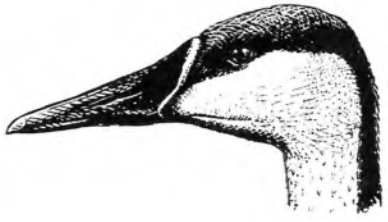
The effects of treatment with Mebendazole on gizzard worm infections in captive Swan Geese *Anser cygnoides*



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Eggs of the gizzard worm Amidostomum anseris *in faeces of a flock of captive Swan Geese in the Tower Pen at Slimbridge were monitored over three months, which included a period of routine worming with the anthelmintic drug Mebendazole. Parasite eggs disappeared from the faeces of all the treated birds which were examined, but reinfection occurred rapidly, eggs reappearing in the faeces between 15 and 28 days of the cessation of worming. It is estimated that prior to worming, the flock was contaminating the Tower Pen with slightly more than 200,000 parasite eggs per day.*

There are several species of nematode parasites which have been recorded as burrowing under the horny lining of the gizzard of wildfowl. One of the commonest species worldwide is *Amidostomum anseris*, which is characteristically a parasite of Anserini although it has also been found in a wide range of other hosts (McDonald 1969). It is frequently found as a parasite of captive geese at Slimbridge and other Trust collections (Avery 1966, Hillgarth *et al.* 1983), and in large numbers may be responsible for ill-health and mortality, especially of young birds (the extensive literature on pathology of the species is listed by McDonald 1969). The life cycle of this parasite is direct, infection usually occurring as a result of the grazing bird swallowing larvae which have hatched from the parasite eggs produced with the faeces (list of references in McDonald 1969).

We monitored the effects of a routine worming of birds with the benzimidazole drug Mebendazole (Mebenvet 5%; Jansen Pharmaceuticals Ltd). For reasons of time and practicability, attention was focused on a flock of Swan Geese *Anser cygnoides* in the Tower Pen at Slimbridge. This species seems to be particularly susceptible to *Amidostomum* infection (for example, all six birds examined *post mortem* at Slimbridge in 1988 harboured the parasite; see also Hillgarth *et al.* 1983). Levels of infection in the Tower Pen Swan Geese were assessed by counting eggs of the parasite in faeces, and followed over a three-month period from mid-November 1988 to the end of February 1989: the birds were treated with Mebendazole in mid-December. Because there have been few systematic and follow-up studies on the effects of worming in wildfowl, the results are presented here.

Materials and methods

The Swan Geese in the Tower Pen were wormed routinely in June and again in December 1988. The 20 birds which were in the pen in November 1988 were marked with colour-coded rings. There were 12 adult birds, three juveniles from Martin Mere and five juveniles from Slimbridge. The latter were wormed immediately prior to their release into the pen on 14 November. Faecal samples were collected from individual birds and stored in a refrigerator overnight. Rapid diagnosis of infection was carried out using a coverslip flotation method, in which a small glass coverslip was placed on the surface of a suspension of faeces in saturated salt solution and left for half an hour. Parasite eggs floating to the surface of the denser salt solution adhere to the coverslip, and can be seen on subsequent microscopic examination. This method is qualitative - it determines whether a bird is infected, but does not give any indication of the number of parasite eggs in the faecal sample. Egg numbers were determined for the more heavily infected samples by the McMaster technique, which involves suspending a weighed sample of faeces in saturated salt solution, pipetting a known volume into the space between two glass microscope slides, and measuring the number of eggs which float to the

under-surface of the upper slide, on which a grid has been etched to determine areas (Urquhart *et al.* 1987). Results are expressed as numbers of eggs per gram of faeces (e.p.g). The figure for e.p.g can be roughly equated with the severity of the infection (i.e. the number of worms present in the bird), but studies have shown that for *Amidostomum* species there can be considerable variation in the egg count from day to day and even at different times of day (Herman & Wehr 1954, confirmed by unpublished data from the present study).

Faecal samples were collected prior to worming and after worming, until the end of February. Worming was carried out by giving Mebendazole with feed for 13 days, from 16 December to 28 December. For logistical reasons, faecal examination could not be carried out during the latter part of worming or for 13 days afterwards.

Results

Faecal samples were collected from individual birds by following them until a pellet was produced. Since birds would not produce pellets 'to order', faeces could be collected from individuals only on an irregular basis. All 12 adult and eight juvenile Swan Geese in the Tower Pen showed a positive egg count at least once during the pre-worming period from 14 November to 15 December. Values for the egg counts of the eight most heavily infected adults are shown in Table 1. Bird number 1455 showed the highest count; it was sampled on 14 separate days, the count ranging from 55 to 1367 e.p.g. Bird number 1712 died on 29 November, and at *post mortem* examination was found to contain 44 worms (there was little damage to the gizzard of this bird). The egg counts on 15 and 17 November had been 67 e.p.g, giving a mean egg output of 1.52 eggs per worm, or 304 eggs per worm per day since an adult Swan Goose produces about 200 g of faeces per day.

