Proc. Natl. Acad. Sci. USA
Vol. 93, pp. 2229–2233, March 1996
Evolution

The contagion indicator hypothesis for parasite-mediated sexual selection

(mate choice/female choice/associatively transmittable parasite/transmittability/direct benefits)

DAVID J. ABLE

Section of Neurobiology and Behavior, Cornell University, Ithaca, NY 14853

Communicated by George C. Williams, State University of New York, Stony Brook, NY, September 25, 1995

ABSTRACT **Hamilton and Zuk [Hamilton, W. D. & Zuk, M. (1982) *Science* 218, 384–387] proposed that females choosing mates based on the degree of expression of male characters obtain heritable parasite resistance for their offspring. Alternatively, the "contagion indicator" hypothesis posits that females choose mates based on the degree of expression of male characters because the latter indicate a male's degree of infestation of parasites and thus the risk that choosing females and their offspring will acquire these parasites. I examined whether parasite transmittability affects the probability that parasite intensity and male mating success are negatively correlated in intraspecific studies of parasite-mediated sexual selection. When females risk infection of themselves or their future offspring as a result of mating with a parasitized male, negative relationships between parasite intensity and male mating success are significantly more likely to occur than when females do not risk such infection. The direct benefit to females of avoiding parasitic infection is proposed to lead to the linkage between variable secondary sexual characters and the intensity of transmittable parasites. The direct benefits of avoiding associatively transmittable parasites should be considered in future studies of parasite-mediated sexual selection.**

The existence of conspicuous male displays in some animal species is puzzling because such traits are apparently detrimental to survival. Darwin (1) recognized that conspicuous male displays such as the antlers of elk and the trains of peacocks could evolve if they increased the mating success of their bearer, relative to others, despite any reduction in survival such traits caused. Under Darwin's theory of sexual selection, individuals could increase their mating success through two processes: intrasexual competition and intersexual choice. In intrasexual competition, the benefit of assessing a rival (usually another male) by his display is avoiding a fight with a superior adversary. In intersexual choice, the benefit to the chooser (usually female) of assessing a potential mate by his display is much less clear despite a great deal of theoretical and empirical research and remains a central problem in behavioral ecology (2).

Hamilton and Zuk (3) proposed a model of sexual selection in which females choose mates on the basis of variable male traits in which the degree of expression indicates heritable parasite resistance. Hamilton and Zuk's hypothesis predicts that, within species, parasite intensity will be inversely correlated with male mating success and with the degree of expression of male characters. A number of studies have borne out these predictions. However, the generality of the Hamilton and Zuk model of parasite-mediated sexual selection is questioned because it fails to explain the results of an approximately equal number of other studies in which no relationship between parasite intensity and male mating success was found (4–7).

A confounding variable may account for the failure of the Hamilton and Zuk model to explain the outcome of some studies. Where parasites are transmittable between mates, it is possible that females choose unparasitized males not because females accrue good genes for parasite resistance for their young, but instead because females choosing unparasitized males obtain the direct benefit of avoiding parasitic infection.

Borgia (8) recognized that avoiding parasitic infection is a potential direct benefit to female mate choice and proposed that selection by female choice has favored male traits that allow females to see ectoparasites. In Borgia's "parasite avoidance" model, the parasite does not affect the production of the male trait—the male trait serves only to make parasites more visible. Thus, the degree of development of the male trait does not vary between males (9), in contrast to the Hamilton and Zuk hypothesis in which male traits are variable. The parasite avoidance hypothesis finds support in male satin bowerbirds, in which females might detect whitish lice on dark male plumage, but in which there is no variation in the degree of development of male plumage. However, variability is a hallmark of sexually selected traits (10). This hypothesis lacks generality as a model of parasite-mediated sexual selection because it does not apply to variable traits.

