# Ecological interactions among protozoan parasites and their avian hosts: an approach

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Ecologist and evolutionary biologist virtually ignored parasites, but nowadays scientists recognize the benefits of using data on the prevalence and distribution of avian haemosporidian in ecological, evolutionary and behavioural studies. We present three different researches on ecological interactions among protozoan parasites and their avian hosts focus on answering whether parasites regulate host populations; and whether parasites act as selective agents that results in adaptative responses in their hosts. Results demonstrate that protozoan malarial parasites can have dramatic effects on their hosts because they are involved in different host's life history traits such as senescence, reproductive success, survival and predation-prey interaction.

**Keywords:** Ecological interaction, Protozoa, malarial parasites, senescence, predation, reproductive success.

Parasitic animal are one of the major cause of the infection diseases which affect man and his domestic stock, and their effects are enormous in terms of mortality, chronic diseases and economic loss. To prevent this many advances in microbiology and parasitology are required with an obvious applied significance because they throw light upon a wide variety of researches, such as immunology, conservation biology, behavioural ecology, biogerontology and evolutionary biology. So, modern researches are directed towards the understanding of parasite's effects on their hosts. Here we present three different researches that allow us to advance in knowledge about parasites and immune responses in wild birds. Most aspects of the life history of hosts have been hypothesised to be affected by parasites, like age of maturity, clutch size and offspring size, but our knowledge about causal relationship is still rudimentary due to a scarcity of experimental manipulation. Malaria is supposed to have strong negative effects of host fitness because this group of intra-cellular parasites cause dramatic effects over the efficiency of metabolism. However, the fitness consequences of malarial infections are generally poorly known. So far only a single study has experimentally treated malarial infections in birds showing a direct effect of avian malaria on reproductive output [1]. But there are more questions that must be answered. Do malarial parasites have effects on clutch size? If so, do these early effects of malarial infection during the reproductive cycle have disproportionately large effects on seasonal reproductive success? So early treatment of individuals host would then be expected to reduce or maintain the levels of infection at a time in the reproductive cycle when infections are normally rapidly increasing.

We experimentally reduced levels and intensity of blood parasites infection, by randomly treating birds (House martin *Delichon urbica*) with an anti-malarial drug (Primaquine. Sigma, St.Louis, Mo) at the beginning of the breeding season [2]. Results showed that clutch size was on average 18% larger in treated birds, while the difference increased to 39% at hatching and 42% at fledging. Our study is the first to demonstrate that there indeed is an improvement in clutch size as a response to a removal of a parasite. The possible mechanisms generating these effect could be a direct impact on foraging ability and therefore rate of level of resource acquisition necessary for production of eggs, or it also would be possible that reduction of clutch size was due to a energy draining by the parasites. In addition, carotenoids used by the immune system for fighting serious infection may also play a role in egg formation [3-5]. If there is a trade-off between use of carotenoids and other antioxidants for egg production and immunity, we hypothesise that control females laid eggs with reduced levels of yolk antioxidants. That could directly lead to reduced hatching success [6]. Alternatively, adult house martins may have been affected by malarial infections, causing a reduction in the efficiency of incubation and provisioning of offspring. To sum up, these findings demonstrate that malarial parasites can have dramatic effects on clutch size and other demographic variables.

Predation is one of the most important causes of natural selection [7]. Although predation often is studied in isolation, several pieces of evidence suggest that different kinds of interespecific interaction may affect each other, so parasites may play an important role in predation-prey interaction. In another study, House sparrow (*Passer domesticus*) were either exposed to a predator (owl) or a control (pigeon) while development of malaria infections was recorded during the following six weeks [8]. We tried to test how perceived predation risk affects the ability of potential prey to produce a response to an immune response (T-cell mediated immune response to a challenge with phytohemaglutinin, PHA), and if there were a long term effects of reduced immune response on malarial infection. The main findings of this study were that predation has significant depressing effect on T-cell response because it was 20 % smaller in the owl (predation) than in pigeon (control) treatment. Figure 1 shows that an increased predation risk was associated with an increase prevalence and intensity of *Haemoproteus* malarial blood parasite.

To the best of our knowledge, this provides the first experimental demonstration of a direct relationship between predation risk and immune function, and the first successful experimental manipulation of infection with a blood parasite, and this could result in an increased risk of mortality [9-12]. In conclusion, predators may interact indirectly with parasites through their effects on immune function in hosts.

Senescence is the progressive lost of functionality accompanied by fertility decreasing and increasing in risk of mortality as time goes by [13]. Immune system deteriorates as ageing, so it causes an ability reduction to fight against parasites and to prevent diseases [14].

