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Anthelminthic treatment negatively affects chick survival in the Eurasian Oystercatcher *Haematopus ostralegus*

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Eurasian Oystercatchers *Haematopus ostralegus* are infested with a wide range of gut parasites, but experimental evidence of their effects on host fitness is scant. We investigated prevalence of parasites, and experimentally tested the effects of gut parasites on chick survival and growth. One hundred and fifty-nine hatchlings from 66 nests were treated with a single dose anthelminthic medicine (0.5 mL Spectril + 0.0025 mL Ivomec) and compared with a sham-treated control group of 163 hatchlings from 66 nests. Unexpectedly, chicks treated with the anthelminthic drug survived less well than control chicks. Fledglings from the treated group were significantly less infected with gut parasites than untreated fledglings, although they were of similar body mass. One possible explanation for these findings is that the treatment interferes with the development of the immune system in the hatchlings. This might have caused mass mortality of treated hatchlings after the drug ceased to work and the treated chicks became susceptible to infections for the first time. Furthermore, all chicks and adults from both saltmarsh and adjacent freshwater habitat appeared free from blood parasites. Thus, in the Eurasian Oystercatcher, we found no support for the hypothesis, based on between-species comparisons, that the presence of blood parasites is related to the saltiness of the environment.

Recent studies show that parasites affect the fitness of birds as hosts (e.g. Hudson 1986, Møller *et al.* 1990, Oppliger *et al.* 1993, Richner *et al.* 1993, McKilligan 1996). Individual differences in fitness have been shown to depend on differences in antiparasite tactics (Clark & Mason 1988, Brown & Brown 1986) or immunity (Saino *et al.* 1995, Møller & Erritzoe 1996).

Several different parasites, notably helminths, infest Eurasian Oystercatchers (Swennen & Duiven 1983, Borgsteede *et al.* 1988, Goater 1989, Goater *et al.* 1995, Galaktionov & Bustnes 1999). Variation in survival and reproduction in Eurasian Oystercatchers (hereon: Oystercatcher) may be related to variation in their resistance to parasites. The birds are infected with gut parasites by eating either free-living stages of parasites or parasitized intermediate hosts (particularly bivalves and Annelids) both in summer and in winter (Borgsteede *et al.* 1988, Goss-Custard *et al.* 1993). Parasite infection may be a continuous threat to both adults and juveniles. The bivalve *Macoma balthica* (Baltic Tellin) and the polychaete worm *Nereis diversicolor* (Ragworm) form the staple food of the Oystercatchers in our study area during the breeding season (Bunskoeke *et al.* 1996, Hulscher *et al.* 1996). *Macoma* is the first and second host of the gymnophallid trematode *Parvatrema affinis* (Swennen & Ching 1974, Hulscher 1982) and is also found in living Oystercatchers. Analysis of frost victims in winter showed high levels of parasite infestation, indicating that parasites might facilitate or induce death, particularly in juveniles (Borgsteede *et al.* 1988). However, on waders, the experimental studies needed to exclude other effects are lacking. Also, the effects of parasites on Oystercatcher reproduction are unknown. Chicks are fed by their parents, and individual differences in prey choice are

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known to exist (de Vlas *et al*. 1996, Ens *et al*. 1996, Goss-Custard 1996). If differential prey with differing infestations is fed to the chicks, dissimilar chick mortality might be the result.

In the first part of this study we test experimentally whether chicks are affected by endoparasites. Does parasite infection lead to increased chick mortality and/or reduced chick growth? Does parasite infection correlate with the breeding status of the parents? In the second part we sought support for the hypothesis, based on between-species comparisons, that the presence of blood parasites is related to the saltiness of the environment. Therefore we tested chicks and adults from both the salty (saltmarsh) and the adjacent freshwater habitat (polder) for the prevalence of blood parasites.

