Hayward AD, Nussey DH, Wilson AJ, Berenos C, Pilkington JG, Watt KA, Pemberton JM, Graham AL. 2014. Natural selection on individual variation in tolerance of gastrointestinal nematode infection. PLoS Biol. 12:e1001917.

Medina I, Langmore NE. 2016. The evolution of acceptance and tolerance in hosts of avian brood parasites. Biol Rev Camb Philos Soc. 91:569–577.

Råberg L. 2014. How to live with the enemy: understanding tolerance to parasites. PLoS Biol. 12:e1001989.

Råberg L, Graham AL, Read AF. 2009. Decomposing health: tolerance and resistance to parasites in animals. Philos Trans R Soc Lond B Biol Sci. 364:37–49.

Conspecific brood parasites can also help us understand the evolution of tolerance: a comment on Avilés

Bruce E. Lyon,[1,] John M. Eadie,[2] and Daizaburo Shizuka[3,]

[1]Department of Ecology and Evolutionary Biology, University of California, Santa Cruz, CA 95064, [2]Ecology and Animal Behavior Graduate Groups, Department of Wildlife, Fish and Conservation Biology, University of California, Davis, CA 95616–8751, and [3]School of Biological Sciences, University of Nebraska–Lincoln, Lincoln, NE 68588-0118, USA

In a thought-provoking review, Avilés (2017) points out that interspecific avian brood parasites have been the focus of intense study for traits that reduce the occurrence of parasitism (resistance) but that traits that reduce the impact of parasitism on host fitness (tolerance) have received little attention. Interestingly, one of the potential tolerance traits that Avilés (2017) discusses, the evolution of host clutch size in response to brood parasitism, has already received theoretical attention for another form of parasitism, brood parasitism within species (conspecific brood parasitism, CBP). CBP is widespread in birds (>230 spp.) and has increasingly been studied in a life history context (Lyon and Eadie 2008), including clutch size responses of hosts (Andersson and Ericksson 1982). We suggest that host adjustment of clutch size in the face of conspecific brood parasitism is directly aligned with the tolerance mechanisms discussed by Avilés (2017), and we suggest that existing models exploring such responses can be informative in understanding the evolution of tolerance generally. Although the magnitude of the costs to hosts often differs between interspecific and conspecific brood parasitism, the qualitative life history responses of hosts should be similar.

In a pioneering paper, Andersson and Eriksson (1982) modeled how CBP might favor the evolution of host clutch size. Optimal host clutch size was modeled assuming a direct cost of parasitism to hosts; specifically, per offspring survival decreased linearly with total brood size. The optimal solution for hosts with this cost function is to reduce their own clutch size by half an egg for each egg parasitic egg added to the nest (Andersson and Eriksson 1982). Although the model was derived simply to explore host clutch size responses generally, the model illustrates how this type of theory can be valuable both in the context of distinguishing between tolerance and resistance, and in evaluating the effectiveness of tolerance. By definition, a mechanism that helps the host by reducing the success of the parasite is resistance, not tolerance. These two mechanisms are not mutually exclusive but any mechanism that does not negatively affect the parasite can be viewed as entirely tolerance.

Further consideration of the Andersson and Eriksson (1982) model indicates that the adaptive host response they modeled involves only tolerance: a reduction in host clutch size actually benefits the parasite because with fewer host eggs the total clutch size is smaller and the per offspring survival is higher for both host and parasite. This specific outcome—tolerance rather than resistance—is likely a result of the specific cost function that was assumed. It would be worth exploring whether there are conditions under which host clutch adjustment also reduces parasite success and would thus be considered resistance. Other models have also examined how host clutch size evolves in response to CBP and it would also be profitable to revisit these models in the context of both tolerance and resistance (Power et al. 1989; Yamauchi 1993; Lyon 1998).

Many studies, including Avilés (2017), highlight that the evolution of tolerance could explain the lack of resistance in some taxa. However, some forms of tolerance might involve shifts in allocation in response to parasitism without actually reducing the costs of parasitism in a large way (i.e. small fitness benefits can drive life history evolution). Here, theory can be helpful to explore the degree to which tolerance mechanisms reduce the lifetime fitness costs of parasitism. As a specific example, the Andersson and Eriksson (1982) model can be used to explore not only optimal host strategies under parasitism, but also the degree to which the host tolerance strategies reduce the fitness costs of parasitism. For example, for a given number of parasitic eggs, one could compare the reproductive success of unparasitized females, parasitized females without tolerance, and females with tolerance. We illustrate this approach using Andersson and Eriksson's (1982) model with 2 different costs of parasitism to the host (the slope parameter a in the function relating per offspring survival to total brood size; see Supplementary Figure). Surprisingly while the model predicts that the optimal host response is to reduce their own clutch size (tolerance), the actual fitness benefits of doing so are quite small (Supplementary Figure). This is interesting for 2 reasons: first, it suggests that careful consideration of how much (or how little) tolerance reduces the impact of parasitism is essential—indeed, tolerance might pay small dividends. Second, this analysis illustrates that models that have already been developed in the context of CBP may be valuable to examine the evolution of tolerance more broadly. Understanding the degree to which tolerance diminishes the costs of parasitism would be informative with respect to the issue of tolerance-resistance trade-offs. Similar comparisons could be done for any models of tolerance. One final factor to keep in mind is the difference between a response that optimizes current reproductive success, as all of the examples discussed here address, and a response that optimizes the trade-off between current and future reproduction (Williams 1966), as modeled by Servedio & Hauber (2006) and Medina et al. (2017) for interspecific brood parasitism.

