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A review of the importance of cryptosporidiosis in farm animals

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Abstract

Cryptosporidium species are coccidian parasites with a large capacity to reproduce and to disseminate. Several species are known to infect farm animals, although the economic importance of cryptosporidiosis is highly host species dependent. This paper reviews the impact of cryptosporidial infections in livestock and poultry. For different farm animals, the *Cryptosporidium* spp. that occur, as well as their clinical and pathological features, and their interactions with other pathogens, are described. In addition, data concerning the prevalence, the transmission and the epidemiology of the disease are mentioned and a description of the economic losses associated with cryptosporidiosis in each of the hosts is given. Cryptosporidiosis seems to be mainly a problem in neonatal ruminants. *Cryptosporidium parvum* is considered to be an important agent in the aetiology of the neonatal diarrhoea syndrome of calves, lambs and goat kids, causing considerable direct and indirect economic losses. Avian cryptosporidiosis is an emerging health problem in poultry, associated with respiratory disease in chickens and other Galliformes, and with intestinal disease in turkeys and quails. Because of limited availability of effective drugs, the control of cryptosporidiosis relies mainly on hygienic measures and good management. © 1999 Australian Society for Parasitology Inc. Published by Elsevier Science Ltd. All rights reserved.

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1. Introduction

The genus *Cryptosporidium* was named at the beginning of this century, but was only recognised as a potential cause of disease in 1955, when it was found to be associated with diarrhoeic turkeys [1]. Although *Cryptosporidium* was subsequently found in a broad range of farm animals, its impact was neglected until the early 1980s when it was found to be a common, serious primary cause of outbreaks of diarrhoea in certain farm mammals [2, 3]. The fact that *Cryptosporidium* was found to infect humans [4], and could cause a life-threatening disease in

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immunodeficient people, especially AIDS patients [5], as well as the association of *Cryptosporidium* with waterborne-related human outbreaks of diarrhoea [6], has certainly given the parasite a more widespread recognition. It has encouraged the scientific work on *Cryptosporidium* in many domains, such as in the veterinary field, to some extent because animal husbandry is seen as a threatening source of infection for humans by the release of tremendous numbers of resistant oocysts in surface waters. Recent studies have revealed the heterogeneity of the *Cryptosporidium* sp. infecting both humans and animals, making the causal connection between animal and human cryptosporidiosis far more complex [7]. Nevertheless, the interest for cryptosporidiosis in the veterinary field arises even more from the fact that it concerns a harmful, difficult to control disease of many farm animals, that results in significant economic losses. This point will be addressed in this review.

2. Life cycle

The life cycle of *Cryptosporidium* is direct and monoxenous and it follows the patterns described for other enteric coccidia, which include a merogonic cycle with two generations of meronts, a gametogonic cycle with macrogametes, microgametes and zygotes and a sporogony (outlined in [8]). Compared with *Eimeria* spp., the life cycle of *Cryptosporidium* is characterised by a number of peculiarities, some of them of major importance for the establishment and spread of the infection and for the treatment of the disease: (i) exposure of *Cryptosporidium* oocysts to reducing conditions, pancreatic enzymes and bile salts, results in a high percentage of excystation. However, in contrast to most other coccidia, *Cryptosporidium* oocysts can liberate their sporozoites in warm aqueous solutions without any of the aforementioned special stimuli [9]. This spontaneous excystation explains, in part, the ability of *Cryptosporidium* to infect tissues other than the intestine, such as the conjunctiva of the eye [10–13] and the respiratory tract [10, 14–19];

(ii) the parasite develops inside the epithelial cell of the digestive or respiratory tract, although on the edge of the host cell cytoplasm and separated from it by a feeder organelle membrane. This intracellular extracytoplasmatic location is unique for the coccidia and might play a major role in the failure of many antimicrobial agents to inhibit the growth of *Cryptosporidium* [20]; (iii) two stages can cause auto-infection: the recycling type I meronts and the thin-walled oocysts. Consequently, in the absence of a protective immune response *Cryptosporidium* may persist inside a single host, even without further exposure to exogenous oocysts; (iv) the thick-walled oocysts are already fully sporulated when they leave the body with the faeces and are therefore immediately infectious. Thus, *Cryptosporidium* seems to have an extraordinary reproductive ability. In addition, the oocysts can travel a considerable distance following runoff [21], can survive for a relatively long time in an aqueous environment [22, 23], and are infectious to a wide range of animals, thus having many potential excretors [20]. As a result, this parasite undoubtedly has an exceptional capacity to disseminate.

3. *Cryptosporidium* infection in cattle

3.1. Historical background

Infection by *Cryptosporidium* spp. in cattle were first reported in the early 1970s [24–26]. However, because of the association with other viral or bacterial enteropathogens, the role of *Cryptosporidium* spp. as primary enteropathogens was uncertain until 1980, when Tzipori et al. [2] attributed an outbreak of neonatal diarrhoea to cryptosporidial infection alone. In the following years methods to free the infective oocysts from other contaminating pathogens became available, which permitted the experimental demonstration that *Cryptosporidium* was capable of causing clinical diarrhoea in calves [27, 28].