METHODS

Study area and population

The study was conducted during the breeding season of 1996 on a population of Oystercatchers nesting on the island of Schiermonnikoog in the Dutch Wadden Sea (Ens *et al*. 1992, Heg 1999, Heg *et al*. 2000, Heg & van der Velde 2001). The birds nest on saltmarsh but depend for food on the adjacent mudflats. Breeding pairs occupy territories of different quality. Pairs in high-quality territories ('residents') defend a nesting territory on the edge of the saltmarsh and have a contiguous feeding territory to which they can take their young to feed them. Pairs in low-quality territories ('leapfrogs') defend a nesting territory further inland and a feeding territory some distance from the shoreline (Ens *et al*. 1992, Heg *et al*. 2000), and thus have to fly over the intervening territories to supply their chicks with food. Residents fledge 3.5 times more chicks than leapfrogs do (Ens *et al*. 1995). For this work we also studied fledged chicks of pairs nesting on intensively grazed grassland 'the Banckspolder' enclosed by dikes and dunes next to the study area. The study area encompasses the area studied by Ens *et al*. (1992, 1995) and Heg *et al*. (2000).

Reproductive performance

From late April until mid July the study area was systematically searched for new nests every other day and the contents of old nests were checked. Partial nest predation induces the parents to continue the clutch in another nestcup. Oystercatchers lay 1–4 eggs. Assuming an incubation time of about 27 days (Hockey 1996), the nests were checked once or twice daily around hatching. All chicks from one brood hatched within 1 or 2 days. Hatching date was calculated as the number of days since 1 January. Hatchlings were marked with rhodamine, picric acid or malachite green on the white under parts to allow identification. In the main part of the study area (Heg *et al*. 2000) the parents were fitted with colour bands, so they were individually recognizable at 200 m. Observations were made from the mudflat and from hides at the edge of the saltmarsh to identify the parents attending the brood and determine the quality of their territory (resident or leapfrog, Ens *et al*. 1992). Around fledging (28–51 days, Kersten & Brenninkmeijer 1995, Heg & van der Velde 2001), the young were tracked from a hide and caught. These fledglings were weighed (in grams) and colour banded. Thus, we could determine the fledging production (the number of fledged chicks) and the fledging success (the number of fledged chicks per hatched egg) per brood.

Experimental manipulation of parasites

As most chicks die in their first days after hatching (Ens *et al*. 1992, Kersten & Brenninkmeijer 1995, Heg & van der Velde 2001) chick manipulation took place directly after hatching.

Broods were matched, as much as possible, by laying date, territory quality and for proximity to each other. Before hatching, one brood in each duo was assigned to the experimental ('treated') and one to the control group ('control') at random. A mix of 0.5 mL Spectril (a product of Mallinckrodt Veterinary Laboratory, containing 1.27 g/100 mL of the active component *levamisole* and 0.3 g *oxyelozanide*/100 mL) and 0.0025 mL Ivomec (a product of MSD-AGVet, containing 1.0 g of the active component *ivermectin*/100 mL) was given orally to each hatchling in the treated group. Spectril and Ivomec are wide-spectrum anthelminthic medicines used in veterinarian practice (Martin *et al*. 1997), *levamisole* and *ivermectin* act on nematodes, *oxyelozanide* acts on trematodes and cestodes (Campbell & Rew 1986). We used the recommended single dosage of levamisole of 20–25 mg/kg (0.08 mL/kg) (Brugere-Picoux & Silim 1992). Chicks weigh about 32 g at hatching, and the dosage of 23.4 mg/kg we applied at hatching is within these margins. In total, 132 broods were in the experiment; treated: 43 leapfrog broods (103 hatchlings), 23 resident broods (56

hatchlings); control: 42 leapfrog broods (99 hatchlings), 24 resident broods (64 hatchlings).

