a; Drumo et al. 2012; Imre et al. 2013; Mueller-Doblies et al. 2008). However, *C. xiaoi* has also been associated with mild to severe diarrhoea and mortality (Díaz et al. 2010b; Navarro-i-Martinez et al. 2007; Rieux et al. 2013), and *C. ubiquitum* too has been found in a few diarrhoeal samples from lambs (Díaz et al. 2010a), indicating that *C. parvum* is not the only pathogenic species in small ruminants.

4.3.3 *Infection Dynamics: Oocyst Excretion and Transmission*

The prepatent period is 3–4 days in goat kids (Paraud et al. 2010) and 2–7 days in lambs (de Graaf et al. 1999). The patent period can last for at least 13 days (Paraud et al. 2010). Shedding peaks a few days to a week into the patent period, and maximum shedding can be as high as 2×10^7 OPG (Rieux et al. 2013). The length of the patent period and shedding intensity are determined by age, immune status and infection dose (de Graaf et al. 1999). A natural age-related resistance to infection seems to be present. In one study, the prepatent period increased and intensity of shedding decreased in lambs with increasing age at infection (Ortega-Mora and Wright 1994). In another study, one naturally infected group of goat kids started shedding at 17 days of age and excretion peaked at a mean of 23×10^4 OPG 5–11 days later (Rieux et al. 2013), whereas another group of animals studied by the same authors started shedding at the age of 10 days, with a mean peak of 3×10^6

OPG 0–4 days later, indicating higher virulence, infection pressure or an age-related higher sensitivity in the latter group.

Factors such as hygienic conditions, milking practices, herd size (population density), season, climatic zone (within a country), and lambing/kidding season are factors that have been associated with prevalence of infection, prevalence of clinical cryptosporidiosis and intensity of oocyst shedding (Alonso-Fresan et al. 2009; Bomfim et al. 2005; Craig et al. 2007; Delafosse et al. 2006; Giadinis et al. 2012; Maurya et al. 2013; Noordeen et al. 2000, 2001). However, factors associated with infection and shedding intensity are also impacted by different management systems and climatic conditions; results from small farms in, for example, India cannot be extrapolated to large-scale farming in, for example, the United Kingdom.

4.3.4 Zoonotic Transmission

The significance of sheep and goats as reservoirs for zoonotic cryptosporidiosis is unclear (Robertson 2009). The first case of suspected zoonotic transmission from sheep was described in 1989 (Casemore 1989), but at that time diagnosis was based solely on microscopy and thus zoonotic transmission cannot be confirmed. However, since the introduction of molecular tools in diagnostics, a number of cases and outbreaks with suspected or confirmed zoonotic transfer from sheep have been described (Cacciò et al. 2013; Gormley et al. 2011). In the UK, where sheep farming is an important industry, a seasonal pattern with spring and autumn peaks of human cryptosporidiosis cases is observed, with the spring peak concurring with the lambing season (Anonymous 2002; Gormley et al. 2011; McLauchlin et al. 2000; Nichols et al. 2006). Lambs in petting zoos seem to be a common infection source (Chalmers et al. 2005; Elwin et al. 2001; Gormley et al. 2011; Pritchard et al. 2007). The incidence of human cryptosporidiosis, especially due to *C. parvum*, dropped significantly during the foot-and-mouth outbreak in the spring and summer 2001 (Hunter et al. 2003) when >6 million livestock animals (~4.9 million sheep; Anonymous 2001) were slaughtered and there were restrictions in animal movements and farm visits, providing further evidence of the importance of zoonotic transmission in this region.

In 2012, an outbreak occurred in Norwegian schoolchildren visiting a farm raising several animal species. *Cryptosporidium parvum* oocysts of an identical and unusual GP60 subtype were identified in faecal samples from six human patients, two lambs and one goat kid. Another human outbreak with the same *C. parvum* subtype had occurred at the same farm 3 years previously, but at that time very few oocysts were detected in animal faecal samples and molecular analyses were not conducted (Lange et al. submitted). Cacciò et al. (2013) described a case where a farmer's son fell ill with cryptosporidiosis, being infected with the same and unusual *C. parvum* subtype that caused high morbidity and mortality in the farm's lambs (Table 4.7).

Studies on *C. parvum* GP60 subtypes have been performed with sheep and goat isolates, and all isolates were found to belong to the IIa and IId families (Table 4.7).

Table 4.7 *Cryptosporidium parvum* GP60 subtypes identified in sheep and goats

Host Country	GP60 subtype	Number of samples	Reference
Sheep			
Australia[a]	IIa	1	(O'Brien et al. 2008)
Italy	IIaA20G2R1	3	(Cacciò et al. 2013)
	IIaA15G2R1	1	
Belgium	IIaA15G2R1	1	(Geurden et al. 2008)
Norway	IIaA19G1R1	2	(Lange et al. submitted)
Romania	IIaA17G1R1	2	(Imre et al. 2013)
	IIaA16G1R1	1	
	IIdA20G1	2	
	IIdA24G1	1	
	IIdA22G2R1	1	
Spain	IIaA15G2R1	3	(Díaz et al. 2010a)
	IIaA16G3R1	7	
Spain	IIaA15G2R1	2	(Quílez et al. 2008a)
	IIaA18G3R1	1	
	IIdA14G1	2	
	IIdA15G1	3	
	IIdA17G1a	44	
	IIdA17G1b	26	
	IIdA18G1	15	
	IIdA19G1	33	
	IIdA21G1	1	
	IIdA22G1	2	
	IIdA24G1	2	
United Kingdom	IIaA15G2R1	3	(Chalmers et al. 2005)
United Kingdom	IIaA15G2R1	1	(Smith et al. 2010)
	IIaA17G1R1	9	
	IIaA17G2R1	1	
Goats			
Belgium	IIaA15G2R1	3	(Geurden et al. 2008)
	IIdA22G1	8	
Norway	IIaA19G1R1	1	(Lange et al. submitted)
Spain	IIdA17G1a	8	(Quílez et al. 2008a)
	IIdA19G1	4	
	IIdA25G1	2	
	IIdA26G1	3	
Italy[a]	IIa	1	(O'Brien et al. 2008)

[a]IIaA12G2R1 or IIaA15G2R1 or IIaA19G4R1 were identified in a sheep, a goat and an alpaca isolate, but it is not reported which subtype was identified in which sample

Several studies using multi-locus genotyping (MLG) have found evidence of specific host associated *C. parvum* populations (Drumo et al. 2012; Mallon et al. 2003a, b; Morrison et al. 2008). In the United Kingdom, sheep MLGs clustered with human and bovine isolates (Mallon et al. 2003a, b), indicating frequent zoonotic transmission, whereas only one of the 34 MLGs identified in

sheep/goats in Italy was also identified in human samples (Drumo et al. 2012), indicating a low rate of zoonotic transmission. However, the latter study included very few human isolates.

Zoonotic transmission is commonly observed with *C. parvum*, but *C. ubiquitum* has also been identified in a number of sporadic human cases (Cieloszyk et al. 2012; Elwin et al. 2012; Feltus et al. 2006; Leoni et al. 2006; Ong et al. 2002; Soba et al. 2006; Trotz-Williams et al. 2006). Oocysts of this species have been identified in storm water, wastewater, raw water and drinking water (Jiang et al. 2005; Liu et al. 2011; Nichols et al. 2010; Van Dyke et al. 2012). In Scotland, *C. ubiquitum* was the third most common species in raw water and the most common species identified in drinking water (Nichols et al. 2010). Thus, in areas where *C. ubiquitum* is common in sheep and goats, this species could be a more important cause of zoonotic infection than *C. parvum*. In addition, the relatively common presence of this species in water indicates a potential for waterborne outbreaks.

Natural infection with *C. hominis* has been reported in one goat and two sheep (Giles et al. 2009; Ryan et al. 2005) and in three lambs following experimental infection (Ebeid et al. 2003; Giles et al. 2001), but since animals are not natural hosts for this species, risk of zoonotic transmission with this species should be negligible compared with the risk of human-to-human transmission.

It is important to note that because *Cryptosporidium* infection can be subclinical, the zoonotic potential is not restricted to contact with diarrhoeic *Cryptosporidium*-infected animals (Pritchard et al. 2007).

4.4 *Cryptosporidium* spp. in Pigs

Since domestication around 4900 BC in China, the pig has been an important food source (Moeller and Crespo 2009). Pigs are farmed worldwide, with the global swine inventory estimated at over 800 million in 2002. Because Asian countries are major consumers but do not produce sufficient pigs for their needs, there is a significant international trade in live and slaughtered pigs. China has the world's largest pig population, mostly small herds consisting of only a few animals, and is a net importer of pigs. The United States, European Union, and Canada are major exporters with relatively few but very large production units (Moeller and Crespo 2009). The global trend is for fewer producers responsible for larger numbers of pigs, and more concentration within the swine industry.

4.4.1 Occurrence (Prevalence)

Pigs are the primary host for *C. suis* (Ryan et al. 2004) formerly identified as *Cryptosporidium* pig genotype I and for *C. scrofarum* (Kváč et al. 2013) formerly identified as *Cryptosporidium* pig genotype II. Farm pigs have also been found

infected with *C. parvum*, *C. muris*, on one occasion with *C. tyzzeri*, a species common to mice, and with the novel genotype isolate Eire 65.5 (Kváč et al. 2013). Cryptosporidiosis occurs in pigs of all ages in 21 countries on 6 continents (Table 4.8). Before molecular methods were developed *C. parvum* was thought to infect 152 species of mammals and to consist of several genotypes. Consequently some early studies erroneously reported *C. parvum* infection in pigs based on the identification of oocysts in faeces by microscopy. Subsequent use of molecular methods provided the necessary tools to identify and distinguish species.

Overall, prevalence data for locations, herds and age groups vary greatly and are not directly comparable because some data represent pooled samples (some from litters, others from fecal slurry), some data originate from single farms while other data come from multiple farms. Some surveys have studied individual pigs at various ages, or only those pigs with diarrhoea, or simply specimens submitted to diagnostic laboratories from unspecified locations (Table 4.8). Even in comparable populations, such as preweaned pigs in the same country or indifferent countries, data differences are too great to draw any conclusions on prevalence. For example: in Australia- reports of 32.7 % versus 6.0 % prevalence (Johnston et al. 2008 vs. Ryan et al. 2003a); in the Czech Republic- reports of 21.8 % versus 5.7 % prevalence (Kváč et al. 2009a vs. Vitovec et al. 2006); or between Serbia and Spain – reports of 32 versus 0 % prevalence (Mišić et al. 2003 vs. Quílez et al. 1996a). Some studies found significant association between the presence of a particular species and the pigs' age, with *C. suis* prominent in piglets and *C. scrofarum* prominent in weaners (Enemark et al. 2003). In contrast, others found no significant association between species and age or housing conditions (Featherstone et al. 2010b). These prevalence data reflect vast differences in management practices from location to location with too many unknown factors to draw valid conclusions on cause and effect or location within the 49 cited studies in Table 4.8 that reported a prevalence of infection between 0.1 % and 100 %. The only variable repeatedly associated with detection of *Cryptosporidium* is age. Most positive samples were from weaners and growers (Table 4.8). Generally, prevalence increased until pigs were 10 weeks of age, then gradually declined.