In cattle two species of the genus *Cryptosporidium* can be distinguished: *Cryptosporidium parvum* infecting the distal small intestine, and *Cryptosporidium muris* infecting the

abomasum. Substantial differences in the size and shape of *C. parvum* (5.0 μm$\times$ 4.5 μm and sperical [29, 30]) and *C. muris* (7.4 μm$\times$5.6 μm and ovoid [30]) oocysts enables the two species to be distinguished readily on microscopical examination. Only *C. parvum* has been associated with neonatal diarrhoea. *Cryptosporidium muris* is much less prevalent and was only found in weaned calves or adult cattle [31–33] and *C. muris* infection is considered to be clinically mild, affecting weight gain [34] and milk production [35].

3.2. Clinical and pathological features

The kinetics of oocyst shedding of experimentally *C. parvum* infected neonatal calves, revealed a prepatent and a patent period ranging from 3–6 and 4–13 days, respectively [36]. In practice, oocyst excretion has been described as early as 3 days of age, which means that calves are already susceptible for infection during or shortly after birth [37–39]. Calves raised in isolation from *Cryptosporidium* remain susceptible to infection at older age, but the clinical signs become less severe [40]. In neonates, a great variability was observed in the severity and duration of diarrhoea due to cryptosporidiosis, even when the animals were exposed to similar conditions. In most calves diarrhoea has already began 3–5 days p.i., and lasted from 4 to 17 days [36]. In fattening units, where the prevalence of *C. parvum* is known to be high (93%), up to 38% of the calves developed a liquid diarrhoea, that could be attributed to cryptosporidiosis [41]. Cryptosporidial diarrhoea is associated with the excretion of tremendous numbers of oocysts [42]. High mortality due to cryptosporidiosis has been reported, even in the absence of other enteropathogens [43], and this would occur more often in the Belgian Blue–White, and the French Limousin and Charolais meat breeds [44].

Cryptosporidium infections are mainly concentrated in the distal small intestine but lesions were also found in the caecum and colon [43, 45], and occasionally in the duodenum [43]. The pathological findings associated with *Cryptosporidium* are a mild to moderate villous atrophy, villous fusion, and changes in the surface epithelium (reviewed in [46]). Further, infiltration of mononuclear cells and neutrophils was seen in the lamina propria [43]. After primary exposure, α/β T-cells, both CD4[+] and CD8[+], and γ/δ T-cells accumulated in the intestinal villi, while in challenged immune animals only an increase in the number of CD8[+] T-cells was found [47]. Respiratory cryptosporidiosis has been reported [19], but can be considered to be of less economic importance than the enteritic form.

3.3. Interactions with other enteropathogens and prevalence

The neonatal diarrhoea syndrome, both in large and small (see Section 4.3) ruminants, is a clear example of a multifactorial disease governed by a wide range of factors related to the animal, conditions of the environment and husbandry, and a variety of viruses, bacteria and protozoan parasites.

In calves, enterotoxigenic *Escherichia coli* (ETEC), with thermolabile and/or thermostable enterotoxins and with colonisation factors [48], are recognised as a common cause of diarrhoea in calves under 3 days old. This age-dependence is caused by a diminished adherence of ETEC to enterocytes after the first few days of life [49].

Between 4 days and 6 weeks of age digestive problems can mostly be attributed to *C. parvum* and/or a variety of viruses, with rotavirus, coronavirus and bovine viral diarrhoea (BVD) virus being the most important. Data from the Veterinary and Agrochemical Research Centre in Brussels, on the proportional occurence of viral enteropathogens and *C. parvum* in samples from diarrhoeic calves (Fig. 1), highlighted the increasing importance of *Cryptosporidium*. During the period July 1982–June 1983 *Cryptosporidium* infections made up only 6.7% (3.9% as a single pathogen, 2.8% in association) of the digestive problems, whereas the impact of cryptosporidiosis was considerably greater in 1992–1993. Indeed, almost 40% of the diarrhoeic calves shed *C. parvum*; in 30.7% of the cases *Cryptosporidium* was the only pathogen present,
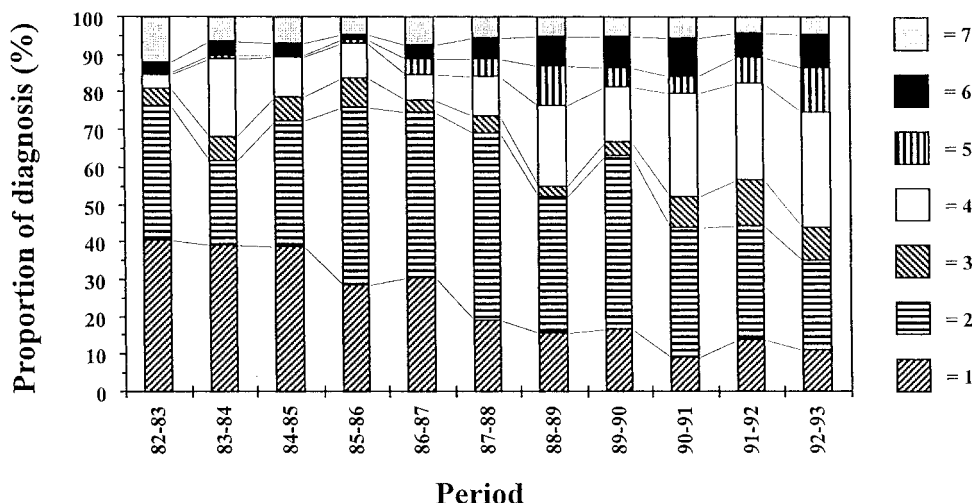
Fig. 1. Proportional occurrence of different enteropathogens in samples from diarrhoeic calves. Over a 10-year period, the number of cases with *Cryptosporidium* as single pathogen present, increased significantly. 1 = rotavirus, 2 = coronavirus, 3 = BVD-virus, 4 = *Cryptosporidium* as single pathogen, 5 = other single pathogens, 6 = *Cryptosporidium* in association with other pathogens, 7 = other associations.