Collection of faecal samples

Faecal samples were collected around fledging. Faeces were frozen at –20 °C upon return to the field lab. The presence of helminth parasites was checked by the occurrence of parasite eggs in the faeces collected. We used the method of coprology by total flotation in mercury iodide (KI, HgI_2, Thienpont *et al*. 1979) to separate the parasite eggs from the rest of the faeces. The procedure is as follows: (1) each faecal sample was weighed and put in a 50-mL tube, such that at least 90% of the volume remained empty for the flotation liquid. (2) The tube was filled with mercury iodide and (3) a slide was placed on top. This method ensures that the parasite eggs float on top of the liquid and attach themselves easily to the slide. (4) The slide was placed upside down on an object glass and observed under a light microscope (×50–100 magnification). All eggs found were identified (if possible to the species level) and counted. Procedures (3) and (4) were repeated every half-hour, until no more eggs occurred on the slides. Identification of eggs follows Thienpont *et al*. (1979), Loos-Frank (1968), Bowers and James (1967) and James (1964). The description of the helminth community of Oystercatchers from Goater (1989) and Borgsteede *et al*. (1988), orientated us in the diagnosis. Chick faecal samples were of comparable mass (two-way ANOVA effects of territorial quality: $F = 0.76$, $df = 1$, $P = 0.39$; treatment: $F = 0.12$, $df = 1$, $P = 0.74$; $n = 37$). However, because of low faecal mass, the number of eggs found per sample was low, so infection level was represented simply as presence or absence of parasite eggs per species per individual. In total, faecal samples were obtained from 17 treated fledglings (seven residents, 10 leapfrogs) and 20 control fledglings (10 residents, 10 leapfrogs). In addition to the two experimental groups, untreated fledglings were sampled from the saltmarsh (five) and from the polder (six).

Collection of blood samples

Chicks were recaptured around fledging and blood was taken from the tarsal vein. A drop of blood was smeared on a slide, slides were air-dried and fixed immediately with 96% methanol on return to the field laboratory. Slides were coloured with Hemacolor (product of Merck KGaA). The slides were systematically screened for the presence of adult blood parasites under a light microscope (10 × 100), by checking 300 fields (each field contained 20 red blood cells on average). Smears were checked for parasites following the nomenclature of Campbell (1995). In total, blood samples were taken from 30 treated chicks (10 residents, 20 leapfrogs) and 51 control chicks (23 residents, 28 leapfrogs). In addition, untreated fledglings were sampled from the saltmarsh ($n = 11$ leapfrogs, two residents) and from the polder ($n = 16$).

Statistical analysis

Statistical analyses were performed using SPSS/PC+, versions 4.0–6.1. Data on infections per species and treatment were analysed using Hierarchical Loglinear analysis with backward elimination of terms from the full model containing all interactions and main effects. Deletion from the model was based on the likelihood ratio (Norusis 1990). Data on counts were analysed using GLIM 4.0 using Poisson regressions, and data on proportions using weighted Binomial regressions (Crawley 1993). GLIM uses a log-link function to ensure model fits result in positive counts and percentages only. The Null model indicates the overall deviance in the dependent variable. Full models were generated including all the effects and interactions. Non-significant effects were subsequently deleted from this model using the maximum-likelihood method, until the final model was reached. The final model is the minimum adequate model containing only significant terms. Significant terms are indicated with their effects on the deviance when removed from the final model (Crawley 1993). Statistical tests are two-tailed; 'not significant at the $P = 0.05$ level' is abbreviated 'ns' throughout.

RESULTS

Effectiveness of the antiparasite medicine

Forty-eight chick faecal samples were analysed for the presence of helminth parasite eggs. Treated chicks were significantly less infected than control chicks (Table 1). At least eight helminth species were detected, trematodes (notably *Parvatrema* sp. and *Psilostomum brevicollis*), *Nematode* sp., *Cestode* sp. and *Acanthocephale* sp. Some parasite species were significantly more prevalent than others. We

Table 1. Helminth infestion of Oystercatcher fledglings. A bird was considered infested when helminth eggs were found during faeces examination. Given are the numbers of infested fledglings (prevalence in percentage) depending on anthelminthic treatment at hatching. Additional untreated chicks were sampled in the saltmarsh (Control I) and in the polder (Polder).

	Trematodes					Nematodes	Cestodes	Acanthocephales
Treatment (*n*)	*Psilostomum brevicolle*	*Meiogymonophallus* sp.	*Parvatrema* sp.	*Notocotylus* sp.	*Trematode* sp.	*Nematode* sp.	*Cestode* sp.	*Acanthocephale* sp.
Treated (17)	3 (18)	0 (0)	3 (18)	0 (0)	0 (0)	0 (0)	3 (18)	0 (0)
Control (20)	7 (35)	3 (15)	7 (35)	2 (10)	1 (5)	2 (5)	4 (20)	1 (5)
Control I (5)	0 (0)	0 (0)	1 (20)	0 (0)	1 (20)	0 (0)	1 (20)	0 (0)
Polder (6)	0 (0)	4 (67)	4 (67)	1 (17)	0 (0)	1 (17)	0 (0)	0 (0)

