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Reduced disease in offspring: a benefit of coloniality in sunfish

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Summary. Increased disease and parasitism are a well-documented cost of group living for colonial birds and mammals, but we now show that disease in offspring of fish may be reduced by nesting in colonies. The aquatic fungus *Saprolegnia* sp., which is a common cause of egg mortality among freshwater fishes, is more prevalent in the nests of solitary than colonial male bluegill sunfish (*Lepomis macrochirus*). Moreover, fungal infection decreases with nest density in colonies. This may be due in part to a behavioural advantage since colonial males can devote less time to defending eggs and more time to fanning them, which reduces fungal infection. In addition, we demonstrate experimentally that solitary nests become infected at higher rates than colonial nests, even in the absence of parental males. This suggests that colonies are encountered by spores at a lower rate and/or that the large number of nests in colonies dilutes the number of fungal spores per nest. Through one or all of these mechanisms, egg mortality in colonial nests is lowered significantly. Therefore, in some cases, disease may select for group living.

Key words: Colony – Sunfish – Disease – *Saprolegnia*

Introduction

The risk of contracting infectious diseases and parasites is thought to be a major cost of group living (Alexander 1974; Freeland 1976). This idea holds true for many colonial birds and mammals (Hoogland and Sherman 1976; Hoogland 1979; Brown and Brown 1986; Møller 1987; Shields and Crook 1987; Rubenstein and Hohmann 1989). In these vertebrates, parasite load or mortality due to disease increases with breeding group size, and is generally higher than for solitary individuals.

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Colonial breeding occurs sporadically throughout the class Pisces and is particularly common among centrarchid sunfishes (Carlander 1977; Thresher 1984). Many sunfishes nest where aquatic fungi (Phycomycetes: Saprolegniaceae) cause important egg mortality, yet the effect of fungal infection and its relationship to group breeding have never been studied. In this paper, we document fungal infection in broods of bluegill sunfish (*Lepomis macrochirus*) in relation to nest dispersion, and report the surprising result that infection is more prevalent in the nests of solitary than colonial individuals.

Bluegill breed in freshwaters in eastern and central North America (Scott and Crossman 1979). In summer, males dig circular nests in colonies located in shallow water. Some males also nest solitarily, that is, more than 1.5 m from colonies, a distance that precludes social interaction with other males (Gross and MacMillan 1981). Gravid females visit nests to spawn and release thousands of 1-mm-diameter eggs that adhere to the nest bottom. Through multiple matings, nests can accumulate over 50,000 eggs (Carlander 1977). Females prefer to spawn in central nests in colonies, but appear indifferent to male phenotype (I.M. Côté et al. unpublished). Female choice for these nests is thought to result from the lower rate of predation on their eggs relative to peripheral sites (Gross and MacMillan 1981). Only parental males remain after spawning to care for the eggs and larvae in their nest.

The eggs require two mutually exclusive forms of care: guarding and fanning. In guarding, parental males must actively repel predatory fish from their nest. They do so by vigorous chasing and biting, often leaving the nest in order to drive the predator away. Predatory conspecifics and congeners nevertheless remove more than 10% of the brood (Gross and MacMillan 1981). Parental males are not a significant source of predation on each other's broods (Gross and MacMillan 1981; personal observation). During fanning, males generate water flow over the eggs with their pectoral fins, which promotes gas exchange necessary for eggs to develop. Males fan nearly continually during the 2–4-day egg stage (Coleman and

Fischer 1991). However, fanning may be interrupted for several minutes when parental males must chase egg predators. Parental bluegill also sometimes eat a few eggs in their nests, perhaps to remove diseased ones, but such behaviour is uncommon, probably because it is difficult for males to discriminate between healthy and dead eggs in their multi-layered egg mass.

After egg hatching, the larvae remain in the nest under the male's protection for an additional 2–5 days, after which both male and fry leave the nest. Three to six cycles of breeding, which include nest building, spawning, guarding, and abandonment of the colony, occur every season (Gross 1980; Claussen 1991).