and in 8.9% it was found in association with a viral infection. For comparison: rotavirus, coronavirus and BVD-virus were found as a single pathogen in, respectively, 11.2, 23.7 and 8.9% of the diagnosed samples. The decreased number of cases of rotavirus, shown in Fig. 1, is probably due to the use of commercialised rotavirus vaccines that became available halfway during the studied period (Vanopdenbosch, personal communication). The proportional occurrence of these pathogens may differ according to geographic parameters and the studied period, but in the last decade several studies described *C. parvum* as the most commonly detected enteropathogenic agent in calves [50–52].

Attaching and effacing *E. coli* have been implicated in diarrhoea and dysentery in 2- to 8-week-old calves [53, 54], with a calculated mean age of 5 weeks [55]. Verotoxigenic *E. coli*, a group of bacteria that produces potent cytotoxins known as verocytotoxins, were found to be occasionally associated with calf diarrhoea [56]. They were detected in 6.0 and 9% of diarrhoeic calves in Germany [57] and Spain [58], respectively. There are reports of viruses other than the aforementioned ones, populating the intestine of young calves (picobirnavirus [59], calici- and astrovirus [60], adenovirus [61], enterovirus [62] and parvovirus [63]), but their pathogenicity is still uncertain. *Giardia* infections have also been found to be exceptionally frequent in suckling and weanling calves, although the role of this protozoan parasite in the aetiology of diarrhoea in calves remains unclear [39]. Finally, the problems related to clinical salmonellosis are only minor in dairy, beef [64] and veal units [65].

Cryptosporidium parvum oocysts were detected in bovines ranging from 3 days old to adults, although the prevalence was significantly higher in suckling calves [39, 66–68]. In a Spanish epidemiological study on the prevalence and age distribution of *C. parvum* infections, as many as 44.4% of calves aged 3–4 days were infected, but infection rates peaked (76.7%) at 6–15 days of age. Prevalence was also high in weanling calves aged 1.5–4 months (14%), fattening calves and heifers 4–24 months old (7.7%) and adults (17.8%). However, *Cryptosporidium* infection was only statistically associated with diarrhoea in suckling calves [39].

3.4. Transmission and epidemiological factors

The transmission occurs by the faecal-oral route. The majority of adult cattle can be described as excretors of *C. parvum* oocysts when sensitive detection methods are used. Nevertheless, their importance in the transmission of the disease remains questionable, since oocyst excretion by adult cattle was similar in herds with serious problems of cryptosporidial neonatal diarrhoea and in those without [68]. So far, in cattle, no increased output of *C. parvum* oocysts around parturition has been observed [67–69]. However, these findings should be considered with great caution, as it was shown that more frequent samplings and more sensitive detection methods in sheep revealed a periparturient rise in oocyst score (Requejo-Fernandez JA, Pereira-Bueno J, Pilar-Izquierdo M, Rojo-Vazquez FA, Ortego-Mora LM. Periparturient rise in ovine cryptosporidiosis. In: Proc COST 820 WG3 Meeting. Brussels, 1997, p. 5).

Nevertheless, infected newborn calves excrete oocyst numbers of the order of 10^6 to $10^7\ \mathrm{g}^{-1}$ of faeces [42] and were considered to be a more dangerous source of infection. Infection can rapidly spread from calf to calf when animals are communally housed and overcrowded, or from cow to calf via the udders when they are contaminated with infected calf faeces in the lying area of the dams [46]. This might explain the association between cryptosporidial diarrhoea and the farm type and/or its specific hygienic conditions. *Cryptosporidium* infections were more common in single or multiple suckler beef herds [70] and dairy farms with multiple-cow maternity facilities [71]. In the so-called fattening units, where calves are purchased through markets, almost 100% of the animals become infected during transit from the market to the rearing unit or soon after their arrival [41].

Other potential risk factors are herd size [71] and season. In a Canadian study of beef calves, higher prevalence was found in winter and spring, the period related to calving season and consequently the period with the greatest number of calves in the high risk group (1- to 3-weeks-old) [72]. However, in American dairy farms, where calvings tend to be year-round, and environmental contamination level is less subjected to fluctuations, cryptosporidiosis was more prevalent in the summer [71].

Little information is available on differences in infectivity, excretion patterns, virulence or immunogenicity among different isolates of *C. parvum* from cattle or other sources [36]. This is at least partially due to the lack of single oocyst cloned isolates since all reported isolates are heterogenic mixtures.

3.5. Economic losses

The economic losses due to cryptosporidial infections of neonatal calves are related to diarrhoea: dehydratation, growth retardation and to a lesser extent mortality [43]. Diarrhoeic problems of calves demand special care: feeding of electrolyte solutions, i.v. fluid therapy, drug administration, hygienic measures, etc. which are costly as well as labour and time-consuming. In Belgium, mortality due to the neonatal diarrhoea syndrome is estimated between 5–10% (Vanopdenbosch, personal communication). *Cryptosporidium parvum* is considered as the most commonly found enteropathogen in calves during their first weeks of life [50–52]. The parasite frequently acts alone, but the losses are more severe when concurrent infections occur (Vanopdenbosch, personal communication), as has been demonstrated for viral and bacterial enteropathogens [73].

