Proc. Natl. Acad. Sci. USA
Vol. 92, pp. 1192–1194, February 1995
Evolution

Paternal investment affects prevalence of malaria

(reproductive investment/parasite/life history/great tit)

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Communicated by Gordon H. Orians, University of Washington, Seattle, WA, November 15, 1994 (received for review September 7, 1994)

ABSTRACT **Both reproduction and parasite defense can be costly, and an animal may face a trade-off between investing in offspring or in parasite defense. In contrast to the findings from nonexperimental studies that the poorly reproducing individuals are often the ones with high parasite loads, this life-history view predicts that individuals with high reproductive investment will show high parasite prevalence. Here we provide an experimental confirmation of a positive association between parental investment levels of male great tits *Parus major* and the prevalence of *Plasmodium* spp, a hematozoa causing malaria in various bird species. We manipulated brood size, measured feeding effort of both males and females, and assessed the prevalence of the hemoparasite from blood smears. In enlarged broods the males, but not the females, showed significantly higher rates of food provisioning to the chicks, and the rate of malarial infection was found to be more than double in male, but not female, parents of enlarged broods. The findings show that there may be a trade-off between reproductive effort and parasite defense of the host and also suggest a mechanism for the well documented trade-off between current reproductive effort and parental survival.**

What is the cost of working hard for one's offspring? For birds and mammals it has been shown that a high parental investment in the current offspring can affect survival of the parents (1–4), their future fecundity (5), or both (6). Both reproduction and parasite defense are costly (7–10), and a fitness reduction through reduced survival or fecundity may arise if there is a coupling between investment levels and parasite prevalence in a host—that is, if increased levels of investment render hosts more susceptible to parasites or increase their exposure to the parasite vector. For an experimental test of this hypothesis we manipulated parental effort of great tits and assessed the prevalence of a common hemoparasite, *Plasmodium* spp., in our host population. Because the sexes can differ in several ways that could affect their parasite load (11), including their readiness to work harder for a larger brood, their exposure to the parasites or to the vectors (12), or their endocrine types and endocrine levels that may interact with immunocompetence (13–16), we assessed both maternal and paternal efforts for all manipulated broods and recorded the prevalence of the hemoparasite in male and female parents.

METHODS

In our study population of great tits (*Parus major*) near Lausanne, Switzerland, we manipulated, in spring 1993, the brood size of great tits on the day of hatching by removing two chicks from a brood and adding them to a brood that hatched within a few hours of the first brood. When an odd number of broods hatched the same day, these extra broods were left unmanipulated and were monitored for comparison with the reduced and enlarged broods. By this exchange of two chicks between broods we created enlarged broods that held significantly more chicks than the reduced broods (9.5 ± 1.6 SD versus 5.6 ± 2.2 SD chicks; $t_{23,22} = 6.1$, $P < 0.001$).

At the peak energy demand of the brood, which occurs 13 days after hatching (17), we measured the rate at which males and females provided food to the nestlings. Recordings were made by using infrared cameras mounted in nest boxes. Each nest box was filmed for 3 hr between 9 a.m. and 4 p.m. After the recordings we captured both parents. Time of capture was randomized with respect to the experimental groups. A blood sample was taken from the brachial vein to assess malarial hemoparasite infection from a blood smear. The slides were air-dried, fixed with absolute methanol, and then stained with Giemsa stain. Each slide was examined for 10 min using oil immersion (×1000).

We further assessed natural prevalence of parasites without manipulation of brood size and return rates to the breeding area of unmanipulated birds with respect to their infection status. A sample of 108 great tit broods captured in 1992 showed that *Plasmodium* spp. prevalence differed significantly between male (36% of males naturally infested) and female (19% infested) parents (logarithm likelihood ratio test, $G = 4.2$, df = 1, $P = 0.04$). A part of this area holding the 108 pairs was monitored again in 1993. This sample was used to assess the return rate of breeding males with respect to their status of infection in 1992. The return rate of birds in this unmanipulated sample is therefore not confounded with other effects that could arise from brood-size manipulation.

A small sample of birds was also filmed at dawn and at dusk to determine the timing of the first and the last feeding trip of a day. For all filming the cameras were started and stopped automatically by an electronic timer so as not to disturb the birds at the nest box, as could occur were cameras manually operated.

RESULTS

Feeding Rates. Males of enlarged broods fed their chicks at a rate ≈50% higher than males of reduced and unmanipulated broods (Fig. 1). The feeding rates of males of reduced broods were not significantly different from the rates of unmanipulated broods (Fisher's least-significant-difference test, $P = 0.83$), but males in enlarged broods fed significantly more than males in unmanipulated ($P = 0.03$) or reduced ($P = 0.008$) broods. In contrast, the females' rate of feeding visits to nest boxes was not significantly higher (all P values > 0.2) in enlarged broods than in reduced or unmanipulated broods.

