# Section I: Introduction

# 1
# Parasitism: Costs and Effects

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Parasitism has been defined in many ways, but in terms of wildlife disease, it is usually taken to mean an obligatory trophic association between individuals of two species in which one (the parasite) derives its food from a living organism of the other species (the host). An individual host bird can be viewed as an island of habitat that provides resources for parasites, with the parasites deriving benefits while the host is harmed or bears some cost. Parasitism is common in nature; for example, Price (1980) estimated that half of all animal taxa are parasitic. Parasitism is ubiquitous in wild birds and individual birds are affected by many different parasites during their lifetime, but our understanding of the parasites that occur in wild birds is fragmentary.

Moore and Clayton (1997) concluded that the majority of parasites of wild birds have yet to be described taxonomically. Some groups, such as blood-inhabiting protozoa (the hematozoa), have been studied widely, perhaps because of the ease with which blood can be collected from living birds, while little is known about other groups such as intestinal flagellates. But even within the hematozoa, species diversity has probably been greatly underestimated (Bensch et al. 2007). Similarly, more is known about the effects of arthropod ectoparasites than about the effect of protozoa and helminths on birds, and cavity nesting birds have been studied more extensively than most other species because of the relative ease in capturing, examining, and following these birds.

Studying parasitism in wild birds is subject to a number of constraints that make working with disease in any free-ranging species more difficult than studying humans or domestic animals. These include:

- inadequate baseline information about the host species. Knowledge of avian life history traits is rudimentary (Zera and Harshman 2001), and so one often must extrapolate from other species and collect information about the basic biology of the host while trying to understand a host–parasite relationship;
- difficulty in quantifying factors related to disease. It is impossible to assess the significance of a parasite for a population without the ability to calculate basic epidemiological proportions such as prevalence, incidence, morbidity, and mortality rates. The number of individuals affected by a parasite (the numerator for such calculations) is usually difficult to determine and the population at risk (the denominator) rarely can be measured adequately;
- the need to consider the long-term effect of a parasite in wild birds. This may be very difficult, even when the number affected and the population at risk can be determined. If a disease, such as coccidiosis, occurs in a flock of chickens and 15% die, the significance of the disease is that 15% fewer chickens go to market. However, a similar 15% loss in a wild bird population might result in more resources per capita for the remaining birds, leading to reduced mortality from other factors and/or improved reproduction. The potential for compensation or other delayed effects may be very important in assessing the impact of a parasite on wild birds at the population level;
- the sample of wild birds available for study is usually biased by the method of collection and may not represent the actual state of nature. Depending on the method of collection, affected birds may be under- or overrepresented, even in groups collected by mass-capture methods (Sulzbach and Cooke 1978); and
- the anonymity of wild birds, except for the small number marked by the researcher. For instance, although age is an important disease determinant, the age of wild birds often cannot be determined except to differentiate hatch-year from after-hatch-year birds. Individuals seldom can be traced back in time to discover previous exposure to disease agents or forward in time to discover

their fate. Commonly used techniques such as retrospective and prospective case–control studies that are useful in human and veterinary epidemiology are impossible except in unusual circumstances, such as in birds with a high degree of nest site fidelity.

A fundamental feature of parasitism is that the presence of a parasite involves a cost to the host. The costs of parasitism may include:

- loss of resources extracted by the parasite directly from the host, for example, loss of blood to blood-feeding ectoparasites;
- competition between the parasite and the host for resources, as occurs with cestodes that absorb nutrients from the host's gut content;
- costs to the host for defense against parasites. These may include foregoing resource-rich areas to avoid areas where parasites may be present, costs for grooming, moving away from parasites, or abandoning a nest, costs to develop and maintain innate and acquired resistance, and costs to activate these systems;
- costs resulting from tissue injury related to the parasite. This may be direct damage caused by the parasite or, more often, injury from the inflammatory and immune response to the parasite. Some injuries may result in dysfunction, such as reduced mobility, reduced digestive efficiency, or increased loss of nutrients through intestinal or kidney injury, that interfere with obtaining or retaining resources;
- costs related to improper development as a result of parasitism early in life (e.g., Spencer et al. 2005); and
- costs to repair or replace damaged tissues.

