



# Is *Giardia* a significant pathogen in production animals?

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## ABSTRACT

Although *Giardia duodenalis* is recognised worldwide as the most important parasitic cause of gastrointestinal disorder in human patients, the relevance of infection in production animals is prone to debate. Since the 1980s, clinical disease has been associated with giardiasis in production animals, both in natural conditions and in experimental studies. However, most *Giardia* research is focussed on the relevance of production animals as a reservoir for zoonotic transmission. In this study, the current knowledge on clinical relevance of giardiasis in production animals is reviewed, along with the diagnosis, treatment and control of infection. Furthermore, future research objectives are discussed.

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## 1. Introduction

Although the first detailed description of the parasite *Giardia* dates from 1859, the clinical relevance of giardiasis was not acknowledged until late in the 20th century. In human patients, *Giardia* is nowadays recognised as the most common parasitological cause of diarrhea, with 280 million infections per year. Giardiasis is also a frequently diagnosed waterborne infection and a major concern to drinking water authorities. Because of the impact on socio-economic development, especially in developing countries, since 2004 *Giardia* is included in the 'Neglected Disease Initiative' of the World Health Organization (Lane and Lloyd, 2002; Savioli et al., 2006).

In veterinary medicine, the increased interest in *Giardia* since the 1990s was mainly driven by public health concerns, and to a lesser extent by a veterinary perspective. Research is therefore focussed on parasite prevalence and on the molecular characterisation of isolates from different hosts to elucidate the zoonotic hazard, as production animals have long been considered as a potential zoonotic reservoir for human *Giardia* infections. More recently, molecular epidemiological studies and subgenotype analysis have provided a more differentiated and detailed insight into the zoonotic potential of *Giardia* isolates from production animals. Due to this focus on transmission patterns, the clinical relevance of a *Giardia* infection in animals was studied only to a limited extent. In companion animals, veterinarians do consider *Giardia* as a potential cause of diarrhea (Zajac, 1992). Despite the higher prevalence compared to

companion animals, the relevance of infection in production animals is however not widely recognised, mostly due to the vagueness of the symptoms associated with infection. The aim of this review is therefore to present an overview of the current knowledge on *Giardia* in production animals, with emphasis on prevalence, clinical outcome, diagnosis, treatment and control.

## 2. Parasite background

Since 1859, over 50 different *Giardia* species have been described, primarily based on the host specificity. This host-specific taxonomy was later replaced by a morphological taxonomy based on the morphological characteristics such as shape and length of the trophozoite and median bodies (Filice, 1952). Three distinct groups or *Giardia* species were described, including *G. duodenalis* with a wide mammalian host range. Molecular characterisation has since revealed that *G. duodenalis* is in fact a species complex, comprising seven assemblages (A–G), some of which have distinct host preferences or a limited host range (Thompson and Monis, 2004). In addition to the assemblages A and B which are also prevalent in human patients, several host-specific assemblages have been identified in animals, of which assemblage E or the hoofed livestock assemblage is identified in production animals. In addition to the livestock-specific assemblage E, the zoonotic assemblage A and occasionally assemblage B have been described in production animals.

To understand the epidemiology and pathogenesis of *Giardia*, the life cycle should be considered. There are two stages: an infectious cyst which is resistant in the environment, and the trophozoite

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which colonizes the intestinal epithelium of the host and causes disease. After oral ingestion, cysts release the trophozoites in the upper part of the small intestine. For the colonisation of the small intestine, attachment to epithelial cells is essential, and the trophozoites have a ventral adhesive disk that is used to attach to the intestinal mucosa. The trophozoites multiply by binary fission in the lumen of the small intestine, although sexual reproduction has been suggested (Meloni et al., 1989). Finally, exposure to biliary salts leads to encystation of trophozoites. Cysts are passed in the faeces and are immediately infectious upon excretion allowing completion of the life cycle within 72 h (Thompson et al., 1993). Early experimental studies suggested that the prepatent period is somewhat longer in ruminants and varies from 6 to 21 days (Taminelli et al., 1989; Koudela and Vitovec, 1998), but this was probably due to the method used for diagnosis as other studies indicated a prepatent period of around 3–10 days (Xiao and Herd, 1994; Geurden et al., 2006a).

### 3. Prevalence

*Giardia* has been reported in production animals worldwide, although prevalence data are mainly available for cattle, and to a lesser extent for other ruminants and pigs. In Tables 1 and 2, an overview of the most recent or large-scale prevalence studies in different production animals around the world is provided. In these studies, both the animal and the farm prevalence vary considerably. In cattle for example, the animal prevalence ranges from 9 to 73% and the farm prevalence ranges from 45 to 100%. In other production animals, a similar variability is observed. Although management, geographical and climatological parameters partially account for this variation, differences in study design also need to be considered, such as the number of animals or farms included in the study and the assay used for diagnosis. Since there is no gold standard reference test for the diagnosis of *Giardia* in production animals, the use of diagnostic techniques with different sensitivity and specificity, might thwart comparison between prevalence

studies. In calves, the prevalence estimate using three different diagnostic assays resulted in a different estimate for each assay (Geurden et al., 2004). Similarly, PCR provided a higher prevalence estimate in post-weaned calves compared to immunofluorescence assay (Trout et al., 2005).