Statistics comparing treated with control group: Hierarchical Loglinear Analysis with backward elimination of terms from the fully saturated model. Treatment*Parasite*Infection. Final Model $\chi^2 = 6.8$, $df = 14$, $P = 0.94$: Species*Infection $\chi^2 = 26.5$, $df = 7$, $P = 0.0004$, Treatment*Infection $\chi^2 = 7.6$, $df = 1$, $P = 0.006$. (Note: Treatment*Parasite*Infection was not significant, $\chi^2 = 6.1$, $df = 7$, $P = 0.53$). Statistics comparing treated, control, control I and polder group: Hierarchical Loglinear Analysis with backward elimination of terms from the fully saturated model Treatment*Parasite*Infection. Final Model $\chi^2 = 32.6$, $df = 42$, $P = 0.85$: Parasite*Infection $\chi^2 = 30.1$, $df = 7$, $P = 0.0001$, Treatment*Infection $\chi^2 = 11.2$, $df = 3$, $P = 0.01$ (Note: Treatment*Parasite*Infection was almost significant, $\chi^2 = 31.7$, $df = 21$, $P = 0.06$).

failed to detect a significant interaction between treatment, parasite species and infection, indicating that the anthelminthic drug affected all parasite species equally (Table 1). Polder chicks harboured a significantly different parasite fauna to untreated saltmarsh chicks. They were more infected by *Meiogymnophallus* sp. (the Cockle C*erastoderma edule* is the intermediate host [IH]) and *Parvatrema* (IH: *Macoma baltica*) and less infected with *Psilostomum* (IH: C*erastoderma edule* and the edible mussel (*Mytilus edulis*), Table 1, control and control I lumped: Hierarchical Loglinear analysis, territory quality*parasite species*infection $\chi^2 = 16.4$, $df = 7$, $P = 0.02$). No significant difference between the treated and untreated chicks could be detected in the parasite fauna (Hierarchical Loglinear analysis, territory quality*treatment*parasite species $\chi^2 = 0.954$, $df = 21$, $P = 1.0$, no interactions or main effects significant). At fledging, treated chicks had significantly fewer parasite species than did control chicks, both in high- and low-quality territories (Table 2). However, although the anti parasite drug could eliminate most of the gut parasites effectively throughout the chick-rearing period (up to 28–45 days), not all parasites were eradicated.

Table 2. Parasite infestion (% chicks infested) and the number of parasite species per individual (mean) for the treated and control fledglings from high- and low-quality territories. Chicks were treated at hatching, faeces were examined at fledging.

Experiment	Territory quality	*n*	% Infested by parasites	No. of parasite species
Control	Low	10	60.0	1.5 ± 0.5
Control	High	10	60.0	1.2 ± 0.5
Treated	Low	10	30.0	0.3 ± 0.2
Treated	High	7	57.1	0.9 ± 0.4
Statistic	*Treatment*		ns[a]	< 0.005[b]
	Territory quality		ns[a]	ns[b]
	Interaction		ns[a]	ns[b]

[a]Significance when indicated effect was removed from the logistic regression model: Full Model Deviance = 50.4, $df = 3$; Final Model Deviance = 52.7, $df = 36$.
[b]Significance when indicated effect was removed from the Poisson regression model: Null Model Deviance = 63.7, $df = 37$; Full Model Deviance = 55.2, $df = 34$; Final Model Deviance; Treatment Deviance = 6.0, $df = 1$.

Reproductive success

Since the control and treated groups were matched for equal situations at hatching, the hatching date, clutch size, number of hatchlings and hatching success were comparable (Table 3). From hatching onwards we expected the treated broods to perform better. However, contrary to our expectation the parents with treated chicks produced significantly fewer fledglings, in terms of both fledging production and fledging success. At fledging, territory quality, but not treatment, affected fledgling mass.

Prevalence of blood parasites

No blood parasites were detected in the blood smears: old chicks and fledglings from the saltmarsh

Table 3. The hatching date, clutch size, number of hatchlings, hatching success, number of fledglings, fledging success and fledging weight (mean ± se) for the treated and the control broods from high- and low-quality territories. Significance of the treatment, territory quality and their interaction are indicated in the bottom rows. *n* = Number of broods. Hatching date = the number of days since 1 January. Sample sizes for fledging weight (number of chicks) are indicated separately in parentheses.

