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NATURAL AND EXPERIMENTAL INFECTIONS OF *EUSTRONGYLIDES IGNOTUS*: EFFECT ON GROWTH AND SURVIVAL OF NESTLING WADING BIRDS

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ABSTRACT.—One of three Tricolored Heron (*Egretta tricolor*) nestlings experimentally infected with *Eustrongylides ignotus* larvae died two days postinfection. During eight days postinfection, the other infected birds consumed less food per meal, consumed less food in relation to body mass, regurgitated more frequently, and exhibited lower bill and mass growth rates than did three uninfected control birds. Prevalence of eustrongylidosis among free-ranging Great Egret (*Casmerodius albus*) nestlings from Lake Okeechobee ranged from less than 5% in 1989 and 1991 to 28.4% in 1990. During 1990, infected nestlings suffered 10.6 to 17.9% higher mortality than uninfected birds, with younger nestlings suffering proportionately greater mortality. Summarized across colonies and nestling ranks (i.e. hatching order), infected nestlings averaged 2.9% shorter bills, 4.1% shorter tarsi, and 6.9% shorter wings than noninfected birds. Among rank 1 (oldest) and 3 (youngest) nestlings, infected birds were lighter (6.3-9.0%), but among rank 2 nestlings from both colonies infected birds averaged heavier than their noninfected counterparts. These results for mass may be due to the interactive effects of sibling rivalry. Eustrongylidosis generally had the greatest impact on those nestlings otherwise disposed to lower growth rates, whether due to colony location or nestling rank. The results emphasize the importance of monitoring diseases whenever prefledging growth and survival are of interest. *Received 5 May 1993, accepted 17 November 1993.*

EUSTRONGYLIDES IGNOTUS INFECTIONS in nestling ciconiiforms, especially those of the family Ardeidae, appear to be associated with anthropogenic habitat alteration (Spalding et al. 1993), and can have a devastating effect upon reproductive success (Weise et al. 1977, Roffe 1988, Spalding et al. 1993, Spalding and Forrester 1993). In this study, we examined the effect of this nematode parasite, which burrows through the stomach wall, on the growth and survival of nestling ardeids in the laboratory and in the field. We experimentally infected Tricolored Heron (*Egretta tricolor*) nestlings to gather details about the effects of parasitism on young nestlings. We also monitored nestlings at Lake Okeechobee, Florida from 1989 to 1991 to gain information about prevalence and to measure the effects that eustrongylidosis might have on the growth and survival of nestlings in the wild. We provide evidence concerning both the mechanisms and magnitude of the effect of eustrongylidosis on ardeid nestlings and show that the impact on growth and survival results can be significant even during periods of only moderate prevalence.

METHODS

Experimental infections.—Six Tricolored Heron nestlings were collected from Frank Key, Florida Bay (25°06.3'N, 80°54.7'W) on 21 May 1990. Each was the intermediate-sized nestling of three siblings and was estimated to be between two to four days of age based on bill length. Nestlings were marked and placed in individual containers within an incubator. They were fed *ad libitum* four times each day with thawed fish or with fresh fish that had been examined and found to be free of eustrongylid larvae. Nestlings could take fish directly from a dish of shallow water or, if necessary, were offered fish held with forceps. They were never force fed. On the evening of the second day, three randomly selected birds (no. 2, 3, and 6) were each given four eustrongylid larvae per os prior to feeding. An average of 2.7 larvae per bird (range 1-10) have been found in very young naturally exposed birds (Spalding and Forrester 1993). Larvae were obtained from naturally infected mosquitofish (*Gambusia holbrooki*) collected in a canal in Gainesville, Alachua County, Florida, and maintained alive in the laboratory. Larvae were extracted from fish, placed in a gelatin capsule, and fed to nestlings immediately. Uninfected control birds (no. 1, 4, and 5) were given empty capsules. The nest container was examined

carefully during the following two days for evidence of regurgitated food and parasites. At each feeding, birds were examined visually and by palpation (Spalding 1990) for evidence of parasitism. Bill lengths (standard culmen length to the nearest 1 mm) were measured at the morning feeding and masses were recorded (using a digital balance accurate to 0.01 g) before and after each feeding. Surviving birds were killed humanely on day 8 postinfection and necropsy examinations were conducted. Lesions have been described elsewhere (Spalding and Forrester 1993).

We used a repeated-measures ANOVA (RMA) with infection status as a grouping variable in SYSTAT (Wilkinson 1990) to compare trends in variables such as food intake rate, assimilation efficiency, and growth statistics.