4.4.2 Association of Infection with Clinical Disease

The first reports of cryptosporidiosis in pigs found one piglet among 81 herds of nursing piglets with necrotic enteritis, but the significance of this finding was described as unknown (Bergeland 1977) and *Cryptosporidium* was found at necropsy in three pigs without clinical signs (Kennedy et al. 1977).

Although a higher prevalence of diarrhoea was found in *Cryptosporidium*-infected pigs than in uninfected pigs (Hamnes et al. 2007), others found no significant relationship between infection and diarrhoea (Quílez et al. 1996b; Guselle et al. 2003; Maddox-Hyttel et al. 2006; Vitovec et al. 2006; Suárez-Luengas et al. 2007).

Table 4.8 Location, age, and prevalence of *Cryptosporidium* in pigs identified by microscopy and molecular methods

Location	Age	No. of animals/farms or locations	Prevalence (% positive)	Microscopy (M)	Molecular identification/species or genotype (no. or % identified)[a]	Reference
Australia	Not specified	78/diagnostic lab	0	M	No/unknown	O'Donoghue et al. 1987
Australia	3–8 weeks	Samples from 3 herds with clinical disease	3 herds: 2, 5, and 4 positive samples	M	18S rRNA/*C. suis* (8) *C. parvum* (4)	Morgan et al. 1999
Australia	1–8 weeks	648/22 indoor and outdoor herds	6.03 0.5 17.2	M	18S rRNA/*C. suis* *C. scrofarum*	Ryan et al. 2003a
Australia	pre-weaned weaned	156 123/4 piggeries	32.7 10.6	M	18S rRNA *C. suis* (6) *C. scofarum* (32)	Johnson et al. 2008
Brazil São Paulo	Not specified	25	0	M	No/unknown	Sevá et al. 2010
Brazil Rio de Janeiro	Not specified	91/10 piggeries	2.2	Not indicated	18S rRNA *C. scrofarum*	Fiuza et al. 2011b
Canada	5–15 days	1,453 diarrhoeic samples/diagnostic lab	0.5	M	No/unknown	Sanford 1983
Canada	1–30 weeks	3491samples/diagnostic lab	5.3	M	No/unknown	Sanford 1987
Canada	Not specified	100/5 farms	11	M	No/unknown	Olson et al. 1997b
Canada Alberta	Not specified	25–50 samples/1 farm	0	M	No/unknown	Heitman et al. 2002
Canada	21 days–6 months	33/1 farm	100 longitudinal study, cumulative prevalence	M	HSP70/*C. suis* (10)	Guselle et al. 2003

Canada Ontario		122 pooled samples/ 10 farms	55.7	M	18S rRNA/*C. parvum* *C. scrofarum*	Farzan et al. 2011
Canada Prince Edward Island		633/21 herds	26	M	18S rDNA HSP70/*C. suis*, *S. scofarum*, *C. parvum*, *C. tyzzeri*	Budu-Amoako et al. 2012d
China Henan	<1 month 1–2 months 3–6 months >6 months	1,350/14 farms in 10 prefectures	10.2 20.6 0 1.4 Avg 8.2	M	18S rRNA *C. suis* *C. scrofarum*	Wang et al. 2010b
China Chongqing	Boars and sows fatteners, growers, weaners	2,971/14 intensive 29 extensive farms in 13 counties	6.6	M	No/unknown	Lai et al. 2011
China Shanghai	60–180 days of age	2,323/8 farms	34.4	M	18S rRNA *C. suis* (82.6 %) *C. scrofarum* (8.7 %) mixed infection (8.7 %)	Chen et al. 2011
China Yangtze River Delta	Not specified	94/6 farms	14.3–25.0	Not indicated	18S rRNA *C. scrofarum*	Yin et al. 2011
Czech Republic	Sows Pre-weaned weaned	135 3,368 835/8 farms	0 5.7 24.1	M	18S rRNA *C. suis* (394)	Vitovec et al. 2006
Czech Republic	Pre-weaners Starters Pre-growers Sows	119 131 123 40 Total 413/1 farm	21.8 29.0 17.1 2.5 Avg 21.2	M	18S rRNA *C. suis*, *C. muris* *C. scrofarum*	Kváč et al. 2009a
Czech Republic South Bohemia	Abattoir	Finishers sows	123/14 farms	M	18S rRNA GP60 *C. suis*, *C. parvum* *C. scrofarum*	Kváč et al. 2009b

(continued)

Table 4.8 (continued)

Location	Age	No. of animals/farms or locations	Prevalence (% positive)	Microscopy (M)	Molecular identification/species or genotype (no. or % identified)[a]	Reference
Croatia	Not specified	5 pigs each/ 38 production units	65.8 of all units	M	No/unknown	Bilic and Bilkei 2006
Cuba Villa Clara	Suckling weaned	45	6.7	E	No/*C. parvum* based on DAS/ELISA	de la Fe et al. 2013
		45	13.3			
		All diarrhoeic/ 6 piggeries				
Denmark	<1 month	488	6	M	No/unknown	Maddox-Hyttel et al. 2006
	1–4 months	504	71			
	sows	245/50 herds	4			
Denmark	Same as above	1,237/50 herds	Same as above	Not indicated	18S rRNA, HSP70 *C. suis* (50) *C. scofarum* (133)	Langkjaer et al. 2007
England East Anglia	Suckler	20	20.0	M	*18S rRNA*	Featherstone et al. 2010b
	Weaner	36	38.9		*C. scrofarum* (64.1 %)	
	Grower	93	48.4			
	Finisher	119	36.9		*C parvum* (20.5 %)	
	Adult	39	30.8		*C suis* (15.4 %)/39 samples	
		Total 308/72 farms	Avg 38.6			
		Diagnostic lab				
Germany	1–42 days	287 diarrhoeic samples/ 24 farms	1.4	M	No/unknown	Wieler et al. 2001
Germany	Not specified	1,427 samples/ diagnostic lab	0.1	M	No/unknown	Epe et al. 2004

Ireland	Not applicable	56 slurry samples/ 33 farms	50 at 1 farm 40.6 at all other farms	M	No/unknown	Xiao et al. 2006
Ireland	Weaners Finishers Sows Gilts Boars	342/5 units in 4 counties	15.0 7.4 13.3 Rare Rare Mean: 11.4	Not indicated	18S rRNA *C. suis*, *C. scofarum*, *C. parvum*, *C. muris*	Zintl et al. 2007
Italy	Piglets	200 litters (pooled feces from each litter)	0.5	M	No/unknown	Canestri-Trotti et al. 1984
Japan Kanagawa	1–3 months 6 months	232 252/8 farms	33.2 NS	M	No/unknown	Izumiyama et al. 2001
Japan Hokkaido	6 months	108/abattoir	0	M	No/unknown	Koyama et al. 2005
Korea	6–8 months	500/location(s) not specified	19.6	M	No/unknown	Rhee et al. 1991
Korea	Not specified	589/4 slaughter locations	62	M	No/unknown	Yu and Seo 2004
Malawi	Not specified	92/2 regions over 3 seasons	17.7–60	M	No/unknown	Banda et al. 2009
Norway	4–33 days	684 litters (each pooled)/100 indoor farms	8.3	M	18S rRNA COWP, Actin/*C. parvum* *C. suis* *C. scrofarum*	Hamnes et al. 2007
Serbia	1–30 days 1–3 months 3–6 months	50 40 38	32 62.5 44.7	M	No/unknown	Mišić et al. 2003

(continued)

Table 4.8 (continued)

Location	Age	No. of animals/farms or locations	Prevalence (% positive)	Microscopy (M)	Molecular identification/species or genotype (no. or % identified)[a]	Reference
	6–9 months	45	31.1			
	9–12 months	42	23.8			
	>12 months	45	15.5			
		Total 260				
Spain	<2 months	329/farms and abattoirs	3 (all in 1 litter of ten 50-day-old pigs)	M	No/unknown	Villacorta et al. 1991
Spain	1–2 months	36	36	M	No/unknown	Fleta et al. 1995
	2–6 months	11	9			
	mature	10 abattoirs	0			
Spain Aragon	<1 months	49	0	M	No/unknown	Quílez et al. 1996a
	1–2 months	49	59.2			
	2–6 months	312	34.3			
	mature	210	0			
		Total 620/27 farms				
Spain Aragon	<1 months	14	0	M	No/unknown	Quílez et al. 1996b
	1–2 months	16	87.5			
	2–6 months	30	56.7			
	Mature	30	0			
		Total 90				
Spain Zaragosa	Weaned (1–2 months)	75	30.7	M	18S rRNA *C. suis* *C. scrofarum*	Suárez-Luengas et al. 2007
	Fattening (2–6 months)	42	11.9			
	Sows	25	16.0			
		Total 142/24 farms	22.5			
Trinidad and Tobago	Not specified	275/locations not specified	19.6	M	No/unknown	Kaminjolo et al. 1993

Trinidad	Not specified	52/3 locations	1.9	M	No/unknown	Adesiyun et al. 2001
USA-California	Not specified	200/stockyard	5	M	No/unknown	Tacal et al. 1987
USA Ohio	7–25 days	44 l	15.9 of litters	M	No/unknown	Xiao et al. 1994
	5–8 weeks	176	8			
	Sows	62/2 farms	0			
USA-California	≤8 months	62	11	M	No/unknown	Atwill et al. 1997
	≥9 months	159/10 feral groups	3			
USA Georgia	Separate farrowing, nursing, finishing, and gestation effluents	10 swine waste lagoons; 12 monthly samples from each	100 (every sample was positive)	M	18S rRNA/*C. suis* and *C. scofarum* (95–100 %); also *C. muris* and *C. parvum*	Jenkins et al. 2010
Vietnam	4–8 weeks	farms	23.5	M	No/unknown	Koudela et al. 1986

[a]The species names *C. suis* and *C. scrofarum* are used throughout. In some places they replaced the former designations *Cryptosporidium* pig genotype I and II that appeared in the original references published before these species were named

Cryptosporidium was detected histologically in the microvillus brush border of 5.3 % of 3,491 pigs from 133 farms examined for routine diagnostic evaluation (Sanford 1987). Most infected pigs were 6–12 weeks old. Organisms were detected in the jejunum, ileum, caecum, and colon, but primarily in microvilli of dome epithelium in the ileum. Twenty six percent of *Cryptosporidium*-infected pigs had diarrhoea but most of these also had other primary agents capable of causing diarrhoea. Similar observations have been made by others. Whereas most infections are asymptomatic or cause only mild, non-specific colitis (Higgins 1999), pigs known to be naturally infected with *C. suis* or *C. scrofarum* have not been found with clinical signs of infection while pigs infected with *C. parvum* or co-infected other enteropathogens such as rotavirus, *Salmonella*, or *Isospora* have had associated diarrhoea and some have died (Enemark et al. 2003; Núñez et al. 2003; Hamnes et al. 2007).

Experimental infections with different species have helped to clarify the relationship of species with clinical disease. Pigs experimentally infected with *C. suis* (Enemark et al. 2003) or *C. scrofarum* (Kváč et al. 2013) showed no clinical signs. The pathogenicity of *C. parvum* isolated from calves was demonstrated in early transmission studies to pigs (Moon and Bemrick 1981; Tzipori et al. 1981b, 1982; Argenzio et al. 1990; Vitovec and Koudela 1992; Pereira et al. 2002). Experimental infection with the avian species, *C. meleagridis*, obtained from a human infection, consistently resulted in oocyst excretion and diarrhoea in pigs, although mucosal changes were milder than those described for *C. parvum* (Akiyoshi et al. 2003). Piglets infected with *C. suis* had mild or no clinical signs despite excreting large numbers of oocysts, in contrast to those infected with *C. parvum* that had diarrhoea for a mean duration of 3.5 days and developed inappetence, depression and vomiting (Enemark et al. 2003).