During the egg stage, dead eggs are highly susceptible to infection by the water-borne spores of the fungus *Saprolegnia* sp., also known as water mould. Only dead eggs are colonized by zoospores, while both dead and living eggs are exploited by the vegetative phase (or hyphae) of the fungus (Smith et al. 1985). After a spore has initially infected a dead egg, the fungus spreads rapidly to neighbouring healthy eggs through hyphal growth. These eggs become covered with a highly visible crown of white hyphal filaments and do not hatch. Although the colonizing zoospores are flagellated, they are almost totally dependent on water movement. In the laboratory, under relatively still conditions, spores use chemotaxis to orient to a host within 2 cm, but their swimming ability is greatly reduced even in slow water flow (Smith et al. 1984, 1985). Thus, in the field, where water turbulence and currents are ubiquitous, fungal spores are unlikely to maintain directed movement over a long distance.

Fanning in fish and the development of fungus are inversely related: unfanned eggs become infected and die more quickly than fanned eggs (van Iersel 1953; Gross 1980; Zoran and Ward 1983). Thus, by keeping the eggs alive, parental fanning also reduces the number of sites (i.e. dead eggs) from which infection can spread in a nest. This occurs even if the water movement from fanning inadvertently increases the number of spores that come into contact with the eggs because live eggs are not infected directly by fungal spores as are dead eggs (Smith et al. 1985).

The objectives of this paper are twofold. First, we document the prevalence and intensity of fungal infection in nests of solitary and colonial bluegill in nature. Prevalence is defined as the proportion of infected nests per colony, and intensity as the proportion of infected eggs per nest (Margolis et al. 1982). Second, we examine some of the potential causes of the observed patterns of distribution of fungal infection. This is done by comparing solitary and colonial males in terms of phenotype, fertilization success, and parental care behaviour, and by using experimental nests to measure, under controlled conditions, rates of fungal infection in relation to nest dispersion.

Methods

Study population and data collection. We studied a natural bluegill population at Lake Opinicon, Ontario, Canada, between 14 June and 15 July, from 1988 to 1990. Each year, 3–4% of males on the study site nested solitarily. Data on fungal infection were obtained in 1989 and 1990 from 12 solitary nests and 456 nests in 22 colonies, ranging in size from 2 to 258 nests and in density from 0.65 to 2.92 nests/m^2. Similar numbers of solitary nests (1–3) were sampled in each of the three spawning bouts occurring each year. All nests occupied a common habitat, a 900-m^2 homogeneous sand bar at a depth of 1–1.5 m (Birch Bay bar, map in Gross and Nowell 1980). The nests of colonial males were separated on average by 5 ± 0.41 cm (range: 0–120 cm, $n = 1360$) from their nearest neighbours while this distance averaged 209 ± 18 cm (range: 150–510 cm, $n = 25$) for solitary males.

Data on male phenotype and mating success were collected for each colony and for most solitary males during the 3 years of the study, individual males being followed for a single season. On the day of spawning, a numbered tile was placed in each nest to facilitate identification. At the end of spawning, the number of eggs in each nest was estimated visually on a scale from 0 to 5 representing increasing numbers of eggs. This scoring technique correlates well with egg numbers obtained from actual nest counts ($r_s = 0.96$, $n = 32$; data from Claussen 1991). Egg scores of 1, 2, 3, 4, and 5 correspond on average to 1100, 14,000, 38,000, 63,000, and 110,000 eggs respectively (Claussen 1991).

Males were then captured with a dip net, weighed, measured and tagged with a uniquely numbered fine-filament anchor tag into the dorsal musculature (Floy Tagging Co., Seattle, Washington, USA). Two or three scales were removed to estimate age. In 1990, the presence of wounds and signs of fungal infection (e.g. white fuzzy epidermal areas) or parasites were also noted. Males were then released over their nests, which were covered with fine-mesh grids to prevent predation during handling. Total handling time was approximately 2–3 min. Males in all nest locations were handled in a similar manner, and resumed guarding within 5 min of release.