Neither the Hamilton and Zuk hypothesis nor the parasite avoidance hypothesis is sufficiently general to explain variation in results of studies of parasite-mediated sexual selection. When parasites affect the degree of expression of male traits, examining whether a parasite is transmittable in the context of mate choice can explain much of the variation in results of intraspecific tests of parasite-mediated sexual selection.

The Contagion Indicator Hypothesis. I propose that the degree of expression of variable male traits indicates a male's intensity of "associatively transmittable" parasites, and thus the risk a choosing female and her offspring will acquire these parasites. An associatively transmittable parasite is one for which a female, due to courtship, mating, or any postmating association with a male or his territory, risks parasitic infection of herself and/or her present and future offspring with parasites from that male. In the contagion indicator hypothesis, females choosing unparasitized or less-parasitized males receive the direct benefit of avoiding or reducing parasite transmission to themselves and/or their offspring, but only when the parasite is associatively transmittable.

The contagion indicator hypothesis predicts that the intensity of associatively transmittable parasites will be negatively correlated with male mating success and with the degree of development of male characters. Nonassociatively transmittable parasites are predicted to have no effect on male mating success or on variable male characters. In contrast, while the Hamilton and Zuk hypothesis also predicts that parasite intensity is negatively correlated with male mating success and the degree of development of male characters, it makes no distinction between associatively transmittable and nonassociatively transmittable parasites. The parasite avoidance hypothesis makes the same prediction as the contagion indicator hypothesis with respect to male mating success, but it predicts no correlation between parasite intensity and variation in male traits.

Determining whether a parasite is associatively transmittable requires knowledge of the biology and behavior of the host, parasite, and any vector. This section provides the general

conceptual approach for determining whether a particular parasite is associatively transmittable. This conceptual approach is only a guide for use in determining associative transmittability. In most cases, including those on which the conclusions in this paper are based, transmittability in the context of mate choice has yet to be determined experimentally.

Parasites Transmitted by Living Vectors. Vectors may transfer parasites biologically, mechanically, by phoresis, or through ingestion by the host. Parasites that, after ingestion by a vector, undergo a process of multiplication and/or development in the vector before they are infective to the host are said to be biologically transmitted. The resulting delay is critical to the determination of transmittability because this interval to infectivity must be compared to the duration of the association between the male and the vector, and the female and the vector, to determine whether females risk infection. If the vector moves immediately to another host, and if the interval to infectivity is shorter than the duration of a vector's association with a male, the female and/or her offspring risk infection by associating with an infected male. If the interval to infectivity exceeds the duration of the male/vector association, she does not risk infection by parasites from that male unless the vector remains associated with her long enough for the parasite to become infective. This relationship between the interval to infectivity and the duration of association between male and vector also holds for parasites transmitted through ingestion of intermediate hosts.

In mechanically transmittable parasites and those transmitted by phoresis, no development in the vector occurs, so there is no interval to infectivity. If a tissue-feeding vector infected with a mechanically transmittable parasite feeds on an infected male and then feeds on his mate, she can be infected immediately. Whether such a parasite is associatively transmittable is then determined by the likelihood that the vector transfers between the members of a mated host pair.

Parasites Not Transmitted by Living Vectors. Parasites with well-developed locomotory abilities and those that are transmitted by contact between hosts are probably associatively transmittable. For example, bird lice often transfer between hosts during courtship and copulation (11).

Parasites transmitted through ingestion of infective stages are associatively transmittable if the female's tenure at the site where her mate deposited parasites coincides with the period when the parasites are infective and if the female feeds or grooms on the site or otherwise risks ingesting parasites.

If offspring are associated with their father's territory, such as when males provide parental care or oviposition sites, mobile or ingestible parasites may be associatively transmittable provided the period of infectivity coincides with the offsprings' tenure at the site, and the offspring engage in behavior in which they risk infection.