Developmental stages of *Cryptosporidium* have been observed throughout the intestinal tract. Villous atrophy, villous fusion, crypt hyperplasia, and cellular infiltration of the lamina propria have been observed (Kennedy et al. 1977; Moon and Bemrick 1981; Tzipori et al. 1982, 1994; Sanford 1987; Vitovec and Koudela 1992; Argenzio et al. 1990; Pereira et al. 2002; Enemark et al. 2003; Núñez et al. 2003; Vitovec et al. 2006). Lesions caused by *C. parvum* were the most severe, as were clinical signs associated with that species. Changes in the location of stages have been noted. In the first days of infection more stages were found in the proximal intestine, but later more stages were found in distal locations (Tzipori et al. 1982; Vitovec and Koudela 1992). Extra-intestinal infections also have been reported in pigs. In two naturally infected piglets, the gall bladder was infected (Fleta et al. 1995). In experimentally immunosuppressed piglets, the gall bladder, bile ducts, and pancreatic ducts were found infected (Healey et al. 1997). Infections in the trachea and conjunctiva were detected in experimentally infected normal piglets (Heine et al. 1984).

4.4.3 *Infection Dynamics: Oocyst Excretion and Transmission*

A survey of faecal slurry from swine finishing operations in Ireland found *C. suis*, *C. scrofarum* and *C. muris* and concluded that *Cryptosporidium* oocysts can persist in treated slurry and potentially contaminate surface water through improper discharge or uncontrolled runoff (Xiao et al. 2006). Hamnes et al. (2007) reported *C. suis* and *C. scrofarum* in faeces of suckling pigs in Norway and reasoned that farrowing operations were sources of these parasites. Additional data on oocyst concentrations, numbers of oocysts excreted, how long oocysts remain infectious under environmental condition, and modes of transmission of *Cryptosporidium* species and genotypes are rare or non-existent. A year-long investigation was conducted at four types of swine operations (finishing, farrowing, nursery and gestation) in Georgia, USA (Jenkins et al. 2010). Mean oocyst concentrations ranged from 11 to 354 oocysts per ml of lagoon effluent; the nursery had the highest concentration of oocysts and the greatest percentage of viable oocysts (24.2 %), *C. suis* and *C. scrofarum* were the dominant species with some *C. muris* and *C. parvum*. Experimental attempts to transmit *C. scrofarum* to adult SCID mice, adult BALB/c mice, Mongolian gerbils, southern multimammate mice, yellow-necked mice, and guinea pigs were unsuccessful, suggesting that rodents are an unlikely source of transmission of this species under natural conditions (Kváč et al. 2013).

4.4.4 *Zoonotic Transmission*

Cryptosporidium suis was detected by immunofluorescence microscopy and RFLP analysis of PCR products in stools from an HIV patient in Peru (Xiao et al. 2002a). The patient was not severely immunosuppressed and was asymptomatic. He had a dog but reported no contact with other animals or animal faeces, including pigs and pig faeces, so the source and method of transmission are unknown.

4.5 *Cryptosporidium* spp. in Other Farmed Mammals

4.5.1 Cryptosporidium *in Farmed Deer*

Many countries, including Australia, Canada, China, Korea, Norway, Russia, Sweden, UK, USA and Vietnam, have thriving deer farming industries. New Zealand, a country where deer are not native, has the world's largest and most advanced deer farming industry. Although it is difficult to find estimates on the numbers of deer farmed worldwide, more than one million deer were being

farmed in New Zealand in 2011 (Sources: Statistics New Zealand and Deer Industry New Zealand http://www.deernz.org/about-deer-industry/nz-deer-industry) – compared with five million dairy cows – and there are over 2,800 deer farmers. More than 90 % of the New Zealand deer industry's products are exported, with approximately half of the export going to Germany and Benelux.

Species of deer which are commercially farmed varies regionally, but the following species are now being farmed in various parts of the world: red deer (*Cervus elaphus*), wapiti or elk (*Cervus canadensis*), fallow deer (*Dama dama*), sika (*Cervus nippon*), rusa deer (*Rusa timorensis*), and reindeer (*Rangifer tarandus*) (FAO http://www.fao.org/docrep/004/X6529E/X6529E02.htm).

4.5.1.1 Occurrence (Prevalence)

Although farmed deer are an important resource in many countries, much of the published information on *Cryptosporidium* in deer refers to studies on wild or free-ranging cervids (e.g. white-tailed deer in USA, roe deer in Spain, caribou in Canada, and moose, red deer, roe deer and reindeer in Norway; Rickard et al. 1999; Castro-Hermida et al. 2011b; Johnson et al 2010; Hamnes et al. 2002). While these studies on free-ranging cervids may give useful information regarding the species or genotype of *Cryptosporidium* that might infect farmed deer (*C. ubiquitum*, *C. parvum*), as farmed deer probably differ quite substantially from their wild counterparts regarding exposures and stresses, extrapolation of prevalence data from wild to farmed deer may give an incorrect picture. Indeed, a study in Poland found that the prevalence of *Cryptosporidium* was significantly higher in wild red deer than farmed red deer (27 % compared with 4.5 %), and mean oocyst concentration was also five times higher in faecal samples from wild red deer (Paziewska et al 2007). However as the sample size was relatively small (52 wild deer, 66 farmed deer) and from only single locations and as age and symptoms were not indicated, it is not possible to determine the reason for these differences.

The few studies on the prevalence of *Cryptosporidium* infection in different farmed or domesticated cervids are summarised in Table 4.9.

4.5.1.2 Association of Infection with Clinical Disease

The lack of surveys for *Cryptosporidium* infection in farmed deer is surprising, given the clear association of infection with clinical disease in farmed cervids. Some of the first published studies on *Cryptosporidium* infection in cervids are case reports of severe (high mortality) outbreaks among farmed red deer calves. In one outbreak in Scotland, UK among 82 artificially reared red deer calves, 56 developed cryptosporidiosis and 20 subsequently died; 80 % of the calves with diarrhoea and 50 % of apparently asymptomatic calves excreted oocysts and post mortem histopathological examination of the intestines demonstrated lesions similar to

Table 4.9 *Cryptosporidium* in different species of deer identified by microscopy

Location	Host species	Symptoms	Host age	Study design	Prevalence (% positive – no. positive/ no. examined)	Diagnostic technique	Molecular analyses	Reference
Ireland	Red deer	Asymptomatic	Adult hinds and calves	Longitudinal – monthly samples	Hinds: 0–63 % according to month (overall prevalence: 39.3 % – 114/290) Calves: 40–100 % according to month (overall prevalence: 60 % – 21/35)	Water-ether sedimentation followed by sucrose flotation and IFAT	Not conducted	Skerret and Holland 2001
Poland	Red deer	Not stated	Not stated	Semi-longitudinal – seasonal sampling	Overall prevalence: 4.5 % – 3/66	Sheather's flotation followed by mZN and IFAT	Unsuccessful	Paziewska et al 2007
China	Red deer, Pere David's deer, Sika deer	Asymptomatic	4 months–10 years	Cross-sectional study at 4 deer farms	Red deer: 0 (0/8) Pere David's deer: 0 (0/33) Sika deer: 2.4 (2/83)	Sheather's flotation followed by light microscopy	1 sample *C. ubiquitum* – PCR and sequence analysis at 4 genes	Wang et al. 2008a

those seen in other species (Tzipori et al. 1981a). Another outbreak among new-born red deer calves in the UK also resulted in high mortality, with calves dying at 24–72 h of age. However, this outbreak was not characterized by diarrhoea, and terminal uraemia was proposed as the symptom leading to death (Simpson 1992). Outbreaks of cryptosporidiosis in red deer calves have also been reported from New Zealand, again with relatively high mortality (10 out of 10 calves dying within a few days of illness onset in one outbreak, and 7 out of 10 calves dying within 3 days of illness in another outbreak) (Orr et al. 1985). Severe subactute enteritis in the small and large intestine were reported in both outbreaks.

Information from other species of farmed deer is more scanty, but a retrospective study of neonatal mortality in farmed elk (Pople et al 2001) identified *Cryptosporidium* infection as one of the most important causes of enteritis leading to death (7 cases out of 11 of infectious enteritis from a total of 111 cases in which 46 had no specific cause of death identified). Among these 7 cases, 4 were associated with an outbreak on a single farm (Pople et al 2001).

Unfortunately, information on the species of *Cryptosporidium* infection associated with clinical disease in deer and *Cryptosporidium* associated with asymptomatic infection is lacking. One outbreak on a Scottish farm occurred when deer were put to graze on a pasture that had previously been grazed by a *Cryptosporidium*-infected herd of cattle (Angus 1988), and therefore it seems probable that this might indicate infection with *C. parvum*; it might be speculated that infection of deer with deer-adapted *C. ubiquitum* is less likely to cause severe symptoms.

4.5.1.3 Infection Dynamics: Oocyst Excretion and Transmission

Published information about infection dynamics in farmed deer is minimal. However, a longitudinal study in asymptomatic farmed red deer in Ireland (Skerrett and Holland 2001) provides some interesting data. Asymptomatic low-level (<10 OPG) oocyst shedding from adult hinds appeared to continue throughout the year, except during the calving season (May - June), when there was a 2-log increase in oocyst excretion rate; the highest level recorded was 67,590 OPG. The authors speculate that this may be related to hormonal or immunological changes, or perhaps alterations in stress levels. The authors note that this preparturient rise in oocyst shedding results in contamination of the environment for the new born calves. However, in this study, although calves became infected, oocyst excretion was low (not exceeding 150 OPG, and usually less), and clinical disease was not observed. Again, the species of *Cryptosporidium* in these infections is unknown.

4.5.1.4 Zoonotic Transmission

Both *C. parvum* and *C. ubiquitum* have zoonotic potential, but there appear to be no documented cases of proven zoonotic transmission from/to deer. Those studies

(USA and Australia) that have investigated deer as sources of contamination in watersheds, have focused on wild deer only and provided contrasting results (Cinque et al 2008; Jellison et al 2009).

4.5.2 Cryptosporidium *in Farmed Camelids*

Camelids are members of the family *Camelidae*, and include the tribe *Camelini* (including dromedaries and Bactrian camels), and the tribe *Lamini* (llamas, alpacas, vicuñas, and guanacos).

There are two species of camel. Approximately 14 million domesticated one-humped dromedaries (*Camelus dromedarius*) are found in Middle Eastern countries including the Sahel and Horn of Africa, as well as parts of Southern Asia where they provide people with milk, food, and transportation. Nearly two million domesticated two-humped bactrians (*Camelus bactrianus*) are native to the steppes of central Asia the Gobi and Taklamakan Deserts in Mongolia and China.

Alpacas (*Vicugna pacos*) and llamas (*Lama glama*) exist only in the domesticated state and are found worldwide. However, both are native to South America and are raised primarily for fibre production although llamas were once used extensively as work animals. The young of both are called crias.