During the egg period (2–4 days after spawning, depending on water temperature), fungal infection was estimated for all broods (see below). In 1990, we also recorded the time devoted to fanning and guarding for 8 solitary males and 15 males from three colonies of similar densities and stage of egg development. Fanning was defined as a complete stroke of both pectoral fins over the eggs. Chasing was rapid swimming directly towards an intruding fish, leading the parental male beyond his nest boundary. The time spent in each activity during a single 10-min observation period was recorded for each male in the afternoon of the day he spawned. This sampling period, although short relative to the total egg development period, allows us to examine differences among males in fanning and guarding at a fixed development time.

At the end of the brood cycle, the inner diameter of all nests and the distances between nests, from edge to edge, were recorded. From these data, accurate colony maps were drawn, allowing the estimation of colony density (number of nests per m^2).

Fungal infection assessment. The intensity of fungal infection was assessed visually in all nests on a qualitative scale, at approximately the same stage of egg development for all males (i.e. 6–12 h before egg hatching). The four-point scale was: 0, little or no fungal infection; 1, less than one-third of the egg mass covered by fungus; 2: one- to two-thirds covered by fungus; and 3, more than two-thirds covered by fungus. The assessment was made at a distance of 20 cm from the egg mass by a snorkeller wearing a face mask.

The same stage of egg development was often reached on different days after spawning for different colonies, depending on water temperature. However, this variability in post-spawning time among colonies did not appear to influence the degree of fungal infection ($F_{3,18} = 1.02$, $P = 0.40$): eggs taking longer to hatch did not necessarily develop more infection.

To calibrate the scoring technique, glass dishes were set at the bottom of 24 randomly selected nests prior to spawning to collect a portion of the eggs spawned. The dishes remained in the nests to become infected naturally until 6–12 h before hatching, at which time the nests were scored for fungus and the dishes retrieved. The number of infected and uninfected eggs were counted under a microscope.

The field fungus scores correlated linearly with the actual proportion of infected eggs in the dishes from nests ($F_{1,2} = 149.03$, $n = 24$, $P < 0.001$). The data were analysed with a more-than-one-*Y*-per-*X* regression to avoid pseudo-replication: 3–9 nests were sampled for each fungus score (Sokal and Rohlf 1981, p. 477). The regression equation was $Y = 13.62 \times X + 7.47$, where Y is the arcsine-square-root transformed proportion of infected eggs, and X the fungus score. The microscopic evaluation revealed that all seven nests scored as 0 in the field had a few infected eggs (mean intensity: 3%, range: 0.1–5.3%).

Field measurements and manipulations. Two field studies were carried out to identify the cause(s) of the patterns of fungal infection among bluegill nests. We tested for differences in average fertilization success, and thus number of dead eggs, among nests. Open glass Petri dishes (10-cm diameter), placed during spawning into 11 nests (3 solitary, 8 colonial), were removed immediately after spawning and kept in an aquarium filled with flowing lake water. After 6–8 h, when gastrulation was visible, the numbers of fertilized and unfertilized eggs in each dish were counted.

Second, to examine the rate of infection of eggs in relation to nest dispersion, solitary nests and nests in low and high colony densities were simulated using artificial nests consisting of 10-cm-diameter glass dishes filled with unfertilized eggs. We used unfertilized rather than fertilized eggs because with the latter, the potential existed for post-fertilization developmental abnormalities which would have generated uncontrolled variation in the number of dead eggs per dish (i.e. the number of potential infection sites). The use of unfertilized eggs guaranteed constant egg mortality (100%), hence the observed rates of infection reflect the rate at which fungal spores came into contact with eggs.