Natural infections.—Each year from 1989 to 1991 as part of a larger study, we monitored wading bird nests in several mixed-species colonies located on Lake Okeechobee in south-central Florida. Selected nests were tagged with numbered flags and visited at intervals of three to six days until nestlings fledged or the nest failed. In 1989 and 1990, necropsy examinations were performed on all nestlings found dead to determine cause of mortality and to check for eustrongylidosis. In 1990 and 1991, live birds were checked by palpation.

Each season a subsample of marked nests that hatched was chosen for detailed growth and nestling health studies. Nestlings were uniquely marked with nail polish on toenails until old enough to be fitted with U.S. Fish and Wildlife Service silver-colored bands in unique combinations with one to three colored leg bands. At each visit, measurements collected for each nestling included: bill length (from posterior margin of the nostril to tip of upper mandible) and tarsus length taken with a dial caliper accurate to 0.01 mm; flattened wing chord taken with a ruler to the nearest 1 mm; mass taken with a hand-held spring-scale to the nearest 1 g. Nestling ages were either known to the day if discovered in an advanced hatching stage or estimated to within one to two days when first discovered based on size and plumage state. For those nestlings that were measured, age designations were further refined by verification against sample means and graphical examinations for outliers.

In 1990, data for Great Egrets (*Casmerodius albus*) were collected from two colonies. The Eagle Bay Island (EB) colony, a 3-km^2 expanse of willows (*Salix caroliniana*) and other marsh vegetation cutoff from the mainland by a canal, was located at the north end of the lake (27°10.3'N, 80°50.8'W). From 1989–1992, the site was occupied by 800 to 2,000 nesting pairs of nine aquatic-feeding ciconiiform species, plus several thousand nesting pairs of Cattle Egrets (*Bubulcus ibis*). In 1990, 165 pairs of Great Egrets nested at the site. Available foraging habitats included: a variety of mixed-prairie and submerged-vegetation habitats; cattle pasture ponds; ditches and flooded fields; riparian and forested wetlands. Many of the nesting Great Egrets fed along residential canals and panhandled fish scraps at cleaning stations (J.P.S. unpubl. data). The Clewiston Spit (CS) colony, a 700-m^2 rocky dredge-spoil island covered with willow and exotic Brazilian pepper (*Schinus terebenthifolius*) and Australian pine (*Casuarina equisetifolia*), was located on the southwest side of the lake (26°46.5'N, 80°54.6'W). From 1989–1992 the site was occupied by 140 to 160 nesting pairs of three to five aquatic-feeding species, plus 100 to 200 pairs of Cattle Egrets. In 1990, the CS colony supported a total of 87 pairs of Great Egrets. Available foraging habitats included: a more limited variety of mixed-prairie and submerged-vegetation habitats; residential ditches; ponds and canals; and a few panhandling stations; and an extensive network of agricultural ditches, canals and flooded fields (primarily sugar cane fields).

In addition to the regular checks of measured nestlings, unmeasured nestlings from marked nests were examined for eustrongylidosis on three occasions during the 1990 season. These data were not included in the growth rates analysis; however, provided other criteria (see below) were met, these cases were included in the mortality analysis. All statistical analyses of data from Lake Okeechobee were conducted in SYSTAT (Wilkinson 1990) unless noted otherwise.

Mortality analysis.—Only birds from marked nests, examined when seven days or older were included in the mortality analysis. It may take five to six days postinfection for palpable lesions to develop; thus, among the youngest nestlings with acute infections the chance of false-negative diagnoses is high (Spalding 1990). Note, however, that since eustrongylidosis probably causes greatest mortality among the youngest nestlings, and our detection of eustrongylidosis antemortem was poorest for these birds, this analysis likely underestimated mortality among infected nestlings.

Uninfected nestlings that died of apparent clumsiness or accidents that resulted in broken limbs were included in the analysis, but those that perished due to external factors such as predation or being blown out of the nest by high winds were not. Nestlings were considered "fledged" if they were confirmed alive (banded young or direct unequivocal association with a marked nest) at six weeks of age. This is an arbitrary cutoff, but corresponds to the age at first flight given by Meyerricks in Palmer (1962) and in Hancock and Kushlan (1984), and to the minimum age of 40 days at first flight confirmed among banded Okeechobee nestlings. Six weeks probably does not correspond to the age of independence in most cases (62–70 days; Weise 1975), but at Lake Okeechobee did often coincide with the onset of greater movement among nestlings as witnessed by the intermittent absence of nestlings at subsequent nest checks. Of the 121 nestlings designated as fledged, 14 (6 infected) were last confirmed alive at ages less than 40 days,

but in all cases unidentified (unbanded or unconfirmed sighting) young of the appropriate age were seen later in the vicinity of the nests.