4.5.2.1 Occurrence (Prevalence)

Camels and dromedaries: In the relatively few studies of dromedaries in Northern Africa and the Middle East, the prevalence of cryptosporidiosis varied greatly (Table 4.10). None of 23 camels from Iraq (Mahdi and Ali 1992) and none of 110 camels on farms in Tunisia were found positive for *Cryptosporidium* (Soltane et al. 2007). *Cryptosporidium* was detected in one of four camel calves in Egypt (Abou-Eisha 1994). However, in an abattoir in Yazd Province in Iran, microscopic examination of 300 faecal specimens detected 61 (20.3 %) positive for *Cryptosporidium* and 12 (12 %) positive abomasal mucosa specimens. At an abattoir in Isfahan Province in central Iran, of 63 adult male and 40 adult female dromedary camels examined, 39 (37.9 %) were *Cryptosporidium*-positive (Razawi et al. 2009). In northwestern Iran, of 170 faecal samples from camels 17 (10 %) were positive for *Cryptosporidium*-like oocysts (Yakhchali and Moradi 2012). The prevalence was significantly higher (20 %) in calves less than a year old.

Oocysts have also been recovered from wild and zoo-housed camels. Faeces from a 3-year-old Bactrian camel in the Wild Animals Rescue Centre of Henan Province in China were found positive for *C. andersoni* (Wang et al. 2008b). Oocysts from a zoo-housed Bactrian camel (Fayer et al. 1991) were infectious for mice (Anderson 1991) and were identified as *C. muris* (Xiao et al. 1999; Morgan et al. 2000); those from camels in the Czech Republic were identified as *C. andersoni*. Other zoo-housed

Table 4.10 *Cryptosporidium* detected in camels

Location	Host	Age	No. infected/ no. examined	Method	Reference
Basrah, Iraq	Camels	Adults	0/24	Microscopy	Mahdi and Ali 1992
Australia	Camel	Not stated	1	Molecular	Morgan et al. 2000
Tunisia	Camels	Not stated	0/110	Microscopy	Soltane et al. 2007
China	Bactrian camel	3-year-old	1	Molecular	Wang et al. 2008b
Isfahan Province, Iran abattoir	Camels	2–14 years	39/103	Microscopy	Razawi et al. 2009
NW Iran	Dromedary camels	Calves and adults	17/170	Microscopy	Yakhchali and Moradi 2012
Yazd Province, Iran abattoir	Camels	<5–>10 years	61/300 faeces 12/100 abomasums	Microscopy	Sazmand et al. 2012

camels have been found to be infected with *Cryptosporidium* (Abou-Eisha 1994; Gomez et al. 2000; Gracenea et al. 2002).

Alpacas and llamas *Cryptosporidium* oocysts have been detected in both these species. However, in California none of 354 llamas from 33 facilities were found positive (Rulofson et al. 2001) nor were 61 alpacas on two farms in Maryland (Trout et al. 2008). Elsewhere in North America, Europe, and Australia small numbers of alpacas, llamas and guanacos have been examined and a few have been found positive for *Cryptosporidium* (Table 4.11). Most examinations were conducted by microscopy, but those that utilized molecular methods identified only *C. parvum* (Morgan et al. 1998; Starkey et al. 2007; O'Brien et al. 2008; Twomey et al. 2008). The exception is a study in which a cria was found infected with *C. ubiquitum* (Gomez-Couso et al. 2012). A national survey of 5,163 1–15-day-old alpacas in 105 Andean herds in Peru, the natural habitat for nearly 80 % of the world's alpacas, found 2 % of the youngest alpacas increasing to 20 % of the oldest alpacas, infected with *Cryptosporidium* spp., with an overall prevalence of 13 % (Lopez-Urbina et al. 2009). More recently in Peru, 4.4 % of 274 alpacas from 12 herds were found positive for *Cryptosporidium* spp. (Gomez-Couso et al. 2012). Herd prevalence was 58.3 % (7/12 herds) for *Cryptosporidium*. The highest prevalence (20 %) was found in the 8-week-old group (Gomez-Couso et al. 2012).

4.5.2.2 Association of Infection with Clinical Disease

Camels and dromedaries Few data are available on the subject of clinical illness associated with cryptosporidiosis in camels. Of 170 faecal samples, 17 camels (10 %) were positive for *Cryptosporidium*-like organisms (Yakchali and Moradi 2012). The prevalence was significantly higher in camel calves (<1 years old) (20 %) than other age groups, in which the diarrhoeic calves had a prevalence of 16 %.

Table 4.11 *Cryptosporidium* in llamas and alpacas

Location	Host	Age	No. infected/ no. examined	Detection method	Reference
Wisconsin, USA	Llama	Cria	1/1	Not stated	Hovda et al.1990
Peru	Alpaca	Not given	1	Molecular	Spano et al. 1997 Morgan et al. 1998
England	Alpaca	9–30 days old	3/3	Microscopy	Bidewell and Cattell 1998
California, USA	Llama	Crias and Adults	0/354	Microscopy	Rulofson et al 2001
Oregon, USA	Llama and Alpaca	31–210 days old	4/45	Microscopy	Cebra et al 2003
Czech Republic	Alpaca	Not given	1/1	Molecular	Ryan et al. 2003c
Canada	Alpaca	Crias	Multiple (number not given)	Microscopy	Shapiro et al. 2005
Scotland	Alpaca	Not given	Not given	Not stated	Stewart et al. 2005
New York, USA	Alpaca	Crias	8/total not given	Microscopy and Molecular	Starkey et al. 2007
Maryland, USA	Alpaca	10 weeks–10 years	0/61	Molecular	Trout et al. 2008
Ohio	Llama and Alpaca	7–100 days old	15/58	Not stated	Whitehead and Anderson 2008
SW England	Alpaca	Crias; Adults Crias	3/6; 0/24 5/14	Molecular	Twomey et al. 2008
Australia	Alpaca	Not given	1/1	Molecular	O'Brien et al.2008
Washington, USA	Alpaca	8–18 days old	20/20	Microscopy	Waitt et al. 2008
Peru	Alpaca	1–15 days old	24/241 666/5,163	Microscopy	Lopez-Urbina et al. 2009
Peru	Alpaca	≥5 weeks	12/274	Microscopy and Molecular	Gomez-Couso et al. 2012
New York and Pennsylvania, USA	Alpaca	Crias Adults	8/110 9/110	Microscopy	Burton et al. 2012

Alpacas and llamas Not all alpacas and llamas infected with *Cryptosporidium* show clinical signs of infection. Of 110 healthy crias and their 110 dams 7 % and 8 %, respectively, were found excreting oocysts (Burton et al. 2012). Oocysts of *C. parvum* were detected in 4 of 14 faecal samples from healthy crias and in one sample from a cria with diarrhoea (Twomey et al. 2008).

Cryptosporidium was observed in a post-operative neonatal llama with diarrhoea, cachexia, dehydration and electrolyte abnormalities (Hovda et al. 1990). During 8 days that intravenous fluids and nutritional support were provided, these signs were not observed.

Of 20 *Cryptosporidium*-infected alpaca crias with diarrhoea, 15 exhibited weight loss and 5 had a poor appetite (Waitt et al. 2008). Most were 8–18 days old when examined. Additional potential gastrointestinal pathogens were found in 7 of these crias. Sixteen crias recovered after supportive therapy that included intravenous rehydration, with partial parenteral administration of nutrients, antimicrobials, oral nutrients, plasma, insulin and other palliative treatments.

Additional reports of diarrhoea associated with cryptosporidiosis have been reported in alpaca and llama crias (Cebra et al. 2003; Shapiro et al. 2005; Whitehead and Anderson 2006; Starkey et al. 2007). Three fatal cases (2 with diarrhoea) of cryptosporidiosis were reported in alpaca crias less than 30 days of age (Bidewell and Cantell 1998). At necropsy, intestinal congestion and distension were noted, oocysts were detected in Ziehl-Neelsen stained smears, and no other significant organisms or toxins were detected.

In South America, llama and alpaca husbandry is a vital economic activity and neonatal diarrhoea syndrome (NDS) is the most common and costly enteric disease in newborn llamas and alpacas (Lopez-Urbina et al. 2009). However, the role of cryptosporidiosis in NDS has not been clearly identified.

4.5.2.3 Zoonotic Transmission

Camels and dromedaries Only rare circumstantial data of zoonoses are available and the link is very tenuous. In Yazd Province in Iran, 24 of 100 people in long-term contact with camels were found infected with *Cryptosporidium* spp. (Sazmand et al. 2012). Infection was higher in winter than summer (16/50 compared with 8/50).

Alpacas and llamas In New York, *Cryptosporidium parvum* infection was identified in 5 crias, 3 of their caretakers were confirmed to have cryptosporidiosis, and three others were suspected to have cryptosporidiosis, suggesting zoonotic transmission (Starkey et al. 2007).

4.5.3 Cryptosporidium *in Farmed Rabbits*

Rabbit farming (cuniculture) for meat, wool, and fur production occurs in a variety of settings around the world, and mostly involves the European (or common) rabbit (*Oryctolagus cuniculus*). Small-scale backyard cuniculture is common in many

countries (especially in Africa and South America), but commercial operations on a larger scale are found in Europe (particularly Italy, Spain and France) and Asia (particularly China and Indonesia). In the EU, rabbit meat production was estimated to be around 520,000 tonnes carcass-weight equivalent in 2005 (EFSA-AHAW 2005). In addition, rabbits continue to be bred for biomedical purposes – but this type of rabbit breeding will not be considered further in this chapter. Production and consumption of rabbit meat is relatively low in North America. Different rabbit breeds are used for meat, wool, and fur – with the most commonly used meat breeds being New Zealand, Californian, Florida White and Altex, all having good growth rates and desirable reproductive characteristics.

Much of the information presented in this section is derived from a comprehensive review article from 2010 (Robinson and Chalmers 2010).

4.5.3.1 Occurrence (Prevalence)

The majority of published prevalence information on *Cryptosporidium* in rabbits refers to studies on wild rabbits. Nevertheless, there have been several studies on the occurrence of infection in farmed rabbits and also in laboratory rabbits. The majority of these studies (involving both wild and domestic rabbits) are summarised in Robinson and Chalmers (2010). In Table 4.12, selected prevalence studies (rather than case reports) from farmed rabbits only are summarized, including two recent studies from China. Additionally, a further three studies from China and referenced in Zhang et al. (2012) are not included in Table 4.12 due to inaccessibility of the original publications. Zhang et al. (2012) do not provide any details on these studies and it is not certain that they refer to farmed rabbits. Although some surveys refer to *Cryptosporidium parvum*, all those studies in which genotyping has been used (including from wild rabbits; e.g. Nolan et al. 2010) suggest that the majority of natural infections in rabbits, if not all, are caused by *C. cuniculus*. Nevertheless, experimental infections with other species of *Cryptosporidium* have been established in rabbits, as summarised by Robinson and Chalmers (2010).

4.5.3.2 Association of Infection with Clinical Disease

Although the majority of surveys do not report symptoms associated with cryptosporidiosis in rabbits, experimental infections in preweaned rabbits have been associated with diarrhoea and high mortality (e.g. as reported by Robinson and Chalmers 2010; Mosier et al 1997) and also as described by Pavlásek et al. (1996) in farmed rabbits. However, even asymptomatic infection may result in some pathology, as noted by Inman and Takeuchi (1979), who reported blunted villi, a decrease in villus-crypt ratio, and mild oedema in the lamina propria in an apparently asymptomatic adult rabbit. Thus, even asymptomatic infection may reduce stock productivity.