METHODS

The biology and behavior of the host, parasite, and any intermediate host or vector must be taken into account in determining a parasite's transmittability. Due to the novelty of this approach and the considerable literature research required, transmittability was determined by the author, and therefore not blindly. Small sample size and the possibility of bias warrant a close examination of which studies of parasite-mediated sexual selection are included and excluded in this analysis. Rationales for transmittability assignments are found in the legend of Table 2. Host species were included if the relationship between male mating success and parasite intensity had been examined, whether or not particular male traits on which females base their choices are known. Also included were studies that determined the effect of parasites on male traits already known to be important in female choice. Excluded were studies for which not enough information is known about host/parasite/vector relationships to determine whether the parasite is associatively transmittable.

I tested the contagion indicator hypothesis by analyzing studies of parasite-mediated sexual selection in 15 host species (see Table 2). The G test with Williams's correction (12) was used to determine whether transmittability and the type of correlation found between parasite intensity and male mating success are independent.

RESULTS

Of the studies involving parasites that are associatively transmittable, six of eight found a negative correlation between parasite intensity and male mating success, while of the studies using parasites that are not associatively transmittable, only one of seven found a negative correlation (Table 1; G with Williams's correction = 5.988; $0.01 < P < 0.025$). The interaction between the type of correlation between male mating success and parasite intensity is a critical prediction of the contagion indicator hypothesis and is not predicted by the Hamilton and Zuk hypothesis. In every case in which there was found a significant negative correlation between parasite intensity and mating success, there was also found a significant negative correlation between parasite intensity and the degree of expression of the male trait examined. This is a critical prediction of the contagion indicator and Hamilton and Zuk hypotheses but is not predicted by the parasite avoidance hypothesis.

In some studies, more than one parasite was examined per host. To reduce the risk of pseudoreplication, only one parasite per host was included in this analysis. Two parasites of the sage grouse, four parasites of the gray treefrog, and one parasite of the zebra finch were eliminated. Six of the seven host/parasite relationships thus eliminated favor the contagion indicator hypothesis. Therefore, the actual probability of rejecting a true null hypothesis may be less than the reported P value of $0.01 < P < 0.025$. If these seven parasites are included in the analysis, $0.001 < P < 0.005$ (G with Williams's correction = 9.500; $n = 22$). The true P value probably lies between 0.001 and 0.025.

DISCUSSION

The contagion indicator hypothesis predicts that male characters important in female choice indicate the presence of associatively transmittable parasites, while the presence of nonassociatively transmittable parasites is not indicated by male characters used in female choice. How might this difference evolve? To the extent that benefits to female choice are responsible for the maintenance of male traits, selection should favor female preferences that result in greater fitness payoffs to mate choice (13). For a choosing female, avoiding males with associatively versus nonassociatively transmittable parasites probably has different fitness payoffs. A female avoiding mating with a male that is infected with associatively transmittable parasites accrues the direct benefit of avoiding

Table 1. Summary of relationship between parasite transmittability and the type of correlation between parasite intensity and male mating success

Type of correlation between parasite intensity and male mating success	Transmittability	
	Associatively transmittable	Nonassociatively transmittable
Significant negative correlation	6	1
No significant negative correlation	2	6

G test with Williams's correction, $0.01 < P < 0.025$.

parasitic infection. Such a payoff is immediate and has a high probability of occurring, relative to the genetic payoff for a female of avoiding a male with nonassociatively transmittable parasites. Any genetic benefit accruing to the latter female will be realized only if the offspring are challenged with the parasite, and if heritable variation in resistance exists.

Virulence of parasites is of prime importance in studies of parasite-mediated sexual selection because the degree of virulence is expected to affect the strength of selective pressure on females for choosing mates based on their parasite intensity. In many of the studies in this analysis in which no significant negative relationship between parasite intensity and male mating success was found, the parasites are apparently not highly virulent (Table 2). If these parasites are truly less virulent, this analysis cannot be used as evidence against the Hamilton and Zuk hypothesis because Hamilton and Zuk predict no effect when parasites are avirulent. However, there is reason to believe that, in these species, the virulence presented in Table 2 does not reflect their true virulence for two reasons.