No eggs were found in faeces of the birds sampled 14 days after treatment had ceased (11 January); there was one positive result the following day (Table 2). Thereafter rapid increases both in the proportion of birds positive and in the egg counts from individuals were seen: 62 days after treatment, all seven of the birds sampled were positive, having counts which ranged from 8 to 279 e.p.g (Table 2).

A similar increase in infection had been observed in the five juvenile birds from Slimbridge released into the pen on 14 November, although egg counts were low and could in most cases be recorded only as positive/negative (Table 3). No positive result was seen before Day 23, but all five birds had been recorded as positive by Day 28. The three birds from Martin Mere had not been wormed prior to release, and so are not included in Table 2: all faecal counts were in fact negative.

Table 1. Parasite egg counts from eight adult captive Swan Geese during the period 14 November to 15 December. + indicates samples which were positive, but in which egg numbers were too small for a count to be possible.

Bird number	Sex	Mean egg count (e.p.g)	Range	Times sampled (*n*)	Times positive
1455	M	349	55 - 1367	14	14
0363	M	178	0 - 753	11	9
1711	F	67	+ - 134	2	1
1712	M	67	-	2	2
0371	M	40	0 - 119	9	5
0271	M	16	-	1	1
0617	F	7	0 - 23	8	4
0313	M	3	+ - 9	3	2

Table 2. Parasite egg counts from eleven adult Swan Geese after worming. + indicates samples which were positive, but in which egg numbers were too small for a count to be possible.

Bird number	Sex	Days after completion of worming 14	15	19	22	29	36	62
0271	M		0					150
0313	M		0	+	69	+		80
0363	M		0					
0370	M			0	+			
0371	M	0	0		+	35	21	90
0609	M	0	0	0	+	+		78
0617	F		0		+		115	
1289	F	0	0	+		33		33
1292	F		0					8
1455	M	0		+	+			279
1711	M	0	+			+		

Table 3. Parasite egg counts from five previously-wormed juvenile female Swan Geese released in the Tower Pen at Slimbridge on 14 November. + indicates samples which were positive, but in which egg numbers were too small for a count to be possible.

Bird number	Days after release 1	3	7	10	23	28	31	34
1898			0		0	+		
1899	0			0	0	+	+	13
1900		0		0	+		74	
1901	0			0	0	+	+	
1902			0	0	+			

Discussion

The striking finding of this study was the rapid reinfection of birds once they had been wormed; this was seen in adults and juveniles in January after worming in December (Table 2) and in juveniles wormed before release in November (Table 3). Experiments in which geese were artificially infected with the parasite showed that the time which elapsed before eggs appeared in the faeces varied from bird to bird, ranging from 14 to 25 days (Cowan 1955). The minimum period between completion of worming and the appearance of eggs in the Swan Geese in the Tower Pen was 15 days, and half of the birds were reinfected by 22 days (Table 2). The potential for infection or reinfection in this environment must therefore have been high. This is perhaps not surprising; the adult worms can live for more than 18 months in the host and the infective larvae can survive both on soil and in water for long periods (many months in some circumstances: Lozovskii 1949, Leiby & Olsen 1965 and other authors listed in McDonald 1969).

A simple calculation shows that contamination of the environment must have been considerable. Adult Swan Geese produce about 200 g, and juveniles about 150 g, of faeces per day. During the period immediately prior to worming, there were eight adult and eight juvenile Swan Geese in the Tower Pen with light infections (say a mean of 25 e.p.g), two adults with medium infections (say 100 e.p.g) and two adults with fairly heavy infections (say 250 e.p.g). This adds up to a total estimated egg output into the pen of 210,000 eggs per day. Contamination of a pen must eventually reach an equilibrium at which additions to the population of parasite larvae hatching from the eggs in faeces are balanced by losses due to egg and larval mortality. The value of this equilibrium is not known, but it must have been many millions of larvae. A high proportion of these would have been available to reinfect birds once treatment with anthelmintics had ceased.

The close correspondence between the minimum possible time for reappearance of eggs in faeces (i.e. the experimentally-determined time from ingestion of larvae to development of adult worms) and their actual reappearance in the Tower Pen birds provides strong circumstantial evidence that the effect of Mebendazole treatment was to remove worms. The alternative is that the drug inhibited their egg production. There is no other reason for supposing that this was the case, but the possibility has not been rigorously eliminated, and it should perhaps be noted that the diluting effects of the plant diet may reduce the efficacy of some anthelmintic drugs in geese.

The implications of these observations for husbandry are clear. Treatment with anthelmintic drugs can have great value in reducing the pathological effects of heavy worm burdens in individual birds, and in reducing the overall levels of environmental contamination. In parasite species in which environmental contamination persists, however (as in *A. anseris*), worming will not prevent rapid reinfection. Anthelmintic treatment must therefore be frequent. A similar conclusion was reached in relation to infections with intestinal worms in calves by Michel (1985). In these cases, treatment must where possible include measures to reduce subsequent reinfection. These can include removal of birds to less heavily contaminated environments, perhaps with rotational grazing; removal of other species of wildfowl which may act as reservoirs for the infection; prevention of excessive faecal contamination; drainage (which reduces survival of the parasite larvae). The extent to which these measures are practicable will depend on individual circumstances.

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