Table 4.12 *Cryptosporidium* in farmed rabbits

Location	Breed	Symptoms	Host age	Study design	Prevalence (% positive – no. positive/no. examined)	Diagnostic technique	Molecular analyses	Reference
Czech Republic	Broiler rabbits of 6 different breeds or crossbreeds	Variable, including diarrhoea and inappetence. Seven deaths recorded	23–33 days and 82–92 days (all post-weaning)	Longitudinal – with pooled samples	Variable throughout study, but at peak pooled samples from 12/28 cages	Giemsa staining in faeces; post mortem examination of digesta and intestinal scrapings. Histology	Not conducted	Pavlásek et al. 1996
Tunisia	Not stated	Not stated	Not stated	Cross-sectional at 1 farm	Overall prevalence: 0 % – 0/178	Formol-ether sedimentation followed by mZN	Not applicable	Soltane et al 2007
China (Henan Province)	Various, including Standard Rex and New Zealand White	Asymptomatic	5 age groups:	Cross-sectional at 8 farms	Overall prevalence: 3.4 % – –37/1,081	Sheather's flotation followed by modified acid-fast stain	All (36/37successful) *C. cuniculus*[a] – PCR and RFLP and sequence analysis at 18S rRNA gene. 8 samples further analysed at 3 other genes	Shi et al. 2010
			<1 month,		<1 month: 4.1 %–3/73			
			1–3 months,		1–3 months: 10.9 %–27/247			
			4–6 months,		4–6 months: 1.3 %–6/474		GP60 subtyping – 30 of 37 successful; VbA29 (18 samples), VbA35 (4 samples), VbA36 (8 samples)	
			7–12 months,		7–12 months: 0.4 %–1/230			
			>12 months		>12 months: 0 %–0/57			
China (Heilongjiang province	Not stated	Not stated	4–6 months	Cross-sectional at 8 farms	Overall prevalence: 2.4 %–9/378 (positive samples from 4 farms only)	Sheather's flotation followed by bright-field microscopy	All (9/9successful) *C. cuniculus* – PCR and sequence analysis at 18S rRNA gene GP60 subtyping – 9 of 9 successful; VbA21 (6 samples), VbA32 (3 samples)	Zhang et al 2012

[a]Described as rabbit genotype in publication

Although no outbreaks of cryptosporidiosis in rabbit farms have been documented in the literature, acute outbreaks of diarrhoea with high mortality rates are frequently observed in rabbits (Banerjee et al 1987). Although bacterial agents are frequently considered to be the aetiological agent, it seems probable that some may be due to undiagnosed cryptosporidiosis. For example, the parasitological techniques (direct microscopy and flotation) used for investigating epizootic outbreaks of diarrhoea, characterized by a high morbidity and mortality, in different commercial rabbit farms in Mexico (Rodríguez-De Lara et al 2008) may have been insufficient for detecting *Cryptosporidium* infection, particularly if the operators had little experience in diagnosing this infection.

4.5.3.3 Infection Dynamics: Oocyst Excretion and Transmission

Information on the dynamics of *Cryptosporidium* infection in farmed rabbits is mostly lacking, although low oocyst excretion rates were reported in the majority of studies on rabbits in general (not just farmed rabbits). The studies from the Czech Republic provide some data, but, as the animals were not sampled individually, the data are difficult to interpret, and suggest that the source of infection for young rabbits may be low-level excretion of oocysts from mother rabbits at around parturition (Pavlásek et al. 1996).

4.5.3.4 Zoonotic Transmission

C. cuniculus is rarely, but sporadically, identified in human infections. In 3030 *Cryptosporidium*-positive faecal samples submitted for routine typing in UK between 2007 and 2008, 37 (1.2 %) were identified as *C. cuniculus*, with both GP60 Va and Vb subtype families detected (Chalmers et al 2011). However, the greatest evidence for *C. cuniculus* from rabbits having a significant zoonotic potential came from a waterborne outbreak of cryptosporidiosis in England in 2008 affecting 29 people; *C. cuniculus*, subtype VaA18 was identified in eight patients, a water sample from the implicated supply, and from the colon of a carcass of a rabbit (presumably wild) that was found in a tank at the water treatment works (Chalmers et al 2009). Nevertheless, transmission of *Cryptosporidium* to humans from farmed rabbits has not been recorded, and an investigation exploring associations between farm animals and human patients with cryptosporidiosis did not implicate rabbits as a source of infection (Smith et al 2010).

4.6 *Cryptosporidium* spp. in Poultry

The world stock of birds in production in 2011 was estimated to 22×10^9 animals (FAOSTAT 2013b). Approximately 56 % of the world stock was found in Asia, whereas Europe, North America and South America had approximately 10–11 %

each of the population. The largest group was chickens, with 90 % of the total stock. Ducks, turkeys and geese/guinea fowls constituted 6.1 %, 2.1 % and 1.7 % respectively, and other birds (ratites, pigeons etc.) only constituted 0.1 % of the world stock. The main chicken, duck and goose/guinea fowl production is in Asia (54 %, 90 % and 91 % within each group respectively), and most of the turkey production in North America (54 %), followed by Europe (23 %). For other birds, 50 % of the reported production was located in Asia, 41 % in Africa and 9 % in Europe.

Chickens (*Gallus gallus domesticus*) are descendants of the Red jungle fowl (*Gallus gallus*), with some hybridization with the Grey junglefowl (*G. sonneratii*). Broilers are usually kept in intense systems and reach slaughter size at about 6 weeks of age. Organically bred broilers and broilers kept on free range grow a bit more slowly. Laying hens can produce over 300 eggs in their first production year, but after that production declines rapidly.

Domesticated ducks (*Anas platyrhynchos domesticus*) are, except for the Moscovy duck (*Cairina moschata*), descendants of the Mallard (*Anas platyrhynchos*). The majority of domesticated geese (*Anser anser domesticus*) descend from the Greylag goose (*Anser anser*), but the breeds Chinese goose and African goose are derived from the Swan goose (*Anser cygnoides*). Ducks and geese are bred for meat, eggs and down, and ducks, to a lesser degree, also for the production of foie gras.

The domestic turkey (*Meleagris gallopavo*) is a progeny of the wild turkey, which is found in the wild in the United States http://www.turkeyfed.com.au/Turkey_Info.php. Turkeys are bred for meat production. The breed used is the white broad breasted turkey, introduced into commercial production in the 1950s http://bizfil.com/turkey-raising-primer. As with commercial chicken broiler farming, turkey farming is intense. The poults are extremely fast-growing, and reach approximately 6 kg at 10 weeks of age if given proper nutrition http://bizfil.com/turkey-raising-primer/. The United States has the highest consumption of turkey meat per person, and they are also the largest turkey producer, with 7.32 billion pounds of turkey meat produced in 2011 http://www.agmrc.org/commodities__products/livestock/poultry/turkey.

Among ratites, mainly ostriches (*Struthio camelus*) are farmed, but rheas (*Rhea americana*) and emus (*Dromaius novaehollandiae*) are also kept for production. Ratites are bred for meat, egg, and feather and leather production. Farming for feather production began already in the nineteenth century. Partridges, such as the Grey or English partridge (*Perdix perdix*) and red-legged partridge (*Alectoris rufa*), are gallinaceous birds used as game, and have been introduced in different parts of the world for this purpose. Another gallinaceous bird is the helmeted guinea fowl (*Numida meleagris*). They are used for pest control, eating ticks and other insects, and can be kept as an alarm system among other domesticated birds due to their loud and shrieking warning call. The meat is considered a delicacy. The Japanese quail (*Coturnix japonica*) is bred for meat and eggs. Domestic pigeons (*Columba livia domestica*) are the progeny of the world's oldest domesticated bird, the Rock pigeon. Pigeons are bred for meat, sporting competitions, homing, as exhibition birds or pets.

Cryptosporidium infection has been associated with large morbidity and mortality in different bird species (Bezuidenhout et al. 1993; Hoerr et al. 1986; Pages-Mante et al. 2007; Penrith et al. 1994; Ritter et al. 1986; Santos et al. 2005) and can thus be of great economic importance.

4.6.1 Prevalence

Avian cryptosporidiosis was first described in chickens (Tyzzer 1929). The infection was subclinical and situated in the caecum. Invasive stages looked identical to those of *C. parvum*, but no oocyst description was made, and no name was proposed. Today, three valid species have been identified in poultry. In addition, five genotypes have been identified in wild ducks and geese, and five additional genotypes have been described from other birds.

The *Cryptosporidium* oocysts identified by Slavin in 1955 were morphologically similar to *C. parvum*, described in mice in 1912 (Tyzzer 1912), and the infection site was the distal ileum. Slavin identified this bird *Cryptosporidium* as a unique species, *C. meleagridis*. When molecular methods were introduced as a means of species determination, it was verified that *C. parvum* and *C. meleagridis* were indeed different species (Sreter et al. 2000).

A species with a larger oocyst, first identified in chickens, and infecting the intestine, bursa and cloaca, was described and named *C. baileyi* (Current et al. 1986). This species is also involved in respiratory cryptosporidiosis, infecting the epithelium of sinuses, air sacs, nasopharynx, trachea and bronchi (Itakura et al. 1984; Lindsay et al. 1987). Infection of the conjunctiva (Chvala et al. 2006) and urinary tract, including the kidneys has also been shown (Abbassi et al. 1999; Trampel et al. 2000).

A third species, *C. galli*, infecting the proventriculus of chickens, was described by Pavlásek in 1999 and 2001, and re-described in 2003 (Pavlásek 1999, 2001; Ryan et al. 2003b). The species was probably described in finches already in 1990 (Blagburn et al. 1990) and later the name *C. blagburni* was proposed (Morgan et al. 2001). However, molecular analyses have shown that *C. blagburni* is the same species already described as *C. galli*, and thus the latter is considered to be the valid species name.

In addition, isolates referred to as goose genotypes I-IV have been identified in Canada geese and a duck genotype has been described in a Black duck and Canada geese. Of the other genotypes described in birds (avian genotypes I-IV and the Eurasian woodcock genotype), avian genotype II has been detected in ostriches.

Two proposed species are today considered as *nomen nudum* due to lack of sufficient data. *Cryptosporidium tyzzeri* in chickens was described in 1961 (Levine 1961) and later *C. anserinum*, found in the large intestine of geese was described (Proctor and Kemp 1974).

Based on 18S rDNA phylogeny, *C. galli* and the woodcock genotype belong to the clade of gastric cryptosporidia together with *C. andersoni*, *C. muris* and

C. serpentis, whereas *C. meleagridis*, *C. baileyi*, goose genotypes I and II and the duck genotype belong to the intestinal clade (Xiao et al. 2004). *Cryptosporidium meleagridis* is closely related to the group including *C. parvum* and *C. hominis*; *C. baileyi* is closely related to the snake genotype, goose genotypes I, II and the duck genotype cluster together and are closely related to *C. scrofarum*, *C. bovis*, *C. ryanae* and the deer genotype. Goose genotypes III-IV and avian genotypes I-IV were not included in the phylogenetic tree. In another publication, goose genotypes I (goose #1, 2, 3, 6 and 8), II (goose #9) and the duck genotype (goose #5) are closely related, whereas goose genotypes III (goose #3b) and IV (goose #7) are more distant (Jellison et al. 2004). The avian genotypes are more scattered. Avian genotypes I and II belong to the intestinal clade and are closely related to *C. baileyi*. Genotypes III and IV belong to the gastric clade, where genotype III is closely related to the Eurasian woodcock genotype and *C. serpentis*, and genotype IV is closely related to *C. galli* (Ng et al. 2006).