The infection status of many nestlings included in the mortality analysis was diagnosed only once. All infected nestlings were parasitized within the first four weeks of age. Among nestlings designated uninfected, 43 were last examined prior to 21 days and 41 at greater than 21 days of age. Our analysis, therefore, is best described as examining the influence of eustrongylidosis on mortality among nestlings whose infection status was known at least to near three to four weeks of age.

Prior analyses revealed that linear growth rate was significantly and negatively correlated with age at last measurement, even with ages at last measurement restricted to less than 20 days (J.P.S. unpubl. data). Therefore, instead of analyzing mean growth rates, we compared measurements for the specific age of 20 days. We chose 20 days for three reasons: (1) all infected birds had been infected before this age; (2) the frequency of measurements became more irregular after this age as nestlings gained mobility; and (3) growth rates for most measurements usually began to level off at this stage. When necessary, we used linear interpolation and extrapolation based on the two nearest-age measurements to estimate the 20-day measurements. The last measurement date for some nestlings was less than 20 days, but no less than 17 days. We included only nestlings believed to have fledged in the uninfected group, and included infected birds only if they survived to 17 days or beyond, since the reliability of extrapolating to 20-day measurements would otherwise have been questionable. The resulting sample included 72 uninfected and 32 infected nestlings.

RESULTS

Experimental infections.—One of the experimentally infected birds died on day 2 postinfection from the parasitic infection and was eliminated from all statistical analyses. From day 1 postinfection through day 8 postinfection, the two remaining infected birds ate less per meal than the three uninfected birds (RMA, $F_{1,3} = 16.65$, $P = 0.027$; Fig. 1), and during the first two days postinfection the infected birds regurgitated more frequently (infected, 20 fish; uninfected, 11 fish). By day 7 postinfection, the percent of body mass consumed per day by uninfected birds had declined from about 100% to 50% (Fig. 1); infected birds usually consumed a lower percentage (RMA, $F_{1,3} = 21.41$, $P = 0.019$). Infected birds also gained less mass per day than uninfected birds (RMA, $F_{1,3} = 126.79$, $P = 0.002$; Fig. 1). In two cases, the infected birds lost mass; bird 3 lost 4.2% of its mass on day 1 postinfection and bird 6 lost 12.3% of its mass on day 4 postinfection. Both mass losses were associated with palpable changes in the abdomen. Bird 2 consistently gained mass; however, the rate of gain was very low for the first three days postinfection. By day 2 postinfection, infected birds weighed less and had shorter bills than the uninfected birds (Fig. 1); the RMA showed a significant effect on mass ($F_{1,3} = 19.59$, $P = 0.012$), but the effect on bill length only approached significance ($F_{1,3} = 8.15$, $P = 0.065$). A comparison of mean growth rates (average of linear regression estimates for each individual) revealed significant effects in both cases: 6.4 versus 14.2 g/day for mass; 1.6 versus 2.3 mm/day for bill length (t-test; mass, $P = 0.001$; bill, $P = 0.016$). Assimilation efficiency (grams mass gained per gram of food consumed converted to percent) varied from 15.6 to 34.0% in uninfected birds, with no consistent differences between the groups (Fig. 1).

Natural infections.—Fifty of 176 (28.4%) Great Egret nestlings from Lake Okeechobee examined in 1990 were diagnosed positive for eustrongylidosis (Table 1). Of these 149 were examined live (four of these were confirmed later by necropsy examination) and 27 were examined at necropsy only. Of 76 nests examined, 35 (46%) contained at least one infected nestling. In contrast, none of 13 nestlings examined as carcasses in 1989 were infected and, in 1991, only 4 of 94 (4.3%) live nestlings from 4 of 37 (10.8%) nests were infected. During 1990, overall prevalence appeared higher at the CS colony than at the EB colony (Table 1), but the difference was not statistically significant when nestling rank (i.e. hatching order with rank 1 oldest) was used as the stratification variable (Mantel-Haenszel test, $P > 0.05$). There was very little difference in prevalence among nestlings of rank 1 and 3 from CS and no infections among the three rank 4 nestlings from CS. There was a suggestion of a trend for increasing prevalence with decreasing nestling rank among EB nestlings (Table 1), but the differences were not statistically significant (Fisher's exact test, $P > 0.05$).