Address correspondence to B.E. Lyon. E-mail: belyon@ucsc.edu
http://orcid.org/0000-0001-8733-9944

SUPPLEMENTARY MATERIAL

Supplementary material can be found at https://academic.oup.com/beheco/

Editor-in-Chief: Leigh Simmons

Abolins-Abols and Hauber • Applying the framework and concepts of parasitology to avian brood parasitism: a comment on Avilés

523

REFERENCES

Andersson M, Eriksson MOG. 1982. Nest parasitism in goldeneyes *Bucephala clangula* - some evolutionary aspects. Am Nat. 120:1–16.

Avilés JM. 2017. Can hosts tolerate avian brood parasites? An appraisal of mechanisms. Behav Ecol. 29:509–519.

Lyon BE. 1998. Optimal clutch size and conspecific brood parasitism. Nature. 392:380–383.

Lyon BE, Eadie JM. 2008. Conspecific brood parasitism in birds: a life history perspective. Annu Rev Ecol Evol Syst. 39:343–363.

Medina I, Langmore NE, Lanfear R, Kokko H. 2017. The evolution of clutch size in hosts of avian brood parasites. Am Nat. 190: E112–E123.

Power HW, Kennedy ED, Romagnano LC, Lombardo MP, Hoffenberg AS, Stouffer PC, McGuire TR. 1989. The parasitism insurance hypothesis - why starlings leave space for parasitic eggs. Condor. 91: 753–765.

Servedio MR, Hauber ME. 2006. To eject or to abandon? Life history traits of hosts and parasites interact to influence the fitness payoffs of alternative anti-parasite strategies. J Evol Biol. 19:1585–1594.

Williams GC. 1966. Natural selection, the costs of reproduction, and a refinement of Lack's principle. Am Nat. 100:687–690.

Yamauchi A. 1993. Theory of intraspecific nest parasitism in birds. Anim Behav. 46:335–345.

Tolerance in hosts of brood parasites: a comment on Avilés

Iliana Medina[a,ORCID] and Naomi E. Langmore[b,ORCID]

[a]School of Biosciences, University of Melbourne, Royal Parade, Melbourne, VIC, 3010, Australia and [b]Department of Ecology and Evolution, Australian National University, Daley Road, Canberra, ACT, 0200, Australia

Biologists have long been fascinated by coevolutionary interactions, such as those between pollinators and flowers, predators and prey or parasites and hosts. These interactions have caused great interest because they are important drivers of the evolution of traits in many taxa. For instance, in antagonistic coevolution exploiters select for the evolution of defences in their victims, which in turn select for counter-adaptations in the exploiters, giving rise to an evolutionary arms race. But what happens if the exploiter "wins" the arms race, or if defences are too costly for victims to evolve? Another possible outcome of antagonistic coevolution is the evolution of tolerance in victims; instead of evolving defences to reduce the number of successful enemy attacks, victims evolve ways to minimize the impact of attacks (Svensson and Råberg 2010). For instance, many plants invest in the production of a massive number of seeds instead of investing in the production of thorns or chemicals to deter herbivores. The evolution of tolerance in hosts of avian brood parasites is the focus of the new review by Avilés (2017).

Brood parasitism is a classic example of coevolution between hosts and parasites. Brood parasites lay their eggs in the nests of their hosts and abandon their young entirely to the care of the host (Davies 2000). Parasitism often results in the death of the host's young, and this high cost has selected for a suite of defences in hosts, including mobbing of adult brood parasites (Feeney

et al. 2012), and rejection of parasite eggs and chicks (Langmore et al. 2003). However, a long-standing puzzle is why some hosts fail to evolve defences despite experiencing high costs of parasitism. A possible solution to this puzzle is that hosts have evolved tolerance towards parasites and thereby significantly reduced the costs of parasitism (Svensson and Råberg 2010; Medina and Langmore 2016). However, evidence of tolerance in hosts of brood parasites is scarce. Avilés (2017) argues that there are two main reasons for this. First, most of the literature has focused on the arms race between hosts and parasites, which is mainly driven by resistance, not tolerance, strategies. Second, the literature tends to focus on highly virulent parasites, which kill all the progeny of the host. In this case, there is very little space for tolerance to operate, because it is difficult to decrease the costs of such a negative interaction. However, Avilés (2017) proposes some mechanisms related to clutch size adjustment and breeding frequency that might have evolved as tolerance adaptations. These mechanisms have been suggested previously in the literature, but they have not been tested explicitly and recent evidence suggests that the logic behind some of these arguments may be flawed (Medina et al. 2017).