First, knowledge of a parasite's effect on a host is incomplete, because of differences in ease of studying different parasites and differences in the economic importance of host species. Parasites of economically important species of farm animals, game animals, and pets are much more likely to be

Table 2. Relationship between parasite transmittability, male mating success, and degree of expression of male secondary sexual characters

Host	Parasite	Transmission via	Associatively transmittable parasite?	Relationship: Parasite intensity and mating success	Relationship: Parasite intensity and trait	Detrimental effects of infection or evidence for virulence
Ring-necked pheasant[a] (14)	Coccidia: Eimeriidae[p]	Ingested cysts	Yes[ee]	Negative*	Negative*	Damages host intestines severely, esp. in young birds (14)
Red jungle fowl[b] (15)	Nematoda: Secernentea[q]	Ingested eggs	Yes[ff]	Negative*	Negative*	Juvenile mortality; see refs. in ref. 15
Rock dove[c] (16)	Mallophaga: Menoponidae, two species[r]	Parasite locomotion	Yes[gg]	Negative*	Negative*	Destruction of insulating feathers, loss of mass (17)
Guppy[d] (18)	Trematoda: Monogenea[s]	Parasite locomotion	Yes[hh]	Negative*	Negative*	Mortality; see refs. in ref. 18
Three-spined stickleback[e] (19)	Ciliata: Holotrichidae[t]	Parasite locomotion	Yes[ii]	Negative*	Negative*	Mortality; see refs. in ref. 19
Barn swallow[f] (20)	Dermanyssid mite[u]	Parasite locomotion	Yes[jj]	Negative*	Negative*	Juvenile mortality (20, 21)
Spadefoot toad[g] (22)	Trematoda: Monogenea[v]	Parasite locomotion	Yes[kk]	None	None	Depletion of fat stores, reduction of hematocrit (23)
Red-spotted newt[h] (unpublished data)	Mastigophora: Trypanosomatidae[w]	Vector: leech	No[ll]	None	None	Reduction in egg numbers (24)
Black grouse[i] (25)	Sporozoa: Haemoproteidae[x]	Vector: diptera	No[ll]	None	None	Indirect evidence of virulence (26):
Redwinged blackbird[j] (27)	Sporozoan: Haemoproteidae and Plasmodiidae[y]	Vector: diptera	No[ll]	None	None	(*i*) High intensity infections in dead and moribund animals,
Redpoll finch[k] (28)	Sporozoan: Haemoproteidae and Plasmodiidae[z]	Vector: diptera	No[ll]	None	None	(*ii*) Severe and fatal infections in domestic fowl
Sage grouse[l] (29)	Sporozoa: Haemoproteidae[aa]	Vector: diptera	No[ll]	Negative*	Negative*	
Zebra finch[m] (30)	Mallophaga: Menoponidae, two species[bb]	Parasite locomotion	Yes[gg]	Positive	Positive	Destruction of insulating feathers (31)
Fence lizard[n] (32)	Sporozoa: Plasmodiidae[cc]	Vector: diptera	No[ll]	None	Positive	Physiological, behavioral, reproductive pathology (see refs. in ref. 32)
Gray treefrog[o] (33)	Trematoda: Monogenea[dd]	Parasite locomotion	No[mm]	None	None	Similar trematode depletes fat stores and reduces hematocrit (34)