Independent contingency table analyses of mortality levels within infection-status/nestling-rank groups resulted in no significant differences due to colony location (Fisher's exact test, $P > 0.05$). Among noninfected nestlings, first- and second-ranked birds had significantly

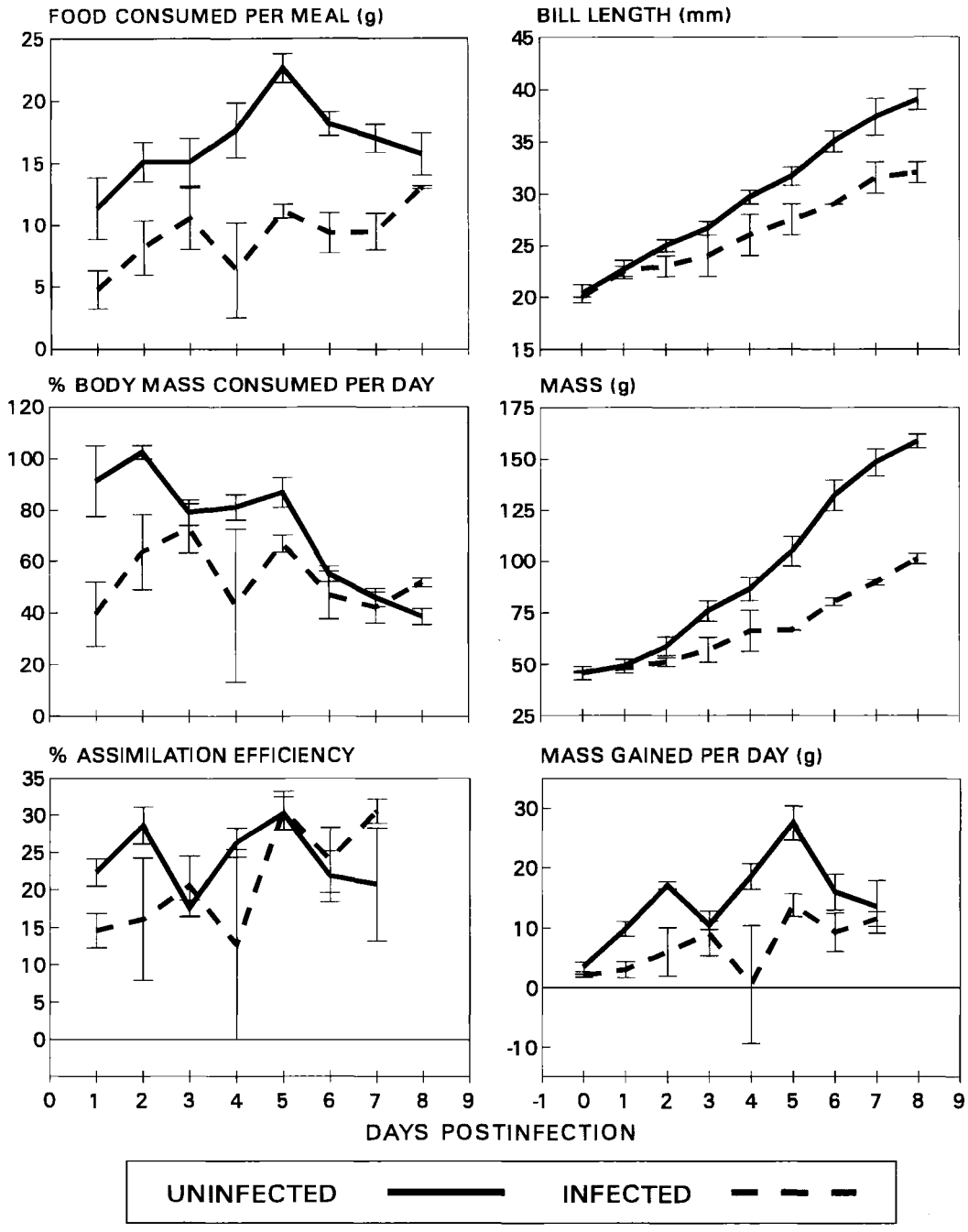

Fig. 1. Food-consumption rates, assimilation efficiency, and growth rates of two infected and three uninfected Tricolored Heron nestlings under experimental conditions. Plots indicate $\bar{x} \pm$ SE. Food consumed per meal is daily average for four meals. Assimilation efficiency is grams food consumed per gram body mass gained per day.

TABLE 1. *Eustrongylides ignotus* prevalence among Great Egret nestlings at Lake Okeechobee in 1990.