4. *Cryptosporidium* infection in sheep and goat

4.1. Historical background

In sheep, infection by *Cryptosporidium* was first described in Australia in 1- to 3-week-old lambs with diarrhoea [25]. Its role as a primary aetiological agent was confirmed in the early 1980s in studies on experimental infections in the absence of other enteropathogenic agents [3, 74]. Since then, *Cryptosporidium* has been attributed an increasingly important role in neonatal diarrhoea syndrome in this domestic species and is

currently associated with high morbidity rates and, depending on environmental conditions and the presence of other intestinal pathogens, mortality [46, 75, 76].

In goats, infection by this agent was also first described in Australia, in a 2-week-old kid with diarrhoea [77]. Since then, the infection has been diagnosed in outbreaks of diarrhoea in goat kids in several European countries [78–80] and is now considered to be one of the principal enteropathogens in these animals [76].

Only one of the two species of the genus *Cryptosporidium* described in domestic ruminants, *C. parvum*, has been associated with diarrhoea in small ruminants. In a recent work, a bovine isolate of *C. muris* did not produce infection in goats [81].

4.2. Clinical and pathological features

In lambs, the prepatent period oscillated between 2 and 7 days [82] and around 4 days in goat kids [83]. This period increases with a reduction in the dose of the infectious agent [84] or with an increase in the age of the animal [85]. The main clinical manifestations of cryptosporidiosis in neonate small ruminants are: apathy and depression, anorexia, abdominal pain, and mainly diarrhoea accompanied by the shedding of a large number of oocysts [74, 83, 85–88]. The faeces are usually yellow, have a soft or liquid consistency and give off a strong unpleasant odour. In experimental infections in lambs [85] the dry weight of the faeces can drop from 24 to 10%. In milder cases of the disease the animals have diarrhoea for 3 to 5 days and in more severe cases for 1 to 2 weeks. The diarrhoea usually coincides with the period of oocyst shedding. The duration of the oocyst shedding depends on factors such as the age or immune status of the animals. The kinetics of the shedding is usually similar in experimentally and naturally infected animals. After the first 2 or 3 days of the patent period there is a progressive increase in the number of oocysts shed which reaches a maximum 5–6 days p.i. and drops sharply between days 10 and 15 p.i. [83, 85, 89]. In addition to the diarrhoea and oocyst shedding the animals characteristically manifest anorexia [74, 83, 85] which results in weight loss and retarded growth during the first few weeks of life [90]. Studies on the development of the lesions over the incubation period and the clinical course of the disease [74, 83] have shown that, over this period, the parasite mainly proliferates in the jejunum and the ileum. After the start of oocyst shedding (3 days after the start of infection) the lesions can spread to other parts of the small and large intestine.

4.3. Interactions with other enteropathogens and prevalence

A recent study showed that the most frequent aetiologic agent involved in outbreaks of diarrhoea in lambs was *C. parvum* (65% of the outbreaks and 45% of the individuals) followed by *E. coli* potentially pathogenic (61% of the outbreaks and 30% of the individuals). Other agents such as rotavirus were less important (7% of the outbreaks and 2% of the individuals). In goat kids, *C. parvum* was present in 40% of the outbreaks and 42% of the individuals, followed by *E. coli* (36 and 22%), rotavirus (14 and 22%), *Clostridium perfringens* (20 and 11%) and *Salmonella* spp. (7 and 3%) [76]. In a parallel study, it has been shown that *E. coli* strains isolated from diarrhoeic lambs and kids older than 4 days (mainly between 7 and 15 days old) are not generally toxigenic and belong to a large number of serogroups [91, 92]. In small ruminants older than 4 weeks, infections by *Eimeria* spp. are increasingly important accompanied by dietary changes and situations of stress [93]. The role of other intestinal protozoa such as *Giardia* sp. is controversial and although infection by this parasite is often diagnosed during the first month of life [94] it is not clearly associated with the production of diarrhoea [39, 95].

The prevalence of infection by *C. parvum* in lambs and goat kids has been studied in both outbreaks of diarrhoea and in randomly selected farms (Table 1), although more research has been done on cattle. In outbreaks of diarrhoea, morbidity can be very high in lambs [98, 100] and goat kids [86, 102–104]. Mortality increases when

Table 1
Prevalence of C. parvum infections in lambs and kid goats

Host species	Country	Type of study*	Prevalence % (number infected/number sampled)		Reference
			Flock	Individual	
Sheep	Canada	3		24 (21/89)	[95]
Sheep	Iran	3	3 (9/280)	4 (18/433)	[96]
Sheep	Italy	1	8 (5/59)	12 (18/156)	[97]
Sheep	Spain	2	73 (16/22)	40 (53/132)	[98]
Sheep	Spain	1	47 (43/92)	15 (331/2,204)	[80]
Sheep	Spain	2	65 (30/46)	45 (82/183)	[76]
Sheep	Trinidad and Tobago	3		20 (18/90)	[99]
Sheep	USA	2		85 (27/32)	[100]
Goat	Hungary	2		25 (10/40)	[79]
Goat	Italy	1	13 (2/15)	19 (8/42)	[97]
Goat	Spain	1	35 (11/31)	11 (40/367)	[80]
Goat	Spain	2	40 (6/15)	42 (15/36)	[76]
Goat	Trinidad and Tobago	3		20 (4/20)	[99]
Goat	USA	3		26 (5/19)	[101]

* 1 = Cross-sectional study, 2 = diarrhoea outbreak, 3 = other.

the disease is associated with concurrent infections or deficiencies in nutrition or husbandry [82, 87, 100]. In goat herds in France and Hungary, C. parvum was considered to be the predominant aetiological agent in neonate goat kids with diarrhoea [79, 102]. In Spain, in a study on 97 sheep farms and 31 goat farms, all randomly selected, corresponding to a total of 2204 lambs and 367 goat kids under 5 weeks old, flock prevalence was 47 and 36% and individual prevalence was 15 and 11% for lambs and goat kids, respectively [80].