Experiment	*n*	Territory quality	Hatching date	Clutch size	No. of hatchlings	Hatching success (%)	No. of fledglings	Fledging success (%)	Fledging weight in g (*n*)
Control	42	Low	167.8 ± 1.6	2.8 ± 0.1	2.3 ± 0.1	82.4 ± 3.5	0.6 ± 0.1	28.6 ± 4.8	256 ± 17 (18)
Control	24	High	169.2 ± 2.1	2.9 ± 0.1	2.6 ± 0.2	88.2 ± 4.1	1.0 ± 0.2	37.8 ± 6.4	325 ± 12 (16)
Treated	43	Low	167.8 ± 1.6	2.9 ± 0.1	2.3 ± 0.2	80.8 ± 3.8	0.3 ± 0.1	18.2 ± 4.4	274 ± 18 (14)
Treated	23	High	171.0 ± 1.9	2.9 ± 0.1	2.4 ± 0.3	84.3 ± 4.4	0.5 ± 0.1	19.6 ± 5.9	336 ± 28 (8)
Statistic	*Treatment*	*P*	ns[a]	ns[b]	ns[c]	ns[d]	< 0.005[e]	< 0.005[f]	ns[g]
	Territory quality	*P*	ns[a]	ns[b]	ns[c]	ns[d]	< 0.05[e]	< 0.05[f]	< 0.001[g]
	Interaction	*P*	ns[a]	ns[b]	ns[c]	ns[d]	ns[e]	ns[f]	ns[g]

[a]Two way ANOVA, effect of treatment $F = 0.3$, $df = 1$, $P = 0.6$; territory quality $F = 1.6$, $df = 1$, $P = 0.2$; interaction $F = 0.2$, $df = 1$, $P = 0.6$.
[b]Significance when indicated effect is removed from the Poisson regression model: Null Model Deviance = 21.5, $df = 136$; Full Model Deviance = 21.4, $df = 133$.
[c]Significance when indicated effect is removed from the Poisson regression model: Null Model Deviance = 44.7, $df = 136$; Full Model Deviance = 44.7, $df = 133$.
[d]Significance when indicated effect is removed from the weighted binomial regression model: Null Model Deviance = 159.5, $df = 136$; Full Model Deviance = 157.2, $df = 133$.
[e]Significance when indicated effect is removed from the Poisson regression model: Null Model Deviance = 123.2, $df = 136$; Full Model Deviance = 110.8, $df = 133$; Final Model Deviance (which excludes the interaction) = 111.2, $df = 134$, treatment Deviance = 8.1, $df = 1$; territory quality Deviance = 4.2, $df = 1$.
[f]Significance when indicated effect is removed from the weighted binomial regression model: Null Model Deviance = 162.7, $df = 136$; Full Model Deviance = 148.7, $df = 133$; Final Model Deviance (which excludes the interaction) = 149.2, $df = 134$, treatment Deviance = 9.9, $df = 1$, territory quality Deviance = 3.8, $df = 1$.
[g]Two way ANOVA, effect of treatment $F = 0.7$, $df = 1$, $P = 0.4$; territory quality $F = 14.1$, $df = 1$, $P < 0.001$; interaction $F = 0.03$, $df = 1$, $P = 0.9$.

(*n* = 58 leapfrog, 35 resident chicks), chicks from the polder (*n* = 16), adult breeders from the saltmarsh (*n* = 26 leapfrog males, 27 leapfrog females, 18 resident males, 18 resident females) and adult breeders from the polder (two males and two females), all appeared free from blood parasites.

DISCUSSION

Many studies have found that chick survival increased when parasite infections were experimentally reduced (e.g. Arendt 1985, Brown & Brown 1986, Møller 1990, Johnson & Albrecht 1993, Richner *et al.* 1993, McKilligan 1996). We attempted to reduce the gut parasite load from hatching onwards, and sampling of the parasite eggs in the faeces of fledglings suggested that our treatment worked. Fledglings treated at hatching were less infected than fledglings untreated at hatching, so we expected higher fledgling rates of treated hatchlings. To our surprise we found the opposite effect: treated hatchlings survived less well compared to untreated hatchlings. How could this come about? We arrived at five different hypotheses explaining this unexpected result.