The diversity of parasites and the variety of ways that they interact with hosts make it difficult to measure the cost of a single parasite species; to compare the relative cost of different parasites such as the lice, intestinal coccidia, and tracheal worms, all of which might be infecting a single host; or to understand how these parasites may interact with each other and with other environmental factors to affect a host population. The costs described above are related to resources, and particularly to energy ["*the single common denominator of life*"; "*something that is absolutely essential and involved in every action large or small*" (Odum 1993)]. Energy is a measure of the ability to do work and is a "currency" that can be used to consider the costs of all types of parasitism, at least conceptually if not quantitatively at this time. Four basic features must be considered when using energy as a currency to consider parasitism:

- The supply of energy is limited. Most birds are unable to increase their intake of energy readily, and so they must function within a finite budget. In other words, a bird cannot use more energy than it can assimilate or has in storage.
- The amount of energy available and accessible is not constant or uniform. The energy available to a bird varies with the time of year, weather, habitat conditions, and the number of competitors for that energy. Not all individuals in a population have equal access to the resources that are available; thus, within a group or population some birds may have abundant resources while others do not.
- Use of energy for one purpose reduces the amount available for other uses. Most of the energy assimilated by a bird is used for maintenance, that is, keeping the body functioning, repaired, maintaining a high core temperature, avoiding predators, and defending against disease. Energy that remains can be used for production (growth and reproduction) or stored as fat for future use. If extra energy is used to defend against parasites or to repair tissue injured by parasites, the energy available for reproduction or growth is reduced. For instance, the cost of producing antibody to a novel antigen is equivalent to that of producing half an egg in female House Sparrows (*Passer domesticus*) (Martin et al. 2003) and mounting an immune response resulted in asymmetry of flight feathers in nestling Mountain Chickadees (*Poecile gambeli*) (Whitaker and Fair 2002). Conversely, increased reproductive effort may result in reduced ability to mount a defense against parasites (Deerenberg et al. 1997).
- The need for energy for various purposes is highly variable among individuals and at different times of year.

Because an individual cannot maximize all life history traits simultaneously, life history theory suggests that a bird should adopt a strategy that optimizes energy use among resource-demanding activities, such as defense and reproduction, to maximize lifetime fitness. Ecologists use the term "trade-off" for this process of making physiological choices among competing needs for resources that should maximize the chances of an individual's genes being passed on to the next generation. Individuals that make the wrong choices are less successful or "fit," and this may provide a basis for genetic selection.

As a result of heterogeneity in both the supply of energy and the need for energy, the appropriate physiological trade-offs in relation to parasitism vary among individual birds and for different parasites, and the pattern of trade-offs is different seasonally and annually. For this reason, the reaction to parasites and the effects of parasitism must always be considered in terms of the context in which parasitism is occurring and of how the situation might influence resource trade-offs. For instance, during one season a bird may be in poor nutritional condition and need to direct all its available resources to simply staying alive, with little or no ability to mount an effective defense against parasites or to grow or reproduce. At another time of year the same bird may have ample resources to meet all needs, and so it can afford strong resistance to parasites and still be able to grow and reproduce effectively.

Young birds may have different priorities than adults and the sexes may have different strategies and trade-offs. For instance, Tschirren et al. (2003) suggested that a greater need for carotenoid-based coloration for signaling by male Great Tits (*Parus major*) might lead to a trade-off that results in reduced immunocompetence in males. Privileged individuals within the population, such as birds that possess a territory, may have a totally different context for trade-offs related to parasites than do the "have-nots" within the population. Changes in environmental conditions may change the context; for example, Blow Fly (*Protocalliphora braueri*) larvae had no effect on Sage Thrasher (*Oreoscoptes montanus*) nestling weight, size at fledging, or mean fledgling age, but in a year with cold wet weather, survival and fledging success were markedly reduced among parasitized birds compared to unparasitized birds (Howe 1992).