Eggs were obtained from 16 ripe females (5 dishes/female; mean number of eggs/dish: 239 ± 107, range: 48–524). The 80 dishes were assigned randomly to solitary (1 nest; $n = 5$ replicates), low-density (5 nests; 4 dishes/m^2; $n = 3$ replicates), or high-density treatments (20 nests; 6.5 dishes/m^2; $n = 3$ replicates). The dishes were covered with mesh to prevent predation and placed on the lake bottom in the study site, alternating treatments. They were retrieved after 20 h to count infected eggs. This period is slightly shorter than the shortest incubation period for bluegill eggs (32 h), but this was necessary since dead eggs are infected more rapidly, and a 100% intensity of infection would not be informative.

Statistical analysis. Several variables required transformation to meet the assumptions of parametric statistics. All proportions were angularly transformed, and phenotypic and behavioural data were log-transformed prior to analysis.

Mean fungus scores were obtained for each colony by summing raw fungus scores and dividing by the number of males present. Since the data did not differ statistically among years ($Z = 0.35$, $n = 34$, $P = 0.73$), they were combined for an overall analysis. All colonies were considered to be independent points since colony location and composition varied across years. Non-parametric Mann-Whitney *U*-tests were performed on the untransformed mean scores. The comparisons were re-analysed with randomized *t*-tests (5,000 permutations; Manly 1991), with probability values modified with sequential Bonferroni adjustments (Rice 1989). Since both results were similar, only non-parametric statistics are reported.

Because egg number is known to affect the intensity of parental care (Coleman et al. 1985), we controlled for this effect in the analysis of time budgets. Both time spent fanning eggs and time spent chasing predators increased with the number of eggs in a nest (log fanning time: $r = 0.44$, $n = 23$, $P = 0.04$; log chasing time: $r = 0.45$, $n = 23$, $P = 0.03$). The effect of egg number was therefore removed by obtaining the residuals from the regressions of (log) fanning time versus egg number and (log) chasing time versus egg number. These residuals were then used in subsequent analyses.

Data from the experiment investigating the rates of infection in relation to artificial nest dispersion were analysed with a nested analysis of variance, with replicates of each density treatment nested within density. Because there was a significant relationship between egg number in a dish and the (angularly transformed) proportion of eggs infected ($r = 0.37$, $n = 80$, $P = 0.001$), egg number per dish was included as a covariate in the analysis.

Results

Phenotypic variation among males

Males were significantly longer, heavier, and older in 1990 than in 1989 (*t*-tests, $P < 0.05$ for all traits). Differences in male phenotype among nest positions were thus analysed separately for each year. There were no significant differences in male length, weight, or age among spawning bouts in each year (ANOVAs, $P > 0.05$ for both years). All males were therefore combined within years.

In 1989, solitary males were significantly heavier, although not longer or older, than colonial males (Table 1). This difference was not present in 1990 (Table 1).

Few males showed evidence of fungal infection or body wounds when examined in 1990 (15/272, or 5.5%). In addition, the presence of fungus on parental males did not appear to influence infection of their brood. In three colonies for which the comparison was possible, the intensity of fungus in nests of fungus-infected fish (total $n = 9$) was similar to that of uninfected fish (total $n = 75$; Mann-Whitney *U*-tests, $P > 0.10$ for each colony).

Fungal infection in solitary and colonial nests

All bluegill nests were infected with fungus. However, the intensity of infection was highly variable, ranging from less than 1% to 73% of the eggs in a nest. Infection intensity was significantly higher in solitary than in colonial nests (Table 2): solitary nests averaged twice the proportion of infected eggs of colonial males. In both years, the intensity of infection was similar in central and pe-

Table 1. Male phenotype in relation to nest position

	Solitary	Colonial	*t*	*P*
1989				
Length	191.0 ± 14.3 (8)	185.7 ± 8.1 (266)	1.67	0.10
Weight	135.6 ± 45.9 (8)	108.2 ± 17.4 (266)	3.29	0.001
Age	7.9 ± 1.2 (7)	7.9 ± 0.9 (237)	0.16	0.87
1990				
Length	190.8 ± 6.5 (9)	189.7 ± 8.1 (278)	0.43	0.67
Weight	119.7 ± 16.2 (9)	117.7 ± 16.9 (278)	0.39	0.69
Age	8.5 ± 0.8 (8)	8.3 ± 1.0 (235)	0.53	0.60