[a]*Phasianus colchicus*; [b]*Gallus gallus*; [c]*Columba livia*; [d]*Poecilia reticulata*; [e]*Gasterosteus aculeatus*; [f]*Hirundo rustica*; [g]*Scaphiopus couchii*; [h]*Notophthalmus viridescens*; [i]*Tetrao tetrix*; [j]*Agelaius phoeniceus*; [k]*Carduelis f. flammea*; [l]*Centrocercus urophasianus*; [m]*Taeniopygia guttata castanotis*; [n]*Sceloporus occidentalis*; [o]*Hyla versicolor*; [p]*Eimeria* sp.; [q]*Ascaridia galli*; [r]*Columbicola columbae*, *Campanulotes bidentatus*; [s]*Gyrodactylus* sp.; [t]*Ichthyophthirius multifiliis*; [u]*Ornithonyssus bursa*; [v]*Pseudodiplorchis americanus*; [w]*Trypanosoma diemyctyli*; [x]*Leucocytozoon lovati*; [y]*Plasmodium* sp., *Haemaproteus* sp., *Leucocytozoon* sp.; [z]*Leucocytozoon fringillinarum*, *Plasmodium* sp.; [aa]*Plasmodium pediocetii*.; [bb]*Brueelia* sp., *Myrsidea* sp.; [cc]*Plasmodium mexicanum*; [dd]*Polystoma nearcticum*; [ee]male sheds infective eggs in feces (35), female feeds on male's territory (36); [ff]male sheds infective cysts in feces (37), female and young are present on male's territory (38); [gg]female is exposed to lice during copulation (11), female and offspring are exposed to lice during period of parental care (39); [hh]females exposed to parasite during mating (A. Houde, personal communication); [ii]young remain on male's territory (H. Weeks, personal communication) and are likely to be exposed to mobile stage of parasite from their father (40); [jj]female is exposed to mites during copulation (41), female and offspring are exposed to mites during period of parental care (20); [kk]infective stage of trematode exits the male's bladder at onset of amplexus (42), making infection of female possible; [ll]an interval to infectivity and the lack of a long-term association between the vector and the male make transmission to the female unlikely in the context of mate choice (this paper); [mm]parasite is shed at amplexus but infects only metamorphosing tadpoles (43), which are not likely to be associated spatially with their father or their oviposition site due to the long interval between amplexus and metamorphosis.

*Reported P value, ≤ 0.05.

discovered and studied than those of host species less economically important. Of the host species in which was found a significant negative relationship between parasite intensity and male mating success in this analysis, most are economically important species. In contrast, most of the host species in which no significant negative relationship was found are not economically important. In addition, it is particularly important to know the effect of a parasite on juvenile hosts, as they are often vulnerable to parasites that are controlled by the adult host. Knowledge of a parasite's effect on juvenile hosts is the sort of information that is known in economically important species, and that is unknown in less-studied species.

Second, there are theoretical grounds for concluding that the reported virulence of some of the parasites in Table 2 might not reflect their true virulence. For many of the host species for which no negative relationship between parasite intensity and mating success was found, and for which virulence is reportedly low, the parasites are transmitted by vectors. Evidence gathered by Ewald (44) strongly supports the hypothesis that the evolution of higher virulence is correlated with the greater transmission opportunities that mobile vectors afford. Parasites that rely on host mobility for transmission are expected to be relatively less virulent than parasites that are transmitted by vectors or are otherwise very mobile, because vector-borne and mobile parasites can debilitate hosts without reducing their opportunities for transmission.

Although this analysis supports the contagion indicator hypothesis, it is a correlational study and therefore can only support or fail to support, but not falsify, the hypothesis, since an unmeasured variable might be responsible for the relationship the contagion indicator hypothesis purports to explain. Unfortunately, transmittability itself cannot be experimentally manipulated on an ecological time scale, since females' response to risk of parasite transmission is expected to occur on an evolutionary time scale. The contagion indicator hypothesis is not supported within a species in two ways: (*i*) if debilitating, associatively transmitted parasites do not affect the degree of development of male traits used in female choice and, therefore, male mating success, and (*ii*) if debilitating, nonassociatively transmitted parasites affect the degree of development of male traits used in female choice and, therefore, male mating success.