Prevalence data based on fecal examination could be affected by the time from sampling to analysis. This has been observed when oocyst numbers in chicken faeces dropped to approximately one third in samples stored for a week from first to second analysis, and where first analysis was performed on the day after sampling (C. Axén, unpublished data). It is possible that oocysts die and are quickly degraded by detrimental effects (extreme pH) due to the high ammonium content of bird droppings.

4.6.1.1 Chickens

A flock prevalence of 41 % (23/56), with 10–60 % within-flock prevalence, was reported for *C. baileyi* respiratory infection in broilers in the USA (Goodwin et al. 1996). In Morocco, *Cryptosporidium* sp. were found in 14 (37 %) of 38 investigated flocks. Within-flock prevalence ranged from 14 % to 100 %, and the highest prevalence (52 %) was identified in broilers aged 36–45 days, with no infection prior to 25 days of age (Kichou et al. 1996). Diagnosis was based on histopathology.

An overall *Cryptosporidium* prevalence of 10.6 % for layer chickens and 3.4 % for broiler chickens was shown in a study of faecal samples from 2015 birds in China (Wang et al. 2010a). The highest prevalence (24.6 %) was found in 31–60-day old laying chickens, whereas prevalences in broiler chickens never exceeded 5 %. DNA analysis identified *C. baileyi* as the major species, with 92/95 investigated samples, and only 3 samples were positive for *C. meleagridis* (Wang et al. 2010a). In contrast, another recent study identified *C. meleagridis* as the major species in chickens (Baroudi et al. 2013). The overall *Cryptosporidium* prevalence was 34.4 % by histopathology, and the highest prevalence (46.2 %) was identified in 16–30-day-old chickens, which is in line with the results from Kichou et al. (1996) and Wang et al. (2010a). The majority of the birds were infected with *C. meleagridis* only ($n = 25$). *Cryptosporidium baileyi*

only was detected in four birds and a mixed *C. meleagridis*/*C. baileyi* infection was found in one bird. However, these chickens had died from diarrhoea, which could affect the outcome regarding *Cryptosporidium* sp.

4.6.1.2 Turkeys

A morbidity of 5–10 % due to sinusitis was reported for a flock where *Cryptosporidium* sp. could be isolated from diseased poults (Glisson et al. 1984). It was stated that macroscopic *post mortem* examination of the infraorbital sinuses of healthy birds was normal compared with those of diseased birds, but it was not clearly stated whether *Cryptosporidium* sp. was also identified in the healthy birds and thus the infection prevalence cannot be estimated. Goodwin et al (1988b) identified invasive *Cryptosporidium* stages in turkey poults form a farm where the poults suffered from self-limiting diarrheal of unknown aetiology, but no prevalence estimation was given. Prevalences of 80 % in 17-day-old poults, 38 % in 24-day-old and 0 % in ≥60-day-old poults was found by Woodmansee et al. (1988). Oocysts were identified as *C. meleagridis* based on morphology and infection site. A 35.5 % (17/60) prevalence in diarrhoeic or just unthrifty poults was reported in Iran (Gharagozlou et al. 2006). Prevalence was based on histological examination of intestinal, bursal and cloacal tissues. Examination of faeces revealed that only 29 % of the infected birds shed oocysts. Infection was identified in 1–7-week-old poults, whereas the 43 uninfected poults all were older than 7 weeks. DNA analysis was not performed and oocyst size was not stated in the publication, but based on infection site, host species and symptoms the authors suggested that *C. meleagridis* was the species responsible. A 10.0 %, 10.5 % and 2.5 % pre-slaughter prevalence respectively (age 4–9 weeks) was detected upon faecal examination of three flocks from the same farm (McEvoy and Giddings 2009). One of 59 turkeys was positive at post-slaughter examination (age 14 weeks). Upon DNA analysis, all six positive samples were identified as *C. parvum*. In a recent study, a 43.9 % prevalence of *C. meleagridis* was shown in deceased turkeys, with the highest prevalence (57.9 %) in poults aged 16–30 days (Baroudi et al. 2013).

4.6.1.3 Ducks and Geese

In one study, 73 (57 %) of 128 ducklings and 44 (59 %) of goslings aged 8–35 days were infected with *Cryptosporidium* (Richter et al. 1994). Infection was present in both intestinal and respiratory tract, but oocyst morphology was not described.

In a study on experimental infection with Usutu virus in geese, *Cryptosporidium* developmental stages in tissue samples were an accidental finding. This was further investigated by *in situ*-hybridization, and *Cryptosporidium* infection was detected in 89 % of conjunctival tissue samples and 88 % of bursal tissue samples. DNA analysis revealed presence of *C. baileyi* (Chvala et al. 2006). *C. baileyi* was also identified in two ducks in Rio de Janeiro (Huber et al. 2007).

4.6.1.4 Other Birds

Ratites *Cryptosporidium* infection in ostriches was first described in the early 1990s (Allwright and Wessels 1993; Bezuidenhout et al. 1993; Gajadhar 1993, 1994; Penrith et al. 1994; Penrith and Burger 1993). Infection was first identified in faecal samples from 14 (8.5 %) of 165 ostriches imported from Africa to Canada (Gajadhar 1993). Penrith and Burger (1993) identified invasive stages in a section of the small intestine of a 4-week old chick that has suffered from rectal prolapse, and Allwright and Wessels (1993) identified *Cryptosporidium* in histology sections of the bursa, intestine and pancreatic ducts. In 1994, Gajadhar et al. characterized the isolated oocysts and investigated host specificity. The oocysts were morphologically similar to those of *C. meleagridis*, but attempts to infect suckling mice, chickens, turkeys and quail failed, indicating that this was probably another species. In addition, only faecal samples were investigated, so the infection site was not determined (Gajadhar 1994). As this study was conducted before molecular tools were commonly used for *Cryptosporidium* species determination, the true identity of this isolate will remain unknown.

A low prevalence, with only 2 (0.6 %) of 336 investigated samples from ostriches aged 2 months–5 years being *Cryptosporidium* positive, was found in Greece (Ponce Gordo et al. 2002). Oocysts were of two sizes, 3.8 × 3.8 μm and 5.7 × 4.8 μm, indicating the presence of two different species. In contrast, in a Spanish study a 60 % *Cryptosporidium* prevalence in adult rheas and ostriches was found (Ponce Gordo et al. 2002). The authors reported an oocyst diameter of 3–5 μm, which is similar to the description provided by Gajadhar (1994). Molecular analysis of the isolates was not performed. Oliveira et al. (2008) found 44 % prevalence in 77 ostriches based on microscopy. Oocysts were generally morphologically similar to *C. baileyi* and *Cryptosporidium* avian genotype II (Ryan and Xiao 2008). However, the morphometric variation was so large that the authors suggested that more than one species had been identified (Oliveira et al. 2008), but this was not verified by molecular analysis. An isolate similar to *C. baileyi* in both oocyst morphology and PCR-RFLP banding pattern was described from Brazilian ostriches (Santos et al. 2005). The isolate was characterized as a sister taxon to *C. baileyi* by sequence analysis of the 18S rDNA, HSP70 and actin genes (Meireles et al. 2006), and was named *Cryptosporidium* avian genotype II by another research group (Ng et al. 2006). Experimental infection (oral or intratracheal) with the Brazilian isolate in chickens failed (Meireles et al. 2006). The avian genotype II has also been identified in Vietnam. On a single ostrich farm 110 (23.7 %) of 464 samples were positive for *Cryptosporidium* oocysts. The highest prevalence as well as the highest shedding intensity (35.2 %) was found in 2–3 month-old animals. Of 17 samples used for molecular characterization, all were found to be avian genotype II (Nguyen et al. 2013).

Wang et al (2011b) reported *Cryptosporidium* infection in 53 (11.7 %) of 452 investigated ostrich samples. Prevalence peaked at the age of 4–8 weeks with 16.2 %. No infection was detected in birds younger than 1 week or older than 12 months. Molecular analysis of positive samples identified only *C. baileyi*.

Quails and partridges Enteric cryptosporidiosis in quails, with oocysts similar to *C. meleagridis*, was first described in 1986 (Hoerr et al. 1986; Ritter et al. 1986). Early attempts at experimental infection of quail with *C. baileyi* isolated from chickens failed (Current et al. 1986; Lindsay et al. 1986), but were later successful (Cardozo et al. 2005). Natural infection was first documented in 2001 (Morgan et al. 2001). Since then, natural *C. baileyi* infection has been described in two reports (Murakami et al. 2002; Wang et al. 2012). One large survey of *Cryptosporidium* infection in quails was performed in China (Wang et al. 2012). Out of 1,818 faecal samples, 239 (13.1 %) from 29 (61.7 %) farms were positive. Infection was most common among 72–100-day old quails (23.6 %). DNA analysis revealed *C. baileyi* in 237 samples and *C. meleagridis* in two samples. One case of *Cryptosporidium* infection in partridges was described (Pages-Mante et al. 2007).

Pigeons There are a few reports of cryptosporidiosis in pigeons (Ozkul and Aydin 1994; Qi et al. 2011; Radfar et al. 2012; Rodriguez et al. 1997). Radfar et al. (2012) describe an overall prevalence of 2.9 % in 102 examined adult and nestling birds, with 3.4 % prevalence in adults and 2.3 % prevalence in nestlings. The other articles are case reports (Ozkul and Aydin 1994; Rodriguez et al. 1997) and a study on pet birds in general, where *C. meleagridis* was found in one pigeon (Qi et al. 2011).

4.6.2 Association of Infection with Clinical Disease

4.6.2.1 Chickens

Respiratory as well as intestinal and bursal *Cryptosporidium* infections cause disease in chickens, but infection without clinical symptoms has also been observed (Fletcher et al. 1975; Taylor et al. 1994).

In Spain, a 90 % morbidity due to respiratory infection in one flock was caused by *Cryptosporidium* sp. Weekly mortality rates were 0.9–1.5 % (Fernandez et al. 1990). Infection was detected in the trachea and oesophagus. In another flock investigated in the same study, weight loss was the primary symptom, and bursal cryptosporidiosis was diagnosed (Fernandez et al. 1990). Goodwin et al. (1996) found a correlation between *C. baileyi* infection of the trachea and severity of tracheitis symptoms, airsacculitis and condemnation of birds.

In respiratory cryptosporidiosis, co-infection with other pathogens has been identified in a number of studies. *Cryptosporidium* sp. and concurrent adenovirus infection was identified in a large broiler flock with respiratory disease (Dhillon et al. 1981). In a retrospective study on *post mortem* diagnoses of respiratory cryptosporidiosis, it was found that co-infection with virus or bacteria was common (Goodwin et al. 1988a). In another study, *Cryptosporidium* sp. and *Aspergillus* or bacteria were detected in the lungs of four layer chickens that died from pneumonia. *Cryptosporidium* were also found in the ureters and kidneys (Nakamura and Abe 1988). The effect of *Cryptosporidium* infection alone on development of clinical

symptoms in these cases cannot be estimated, but there is probably a synergistic effect of co-infections, increasing the severity. Such a synergistic effect of co-infection with infectious bronchitis virus or *Escherichia coli* has been reported (Blagburn et al. 1991).