4.4. Transmission and epidemiological factors

Infection occurs mainly by the ingestion of oocysts previously eliminated with the faeces of infected neonates or asymptomatic adult carriers [100]. Daily excretion of oocysts by infected lambs can exceed 2×10^9 and more than 10^{10} oocysts are shed during the patent period [85, 89]. Moreover, adult sheep can act as asymptomatic carriers shedding small numbers of oocysts to the environment which was shown to increase in number in the perinatal period and contribute to maintaining the infection between lambing periods. Between 75 and 100% of lambs born in this environment become infected in the first few weeks of life [105]. In a recent study, the viability of oocysts shed was shown to be greater between days 5 and 11 p.i. than between days 11 and 15 p.i. [106]. Other possible sources of infection, such as farm rodents, should also be considered [107].

The influence of the dose of the infectious agent on the course of the disease has been studied by several authors. There do not seem to be any differences between natural or experimental infections with respect to clinical symptoms, oocyst shedding or serum antibody responses [108]. However, the prepatent period is a few days longer in experimental infections with low infectious doses [84]. In gnotobiotic lambs the minimum infectious dose can even correspond to a single oocyst and the average infectious dose is around five oocysts [84].

At present, no intraspecific differences in pathogenicity have been found between isolates obtained from domestic ruminants. However, in the laboratory of one of the authors (Ortega-Mora, personal communication), two different isolates obtained from diarrhoeic ruminants

showed significant differences in the number of oocysts shed and the severity of the diarrhoea after experimental infection in lambs. However, further research is necessary to confirm this finding.

In both natural and experimental infections the clinical process that accompanies infection has been reported to be more common in lambs and goat kids under 30 days old [76, 82, 85]. Extension of the prepatent period and reduction of oocyst shedding occur as the age of the lambs at infection increases. In a recent study, diarrhoea was only present in lambs infected at 6 days old and in a small percentage of lambs infected at 28 days old, but not in lambs infected at 56 days old. The size of the specific serum (IgG and IgA) and faecal (IgA) humoral response was similar and apparently independent of the age, suggesting the participation of mechanisms other than the humoral response in the development of protection [85]. However, a greater intestinal mucus production and increased glucoprotein concentration in the mucus was observed in animals infected at 28 or 56 days old. The kinetics of the IgA in the intestinal mucus response varied with the age of the animal at infection, the most rapid responses and highest titres occurred in animals which were older when infected (Quintanilla-Gozalo A, Wright S, Pereira-Bueno J, Rojo-Vazquez FA, Ortega-Mora LM. Changes in the intestinal mucus during infection by *Cryptosporidium parvum* in lambs. In: Proc 1995 COST 820 Annual Workshop. Prague, 1995, p. 70).

In natural infections, the majority of the infected lambs and kids were also less than 2 weeks old and the proportion of infected animals underwent a pronounced decrease in animals more than 3 weeks old [76, 80, 109].

4.5. Economic losses

The economic losses associated with this disease are not only due to the resulting mortality, but also to the retarded growth of the animals, the cost of drugs, veterinary assistance and the increased labour involved. In the absence of other enteropathogens, mortality is higher in lambs than in calves [88] and morbidity can reach 100% [46, 75]. The anorexia is very pronounced at the start of the process in both lambs [74, 82, 85] and goat kids [83, 90]. In lambs naturally infected with *C. parvum*, there was a mean weight difference of 2 kg at 4 weeks old compared with non-infected control lambs of this age (Ortega-Mora, personal communication).

5. Cryptosporidiosis in chickens and other birds

5.1. Historical background

First reports of a *Cryptosporidium* sp. in avian species were from Tyzzer [110]. He found a parasite in the caecal epithelium of chickens with structural similarities to *C. parvum*, found in mice. At present, only two *Cryptosporidium* spp. are recognised as valid in avian hosts: (i) *Cryptosporidium meleagridis*, first reported in turkeys [1]; and (ii) *Cryptosporidium baileyi*, isolated from broiler chickens [111]. *Cryptosporidium meleagridis* and *C. baileyi* can be distinguished by morphological differences between their oocysts (length and width) [112]. There is possibly a third species from bobwhite quail [113, 114] and fourth species from ostrich [115]. Infection by *Cryptosporidium* spp. has been detected in over 30 species of birds including domesticated chickens, turkeys, ducks, geese, quails, pheasants, peacocks, and a wide variety of wild and captive birds [116, 117].

5.2. Clinical and pathological features

Cryptosporidium meleagridis may infect the intestinal tract, bursa of Fabricius (BF) and cloaca of turkeys [118] and chickens [112], but infection was only associated with illness, including diarrhoea and moderate mortality in turkeys [119].