The contagion indicator hypothesis is subsumed under a broader general hypothesis for sexual selection—one based on differentially expressed male traits—which might be affected by many different selective pressures. Therefore, the generality of the contagion indicator hypothesis would seem to be reduced. However, the process the contagion indicator hypothesis predicts might still be very general due to the abundance of parasites and pathogens with which every host must contend.

The Hamilton and Zuk hypothesis and the contagion indicator hypothesis are not mutually exclusive. To the extent that there is heritable variation for parasite resistance positively correlated with the degree of expression of male traits, female choice for such male traits will result in female choice for genes for parasite resistance. However, the dearth of negative correlations between parasite intensity and male mating success when the direct benefit of transmission avoidance is eliminated suggests that the Hamilton and Zuk model might not be a general explanation for parasite-mediated sexual selection. Similarly, the parasite avoidance hypothesis and the contagion indicator hypothesis are not mutually exclusive. There is no reason that traits which make ectoparasites more visible could not also be variably expressed. However, the parasite avoidance hypothesis fails to explain those studies in which male traits are variable and so cannot be considered a general model of parasite-mediated sexual selection. The contagion indicator hypothesis thus appears to be the most generally applicable model of parasite-mediated sexual selection.

This analysis suggests that parasite transmittability in the context of mate choice is an important aspect of parasite-mediated sexual selection and that the direct benefit of avoiding contagion in mate choice might be more important than choice for good genes for parasite resistance in most species. The direct benefit to females of avoiding parasitic infection through choice of variably expressed male traits may help explain the maintenance of these traits and female choice for them.

I am grateful to J. Phillips for stimulating this work and to the Cornell Behavior Group and C. Van Riper for helpful criticism and discussions. The manuscript benefited from comments by K. Adler, A. Bouck, D. Clayton, U. Mueller, S. Pratt, H. K. Reeve, P. Sherman, B. Tershy, and G. C. Williams. P. Ewald and an anonymous reviewer improved an earlier draft of this manuscript. This work was done when the author was supported by a National Institutes of Mental Health Neurobiology and Behavior Training Grant and by Department of Agriculture (Hatch) Funds to K. Adler.