A particularly novel contribution of Avilés (2017) review is to propose an experimental framework for detecting the effects of tolerance in future studies. Avilés proposes investigating the repertoire of phenotypic fitness responses of a host genotype along a gradient of intensity of brood parasitism, using a reaction norm approach. Such an approach has been applied to studies of nematode parasites in sheep (Hayward et al. 2014), but has not yet been adopted in studies of avian brood parasitism. He also suggests manipulating perceived risk of parasitism to test for phenotypically plastic tolerance responses.

An interesting point raised in Avilés review is that our current observations might already be the result of tolerance to brood parasites in the past, and the current costs of parasitism are already lower than they were at the beginning of the interaction. This idea is of course very difficult to test, but could explain why demonstrating tolerance has proved difficult. To build on Avilés' point, a potential interesting avenue for future work would be to measure the costs of parasitism in species that have been hosts for a long time, and compare this to the costs of recently parasitized hosts, which presumably have not had as much time to evolve tolerance. If some tolerance mechanisms are behavioral and plastic (e.g., feeding patterns, renesting, hatching synchrony) then they might evolve and spread very rapidly in a population, giving us little opportunity to detect such changes.

Overall, Avilés' review highlights an important and neglected area of brood parasite—host interactions and proposes a promising framework for future investigations. Very little is known about the evolution of tolerance in hosts of brood parasites, but the potential for taking this research in new directions is exciting.

REFERENCES

Avilés JM. 2017. Can hosts tolerate avian brood parasites? An appraisal of mechanisms. Behav. Ecol. 29:509–519.

Davies NB. 2000. Cuckoos, cowbirds and other cheats. T. & A.D. Poyser. Bloomsbury Publishing PLC. p. 310.

Supplemental Figure. We use the model by Andersson and Eriksson (1982) to show how models of optimal host clutch size under within-species (conspecific) brood parasitism can be used to quantitatively explore the effectiveness of clutch size adjustment as a tolerance mechanism. The model was developed for precocial birds like goldeneye ducks (*Bucephala clangula*) and assumed that the proportion of offspring fledged P decreased in a negative linear relationship with the total number of eggs in a nest, b. Thus, the proportion of hatched offspring that survive to fledge is $P = 1 - ab$, where the constant a is the slope of the relationship between total clutch size (host plus parasite eggs) and offspring survival. The larger the value of a, the greater the strength of negative density-dependence within the brood, and the higher the cost of brood parasitism to hosts. With this assumed pattern of offspring survival, the optimal clutch response for a parasitized host is to reduce the number of eggs she lays by half an egg for each egg the parasite lays in her nest (see Andersson & Eriksson 1982). If we now evaluate a female's fitness under two other scenarios—in the absence of parasitism (solve for optimal host clutch size with zero parasite eggs) and with parasitism but without tolerance (the host lays the clutch size that would be optimal in the absence of parasitism but parasitic eggs are also added to the clutch)—we can determine the effectiveness of tolerance for recouping fitness lost due to parasitism. In all three scenarios, once the host's optimal clutch size is determined, her fitness is determined by multiplying her clutch size by the value of P determined for the total number of eggs in the nest, both host and parasite. Comparing these three fitness estimates indicates the degree to which tolerance recoups fitness lost to parasitism and hence the relative fitness benefit of tolerance. Following Andersson & Eriksson's (1982) original analysis, we assumed that parasites lay 6 eggs in the host nest and contrasted host fitness estimates for two different values of the slope parameter, $a = 0.03$ and $a = 0.05$ (in their Figure 2 Andersson and Eriksson explored optimal host clutch size when parasites lay 4 eggs or 8 eggs (we assumed 6 parasitic eggs, the mean of these two values) and they assumed a = 0.05 (we assumed this value and also the less costly value of 0.03)). Under these assumptions, tolerance recoups only a small amount of the fitness costs of brood parasitism (Figure).

References

Andersson M, Eriksson MOG. 1982. Nest parasitism in goldeneyes *Bucephala clangula*—some evolutionary aspects. *Am Nat.* 120:1–16