Nestling rank	Nesting colony[a]	Uninfected	Infected	Percent infected
1	CS	20	11	35.5
	EB	28	6	17.7
2	CS	21	10	32.3
	EB	23	6	20.7
3	CS	15	8	34.8
	EB	15	7	31.8
4	CS	3	0	0.0
	EB	1	2	66.7
Total	CS	59	29	33.0
Total	EB	67	21	23.9

[a] (CS) Clewiston Spit; (EB) Eagle Bay Island.

lower mortality than their younger siblings (Fisher's exact test, $P = 0.004$). Therefore, we stratified cases within the two infection-status groups by nestling rank, combining first- and second-rank nestlings, as well as third- and fourth-rank nestlings to boost sample sizes. The effect of eustrongylidosis on mortality was then determined using the Mantel-Haenszel test with a correction for continuity (Snedecor and Cochran 1989:215). The mortality among infected nestlings averaged 10.6 to 17.9% higher ($P = 0.031$) than among uninfected nestlings (Table 2).

Preliminary analyses restricted to uninfected nestlings revealed that both colony affiliation and nestling rank exerted a significant influence on 20-day measurement statistics. An additional series of saturated and partial ANOVAs revealed no significant interactions involving colony, nestling rank, and infection status ($P > 0.05$). Therefore, the selected ANOVA model was purely additive: 20-day measure = COLONY + RANK + INFECTION STATUS + ERROR. Normal probability plots of residuals and plots of residuals against estimates indicated no significant departures from the ANOVA assumptions of normally distributed residuals and homoscedastic variances, respectively.

For all measurements, colony affiliation was a highly significant factor in the models ($P < 0.001$), with CS nestlings consistently slower growing than those at EB (Fig. 2). Stratifying by nestling rank (with two rank 4 nestlings grouped with those of rank 3) also contributed significantly to the models in all cases ($P < 0.026$; Fig. 2). Posthoc multiple comparisons (Bonferroni-adjusted probabilities) revealed that, with variation due to colony affiliation and infection status accounted for, rank 3 nestlings nearly always averaged smaller at 20 days than either rank 1 or 2 nestlings. The only exception was for bill length where rank 2 and 3 nestlings did not differ significantly ($P > 0.05$). In no case was the effect of differences between rank 1 and 2 nestlings significant, but rank 2 nestlings at CS generally averaged slightly larger than those of rank 1, whereas the opposite was true at EB.

Summarized across the three nestling ranks and two nesting colonies, infected 20-day-old nestlings averaged 1.43 mm or 2.9% shorter bills ($P = 0.016$), 3.15 mm or 4.1% shorter tarsi ($P = 0.019$), and 8.4 mm or 6.9% shorter wings ($P = 0.003$) than noninfected birds (Fig. 2). Infected nestlings of rank 1 and 3 averaged 33.9 g (6.3%) and 46.2 g (9.0%) lighter, respectively, than noninfected birds; however, infected rank 2 nestlings from both colonies were heavier than their noninfected counterparts. Consequently, the overall effect of parasitism on 20-day mass was insignificant ($P = 0.158$; Fig. 2).

Uninfected nestlings from the CS colony exhibited consistently lower growth rates than those from EB (Fig. 2). Among rank 1 and 2 nestlings, eustrongylidosis always had a proportionately greater effect at CS, but the opposite was true for rank 3 nestlings (Fig. 3). At the EB colony, the effect of eustrongylidosis on bill, tarsus, and wing lengths increased with

TABLE 2. Mortality among infected and uninfected nestling Great Egrets at Lake Okeechobee in 1990 with cases stratified by nestling rank (Mantel-Haenszel test corrected for continuity, $z = 1.862$, $P = 0.031$).

Nestling rank	Uninfected			Infected		
	Fledged	Died	Percent mortality	Fledged	Died	Percent mortality
1 and 2	72	2	2.7	26	4	13.3
3 and 4	15	5	25.0	8	6	42.9
Total	87	7	7.4	34	10	22.7