1. Darwin, C. (1874) *Descent of Man, and Selection in Relation to Sex* (Hurst, New York).
2. Ryan, M. J. & Kirkpatrick, M. (1991) *Nature (London)* **350,** 33–38.
3. Hamilton, W. D. & Zuk, M. (1982) *Science* **218,** 384–387.
4. Møller, A. P. (1990) *J. Evol. Biol.* **3,** 319–328.
5. Zuk, M. (1992) *Adv. Study Behav.* **21,** 39–68.
6. Clayton, D. H. (1991) *Parasitol. Today* **4,** 329–334.
7. Sullivan, B. K. (1991) *Herpetologica* **47,** 250–264.
8. Borgia, G. (1986) *Behav. Ecol. Sociobiol.* **19,** 335–358.
9. Borgia, G. & Collis, K. (1990) *Am. Zool.* **30,** 279–285.
10. Ryan, M. J. & Keddy-Hector, A. (1992) *Am. Nat.* **139,** S4–S35.
11. Turner, E. C., Jr. (1971) in *Infectious and Parasitic Diseases in Wild Birds*, eds. Davis, J. W., Anderson, R. C., Karstag, L. & Trainer, D. O. (Iowa State Univ. Press, Ames), pp. 175–185.
12. Sokal, R. R. & Rohlf, J. (1981) *Biometry: The Principles and Practice of Statistics in Biological Research* (Freeman, San Francisco), pp. 706–707.
13. Zahavi, A. (1991) *Anim. Behav.* **42,** 501–503.
14. Hillgarth, N. (1990) *Am. Zool.* **30,** 227–233.
15. Zuk, M., Thornhill, R., Ligon, J. D. & Johnson, K. (1990) *Am. Zool.* **30,** 235–244.
16. Clayton, D. H. (1990) *Am. Zool.* **30,** 251–262.
17. Booth, D. T., Clayton, D. H. & Block, B. A. (1993) *Proc. R. Soc. London B* **253,** 125–129.
18. Houde, A. E. & Torio, A. J. (1992) *Behav. Ecol.* **3,** 346–351.
19. Milinski, M. & Bakker, T. C. M. (1990) *Nature (London)* **344,** 330–333.
20. Møller, A. P. (1990) *Ecology* **71,** 2345–2357.
21. Gjelstrup, P. & Møller, A. P. (1986) *Entomol. Medd* **53,** 119–122.
22. Tinsley, R. C. (1990) *Am. Zool.* **30,** 313–324.
23. Toque, K. (1993) *J. Anim. Ecol.* **63,** 683–693.
24. Mock, B. A. (1983) Ph.D. Thesis (Univ. of Maryland, College Park).
25. Höglund, J., Alatalo, R. V. & Lundberg, A. (1992) *Behav. Ecol. Sociobiol.* **30,** 71–76.
26. Atkinson, C. T. & Van Riper, C., III (1991) in *Bird-Parasite Interactions: Ecology, Evolution, and Behaviour*, eds. Loye, J. E. & Zuk, M. (Oxford Univ. Press, Oxford), pp. 19–48.
27. Weatherhead, P. J. (1990) *Behav. Ecol.* **1,** 125–130.
28. Seutin, G. (1994) *Oikos* **70,** 280–286.
29. Johnson, L. L. & Boyce, M. S. (1991) in *Bird-Parasite Interactions: Ecology, Evolution, and Behaviour*, eds. Loye, J. E. & Zuk, M. (Oxford Univ. Press, Oxford), pp. 377–388.
30. Burley, N., Tidemann, S. C. & Halupka, K. (1991) in *Bird-Parasite Interactions: Ecology, Evolution, and Behaviour*, eds. Loye, J. E. & Zuk, M. (Oxford Univ. Press, Oxford), pp. 359–376.
31. Clayton, D. H. (1990) *Am. Zool.* **30,** 251–262.
32. Ressel, S. & Schall, J. J. (1989) *Oecologia* **78,** 158–164.
33. Hausfater, G., Gerhardt, H. C. & Klump, G. M. (1990) *Am. Zool.* **30,** 299–311.
34. Toque, K. (1993) *J. Anim. Ecol.* **63,** 683–693.
35. Schmidt, G. D. & Roberts, L. S. (1977) *Foundations of Parasitology* (Mosby, St. Louis, MO).
36. Collias, N. E. & Collias, E. C. (1967) *Condor* **69,** 360–386.

37. Schmidt, G. D. & Roberts, L. S. (1977) *Foundations of Parasitology* (Mosby, St. Louis, MO).
38. Hillgarth, N. (1990) *Am. Zool.* **30,** 227–233.
39. Clayton, D. H. (1991) in *Bird-Parasite Interactions: Ecology, Evolution, and Behaviour*, eds. Loye, J. E. & Zuk, M. (Oxford Univ. Press, Oxford), pp. 258–289.
40. Schmidt, G. D. & Roberts, L. S. (1977) *Foundations of Parasitology* (Mosby, St. Louis, MO).
41. Turner, E. C., Jr. (1971) in *Infectious and Parasitic Diseases of Wild Birds*, eds. Davis, J. W., Anderson, R. C., Karstag, L. & Trainer, D. O. (Iowa State Univ. Press, Ames), pp. 175–185.
42. Tinsley, R. C. (1990) *Am. Zool.* **30,** 313–324.
43. Schmidt, G. D. & Roberts, L. S. (1977) *Foundations of Parasitology* (Mosby, St. Louis, MO).
44. Ewald, P. (1994) *The Evolution of Infectious Disease* (Oxford Univ. Press, Oxford).