Cryptosporidium baileyi may infect the respiratory tract (larynx, trachea, primary and secondary bronchi, air sacs), BF and cloaca of chickens [120, 121], turkeys [122] and ducks [123]. It is the most prevalent *Cryptosporidium* sp. in poultry and the most commonly associated with disease in chickens. Following experimental in-

oculation, *C. baileyi* can develop infections in many anatomic sites of its avian hosts, and it seems that the route of oocyst administration strongly determines the site where the infection finally establishes (reviewed in [124]).

A *Cryptosporidium* sp. was responsible for intestinal disease, including diarrhoea, in quails [113, 114]. Sometimes, the disease can be severe and mortality can reach 90% in young quails [125].

Naturally occurring cryptosporidiosis in chickens usually manifests as respiratory disease, and only occasionally as intestinal or renal disease [124]. Symptoms (depression, anorexia, emaciation, coughing, sneezing, dyspnoea), pathological consequences (excessive mucoid exudate, local inflammation, airsacculitis, deciliation, epithelial hypertrophy and hyperplasia), as well as increased mortality are most often associated with respiratory cryptosporidiosis [15, 126, 127]. The primary respiratory disease potential of *C. baileyi* was proven in the absence of other detected pathogens, and the negative effect of cryptosporidiosis on growth performance and carcass pigment was clearly shown [121]. Severe air sac disease [15, 121] which resulted in heavy carcass condemnation at processing, places *C. baileyi* among the agents to be considered in the respiratory disease complex [121].

The infection of immunocompetent birds is self-limiting. Two-week-old broiler chicks inoculated orally with *C. baileyi* oocysts and capable of clearing the endogenous stages from the cloaca and/or BF, were resistant to subsequent oral challenge [128]. Furthermore, there is an age-related resistance to clinical disease. Older chickens are less susceptible to *C. baileyi* infection, exhibiting a longer prepatent and a shorter patent period [129–131]. Unlike younger (7-day-old) chicks [121], food conversion and body weight gain were not influenced by *C. baileyi* infection in 26-day-old broilers [132]. In practice, infections with *Cryptosporidium* are known to occur only in chickens less than 11 weeks of age [133], and not in adult birds [127, 134].

5.3. Interaction with other pathogens and prevalence

When appropriate diagnostic tools are used, *Cryptosporidium* spp. appear to be present wherever avian hosts are raised commercially [135]. *Cryptosporidium baileyi* has been reported in domestic chickens from Asia, Australia, Europe, North America [136] and North Africa [137]. The prevalence of *Cryptosporidium* spp. in chickens and other Galliformes has been studied both in outbreaks of respiratory and/or intestinal disease and in randomly selected flocks (Table 2). Histological observations demonstrated a similar incidence of *Cryptosporidium* infection in broilers and commercial egg-layer-type pullets [134]. However, limited epizootiological data suggest that the infection in the latter [126] is underreported compared with that in broilers [136]. Goodwin et al. [148] found that 41% (23/56) of the northern Georgia broiler flocks had *C. baileyi* tracheitis. Parasitism rates among *C. baileyi*-infected flocks ranged from a low of 10% to a high of 60%. *Cryptosporidium baileyi* tracheitis was very highly correlated to severity of tracheitis, negatively correlated with average body weight, and correlated with airsacculitis and condemnations. This study indicated that *C. baileyi* infection rates in Georgia were much higher than previously suspected.

Avian cryptosporidiosis has been reported in concurrence with immunosupressive viruses like reovirus [113], Marek's disease virus (MDV) [149–151], infectious bursal disease virus [139, 150], and chicken anemia virus [152]. In chickens, the synergistic effect of *C. baileyi* and these viruses was experimentally confirmed (reovirus [153], MDV [Abbassi H, Coudert F, Cherel Y, Naciri M. Effect of oncogenic Marek's disease virus and vaccinal virus on the pathology induced by *Cryptosporidium baileyi*. In: Proc VIIth International Coccidiosis Conference and European Union COST 820 Workshop. Oxford, 1997, pp. 108–109], infectious bursal disease virus [154], chicken anaemia virus [155]). Marek's disease virus may induce the establishment of the parasite in inhabitual sites ([156], Abbassi H, Coudert F, Cherel Y, Brugere-Picoux J,

Table 2
Prevalence of *Cryptosporidium* infections in Galliformes

Host species (type)	Age	Country (period of study)	Diagnostic technique*	Prevalence % (number infected/ number sampled)		Reference
				Flock	Individual	
Chicken		Scotland	3		88 (22/25)	[138]
Chicken (broiler)	4–7 weeks	Scotland	1		19 (26/139)	[139]
Chicken (layer)	31–86 days	Japan (1976–1983)	1, 4		37 (25/68)	[126]
Chicken (broiler)	40–48 days	Japan (1975–1981)	1, 4		33 (4/12)	[126]
Chicken (broiler)	21–42 days	USA, Deleware	1	25 (2/8)		[140]
Chicken (broiler)	49 days	USA (1985)	3	38 (7/18)		[141]
Chicken (broiler)	63 days	USA (1985)	3	50 (9/18)		[141]
Chicken (broiler)	49 days	USA (1987)	3	22 (100/454)		[141]
Chicken (broiler)	5–50 days	Greece	1, 4		24 (17/70)	[142]
Chicken	14–51 days	USA, Georgia (1986)	1		6 (105/1065)	[134]
Chicken (broiler)	25–48 days	USA, North Carolina	2		27 (9/33)	[143]
Chicken (broiler breeder)	27–59 days	USA, North Carolina	2		10 (3/30)	[143]
Chicken (layer)	42 days	USA, North Carolina	2		6 (1/17)	[143]
Turkey	17–24 days	USA, Iowa	2		45 (54/120)	[144]
Chicken, turkey		USA, Georgia (1974–1984)	1		1 (63/6050)	[145]
Chicken, turkey		USA, Georgia (1985–1988)	1		6 (157/2622)	[145]
Chicken	4 weeks	Korea	2		15 (75/500)	[146]
Chicken (broiler)		USA, Georgia (1987–1992)	1		1 (84/7007)	[147]
Chicken (broiler)	49–52 days	USA, Georgia	1	41 (23/56)		[148]
Chicken (broiler)	26–50 days	Marocco	1, 4	37 (14/38)	24 (54/225)	[137]

