

Preliminary Observations on Naturally Acquired Hypobiotic *Cooperia oncophora* Infections in Cattle

Many nematode species, parasitic as well as free-living, have the ability to enter a hypobiotic state of development in the course of their life cycle. Among the parasitic nematodes of cattle, perhaps *Ostertagia ostertagi* is the most renowned in that respect, as the resumed development of hypobiotic larvae is thought to contribute to the Type II ostertagiasis seen in early spring (Armour & Ogbourne 1982). In contrast, only few reports have been made on the occurrence of naturally acquired hypobiotic infections with *Cooperia oncophora* (Brunsdon 1972). The present experiment was originally set up to assess the occurrence in Denmark of hypobiotic *O.ostertagi* larval infections in young cattle pastured on *O.ostertagi* contaminated fields during autumn. However, in the course of these studies *C.oncophora* infection became evident, leading to the observations reported here.

Two groups of parasite-free Jersey bullock calves were grazed for 6 weeks during October and November, 1989. One group (A), comprising 21 calves, was grazed on an *O.ostertagi* and *C.oncophora* contaminated pasture, while the other group (B), comprising 9 animals, grazed a non-contaminated pasture. In the course of the winter housing period from November until April, 12 calves of group A were slaughtered (2 at a time) at monthly intervals, and the worm burdens were assessed according to the method described by Grønvold *et al.* (1989). However, determin-

ations of *C.oncophora* worm burdens were not included in the studies until the third time of slaughtering (February 2). Therefore, only the recoveries from 8 animals could be presented in Table 1.

The following parasitological and immunological parameters were monitored at 2 week intervals: faecal strongylid egg counts (Henriksen & Aagaard 1976), faecal cultures with subsequent larval differentiations (Henriksen 1972) and specific antibody responses of the IgG₁ isotype. The latter were determined in an ELISA according to the method described by Canals & Gasbarre (1990). The employed parasite antigens were crude whole worm extracts derived from adult worms of *C.oncophora*.

Table 1. *Cooperia oncophora* worm burdens recovered from 8 of the 12 group A calves at the sequential killings.

Date Date	C.onc. adults	C.onc. larvae	*Total
Feb. 2	66.7%	33.3%	900
	50.0%	50.0%	800
Mar. 1	88.9%	11.1%	1800
	69.8%	30.2%	430
Mar. 28	26.1%	73.9%	2300
	0.0%	100.0%	500
Apr. 26	75.0%	25.0%	400
	-	-	0

C.onc. = *C.oncophora*

*Total = The total *C.oncophora* worm burden of the individual animals.

Group A calves acquired a moderate initial infection resulting in egg excretions declining from around 200 EPG in December to negligible values in April. The results of the larval differentiations indicated that *C. oncophora* and *O. ostertagi* contributed approximately equally to the EPG values. However, these differentiations were not initiated until January 1990, and the preceding time cannot be accounted for. The anti-*C. oncophora* serum IgG₁ antibody response of group A rose sig-

nificantly above the control group (B) levels soon after turnout on pasture in October 1989 ($p < 0.05$; Bonferroni's t-test). Through the winter housing period, the IgG₁ levels consistently increased until the end of the experiment in late April (Fig. 1). At the sequential killings of group A calves moderate numbers of both adults and larvae of *C. oncophora* were recovered (Table 1).

Although initially intended to be a study of responses to only *O. ostertagi* infections, there

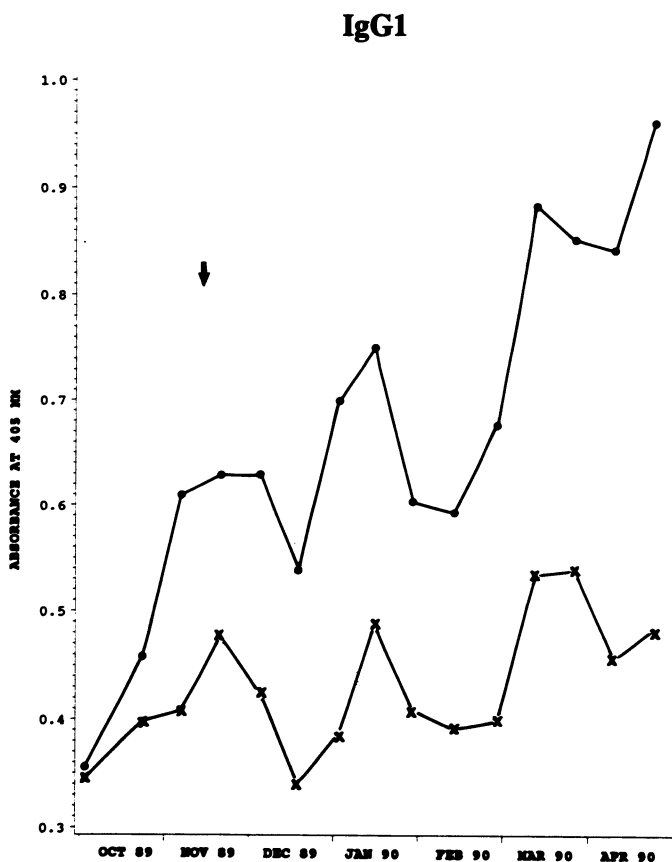


Figure 1. Serum IgG₁ antibody response against *Cooperia oncophora* adult worm-antigen; expressed as group mean values of the absorbance measured at 405 nm (for the 2 × 9 continuously kept calves of grp. A and B). Group A (●) exposed to parasites; group B (×) uninfected controls. ↓ indicates the time of housing.

was an admixture of *C. oncophora* to the previously *O. ostertagi* mono-infected pastures, thus allowing for a preliminary study of the occurrence of hypobiotic *C. oncophora* infections in autumn-pastured cattle in Denmark. The presence of hypobiotic *C. oncophora* infections was indicated by the larval worm burdens recovered from the calves (see Table 1). A continued presence of adult worms, presumably as a result of the resumption of larval development, was further suggested by a persistent (although moderate) *C. oncophora* egg output throughout the housing period. Further indications were inferred by the continuous rise in anti-*C. oncophora* serum IgG₁ antibody responses. When testing the same sera against *O. ostertagi* antigens, a significant increase in anti-*O. ostertagi* IgG₁ antibody levels was not apparent until March/April (unpublished data).

While the occurrence in Denmark of hypobiotic *O. ostertagi* infections leading to Type II ostertagiasis has previously been reported (Hansen 1979), similar descriptions cannot be found for *C. oncophora* infections. However, the propensity of *C. oncophora* to enter hypobiosis has been described elsewhere for naturally acquired (Brunsdon 1972) as well as experimental infections (Borgsteede & Hendriks 1978). It has been concluded that, as for *O. ostertagi*, the environmental factors are of major importance for the induction of arrested development in primary infections (Michel *et al.* 1975).

The occurrence of arrested *Cooperia* may nonetheless be somewhat atypical. Immunity towards *Cooperia* spp. will usually be built up within 2 months, i.e. fairly early in the grazing season, thus preventing large numbers of larvae (including those that are inhibition-prone) to become established later on (Coop *et al.* 1979). However, in the present case susceptible animals were set out on contaminat-

ed pastures, where they acquired substantial infections, when compared to the 'usual' late-season infection.

More experiments should be carried out, employing predominantly *C. oncophora* contaminated pastures, in order to properly assess the proclivity of *C. oncophora* to enter hypobiosis in the course of naturally acquired infections. Additionally, the possible impact of such infections on the course of *O. ostertagi* hypobiotic infections and development of Type II ostertagiasis should be investigated.

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