Respiratory symptoms were reported from chickens that had been experimentally inoculated intra-tracheally with *C. baileyi*, whereas infection was successful but caused no symptoms in orally inoculated chickens (Lindsay et al. 1988).

C. meleagridis infection was associated with diarrhoea and mortality in one study of Algerian chickens (Baroudi et al. 2013). Experimental *C. meleagridis* infection of chickens has been observed to result in the chickens becoming indolent and having soiled feathers. Growth retardation was reported, but compensatory growth occurred after a few weeks (Tumova et al. 2002).

4.6.2.2 Turkeys

Turkey was the first animal species in which clinical cryptosporidiosis was described (Slavin 1955). Infection was associated with diarrhoea at 10–14 days of age, but other parasites (including *Histomonas*, *Trichomonas* and Strongylides) were also detected. Experimental infection (crop inoculation) with *C. meleagridis* produced infection of the ileum, caecum and bursa, but was not associated with clinical symptoms (Bermudez et al. 1988). The isolate used was from symptomatic poults; however, these were simultaneously infected with reovirus (causing enteritis and hepatitis). Co-infection with *Cryptosporidium* and reovirus in turkeys with enteritis and hepatitis, leading to increased mortality, has also been shown in another study (Wages and Ficken 1989). The presence of other pathogens in these studies could indicate a low to moderate primary pathogenicity of *C. meleagridis*. Self-limiting diarrhoea (moderate to severe in character), a slower growth rate and growth deformities were reported from one farm where diseased poults were diagnosed with *Cryptosporidium* infection (Goodwin et al. 1988b). Other pathogens were not excluded, as was also mentioned by the authors.

In dead poults that had suffered from depression and diarrhoea (faeces adhered on the hind part of the body), necropsy revealed lesions in the small intestine. Microscopic investigation identified *Cryptosporidium* sp. in the respiratory tract and kidneys, as well as in the gastrointestinal tract (Tacconi et al. 2001). Diarrhoea, emaciation, lethargy and reduced growth associated with natural *C. meleagridis* infection have been reported from Iran, but the presence of other pathogens was not excluded (Gharagozlou et al. 2006). Of 60 diarrhoeal and/or unthrifty birds, 17 (35.3 %) were identified as *Cryptosporidium* positive by histology, and *C. meleagridis* was reported based on oocyst morphology. Baroudi et al. (2013) identified *C. meleagridis* in 25 (44 %) of 57 examined turkeys that died from diarrhoea, but infection with other pathogens was not investigated.

Respiratory cryptosporidiosis has also been described in turkeys (Ranck and Hoerr 1987; Tarwid et al. 1985). Tarwid et al. (1985) identified *Cryptosporidium* sp. in necropsied birds from two outbreaks of colibacillosis. Colibacillosis is, according to

the authors, a secondary disease in turkeys, and *Cryptosporidium* sp. was identified as the primary pathogen. Symptoms were frothy conjunctivitis and increased mortality. Necropsy revealed pathological changes such as pericarditis, peritonitis and air-sacculitis in addition to the conjunctivitis that was observed in live birds.

Thirteen birds with respiratory disease were all positive for *Cryptosporidium* sp. by histology (Ranck and Hoerr 1987). Microscopy of sinus and/or tracheal exudates revealed oval oocysts in some samples, but oocyst size was not described, and both *C. baileyi* and *C. meleagridis* can appear oval (length/width ratios of 1.05–1.79 and 1.00–1.33 respectively (Ryan and Xiao 2008)). Symptoms such as coughing, rattling, sneezing, frothy eyes and swollen sinuses were reported. Other pathogens were present in all but two of the examined birds, and it is unclear whether the infection with *Cryptosporidium* played a primary role in the pathogenesis or not. Studies on cryptosporidiosis in turkeys, including clinical symptoms, are summarised in Table 4.13.

4.6.2.3 Ducks and Geese

Clinical cryptosporidiosis in ducks and geese seems to be less common and milder (see Table 4.14) than in other poultry. Only mild respiratory symptoms resulted from experimental *C. baileyi* infection (both oral and intratracheal inoculation) in ducks (Lindsay et al. 1989). Respiratory and intestinal infection occurred for both infection routes, but symptoms (sneezing, rales, mild dyspnea) were only present in animals infected by the intratracheal route. Mason (1986) described a case of conjunctival cryptosporidiosis. However, since only one of 97 affected ducks was *Cryptosporidium* positive, the author concluded that the parasite was not the cause of the disease. Similarly, no symptoms occurred in geese in which *Cryptosporidium* infection was detected in the conjunctivas and bursas (Chvala et al. 2006); and Richter et al. (1994) noted that enteritis and upper respiratory tract symptoms were equally present in infected and non-infected ducks and geese. Mortality was not increased in the positive flocks (Richter et al. 1994).

4.6.2.4 Other Birds

Ratites *Cryptosporidium* infection in ostrich chicks has been associated with cloacal and phallus prolapse, leading to high mortality (Bezuidenhout et al. 1993; Penrith et al. 1994; Santos et al. 2005). Bezuidenhout et al (1993) found that prolapsed cloacas were heavily infected, whereas Penrith et al. (1994) described heavy infection of both the bursa and cloaca in affected chicks, but healthy chicks were not infected. Santos et al (2005) also identified *Cryptosporidium* infection in the rectum, coprodeum, urodeum and bursa of two dead chicks with cloacal prolapse, both originating from a farm with high mortality rates in 7–30-day-old chicks. However, the authors did not associate the problems with the infection, since changed management practices decreased clinical symptoms and mortality,

Table 4.13 Studies on *Cryptosporidium* infection in turkeys

Country	Age	Symptoms	Location of parasites	Oocyst size	*Cryptosporidium* sp.	Diagnostic method	Reference
United Kingdom	10–14 days	Diarrhea, mortality	Distal jejunum, ileum	4.5 × 4.0 μm	*C. meleagridis*	Feces (smears) histopathology	(Slavin 1955)
United States	7 weeks	Sinusitis, serous conjunctivitis	Infraorbital sinuses	Not stated	*Cryptosporidium* spp.	Histopathology	(Glisson et al. 1984)
Canada	5 weeks	Bronchopneumonia, conjunctivitis, mortality	Trachea	Not stated	*Cryptosporidium* spp.	Histopathology	(Tarwid et al. 1985)
United States	2.5–11 weeks	Coughing, gasping, sneezing, rattling, sinusitis	Turbinates, sinuses, trachea, bronchi	Not stated	*Cryptosporidium* spp.	Exudate (smears) histopathology	(Ranck and Hoerr 1987)
United States[a]	5–26 days	None	Ileum, caecum, bursa	4.9 μm Ø	*C. meleagridis*	Feces (smears + auramine O) histopathology	(Bermudez et al. 1988)
United States	Not stated	Diarrhea, depression, growth retardation, abnormal feathers, misshapen bones	Mid - to distal small intestine	Not stated, developmental stages 2–4 μm Ø	*C. meleagridis*	Pathology, histopathology	(Goodwin et al. 1988b)
United States	25 days	Enteritis, depression, stunted growth, mortality	Ileum, ileocaecal junction	5 μm	*C. meleagridis*	Feces (smears + auramine O) histopathology	(Wages and Ficken 1989)
Hungary[a]	1 weeks	None stated	Mainly small intestine[b]	4.8 × 4.2 μm	*C. meleagridis*	Histopathology, 18S rDNA PCR +	(Sreter et al. 2000)
Italy	30 days	Diarrhea, depression, huddling	Ileum, caecal tonsil, caecum, rectum, bursa, duodenum, proventriculus, kidney, trachea, lung	4.5–5.0 μm Ø	*C. meleagridis*	mucosal scrapings (flotation + mZN) histopathology	(Tacconi et al. 2001)

Iran	1–7 weeks	diarrhea, emaciation, lethargy, growth retardation	Duodenum, jejunum, ileum, caecum, colon, cloaca, bursa	Not stated, developmental stages <5 µm Ø	*C meleagridis*	Feces (flotation + mZN) pathology, histopathology	(Gharagozlou et al. 2006)
United States	4, 9, 18 weeks	None stated	unknown/ caecum[c]	Not stated	*C. parvum*	18S rDNA analysis of fecal droppings and post-slaughter caecal content	(McEvoy and Giddings 2009)

[a]Experimental infection
[b]Determined in chickens and mice, oocysts first passaged through turkey poults
[c]One caecum-positive

Table 4.14 Studies on *Cryptosporidium* spp. in ducks and geese

Country	Age	Species	Symptoms	Location of parasites	Oocyst size	*Cryptosporidium* sp.	Diagnostic method	Reference
Ducks								
United States[b]	4 days	Domestic ducks	None or mild respiratory disease	Trachea, Bursa	Not stated	*C. baileyi*	Histology	(Lindsay et al. 1987)
Germany	9–35 days	Peking ducks	Not stated	Bursa, cloaca, intestine, respiratory tract, conjunctiva	Not stated	*Cryptosporidium* spp.	Tissue scrapings + mZN, histology + IFA	(Richter et al. 1994)
Australia	Not stated	Black duck (wild)	Not stated	Not stated	Not stated	*Cryptosporidium* duck genotype	18S rDNA PCR + sequencing	(Morgan et al. (2001)
United States	Unknown	Wild ducks	Unknown	Intestine	Not stated	*Cryptosporidium* spp.	Faecal flotation + IFA, 18S rDNA PCR	(Kuhn et al. 2002)
Brazil	Not stated	Domestic ducks	Not stated	Not done	Not stated	*C. baileyi*	18S rDNA PCR-RFLP and sequencing	(Huber et al. 2007)
Geese								
United States	25 days	Domestic geese	Not stated	Large intestine	Not stated Schizont 3.6 μm Macrogamete 4.5 μm	*Cryptosporidium anserium*, nomen nudum	Histology	(Proctor and Kemp 1974)
Germany	8–35 days	Domestic geese (Danish breed)	Not stated	Bursa, gastro-intestinal and respiratory tract	Not stated	*Cryptosporidium* spp.	Tissue scrapings + mZN, histology + IFA	(Richter et al. 1994)

United States	Unknown	Canada geese	Unknown	Not done	Not done	*C. parvum*[c]	TRAP C2 and β-tubulin PCR + genotyping	(Graczyk et al. 1998)
Austria[a]	16–36 days	Domestic geese	Not stated	Bursa, conjunctiva	Not done	*C. baileyi*	Histology with in-situ hybridization, 18S rDNA PCR + sequencing	(Chvala et al. 2006)
United States	Unknown	Canada geese	Unknown	Not done	Not done	*Cryptosporidium* goose genotypes I, II, III, IV, V[d]	18S rDNA PCR and sequencing	(Jellison et al. 2004)
United States	Unknown	Canada geese	Unknown	Not done	Not done	*Cryptosporidium* goose genotypes I, II, *Cryptosporidium* duck genotype, *C. parvum*[c], *C. hominis*[c]	18S rDNA PCR-RFLP and sequencing	(Zhou et al. 2004)

[a]Experimental infection study for Usutu virus, *Cryptosporidium* accidental finding
[b]Experimental infection
[c]Finding reported as passage of oocysts, not manifest infection
[d]Not named in this publication

although *Cryptosporidium* infection was still present on the farm. Enteritis was indicated by the presence of intestinal invasive stages and rectal prolapse in one chick examined by Penrith and Burger (1993). Because diarrhoea was not reported it is unknown whether the prolapse was caused by intense bowel movements or something else. *Cryptosporidium* infection has also been associated with pancreatic necrosis (Allwright and Wessels 1993).