Yearly means are presented ±1 SD with sample sizes in parenthese (see next table). Total length is expressed in mm, weight in g, and age in years. *t* and *P* values are for log-transformed data

Table 2. The proportion of eggs infected by fungus in nests of solitary and colonial bluegill

Year	% Infected eggs		Z	P
	Solitary	Colonial		
1989	40.0±3.2 (6)	17.4±2.7 (11)	3.22	0.001
1990	36.3±4.0 (6)	21.1±3.3 (11)	2.07	0.04
Combined	38.1±3.0 (12)	19.0±2.6 (22)	3.88	<0.001

Yearly and overall means for solitary and colonial nests are presented ±1 SE with sample sizes in parentheses. Sample sizes for colonial males represent the number of colonies sampled (total number of males sampled = 456). Means were converted to real proportions using the regression equation given in the Methods, and analysed with Mann-Whitney *U*-tests

ripheral colonial nests (Mann-Whitney *U*-tests, $P > 0.05$ in both cases).

The difference in infection intensity between solitary and colonial males was not due to initial differences in egg mortality owing to variation in fertilization success. Solitary males fertilized on average 94.4% ± 4.8% (SD) ($n = 3$) of eggs released in their nests, while colonial males fertilized 87.4% ± 12.6% (SD) of their eggs ($t = 0.70, n = 8, P = 0.50$, after angular transformation). Solitary and colonial males obtained similar average numbers of eggs over 3 years (overall mean egg score, solitary: 1.85±0.5 ($n = 26$), colonial: 1.67±0.1 ($n = 1358$); Mann-Whitney *U*-tests, $P > 0.05$ for each bout); thus, the number of infection sites (i.e. unfertilized eggs) would be similar in their nests.

However, there were behavioural differences between solitary and colonial males which could account for the pattern of infection observed. Solitary males spent significantly more time chasing predators and less time fanning their eggs than colonial males (Table 3). Moreover, among colonial males, there was a negative relationship between the time spent fanning eggs and the time spent defending eggs against predators (Fig. 1). Thus, within colonial males, those which chase predators more fan their eggs less often. This trade-off is extreme in solitary males, which spend the highest amount of time chasing egg predators.

Table 3. Time spent fanning eggs and time spent chasing potential predators from the nest by solitary and colonial bluegill sunfish

	Solitary ($n=9$)	Colonial ($n=15$)	t	P
Time spent fanning eggs (s/10 min)	202.8±67.1	274 ±43.7	2.44	0.023
Time spent chasing (s/10 min)	72.8±28.2	16.3±15.4	3.92	0.001

Sample sizes are given in parentheses. *t* and *P* values are for log-transformed data, corrected for egg number (see Methods)

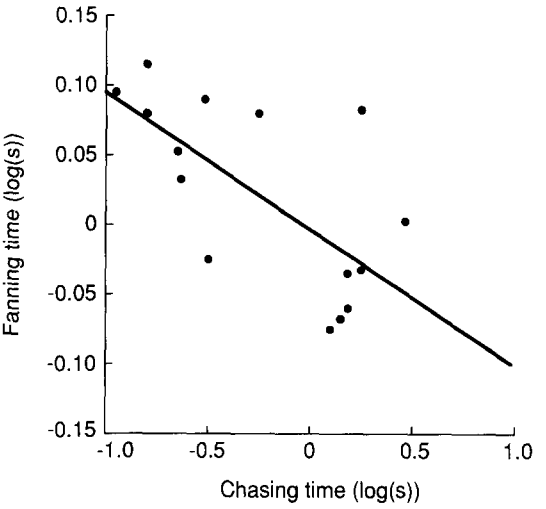

Fig. 1. The total amount of time spent fanning eggs as a function of the amount of time spent chasing egg predators, primarily other fish, from the nest by colonial male bluegill. The regression equation is: $F = -0.096 \times C - 2.89 \times 10^{-17}$, where F is log time spent fanning, adjusted for egg number, and C is log time spent chasing, adjusted for egg number ($r = 0.66, n = 15, P = 0.008$)