Knowledge of how trade-offs occur in relation to parasitism is fragmentary at this time and general rules about which activity (reproduction, growth, defense against predators or parasites) should take precedence for resources are likely subject to many exceptions. For instance, hosts may be selected to develop acquired immunity to only some of the disease agents that they encounter (Boots and Bowers 2004). While mounting a strong defensive response to parasites is likely a "good" thing generally, in some situations it may be adaptive to suppress the defensive response. This may be the case in nesting Common Eiders (*Somateria mollissima*). Female eiders do not feed during breeding and face severe resource restrictions while incubating. Birds that do not begin with adequate resources abandon their nest in order to survive.

Hanssen et al. (2004) immunized incubating female eiders with nonpathogenic antigens, including sheep red blood cells. Not surprisingly, the rate of successful immunization was not very good compared to what would be expected at other times of year. Under these circumstances, it appears that the appropriate choice for many eiders is to use their limited resources to survive and reproduce rather than to mount an immune response. A second part of the same study compared survival of birds that mounted an immune response to that of birds that did not produce antibodies. Both responding and nonresponding eiders had sufficient resources to complete reproduction; however, only about 27% of birds that produced antibody to sheep red blood cells returned to the colony in subsequent years, compared with approximately 72% of birds that did *not* produce antibody. Under these conditions, females that invested in an immune response "*experienced considerably impaired long-term survival*" compared to females that did not respond. This example also serves to illustrate that the effect of a trade-off on fitness may be delayed.

The cost to the host is not obvious for most parasites encountered in wild birds. It is only in a minority of situations, described elsewhere in this book, that parasitism is clearly associated with recognizable functional impairment of the host that we can characterize as disease. The apparently "benign" nature of many parasites could be because:

- the effect of the parasites actually is so trivial as to be undetectable;
- the cost is not trivial but it is tolerable; that is, the bird has sufficient resources to cover the costs without significant negative effects on other functions *under conditions at the time the effect was measured*;
- the cost of parasitism is obscured by other more proximate regulatory factors such as predation and competition. Predation is thought to be a major factor in shaping the life history of birds (Zera and Harshman 2001) and parasitized prey may be taken disproportionately by predators (Temple 1987). In some situations the parasite benefits if the infected host is eaten by an appropriate predator (parasite-induced trophic transmission; Lafferty 1999). But infections in which there is no apparent benefit to the parasite may make animals more susceptible to predators, perhaps because of the pathology induced by the parasite. Hudson et al. (1992a) found that Red Grouse (*Lagopus lagopus scotica*) killed by predators were more heavily parasitized by the cecal nematode (*Trichostrongylus tenuis*) than were hunter-killed birds and that birds with many worms may emit more scent and, hence, be more vulnerable to mammalian predators. In some situations, increased vulnerability to predators may be related to energy trade-offs and reduced resources for

predator vigilance or avoidance. For instance, Common Redshanks (*Tringa totanus*) that are energetically stressed (as might result from parasitism) respond by taking risks that increase the probability of predation (Quinn and Cresswell 2004). The interaction between predation and parasitism is undoubtedly complex. Navarro et al. (2004) found that House Sparrows exposed to potential predators (cat or owl) had reduced T-cell-mediated immune response and a higher prevalence and intensity of infection with *Haemoproteus* spp. than did sparrows exposed to nonthreatening animals (rabbit or pigeon), suggesting that even the threat of predation may alter trade-offs that influence parasitism. Although little is known about the effect of parasitism on intraspecific competition, this may be an important factor. For instance, male Greater Sage-Grouse (*Centrocercus urophasianus*) infested with lice are discriminated against for breeding (Spurrier et al. 1991). Females appear to recognize infected males by the occurrence of petechial hemorrhages on the air sacs and males infested with lice are shunned, and so their reproductive input to the population is minimal; that is, their fitness is very low and there is likely negative selection against their genotype. In a similar manner, male Red Grouse infected with *T. tenuis* may have difficulty defending a territory (Delahay et al. 1995). Consideration of interactions between parasitism and competition must also include competition among species that share parasites, such as the Ring-necked Pheasant (*Phasianus colchicus*) and Gray Partridge (*Perdix perdix*) that share *Heterakis gallinarum,* with asymmetrically severe effects on the partridge (Tompkins et al. 2001b); and