Quails and partridges *Cryptosporidium* infection has been shown in both diarrhoea and respiratory disease in quails (Guy et al. 1987; Hoerr et al. 1986; Murakami et al. 2002; Ritter et al. 1986). Hoerr et al. (1986) reported high mortality rates from 5 days of age in quails infected with *Cryptosporidium* sp., and with no bacterial or viral pathogens detected. Acute fatal diarrhoea with mortality rates of up to 45 % in 0–17-day-old birds was described by Ritter et al (1986). Reovirus was also detected in necropsied birds, but another study reported that experimental infection with reovirus alone did not produce diarrhoea, whereas infection with *Cryptosporidium* sp., either alone or simultaneously with reovirus, resulted in severe diarrhoea and mortality (Guy et al. 1987). A synergistic effect of co-infection was, however, shown, since oocyst shedding was higher and reovirus infection became systemic and liver necrosis occurred (Guy et al. 1987).

Muramaki et al. (2002) reported a daily mortality rate of 5.7 % in one farm, where birds suffered from upper respiratory tract disease and decreased egg production. Respiratory symptoms were head swelling, nasal discharge and increased lacrimation, and necropsy revealed sinusitis, airsacculitis and egg peritonitis. Co-infection of *Cryptosporidium* sp., *Mycoplasma gallisepticum* and other bacteria was shown. The authors concluded that *M. gallisepticum* was the primary pathogen, but that the mixed infections in conjunction with high ammonia concentrations in the air worsened the symptoms. The role of *Cryptosporidium* infection in respiratory disease in quails thus remains unclear. Wang et al. (2012) reported that no clinical symptoms were seen in 1,818 sampled quails, of which 239 were *Cryptosporidium* positive.

C. meleagridis was the only pathogen identified in an outbreak of diarrhoea and cough in red-legged partridge chicks (Pages-Mante et al. 2007). Morbidity rates were 60–70 % and mortality more than 50 %, indicating high pathogenicity. Invasive stages were identified in both the respiratory and intestinal tract, suggesting that not only *C. bailey* might be associated with respiratory avian cryptosporidiosis.

Pigeons Diarrhoea associated with cryptosporidiosis in pigeons has been described in four birds (Ozkul and Aydin 1994; Rodriguez et al. 1997). Rodriguez et al. (1997) described a 40 % morbidity of yellow watery diarrhoea, weight loss, dehydration and weakness in a farm with 280 pigeons. Mortality was 5 % and necropsy of three birds revealed invasive stages of *Cryptosporidium* in the small intestine, caecum, colon, cloaca, and bursa. No viruses or bacteria could be isolated. Ozkul and Aydin (1994) identified invasive stages in the small intestine of a pigeon that had been depressed and had evidence of diarrhoea in the form of faeces in its hind feathers.

4.6.3 *Infection Dynamics: Oocyst Excretion and Transmission*

Isolates of both *C. baileyi* and *C. meleagridis* derived from one domestic bird species have been successfully transmitted to other domestic birds (Current et al. 1986; Lindsay et al. 1987). *C. galli* has not been experimentally transmitted between different domestic birds, but has been shown in finches as well as chickens (Blagburn et al. 1990; Pavlásek 1999, 2001; Ryan et al. 2003b), and thus has the potential to infect different bird species.

4.6.3.1 Chickens

The prepatent period of *C. baileyi* is approximately 4–8 days (Hornok et al. 1998; Lindsay et al. 1988; Rhee et al. 1991; Tumova et al. 2002). However, in the first report on *C. baileyi* infection in chickens, a prepatent period of up to 24 days was described (Current et al. 1986). Older chicks have a slightly longer prepatent periods than younger ones (Lindsay et al. 1988; Rhee et al. 1991; Taylor et al. 1994; Tumova et al. 2002).

The patent period varies more. At oral inoculation of 2-day-old chicks, a patent period of 26 days was seen, whereas it was 11–15 days in chicks inoculated at 14 days of age, 11–12 days in chicks inoculated at 28 days of age and <7 days in chicks inoculated at 42 days of age (Lindsay et al. 1988). With intratracheal inoculation, the same authors described patent periods of 27, 11–19, 10–11 and <7 days in these age groups (Lindsay et al. 1988). Rhee et al. (1991) and Tumova et al. (2002) observed a mean patent period of approximately 14 days. Oocyst excretion peaked on day 12 and days 11–17 post inoculation, respectively (Rhee et al. 1991; Tumova et al. 2002). Taylor et al. (1994) showed shorter patent periods and lower total oocyst output in older than younger chickens. There was also an effect of infection dose, in that oocyst output was higher and declined more slowly with lower infection doses (Taylor et al. 1994). Similar observations were made for 1- and 9-week old chicks (Sreter et al. 1995). In that study, the mean patent period for 1-week old chicks was 32 days, but one chicken shed oocysts for 151 days.

C. meleagridis was shed in the faeces on days 4–7 post infection in two chickens experimentally infected at 6 weeks of age (Woodmansee et al. 1988). Tumova et al. (2002) infected 7-day-old chicks. Oocysts first appeared 3 days later and the patent period lasted for 16–17 days. Shedding rates were significantly lower than in chicks inoculated with the same number of *C. baileyi* oocysts.

The prepatent and patent period of *C. galli* has been described to be 25 and 6 days respectively (Pavlásek 2001), but was later reported as unknown when *C. galli* was redescribed (Ryan et al. 2003b).

4.6.3.2 Turkeys

The prepatent period of *C. meleagridis* in turkeys inoculated at 7–11 days of age was 2–4 days (Bermudez et al. 1988; Sreter et al. 2000; Woodmansee et al. 1988).

Woodmansee et al. (1988) reported that oocysts were shed for only 4 days; Sreter et al. (2000) found the patent period to be 8–10 days, whereas Bermudez et al. (1988) reported oocyst shedding and invasive stages still being present at day 21 post inoculation. Oocyst shedding rates were moderate (Sreter et al. 2000), and low to moderate (Bermudez et al. 1988).

Experimental infection with *C. baileyi* induced mild infection of the bursa (Current et al. 1986). Lindsay et al. (1987) inoculated turkey poults via the intratracheal, oral and intracloacal route. All three experiments caused infection, but only poults inoculated via the trachea developed symptoms (Lindsay et al. 1987).

4.6.3.3 Ducks and Geese

Lindsay et al (1986) described a prepatent period of 5 days and a possible patent period of 9–10 days in experimentally infected Muscovy ducks, based on investigation of pooled faecal samples (Lindsay et al. 1986). Oocyst morphology was not described. The intestine, bursa and cloaca were positive for invasive stages, but these tissues can be infected by both *C. baileyi* and *C. meleagridis* (Table 4.14).

4.6.3.4 Other Birds

For quails, one study describes a prepatent period of 7 days and a patent period of 21 days for *C. baileyi* (Cardozo et al. 2005). Otherwise, no data are available.

4.6.4 Zoonotic Transmission

Only one of the species and genotypes commonly infecting birds – *Cryptosporidium meleagridis* – has, so far, proved to be important in human cryptosporidiosis. This species is the third most common species in human cryptosporidiosis worldwide. In the industrialised world, *C. meleagridis* infection is usually associated with cryptosporidiosis cases in travellers to Asia or Africa (Elwin et al. 2012; Insulander et al. 2013; Leoni et al. 2006), but autochthonous cases have also been described (Elwin et al. 2012; Leoni et al. 2006; Silverlås et al. 2012). Studies on *Cryptosporidium* prevalence and species distribution in humans in South America have identified *C. meleagridis* infection at about the same prevalence as *C. parvum* (Cama et al. 2003, 2007, 2008). Although this is a true zoonotic species, there is only one report in which the bird source has been identified, and in that case,

chickens and not turkeys were involved (Silverlås et al. 2012). It is not known whether anthroponotic transmission occurs with this species, but it has been indicated by the fact that not all *C. meleagridis*-infected patients in an epidemiological investigation had had bird or animal contact (Elwin et al. 2012). *C. meleagridis* has the potential to infect other mammalian species as well, and experimental infection of mice, rats, rabbits, pigs and calves has been reported (Akiyoshi et al. 2003; Darabus and Olariu 2003). Due to the wide host range and the close relationship of *C. meleagridis* to *C. parvum* and *C. hominis*, it has been proposed that this species originated as a mammalian *Cryptosporidium* species, and later adapted to birds (Xiao et al. 2002b, 2004).

One study has identified *C. parvum* in turkeys (McEvoy and Giddings 2009), indicating that this species could play a role in zoonotic transmission. However, only one of 59 birds post-slaughter was positive compared to 2.5–10.5 % of the 5–10-week younger poults, which means risk of transmission via contaminated meat should be very small. The higher prevalence in poults should not pose a risk as long as the flocks are closed to the public. The shedding intensity was not reported, but prevalence indicates that infection rather than just intestinal passage was present. Some studies have identified *C. parvum*, *C. hominis* and *C. hominis*-like isolates in Canada geese (Jellison et al. 2004, 2009; Zhou et al. 2004). The authors conclude that these findings are probably not associated with infection and parasite proliferation, but rather transient carriage. Nevertheless, this indicates that domesticated ducks and geese can potentially act as transmission vehicles for these species.

Infection with *C. baileyi* has been identified in one immunodeficient patient. Diagnosis was based on oocyst morphology and biology – experimental infection of mice failed whereas inoculated chickens developed infection of the intestine, bursa and trachea (Ditrich et al. 1991). Since this patient was immunodeficient and no other reports exist, this species should not be considered as a true zoonotic agent.

4.7 Conclusion

Ever since animals were first domesticated, and humans became dependent upon them for the commodities that they supply, particularly food and fibre, the infections that affect the health and productivity of livestock have been a concern. Cryptosporidiosis was first identified as a disease of veterinary significance in the 1950s (in turkeys) and then in the early 1970s in calves, but major interest in cryptosporidiosis only developed with the first report of a human cases later that decade, and the recognition that *Cryptosporidium* infection was also of medical importance. Since then our knowledge on the veterinary significance of *Cryptosporidium* infection has expanded enormously – particularly in the livestock sector most impacted by cryptosporidiosis – young calves. However, as demonstrated in this chapter, it should not be forgotten almost all farmed animals may be pathologically affected by at least one species of *Cryptosporidium*, often

causing clinical disease that in some instances may be fatal. For some *Cryptosporidium* species in some farmed animal species, transmission may be anthropozoonotic.

Cryptosporidium is a hugely successful parasite, as demonstrated by its host range and wide geographic distribution, and its control has proved challenging. As long as humans raise and depend on animals, there will be a need to control the transmission of cryptosporidiosis amongst livestock species.

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