Prevalence of *Plasmodium*. Given the fact that only males adjusted the feeding rate to the increased demand, we predict a higher prevalence of hematozoa for males of enlarged broods than for males of reduced and unmanipulated broods but predicted no effect on females. The experimental manipulation of brood size was significantly associated with the occurrence of *Plasmodium* spp. in male parents (Fig. 2) but not in females. In reduced and unmanipulated broods prevalence of

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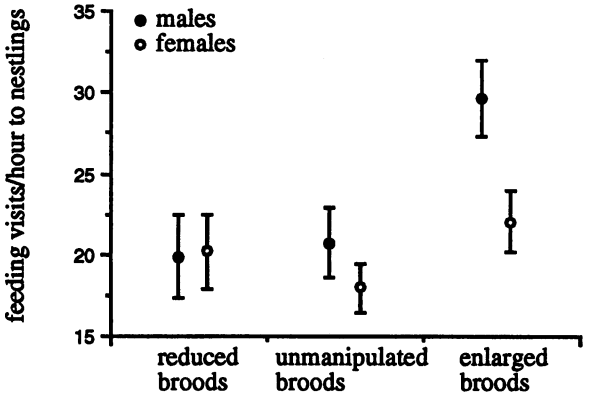

FIG. 1. Number of feeding trips to the nest box per hour (± SE) by male (●) and female (○) parents of reduced (n = 13), unmanipulated (n = 8), and enlarged (n = 13) broods.

Plasmodium in males was 38% and 32%, respectively, whereas in enlarged broods prevalence of *Plasmodium* in males reached 76% (χ^2 = 9.9, df = 2, P = 0.009, n = 61). None of the females in the reduced group, and two females each in the unmanipulated and the enlarged group showed an infestation with *Plasmodium* (χ^2 = 2.3, df = 2, P = 0.31, n = 57).

Return of Breeders. From an unmanipulated sample of great tits we recorded the return rates of adult breeders with known state of malarial infection in spring 1992 as breeders in 1993. Seventeen of the 30 noninfected males (57%) in 1992 returned to the breeding site in 1993, but only 2 of the 14 males (14%) infected with *Plasmodium* in 1992 returned in 1993 (χ^2 = 7.0, df = 1, P = 0.008, n = 44).

DISCUSSION

The findings support the hypothesis that work load affects the prevalence of hematozoan parasites in their hosts. How does higher parasite prevalence in males of enlarged broods arise? If all birds are uninfected at the start of the cycle, the males that work harder may contract the infection through more frequent exposure to *Plasmodium* vectors, perhaps combined with a reduction in their immune response. Alternatively, if birds have chronic but latent infection at the beginning of the reproductive cycle, a reduction in the immune response may lead to a relapse due to *Plasmodium*. Higher exposure to the vectors may occur if the harder working birds are less vigilant for the vectors of *Plasmodium* or if longer feeding hours increase the time that they are available to the vectors. Males of enlarged broods start feeding their chicks slightly, but not significantly ($t_{6,8}$ = 1.8, P = 0.09), earlier in the morning than the males of reduced broods. There is no difference in the timing of the last feeding in the evening between the males of the two groups, and there is no difference in the timing of the first and last feedings of females in reduced and enlarged broods (all P values > 0.2). We therefore cannot discriminate between the possibilities that higher prevalence of malaria in harder working males of the enlarged broods arises as a consequence of increased susceptibility to hematozoan parasites or increased exposure to the vectors or both.

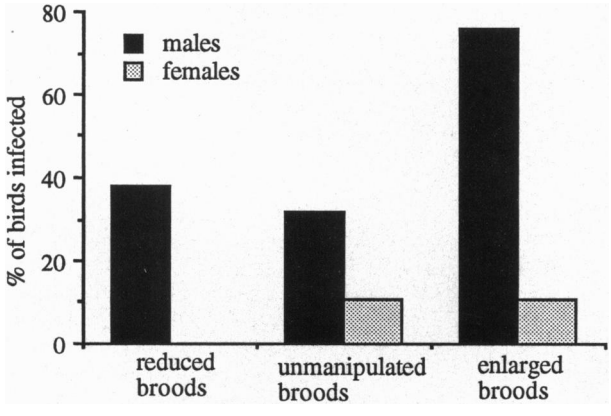

FIG. 2. Prevalence of *Plasmodium* spp. in blood smears of parents rearing reduced (21 males/20 females), unmanipulated (19 males/18 females), and enlarged (21 males/19 females) broods. Brood size significantly affected the prevalence of *Plasmodium* spp. in males.

Plasmodium is common in many bird species (18), and its pathogenic effects include hemolytic anemia, a lower metabolic rate, poorer thermoregulation (19), and reduced survival and fecundity (20, 21). If a large clutch compromises a male's survival through higher parasite susceptibility or exposure, the males should then prefer to care for a brood below the size where there is a high risk of primary infection or a relapse due to the hematozoan parasite. The female, on the other hand, because she does not increase investment, may gain by laying a clutch larger than the male's optimum. The death of her mate after reproduction may have little cost if she can easily find another mate. Why should males but not females of enlarged clutches work harder? From a life-history point of view (9, 22, 23) males would be expected to work harder than females if the current brood is of higher value than future broods for a male, but for a female the future broods are worth more than the current brood. This result may occur, for example, in populations with a male-biased operational sex ratio (e.g., refs. 24 and 25), where the males have a higher yearly variance in offspring production than the females. This situation would also be expected in pairs where the male is of lower-than-average quality and has a lower chance to reproduce in the future than high-quality males.