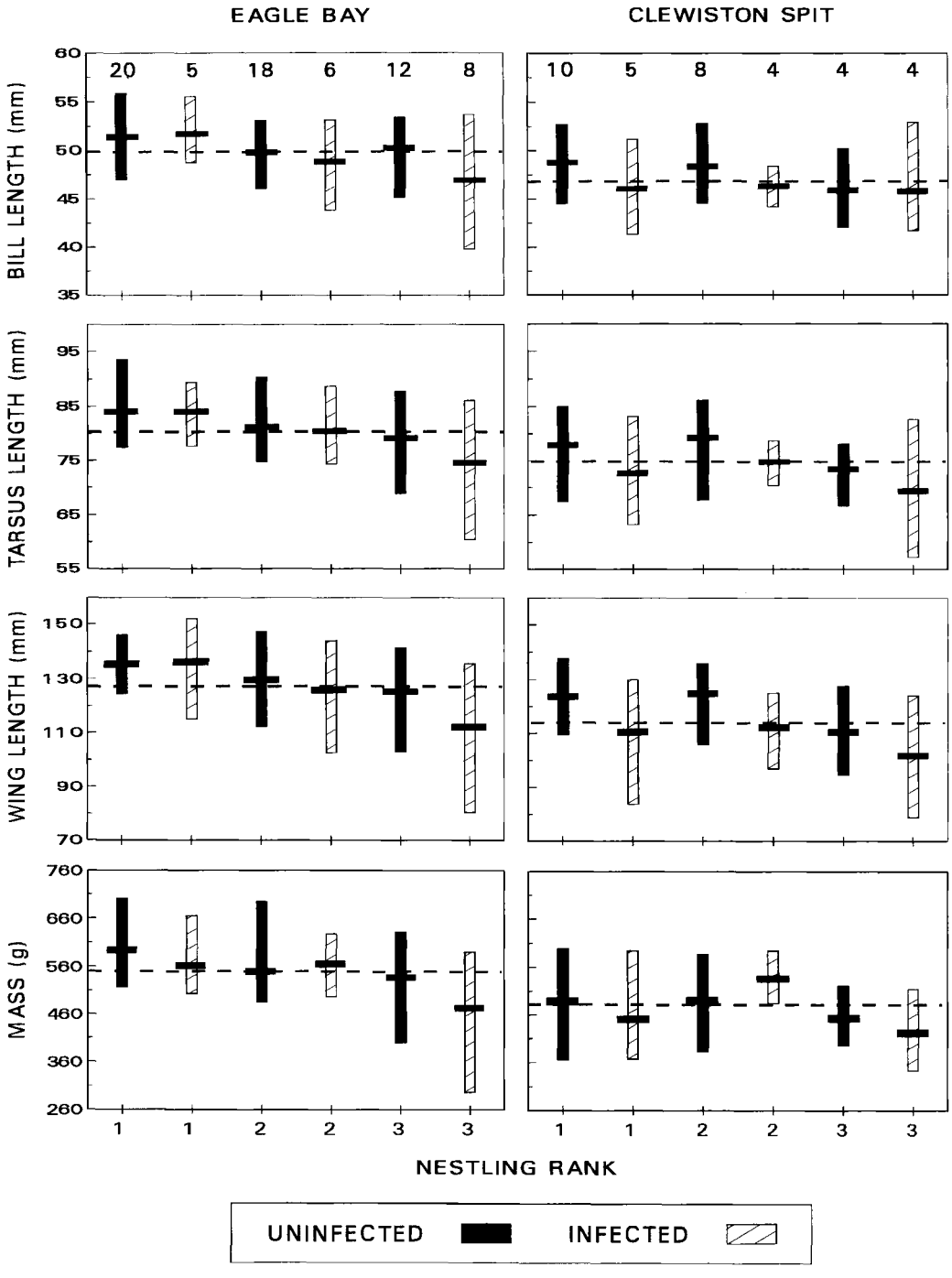

Fig. 2. Measurements at 20 days of age for infected and uninfected Great Egret nestlings from two colonies on Lake Okeechobee. Plots are of means and ranges for each nestling rank. Sample sizes indicated in upper panels. Dashed, horizontal lines represent grand means for each measurement within colonies.