In addition to study design, the age of the animals needs to be taken into account. In calves, both longitudinal and cross-sectional prevalence studies indicate a peak prevalence in animals aged between 1 and 6 months, and a decrease in prevalence from the age of 6 months onwards (Xiao et al., 1994; Nydam et al., 2001; Ralston et al., 2003; Becher et al., 2004). In contrast to *Cryptosporidium* (Ralston et al., 2003), *Giardia* seems to be equally prevalent in dairy and beef calves. Although there are few data on age-related prevalence in other production animals, most authors seem to consider a similar infection pattern.

Despite the high variability in reported prevalences, some conclusions can be drawn from these studies. In cattle, the farm prevalence varies between 45% and 100%. The cumulative incidence on a farm where *Giardia* has been diagnosed, is 100% in cattle and goats (Xiao, 1994; O'Handley et al., 1999; Castro-Hermida et al., 2005) and close to 100% in sheep (Xiao and Herd, 1994), implying that every animal on that farm will get infected. Given the limits of a point prevalence study, including the high variability of animal prevalence estimates throughout the year, the farm prevalence is probably more informative than the animal prevalence to study the occurrence of infection in a large population. Given the high farm prevalence reported in these studies, a high proportion of production animals are at risk of infection. In pigs, there are no data on cumulative incidence, but the high farm prevalences also suggest a widespread occurrence of infection.

### 4. Epidemiology

Hosts are infected by oral intake of infectious cysts, and as soon as 3 days after infection cysts are recovered from the faeces. The

**Table 1**

The animal prevalence ( $P_A$ ) and farm prevalence ( $P_F$ ) of *Giardia* in cattle in different countries. The age of the animals, the number of animals ( $\#_A$ ) and farms ( $\#_F$ ) are presented along with the diagnostic assay (Diag) used in the study (IFA, Immunofluorescence microscopy; PCR, polymerase chain reaction; or ME, microscopical examination). -, not known.

Country	Diag	$\#_A$	$\#_F$	age	$P_A$	$P_F$	Reference
<i>Dairy &lt; 6 m</i>							
Belgium	IFA	499	100	<2.5 m	22	48	Geurden et al. (2008b)
Canada	IFA	386	20	<6 m	73	100	Olson et al. (1997)
Canada	ME	–	505	<6 m	–	45	Ruest et al. (1998)
Denmark	IFA	377	50	<1 m	24	82	Maddox-Hyttel et al. (2006)
New Zealand	IFA	715	12	<2 m	41	100	Hunt et al. (2000)
New Zealand	IFA	–	10	<2 m	–	31	Winkworth et al. (2008)
Norway	IFA	1386	136	<6 m	49	93	Hamnes et al. (2006)
Spain	IFA	734	60	<6 m	29–57	67	Castro-Hermida et al. (2006a)
USA	ME	2943	109	<6 m	20	70	Wade et al. (2000b)
USA	PCR	407	14	<2 m	40	100	Trout et al. (2004)
Vietnam	IFA	68	8	<3 m	50	88	Geurden et al. (2008c)
<i>Dairy &gt; 6 m</i>							
Denmark	IFA	518	50	1–12 m	43	100	Maddox-Hyttel et al. (2006)
Denmark	IFA	255	50	>12 m	40	60	Maddox-Hyttel et al. (2006)
Spain	IFA	734	60	>6 m	25–40	67	Castro-Hermida et al. (2006a)
Spain	IFA	379	60	>36 m	27	97	Castro-Hermida et al. (2007)
Spain	ME	199	30	<24 m	26	53	Quilez et al. (1996)
USA	PCR	456	14	3–11 m	52	100	Trout et al. (2005)
USA	PCR	571	14	12–24 m	36	100	Trout et al. (2006)
USA	PCR	541	14	>24 m	27	100	Trout et al. (2006)
<i>Beef</i>							
Belgium	IFA	333	50	<2.5 m	45	64	Geurden et al. (2008b)
Canada	IFA	193	10	<2.5 m	36	100	McAllister et al. (2005)
Canada	IFA	495	9	<3 m	34	100	Appelbee et al. (2003)
Canada	IFA	605	100	<6 m	23	48	Gow and Waldner (2006)
Canada	IFA	605	100	>24 m	17	69	Gow and Waldner (2006)
Canada	IFA	669	39	>24 m	9	64	McAllister et al. (2005)

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