First, the particular anthelminthic drug administered might be poisonous to Oystercatcher chicks. We consider this unlikely because the drug (*levamisole* or *ivermectin*) has already been applied to other bird species with no detrimental effects on the survival or the growth of the individuals involved. This is shown in studies of adult and young geese by Cencek *et al.* (1992) and Ziomko *et al.* (1992), in a study of Red Grouse *Lagopus lagopus* by Hudson (1986) and a study of poultry by Brugere-Picoux and Silim (1992). In addition, no poisonous effects have been documented in frogs, golden hamsters, sheep, moufflon and camels (Lamka *et al.* 1996, Maqbool *et al.* 1996, Sisodia *et al.* 1996, Iglauer *et al.* 1997, Rajani & Maity 1997). Further, the hatchlings were given a dosage within the safety margins (see Methods). Last and most important, *levamisole* and *ivermectin* act specifically on the biochemistry of invertebrates only. *Levamisole* (a *benzimidazole*) acts as an agonist of the nicotinic acetylcholine receptors of nematodes (Martin *et al.* 1997). *Ivermectin* (an *avermectin*) potentates or gates the opening of glutamate-gated chloride channels found only in invertebrates (Martin *et al.* 1997). Therefore, we think it is highly unlikely that *levamisole* and/or

ivermectin are poisonous to Oystercatcher chicks. Nevertheless, however unlikely, a high susceptibility of this bird species to one or both of the components of this anthelminthic drug, maybe in combination with their low age at administration, cannot be completely ruled out. This hypothesis might need further attention, by monitoring the development of the immune system of treated and untreated chicks under laboratory conditions. Hence, the helminth fauna can be sampled frequently, and the maturation of the immune system can be tested with short-term challenges (Sheldon & Verhulst 1996), without detrimental effects on the survival or health of the chicks.

A second explanation might be that the drug did increase the survival of chicks, but as a side-effect increased the amount of sibling competition in the treated broods. In the end, this might reduce the fledging rate of the treated broods relative to the untreated broods. The only evidence against this explanation is provided by the results of the treated and untreated resident broods. Chicks in large resident broods do not survive less well than small resident broods (Ens *et al*. 1992, Heg & van der Velde 2001), suggesting that sibling competition does not cause increased mortality rates in these large broods. Since, like the leapfrogs, the treated resident chicks survived less well than the untreated resident chicks, and there was no significant interaction between treatment and territory quality, the second explanation seems less likely.

Thirdly, if Oystercatchers are highly infested with parasites, administration of the drug could cause extensive death of the helminths. This might result in the release of toxic products of catabolism, which might intoxicate the treated birds. This is known in domestic animals (Fraser 1991). However, because of the early administration of the drug, it is very unlikely that chicks were already heavily infected.

Fourthly, by keeping the intestines free of parasites, the gut flora might have changed. More harmful organisms (e.g. bacteria and fungi) could have taken advantage of this, which might have led to diseases in the treated group, and therefore to a higher mortality. Evidence against this point is that the surviving birds from the treated group were not in a poorer condition than the untreated birds.

Finally, if the anthelminthic drug was effective, it might have prevented the treated hatchlings from developing their immune system. As soon as the drug ceased to work, they would then be highly susceptible to any infection challenging their essentially undeveloped immune system. We could expect our drug to protect the treated chicks for 15–20 days, and hence infection is likely just before fledging age (28–51 days). Possibly the treated chicks experienced mass mortality soon after the drug ceased to work. At present, we consider this the most likely explanation for why treated chicks survived less well than untreated chicks. Rapid re-infection after treatment with an antihelminth drug has been reported by Bailey *et al*. (1990) in a study of captive Swan Geese *Anser cygnoides*.

Effects of antiparasite treatment on the development of the immune system and on possible internal changes need further study in this and other free-living species. In the future, treatments are only justifiable after the chicks have acquired an effective immune system.