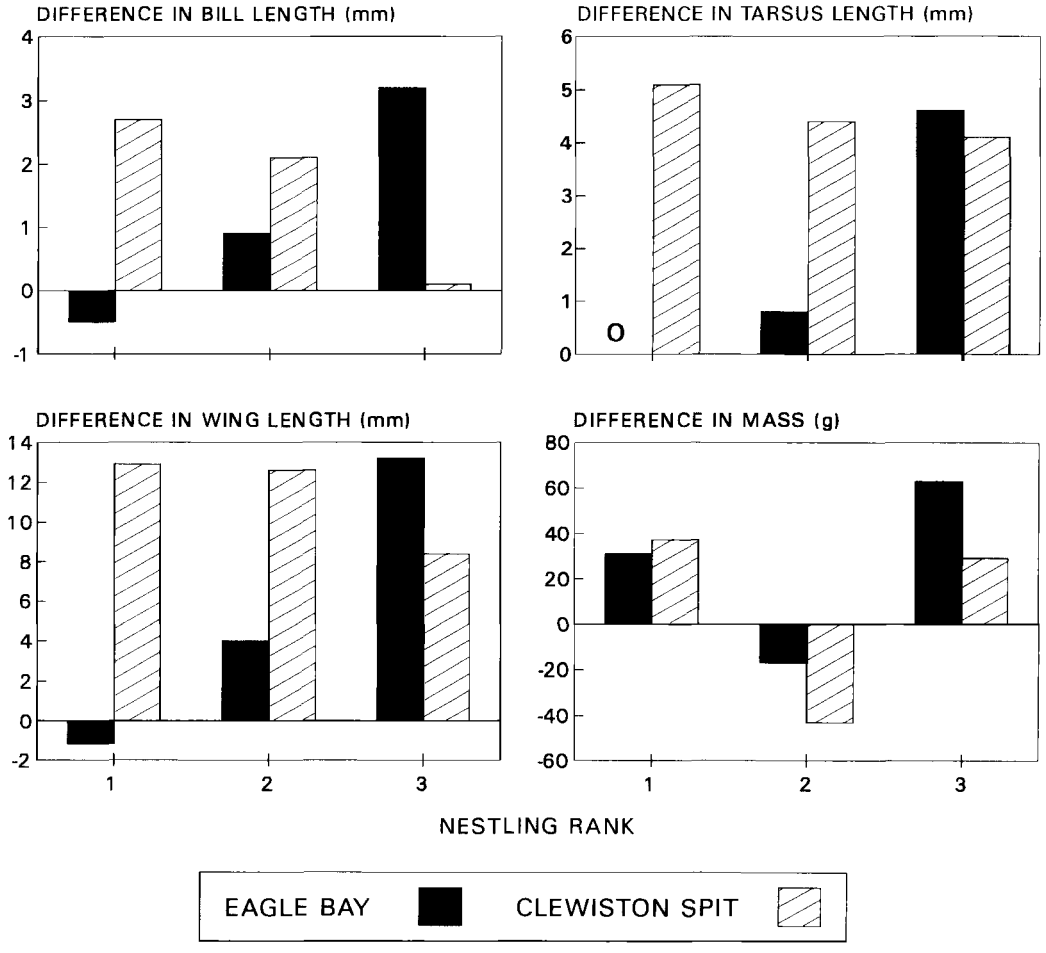

Fig. 3. Differences between mean 20-day measurements of infected and uninfected nestling Great Egrets from Lake Okeechobee by nesting colony and nestling rank. Positive differences indicate a reduction in measurements in infected birds.

decreasing nestling rank, with no reductions in growth evident among infected rank 1 nestlings (Fig. 3). At the CS colony the trends were opposite, albeit less pronounced, and hence the lack of a significant interaction of rank and infection status in the ANOVA model. No clear trends were evident for mass. The range of variation in individual measurements was often larger among infected nestlings (Fig. 2), as might be expected given individual variation in resistance to the effects of the parasite or chronicity of infection.

Discussion

One of us recorded a three-year average bill growth rate from 3 to 14 days of 2.1 mm/day for 133 Tricolored Herons from Lake Okeechobee (least-squares grand mean calculated across years, colonies and nestling ranks; J.P.S. unpubl. data). The mean rate of 2.3 mm/day calculated for the three uninfected control birds, therefore, was high, as might be expected for birds fed *ad libitum*. However, mass gain per day in the uninfected control birds (14.2 g/day) averaged slightly lower than for the natural Lake Okeechobee populations (15.7 g/day). For the two experimentally infected nestlings, both bill and mass growth rates (1.6 mm/day and 6.4 g/day, respectively) were considerably depressed relative to rates for the natural populations.

The mechanism for slower growth among the infected nestlings apparently included a combination of a loss of appetite, a slight decline in assimilation efficiency, and a tendency to re-

gurgitate more in the early stages of infection. The cause of death in the one infected bird was due to hemorrhage into the abdominal cavity and possibly septicemia, both direct consequences of the parasitic infection. *Eustrongylides ignotus* perforate the stomach within 3 to 5 h following ingestion of infected fish (Spalding and Forrester 1993).

Although two of the three experimentally infected birds survived to eight days postinfection, they probably would not have survived in a natural situation. Numerous adhesions and active bacterial infection of the abdominal cavity were present at the time of euthanasia. The experimental birds had free and frequent access to food, unlike wild birds where food is delivered infrequently and competition among siblings can be intense. Siblicidal brood reduction is a common occurrence among ardeids; Mock et al. (1987) demonstrated that among Great Egrets brood reduction increased with decreasing food supply, not because fighting levels increased, but because the younger siblings were more likely to succumb to siblicidal abuse when undernourished. Our experimentally infected birds also were kept in a temperature-controlled environment, unlike a natural situation. Both of the surviving infected birds had accumulated much more fat than was observed among healthy nestlings of the same age examined in the wild.