- the cost is not trivial but it goes undetected because of insensitivity of the methods used to look for effects. For instance, it would be very easy to dismiss the tiny hemorrhages caused by lice as inconsequential to male Greater Sage-Grouse, without even considering that they might have a profound effect on behavior, reproductive success, and natural selection. The costs of parasitism could also be overlooked because the wrong individuals within the population are examined, the interaction between parasite and host is examined in an inappropriate context (e.g., at the wrong time of year or in an experimental situation in which resources are not limited), inappropriate parameters are measured, or because the long-term (lifetime) consequences of parasitism are not measured. Møller (1994) suggested that the cost of parasitism in nestling birds could be paid by the nestlings through reduced growth or survival or by the parents through reduced survival or future reproductive success as a result of having to provide additional resources to the parasitized young. Bize et al. (2003) found that nestling Alpine Swifts (*Tachymarptis melba*) can compensate for early growth retardation by rapid feather growth, so that if measured at fledging no effect might be obvious; however, rapid feather growth may result in poor feather quality with later effects (Dawson et al. 2000). Nutrient shortage in early development can have other serious long-term consequences including effects on adult dominance rank, morphology, and lifespan (Metcalfe and Monaghan 2001). Island Canaries (*Serinus canaria*) infected with plasmodia as nestlings have structural changes in their brain and reduced song repertoire as adults (Spencer et al. 2005). The effects of parasites are usually not distributed evenly or fairly among all members of a population, which complicates measuring their cost. Metazoa characteristically are distributed in an aggregated manner within the host population (Shaw et al. 1998). Most hosts have few or no parasites and a few individuals have many parasites (often referred to as the 20:80 rule: 20% of the population carries 80% of the parasites). Severe effects are likely to be confined to those individuals with many parasites. Measures of central tendency, such as average intensity of infection and average cost of parasitism, may not be helpful in understanding the significance of the parasite if effects are concentrated in a small group of heavily infected individuals. These animals at the extreme end of the distribution are also important as the major source of infection within the population, but samples drawn from the population are unlikely to contain these individuals unless the sample is very large. Much of the information available on the occurrence of parasites in wild birds comes from the study of birds that died of other causes, because it is inappropriate to kill large samples of birds simply to record their parasites. At one extreme, such a sample may primarily consist of the survivors of conditions that were severe, resulting in underestimation of the cost of parasitism. At the opposite extreme, the sample may contain the few significantly affected individuals in the population, and so the cost to the population is overestimated.

"*While the study of specific host–parasite relationships have proven insightful, they reflect only a small part of the wealth of parasites and pathogens in an*

*animal's internal and external environment*" (Lochmiller and Deerenberg 2000). Virtually all the information available about parasites of birds relates to the effects of individual parasite species, but individual birds are host to many different parasites, often simultaneously; for example, a single feather may be infested with 6 species of feather mite (Pérez and Atyeo 1984) and a group of 45 Lesser Scaup (*Aythya affinis*) were infected by almost 1 million individuals of 52 different helminth species (Bush and Holmes 1986). Examining the effect of parasitism as the interaction between two species fails to account for interactions among parasites that might be additive, synergistic, or antagonistic. Almost nothing is known about the effects or dynamics of parasite assemblages or communities in wild birds.

The largest challenge for those interested in parasites of birds is to answer the question "Do parasites influence bird populations?" Most ecologists and wildlife managers have assumed that the answer is "No" (Tompkins et al. 2001a), but modeling suggests that parasites could regulate host populations if they reduce host survival and/or fecundity in a density-dependent manner (Anderson and May 1978; May and Anderson 1978). To understand the effect of a parasite on the host population, one needs to understand the effect of the parasite on the individual host, the prevalence and intensity of parasite infection within the host population, and the context within which the interaction is occurring. Parasites rarely result in obvious piles of dead birds but many studies have concentrated on the direct effect of parasites on mortality although "*. . . highly pathogenic parasites tend not to have an impact at the population level. . . .*" (Hudson and Dobson 1997), because this type of parasite may kill the host rapidly, thus limiting transmission to other individuals. Sublethal effects of chronic infections that are mediated through reduced fecundity are more likely to have an effect at the population level.