Effect of nest density on fungal infection

In addition to differences in fungal infection between solitary and colonial males, the mean intensity of infection decreased significantly with increasing colony density ($r_s = -0.55$, $n = 22$ colonies, $P = 0.012$). Although colony size and density were correlated ($r = 0.47$, $n = 22$, $P = 0.03$), the mean intensity of infection was not correlated with colony size ($r_s = 0.20$, $n = 22$, $P = 0.36$).

Nests were smaller in denser colonies ($r = 0.71$, $n = 22, P < 0.001$). A smaller nest area available for spore colonization could thus explain the lower rate of infection in high-density colonies. However, the mean intensity of fungal infection was not related to nest diameter ($r_s = 0.29, n = 22, P = 0.18$).

The pattern of infection in experimental colonies with artificial nests mirrored that of natural colonies. Significantly more eggs became infected in the "solitary nests" than in "high-density colonies", while "low-density colonies" showed intermediate intensity of fungal infec-

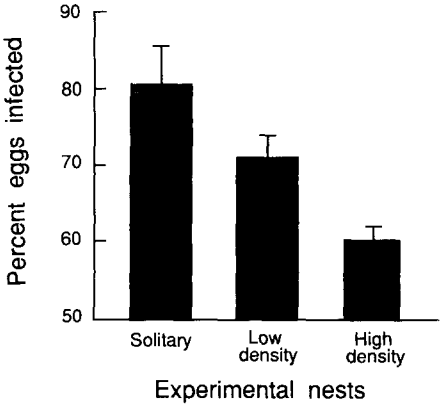

Fig. 2. The average proportion of bluegill eggs infected by fungus in experimental solitary nests and colonies of low and high densities. Mean proportions of eggs infected are presented with SE bars

tion (Fig. 2; nested ANOVA, density: $F_{2,69} = 7.09$, $P = 0.02$; replicates within density: $F_{8,69} = 1.68$, $P = 0.12$; covariate (egg number per dish): $F_{1,69} = 21.13$, $P < 0.001$). Lower fungal infection in colonies than in solitary nests may therefore be achieved without differences in male parental behaviour.

Fungal transmission among and within nests

Finally, we investigated fungal transmission among and within nests. Transmission among nests did not seem to occur: fungus scores of individual males were unrelated to the fungus score of their neighbours in colonies ($r_s = 0.01$, $n = 270$, $P = 0.90$), and nests with high fungus scores appeared randomly scattered throughout each colony. However, transmission occurred within nests, and appeared to be directly related to egg density in the nest. Egg number and fungus score were positively related ($r_s = 0.20$, $n = 270$, $P = 0.002$). In the artificial nests, the proportion of eggs infected was also directly related to the number of eggs present (see above).

Discussion

The adaptive significance of colonial breeding in fish has previously been related to decreases in predation on offspring (Loiselle 1977; Dominey 1981; Gross and MacMillan 1981; Foster 1989). Coloniality may also have evolved in some species because unattractive males gain matings by associating with attractive individuals (Bietz 1980; Dupuis and Keenleyside 1989; Jennings and Philipp 1992). Previous studies did not measure the effects of diseases, but our study shows that a reduction in fungal infection in offspring may be an important selective pressure favouring colonial nesting. Colonial nests, especially nests at high density, suffer less fungal infection than solitary or nests at lower density. Disease must therefore be considered in the cost-benefit equation for the evolution of coloniality in fishes.

In order for any disease to favour group living, a clumped dispersion must not lead to increased transmission of the pathogen. This condition appears to be satisfied in sunfish colonies: the fungus score in any one nest was uncorrelated with the scores in neighbouring nests. Transmission among nests is apparently hampered by the inability of fungal hyphae to extend across the sand between nests. In contrast, transmission within nests is enhanced by high egg density. This was true in both artificial and natural nests. Fungal infection is thus affected in opposite ways by egg density in a nest, and by nest density in a colony, but only the latter is relevant to coloniality.