Blood parasites

Another important result of our study is the apparent absence of blood parasites in both adult and fledged Oystercatchers. It is highly debated why coastal living vertebrates show low levels of blood parasite infections, although they may have many helminth parasites (Bennett *et al*. 1992, Earlé & Underhill 1992, Galaktionov & Bustnes 1999, Piersma 1997). One hypothesis states that the environment is unsuitable for the intermediate hosts of the blood parasites (e.g. the vectors of ectoparasites, Bennett *et al*. 1995) or for the blood parasites themselves (the '*environment hypothesis*', Rytkönen *et al*. 1996, Tarof & Stutchbury 1997). If the hosts lived in another environment, blood parasites would infect them. Another hypothesis states that for some other reason in the evolutionary history of coastal birds, ancestors acquired resistance against parasites in general (the '*phylogenetic hypothesis*'). Since then, derived species have inherited this resistance and largely retained coastal breeding. Thus, today one would falsely infer a causal effect from the negative correlation between parasite prevalence and coastal breeding. If these same species were transferred to a non-coastal habitat, they would remain uninfected by blood parasites. Interestingly, this study provides a test of these hypotheses, since on the island of Schiermonnikoog Oystercatchers can be found breeding in the inland, freshwater habitat (the polder) and the saltmarsh close to each other. Adults from both habitats regularly visited the salty mudflats. Similarly, chicks on the saltmarsh were continuously exposed to the salty environment, i.e. receiving food

from the mudflats, bathing in salt water. The only group not in regular contact with the salty environment was chicks from the polder. These chicks stay in their natal territories in the fertilized grassy pastures, with freshwater ditches to bathe in. They were fed mainly on leatherjackets (tipulid larvae) and earthworms found in these territories, and lived in a predominantly inland habitat. Nevertheless, in both habitats no blood parasites were found in the chicks, in disagreement with the first hypothesis.

Comparative studies of blood parasite prevalence in Oystercatcher chicks from populations breeding at coastal sites and far inland might prove our point that the absence of blood parasites might be related to species, rather than to environment effects. Also, any critical test of these hypotheses must address the consequences of the various blood and gut parasites on the fitness of both chicks and adults. For instance, some blood parasites might show low prevalence in general, but might have large fitness effects (e.g. Dufva 1996). If coastal breeding birds are infested by these parasites, but not by other parasite species, they might nevertheless have to invest similar quantities of resources in immune function to inland breeding birds, which might harbour large numbers of other, but fewer detrimental, parasites. Furthermore, coastal breeding birds might show low levels of blood parasites, but high levels of gut parasites. We have found high levels of gut parasites in both polder ('inland') and saltmarsh ('coastal') chicks, and the parasite fauna differed significantly between these two adjacent sites. This suggests that chicks from both sites have to invest heavily in parasite defence, a conclusion which might not have been drawn if only blood parasites had been sampled.

Many waders, including Oystercatchers, are coastal living for a major part of their lives, and many species rely heavily on *gastropoda* and other *mollusca* for food (Cramp & Simmons 1983). Most of these prey species are intermediate hosts of some severe bird parasites, and high levels of gut parasite infection have been reported (Jensen & Mouritzen 1992, Goater 1993, Goater *et al.* 1995). These waders might avoid gut parasites only in the short breeding season, when many species breed in the high arctic and prey predominantly on insects. However, it is still largely unknown for how many days parasites remain within the gut and for how many days after last infection hosts are affected by these gut parasites. Insects (and arthropods generally) may also be intermediate hosts of other parasites, especially pathogenic species such as *Acanthocephales*. In the Common Eider *Somateria mollissima* and Mute Swan *Cygnus olor* for example, infection with *Acanthocephales* following a switch of diet from the 'traditional' mollusc food to crustaceans can cause widespread mortality (Pennycott 1998). Experimental manipulations of parasite loads of either gut or blood parasites and monitoring their fitness effects would be essential if the effects of parasites are to be understood.

In conclusion, we found a negative effect of anthelminthic treatment on chick survival, maybe caused by immune depression in the treated hatchlings. Future studies manipulating gut parasite loads must address the possibility that the treatment delays the maturation of the immune system, and consequently the possibility of enhanced susceptibility to gut parasites after the treatment has stopped. We did not find support for the '*environment hypothesis*' relating low levels of blood parasites to the coastal environment, but by implication we found support for the '*phylogenetic hypothesis*': some other species-dependent factor might cause the low levels of blood parasites in coastal Oystercatchers.

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