Much of the information available about parasites in birds is descriptive. More than 70 years ago, Aldo Leopold recognized that observational and correlational studies have limited ability to lead to an understanding of disease in wild species (Leopold 1933). Marzal et al. (2005) observed that knowledge of causal relationships of disease caused by parasites of birds "*is still rudimentary due to a scarcity of experimental manipulation*," and Tompkins and Begon (2000) stated that "*regulation by parasites can be established only by experimentally perturbing host/parasite systems away from their equilibrium levels and monitoring subsequent changes in both parasite and host densities relative to control*." Studies that include intervention through treatment of parasites in natural populations, such as by Hudson et al. (1992b, 1998) (*T. tenuis* and Red Grouse), Merino et al. (2000) (hematazoa in Eurasian Blue Tits, *Cyanistes caeruleus*), Hoodless et al. (2002) (ticks and Ring-necked Pheasants) and Marzal et al. (2005) (*Haemoproteus prognei* in House Martins, *Delichon urbicum*), and through experimental infection (e.g., Spencer et al. 2005), have provided insights into parasitism that would be unattainable with traditional observational study. As in all aspects of the study of parasitism, it is important to consider the long-term effects of such interventions. For instance, Hanssen et al. (2003) studied the effect of antiparasite treatment on nesting female eiders. There was no effect of treatment on nest success or on the survival to the next year of birds that nested successfully. However, among the females that were unsuccessful in nesting, 69% of treated birds survived compared with 18% of untreated birds. This suggests that birds that nested successfully were able to tolerate the effects of parasitism, while unsuccessful females were less able to bear the costs from parasites, resulting in a delayed effect on survival. In another example, McCutchan et al. (2004) found that a vaccine significantly protected canaries against natural infection with *Plasmodium relictum* in the year of vaccination. In the following year, survivors in the vaccinated group suffered much higher mortality than unvaccinated birds that had survived exposure in year 1, presumably because vaccine-induced immunity prevented acquisition of protective natural immunity.

Wild birds have developed a suite of trade-offs that allow them to be successful under a particular set of conditions. Environmental cues, such as photoperiod, may guide the timing of these trade-offs. Our world is changing rapidly and dramatically, especially for many wild species. With rapid anthropogenic alterations, such as climate change and environmental contamination, cues that were reliable may no longer be associated with adaptive outcomes (Schlaepfer et al. 2002). If birds are trapped by their evolutionary response to cues, they may find themselves equipped with attributes that are no longer optimal. Schlaepfer et al. (2002) used the term "*evolutionary trap*" for decisions that are now maladaptive because of a sudden anthropogenic disruption. For instance, the optimal time for reproduction by seasonally breeding birds matches peak food supply with peak nestling demand. If birds schedule reproduction based on photoperiod while food supply is determined by temperature, a mismatch in timing may result in peak nestling demand occurring while food supplies are declining, with serious consequences for fitness (e.g., Thomas et al. 2001). The effect of this type of evolutionary trap on parasitism has not been explored, but mismatches between the phenology of parasites or disease vectors and birds, as well as range expansion by parasites as a result of

climate change and interactions among parasites and contaminants, could result in parasites assuming different or greater significance in altered environments.

In summary, although parasitism is a universal phenomenon in wild birds and many parasites have been observed and described, the information is still fragmentary and largely descriptive in nature. Little is known about the effect of most parasites on their hosts and almost nothing is known about interactions among the parasites that make up parasite assemblages or communities. The cost of parasites to their hosts is difficult to measure, but using energy as a currency may be a fruitful way to understand how costs are incurred, why birds must make trade-offs that influence both their exposure and resistance to parasites, and how being parasitized may affect basic life history traits including reproduction and susceptibility to predation. Parasitism can never be considered in isolation; it must always be considered in terms of the context in which it is occurring and this consideration must include the potential effects of anthropogenic changes.

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