Observational studies have often found a negative association between reproductive success and prevalence of parasites—that is, the poorly reproducing individuals are the ones carrying a high parasite load (26–29). This result may arise when phenotypes differ in quality. Both high parasite prevalence and low reproductive success may then be seen in low-quality phenotypes, and the opposite would be observed in high-quality phenotypes, leading to the negative correlation. Our experiment with subtle manipulations of reproductive effort within phenotypes shows that there is a positive association between reproductive effort and parasite prevalence, as predicted by life-history theory. Our work shows that, for a host, there may be a trade-off between investment in reproduction and investment in parasite defense and also suggests that this trade-off is dissimilar for males and females. These results further suggest a mechanism for the expected and often observed trade-off (30) between reproduction and survival.

We thank V. Apanius, J. Blondel, L. Keller, K. Lessels, M. Milinski, K. Norris, A. Read, C. Wedekind, and two anonymous referees for their comments. The work is supported by the Swiss National Science Foundation, Grants 31-26606.89 (H.R.) and 31-34020.92 (H.R.).

1. Askenmo, C. (1979) *Am. Nat.* **114,** 748–755.
2. Linden, M. (1988) *Oikos* **51,** 285–290.
3. Dijkstra, C., Bult, A., Bijlsma, S., Daan, S., Meijer, T. & Zijlstra, M. (1990) *J. Anim. Ecol.* **59,** 269–286.
4. Møller, A. P. (1993) *J. Anim. Ecol.* **62,** 309–322.
5. Gustafsson, L. & Sutherland, W. J. (1988) *Nature (London)* **335,** 813–815.
6. Clutton-Brock, T. H., Guiness, F. E. & Albon, S. D. (1982) *Red Deer: Behaviour and Ecology of Two Sexes* (Univ. of Chicago Press, Chicago).
7. Linden, M. & Møller, A. P. (1989) *Trends Ecol. Evol.* **4,** 367–371.
8. Drent, R. H. & Daan, S. (1981) *Ardea* **68,** 225–252.
9. Lessels, C. M. (1991) in *Behavioural Ecology: An Evolutionary Approach*, eds. Krebs, J. R. & Davies, N. B. (Blackwell, Oxford), pp. 32–68.

10. Keymer, A. E. & Read, A. F. (1991) in *Parasite-Host Associations: Coexistence or Conflict*, eds. Toft, C. A., Aeschlimann, A. & Bolis, L. (Oxford Univ. Press, New York), pp. 37–61.
11. Zuk, M. (1990) *Parasitol. Today* **6,** 231–233.
12. Bundi, D. A. P. (1988) *Parasitol. Today* **4,** 186–189.
13. Grossman, C. J. & Roselle, G. A. (1986) *Prog. Clin. Biochem. Med.* **4,** 9–56.
14. Alexander, J. & Stimson, W. H. (1988) *Parasitol. Today* **4,** 189–193.
15. Grossman, C. J. (1988) *Immunol. Allergy Pract.* **10,** 104–112.
16. Folstad, I. & Karter, A. J. (1992) *Am. Nat.* **139,** 603–622.
17. VanBalen, J. H. (1973) *Ardea* **61,** 1–93.
18. Seed, T. M. & Manwell, R. D. (1977) in *Parasitic Protozoa*, ed. Kreier, J. P. (Academic, New York), pp. 311–357.
19. Hayworth, A. M., Van Riper, C., III, & Weathers, W. W. (1987) *J. Parasitol.* **73,** 850–853.
20. Van Riper, C., III, Van Riper, S. G., Goff, M. L. & Laird, M. (1986) *Ecol. Monogr.* **56,** 327–344.
21. Atkinson, C. T. & Van Riper, C. (1991) in *Bird-Parasite Interactions*, eds. Loye, J. E. & Zuk, M. (Oxford Univ. Press, Oxford), pp. 19–48.
22. Williams, G. C. (1966) *Am. Nat.* **100,** 687–690.
23. Stearns, S. C. (1992) *The Evolution of Life Histories* (Oxford Univ. Press, New York).
24. Bulmer, M. G. & Perrins, C. M. (1973) *Ibis* **115,** 277–281.
25. Perrins, C. M. (1979) *British Tits* (Collins, London).
26. Møller, A. P., Allander, K. & Dufva, R. (1990) in *Population Biology of Passerine Birds*, eds. Blondel, J., Gosler, A., Lebreton, J. D. & McCleery, R. H. (Springer, Berlin), pp. 269–280.
27. Møller, A. P. (1990) *Ecology* **71,** 2345–2357.
28. Richner, H., Oppliger, A. & Christe, P. (1993) *J. Anim. Ecol.* **62,** 703–710.
29. Loye, J. E. & Zuk, M. (1991) *Bird-Parasite Interactions* (Oxford Univ. Press, Oxford).
30. Charnov, E. L. & Krebs, J. R. (1974) *Ibis* **116,** 217–219.