Although fungus can spread to healthy eggs once established in the nest, the initial target for fungal infection is dead eggs (Smith et al. 1985). These can originate from fertilization failure, but we found no evidence for differing rates of fertilization between solitary and colonial males. Moreover, the difference in fungal infection between solitary and colonial nests is not rooted in phenotypic differences among males which would affect the efficiency of parental care. Solitary males were heavier than colonial males in one year of the study, but no difference was observed the following year, while discrepancies in fungal infection remained. In addition, in the artificial nests where males were absent, differences in fungal infection between solitary and grouped nests were similar to those found under natural conditions with males present. This suggests that phenotypic differences among males are not sufficient to explain the observed pattern of fungal distribution.

The intensity of fungal infection not only varied between solitary and colonial males, it also decreased as colony density increased. Two non-mutually-exclusive mechanisms may act to explain these observations: (1) a parental care trade-off, and (2) a statistical abatement effect.

Solitary males spent more time chasing predators from their nest, at the expense of fanning. This apparently occurs because nesting close to neighbours increases the overlap in defence zones around nests, thus reducing the amount of exclusive area an individual male has to defend (Gross and MacMillan 1981). Colonial males can thus decrease investment in nest defence by sharing it with neighbours, and can increase fanning time which leads to better egg survivorship and reduced fungal infection.

This behavioural trade-off may be amplified at high nest densities. The frequency of nest defence behaviour is known to decrease with increasing colony density (Gross and MacMillan 1981), thus making more time available for fanning eggs in denser colonies, leading to lower fungal infection.

The advantage of decreased infection in colonies may also come about by two statistical means, similar to those invoked for predation benefits of group living (Turner and Pitcher 1986). An "avoidance" effect may occur whereby nests in a colony are less likely to be encountered by disease agents than a similar number of nests scattered throughout the habitat (e.g. Treisman 1975; Rasmussen and Downing 1988). A "dilution" effect may operate when the likelihood of a nest becoming infected after being encountered decreases with group size/density (e.g. Hamilton 1971; Gross and MacMillan 1981; Morgan and Godin 1985; Poulin and FitzGerald 1989). Turner and Pitcher (1986) pointed out that both effects (=attack abatement) must operate to result in an overall fitness advantage of group living. This has been verified in recent empirical studies (e.g. Arnqvist and Byström 1991; Wrona and Dixon 1991).

Are these statistical mechanisms acting to reduce fungal parasitism in colonies, particularly those with high nest density, relative to solitary nests? Fungal spores are probably largely random in their search for eggs, given their reliance on water currents for long-distance movements. But this does not preclude an avoidance effect. There are empirically documented cases of prey aggregation reducing the rate of encounter of random-search predators (e.g. Folt 1987; Rasmussen and Downing 1988). A dilution effect may also occur because the number of spores in the water column must be limited, and as the number of nests per square metre increases, the number of potential spores per nest must decrease. Infection probability can thus potentially be reduced in denser colonies.

Thus, in the present system, avoidance and dilution effects could result in a negative relationship between

intensity of infection and colony density. While our experiment with artificial nests showed that the natural pattern of fungal infection was not solely the result of differences in male parental behaviour, it can unfortunately not differentiate between these two statistical processes, nor determine whether both are operating. Further experiments will be required to resolve these questions, and to determine the relative importance of statistical effects and parental care differences in the patterns discovered.

Finally, our finding of a reduction in egg disease in colonies has implications beyond issues of male dispersion. For example, female mate choice should be influenced by the differential levels of disease-induced egg mortality at solitary and colonial nests. However, our mating success data suggest that females do not discriminate against solitary males. Clearly, other factors unrelated to fungal infection must impinge on female choice decisions. Similarly, since high egg density in nests appears to favour rapid spread of fungal infection, males may be selected to limit the number of eggs in their nests and thus become more selective of their mates.

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