Rank 3 and 4 Great Egret nestlings suffered greater mortality than their older siblings and even greater mortality when infected with *Eustrongylides*. The younger nestlings, already at a size disadvantage when competing for food and defending themselves against attacks from older siblings, are apparently also less able to resist the effects of parasitism. At EB, a similar effect was suggested for growth rates versus nestling rank, with growth rates decreasing with decreasing rank and a proportionately greater effect of eustrongylidosis among lower-ranked/younger nestlings. However, while the lowest-ranked nestlings at CS exhibited lower growth rates, the effect of eustrongylidosis generally was least pronounced among the lowest-ranked nestlings. Growth rates at CS were depressed relative to EB for both infected and uninfected birds, suggesting that factors other than eustrongylidosis were involved; perhaps the growth rates were already so low as to preclude further reduction due to parasitism among the slowest growing rank 3 nestlings. Conversely, the healthier, higher-ranked nestlings at EB may have been strong enough to resist the negative effects of parasitism.

While eustrongylidosis resulted in the expected decrease in 20-day mass for rank 1 and 3 infected nestlings, the effect on rank 2 nestlings was opposite at both colonies. Mass estimates are subject to large fluctuations in relation to time of feeding (i.e. some birds may have been fed before weighing, others after). The infection itself could lead to increased mass, either due to the presence of inflammatory tissue or intestinal obstruction. However, neither of these effects should act on rank 2 nestlings alone. Mock (pers. comm.) has suggested that rank 2 ardeid nestlings may experience overall higher energetic costs and, therefore, may be the most selfish and on average consume the most food among broods of three chicks to buffer their control of the rank 2 position (i.e. eat more to keep nestling 3 from getting it). While this tactic may not yield higher growth rates due to the energetic costs of competing with nestling 1, nestling 2 may often secure more food. This might increase the chances of obtaining high mass estimates as a result of nestlings having full stomachs when weighed, but the effect should be evident among uninfected nestlings as well. While no unequivocal trend was evident, rank 2 nestlings were heavier than rank 1 nestlings in 5 of 10 nests within which nestlings of both ranks were uninfected. If rank 2 nestlings do succeed in securing more food they might also acquire larger numbers of parasites which could lead to intestinal obstruction and more persistent increases in mass. However, if rank 2 nestlings are obtaining food that would normally go to the rank 3 nestling then they should also experience a higher prevalence of eustrongylidosis. Although not significant, the opposite trend was seen.

Our field results demonstrate that eustrongylidosis can have a significant impact on nestling survival and development. Epizootic events such as those documented by Weise et al. (1977), Roffe (1988), and Spalding et al. (1993) would go unnoticed without postmortem examination of carcasses. The infection and mortality levels observed at Lake Okeechobee during our study were relatively low and the impact of such parasitism might easily have been ignored by investigators not concerned about this disease. Unhealthy or dead nestlings might routinely have been categorized as those experiencing malnutrition or climatic stress. The potential impact of such infections on growth and sur-

vival statistics must be considered to accurately interpret results.

Spalding et al. (1993) sampled fish throughout southern and central Florida, including 997 fish from the Lake Okeechobee region during the same years as this study. None of the sites containing infected fish were within foraging range of the Lake Okeechobee colonies. In that study infected fish were found only at infrequent point sources rather than uniformly distributed at low densities. They further reported that all of those point sources had evidence of nutrient pollution; however, not all nutrient polluted sites had infected fish.

Urban development, livestock, and agricultural operations are common in the Okeechobee area and are all sources of nutrient pollution. Since prevalences were low among Okeechobee nestlings, it is likely that either persistent sources of infected fish were rarely visited by foraging adults (i.e. sites either featuring generally undesirable or inaccessible foraging habitats or far away from the colony), or that the sources were ephemeral wetlands and were only periodically exploited. Infected sites or higher concentrations of infected fish apparently were somewhat more available to birds from the CS colony than to those from EB.

Lake Okeechobee and the adjacent wetlands typically support 2,000 to 10,000 nesting pairs of up to nine species of aquatic-feeding ciconiiform wading birds each year, plus another 2,000 to 10,000 pairs of Cattle Egrets (Zaffke 1984; J.P.S. unpubl. data). Usually 6 to 10 mixed-species colonies form each year (which during our study always included the EB and CS colonies); 40 colony sites were used during this study. Lake Okeechobee, thus, is an important source for wading bird recruitment in Florida. The area is being developed rapidly, and sources of nutrient pollution are numerous. This population, therefore, would be extremely susceptible to epizootic mortality such as has occurred elsewhere in Florida. For this reason, the location and management of sources of infected fish is of great importance to the conservation of wading birds.

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