* 1 = Histology, 2 = faecal examination, 3 = serology, 4 = other.

Naciri M. Experimental reproduction of renal cryptosporidiosis (*C. baileyi*) in SPF chickens after oral inoculation of parasite. In: AAAP Meeting. Baltimore, 1998, pp. 210–211). The severity of respiratory disease induced by *C. baileyi* can be enhanced by other respiratory pathogens, such as infectious bronchitis virus and *E. coli* (157, Hoerr FJ, Blagburn BL, Lindsay DS, Giambrone JJ. Interactions of *Cryptosporidium* with other infectious pathogens in chickens. In: Proc of the 124th Annual Meeting of the Am Vet Med Assoc, Chicago, USA, 1987, p. 134). Moreover, *C. baileyi* infection affects the development of humoral immunity to heterologeous antigens [121, 158, 159], and can diminish cell-mediated immunity, as shown by decreased delayed hypersensitivity indices of infected chickens [121].

5.4. Transmission and epidemiological factors

Chickens can become infected by ingestion or inhalation and/or aspiration of oocysts present in the environment (litter, faeces, water, breeding materials, dust, etc.). As few as 100 oocysts can result in intestinal or respiratory infections [135]. Since *C. baileyi* can infect a variety of wild birds they must be considered as possible sources of infection. Although *C. baileyi* does not infect rodents (mice and rats) or insects, they may serve as mechanical carriers of oocysts [135].

Hygienic conditions and management strongly influences the incidence and persistance of avian cryptosporidiosis. The disease was shown to be more prevalent in flocks with defective hygiene [137]. Goodwin et al. [145], suggested that the increase of intestinal and respiratory cryptosporidiosis infection from 1% (63/6050)

during 1974–1984 to 6% (157/2622) during 1984–1988, could be explained in part by the relatively widespread practice of cleaning out poultry houses less frequently. The increased use of 'built-up litters' could increase exposure to and infection of chicks by *Cryptosporidium* [145]. In quails, rigorous clean up and disinfection with hypochlorite acid was effective in preventing continued infection, morbidity and mortality attributed to *Cryptosporidium* sp. [125].

Furthermore, outbreaks of cryptosporidiosis seem to be related to climatologic parameters. It was found that the percentage of cryptosporidiosis cases in winter was significantly lower than in other seasons [134]. Differences in the incidence between geographic locations were related to the number of days of frost [147].

5.5. Economic losses

For a long time, avian cryptosporidiosis was considered to be an opportunistic disease, but it is now recognised as a first order disease [120, 121, 127]. The economic losses associated with this disease are due to poor flock performance (growth retardation and increased consumption index) and/or mortality [121, 140, 141, 148]. In addition, cost of therapy, although most drugs are inefficient [122, 125, 142, 160], and carcass condemnation at processing due to air sacculitis [15, 121], should also be taken into account. When *C. baileyi* occurs concurrently with immunosuppressive or other respiratory pathogenic agents, the consequences are more serious, and the losses more important [15, 127, 157]. Vaccination with the attenuated serotype 1 MDV can enhance the severity of subsequent cryptosporidial respiratory disease, resulting in unexpected losses (Abbassi H, Couder F, Cherel Y, Brugere-Picoux J, Naciri M. Effect of *Cryptosporidium baileyi* on the development of vaccinal immunity to Marek's disease in SPF chickens. In: 5th Avian Immunology Research Group Meeting. Turku, 1998, p. 7.4). Thus, the role this parasite plays in the pathogenesis of respiratory disease and the related production losses could be unexpect-

edly large. Therefore, avian cryptosporidiosis should not be neglected or overlooked during diagnosis.

6. *Cryptosporidium* infection in pigs and rabbits

Cryptosporidial infection was first reported in pigs by Bergeland [161] and Kennedy et al. [162]. Thereafter, naturally occurring cryptosporidiosis in pigs has been described worldwide [163]. Experimental infections of piglets resulted only in diarrhoeic problems when the animals were inoculated before 2 weeks of age [164–166]. In older piglets, experimental *Cryptosporidium* infection did not cause clinical manifestations [167–169]. The prevalence of cryptosporidiosis was reported to be very low in nursing piglets [168, 170, 171]. Several studies demonstrated that naturally occurring cryptosporidiosis is delayed until after weaning [146, 169, 171–173]. The infections are mostly asymptomatic and usually of low intensity, and no statistical association between infection and clinical symptoms has been found [171, 174]. Previous studies have confirmed that diarrhoea in suckling and weaned piglets is usually a multifactorial problem, where mixed infections between *C. parvum* and *E. coli* or rotavirus are frequent. It has been suggested that *Cryptosporidium* is not an important primary agent of diarrhoea in piglets but it may be a copathogen in the multifactorial aetiology [163]. Recently, genetic analysis of the *Cryptosporidium* isolates from pig herds revealed that the animals harboured two distinct genotypes: a porcine genotype and a bovine genotype with distinct virulence in nude mice [175]. The zoonotic potential of the porcine genotype is still uncertain and requires further study.