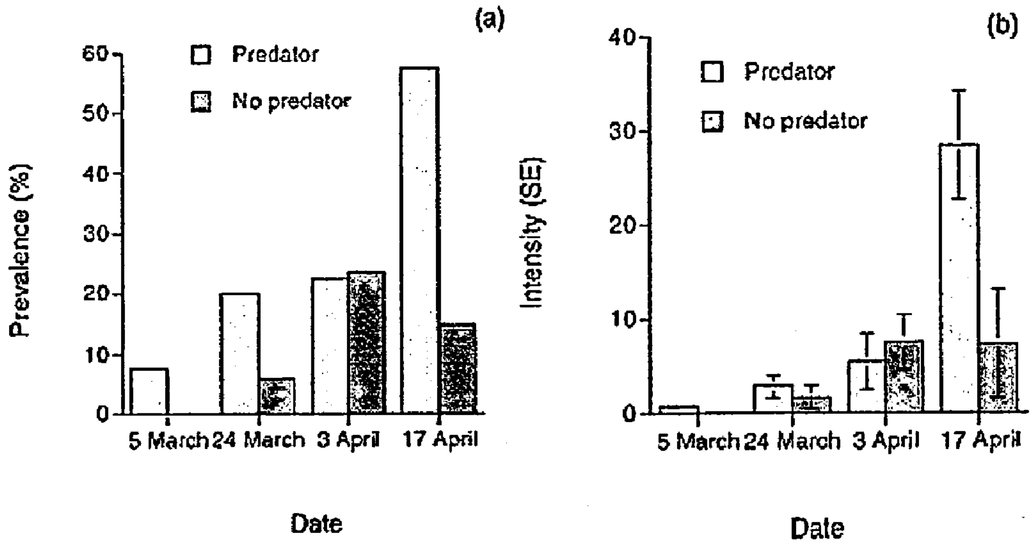


**Figure 1.** Prevalence (a) and intensity (b) of infection with *Haemoproteus* blood parasites in relation to date and treatment. Error bars are SE. Sample sizes are 40 for predator exposure and 34 for control treatment.

This is the reason why parasitism have been involved in the expression on the senescence for a long time [15, 16]. So, must we expect higher blood parasites infection in wild birds as ageing? Many studies tried to show an increasing in parasites infection in wild populations of migratory birds. The main problem of all these researches was the impossibility to study the same individual consecutive years. In the third study we made a comparative test for prevalence of malarial infection between two avian species (House martin and Barn swallow), capturing the same individual every year. As we can see at figure 2 we found significance differences in the number of infected adult birds between years in both species.

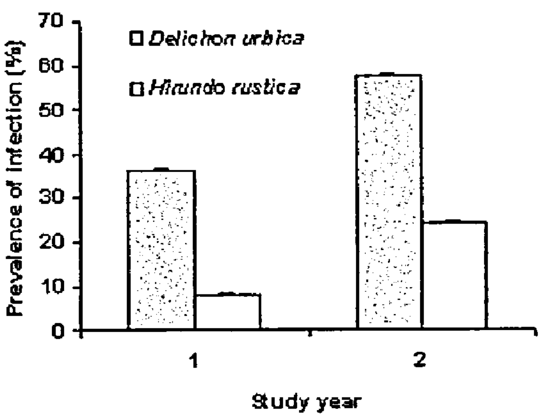


**Figure 2.** Prevalence of infection with *Haemoproteus* and *Trypanosoma* blood parasites in relation to study year. Error bars are SE. Sample sizes are 127 for martins (*Delichon urbica*) and 50 for swallows (*Hirundo rustica*).

Moreover, many individuals significantly change their status from non infected to infected year after year. This could be explained because the host's ability loss to fight against parasites as growing old [17], a spring relapse in blood parasites [18], a higher vector

exposition [19] or a immune function deterioration associated to senescence [20, 21]. In resume, protozoan parasites are related to the expression of their hosts senescence in wild populations of avian species because blood parasites infection levels increase as their hosts grow old.
All these findings experimentally demonstrate that protozoan malarial parasites affects their hosts in many different ways, such as senescence, reproductive success, immune function and mortality. Thus, the study of parasites is a field that must to pay attention because it gives many answers to unsolved questions in behavioural ecology and evolutionary biology.

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## References

[1] S. Merino, J. Moreno, J.J. Sanz, & E. Arriero. Proc. R. Soc. Lond. B **267** p.2507 (2001)
[2] A. Marzal, F. de Lope, C. Navarro and A. P. Møller Oecología **142** p.541(2005)
[3] J.D. Blount, D.C. Houston, P.F. Surai & A.P. Møller Proc R Soc Lond Ser B **271** [Suppl] p. 79 (2004)
[4] N. Saino, R. Ferrari, M. Romano, R. Martinelli & A.P.Møller. Proc R Soc Lond Ser B **270** p.2485 (2003)
[5] D. Wakelin, D. Immunity to parasites: How parasitic infections are controlled. Cambridge University Press, Cambridge, U. K.(1996)
[6] P.F. Surai. Natural antioxidants in avian nutrition and reproduction. Nottingham University Press, Nottingham, U. K. (2003)
[7] J.A. Endler. Natural selection in the wild. Princeton, Princeton University Press (1986)
[8] C. Navarro, F. de Lope, A. Marzal, and A. P. Møller Behavioural Ecology **15** p. 629 (2004)
[9] P. Christe, A.P. Møller, N. Saino, F. de Lope. Heredity **85** p. 75 (2000)
[10] G. González, G. Sorci & F. de Lope. Behavioral Ecology and Sociobiology **46** p.117 (1999)
[11] S. Merino, A.P. Møller, F. de Lope. Oikos **90** p.327 (2000)
[12] M. Soler, M. Martín-Vivaldi, J.M. Marín & A.P. Møller. Behavioral Ecology **10** p.281 (1999)
[13] T.B. Kirkwood & S.N.Austad. Nature **408** p. 233 (2000)
[14] R.A. Miller. Science **273** p. 70 (1996)
[15] P.B. Medawar. An Unsolved Problem in Biology. H. K. Lewis, London (1952)
[16] G.C. Williams. Evolution **11** p.398 (1957)
[17] A.P. Møller & F. de Lope. Journal of Animal Ecology **68** p.163 (1999):
[18] C. Atkinson. & C. van Riper III. In Bird-Parasite interacctions (Loyle & Zuk eds.) Oxford University Press. Oxford pp. 19-48 (1991)
[19] K. Allander & G.F. Bennet. Journal of Avian Biology **25** p.69 (1994).
[20] M. Cichon, J. Sendecka, L. Gustafsson. Journal of Evolutionary Biology **16** (6) p. 1205 (2003)
[21] N. Saino, R. P. Ferrari, M. Romano, D. Rubolini & A. P. Møller Journal of Evolutionary Biology **16** (6) p. 1127 (2003)