In commercial rabbits, digestive disorders are the predominant cause of mortality. Mainly weaned rabbits of 4 to 8 weeks of age are affected, although another peak may occur in suckling rabbits of 8 to 12 days old [176]. Experimental *Cryptosporidium* infection of rabbits resulted in high mortality and liquid diarrhoea in suckling 3-day-old rabbits, but no mortality and very discrete diarrhoea in weaned

animals [177]. The parasite was found in 2–11% of weaned diarrhoeic Belgian rabbits [176]. In general, field outbreaks of cryptosporidiosis in suckling rabbits are rarely detected and in weaned rabbits the parasite causes only subclinical enteritis. When concurrent infections with immunosuppressive agents or respiratory pathogens occur, the impact of *Cryptosporidium* infection can be more significant (Peeters, personal communication).

7. Treatment and control

Aetiological treatment of cryptosporidiosis has only recently been made possible. Drugs demonstrated to be partially effective in the treatment and prophylaxis of cryptosporidiosis in ruminants include halofuginone lactate, paromomycin and decoquinate (Table 3). However, the commercial availability of some of these products is still a problem in many countries. In poultry, so far none of the tested drugs showed a satisfactory anti-cryptosporidial activity, unless at toxic dosage [122, 125, 142, 160]. Recently, it was shown that the oral infection of hens with *C. baileyi* at the beginning of their laying period, resulted in partial protection of their progeny [185].

The importance of colostrum in protecting neonatal ruminants against infection by *C. parvum* is a very valuable point to address. In field conditions, passively acquired antibodies did not protect calves [44, 186] and lambs against naturally acquired infection [108]. However, both calves [187] and lambs [188] fed with colostrum from immunised mothers with high titres of specific antibodies were partially protected against infection. Immunoprophylaxis of cryptosporidiosis is more thoroughly discussed in another contribution of this Special Issue of the *International Journal for Parasitology* [189].

From a perspective of disease control, preventive hygiene measures are the most important tools in the struggle against cryptosporidiosis in farm animals, the objective being to destroy external forms of the parasite and to prevent their transmission among animals and from the environment to the host [46]. In ruminant husbandry, the destruction of oocysts in the pens and buildings used for parturition by applying moist heat and/or chemical disinfectants, the use of abundant clean straw beds, avoidance of high stocking rates in the parturition area and the separation of healthy and ill animals during outbreaks of diarrhoea, in addition to the administration of appropriated supplies of colostrum to neonates, all help to prevent outbreaks of cryptosporidiosis and to minimise mortality and morbidity in infected herds.

Table 3
Efficacy of different drugs against cryptosporidiosis in ruminants

| Drug | Animal species | Dose* | Administration period | Best results | | Reference |
				Oocyst shedding	Episode of diarrhoea[1] or stool consistence[2]	
Halofuginone lactate	lamb	500 μg	1–5 days	prevented	prevented[1]	[178]
Halofuginone lactate	calf	30–500 μg	3–14 days	prevented	prevented[1]	[41, 179, 180]
Paromomycin	calf	25–100 mg	11 days	prevented	reduced[1], improved[2]	[181]
Paromomycin	goat kid	100 mg	12 days	prevented	improved[2]	[182]
Decoquinate	calf	2.5–10 mg	8 weeks	decreased	improved[2]	[183]
Decoquinate	goat kid	2.5 mg	21 days	decreased	prevented[1]	[184]

* Per kg body weight.

8. Conclusion

Cryptosporidiosis is mainly a problem in neonatal ruminants. *Cryptosporidium parvum* is the most commonly found enteropathogen during the first weeks of the life of calves, lambs and goat kids and is considered to be an important agent in the aetiology of the neonatal diarrhoea syndrome. The parasite frequently acts alone, but the losses are more pronounced when concurrent enteropathogens are present. Economic losses associated with cryptosporidiosis are retarded growth and mortality, and a number of hard to estimate costs resulting from interventions necessitated by diarrhoeic problems. Especially in small ruminants, the direct losses due to mortality caused by cryptosporidiosis alone was reported to be high. Because of the limited availability of effective drugs, hygienic measures and good management are the most valid weapons in the struggle against this disease. Avian cryptosporidiosis is an emerging health problem. In chickens and other Galliformes, cryptosporidiosis is mostly manifest as respiratory disease, although in turkeys and quails enteric forms of the disease were also reported to be responsible for morbidity and mortality. It was alarming to ascertain the unexpectedly high prevalence of *C. baileyi* in chicken flocks and the enhanced pathogenicity when immunosuppresive or other respiratory pathogens are present, or when birds are subjected to vaccination against other pathogens. In pigs and rabbits, *Cryptosporidium* spp. were reported to cause only clinical disease when administered experimentally to nursing neonates, but in these farm animals naturally occurring cryptosporidiosis is mostly asymptomatic.

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