EVOLUTION IN A HOST-PARASITE SYSTEM

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Some organisms employ multiple defence strategies against their enemies, while others fail to employ a defence that seems obvious. We shall investigate three questions for host-parasite systems.

(1) Under what circumstances does it pay for a host to employ a given defence strategy against one of its parasites?
(2) If alternative strategies are available, how is the appropriate strategy chosen?
(3) When is it appropriate to employ multiple defence strategies against an enemy?

We shall illustrate our results in two cases of brood parasites and their hosts. The paper by Britton *et al.* (2007) contains more background details on the basic model and the analysis but the extensions to the model and some of the results are new.

1. Introduction

1.1. *General introduction*

Flax (*Linum usitatissimum*) has twenty-six defensive genes conferring resistance to flax rust (*Melampsora lini*), but each such gene is countered by an attacking gene in the rust (Flor 1956). This situation may have come about through an *arms race* (Dawkins and Krebs, 1979), a succession of defensive gambits in the flax each countered by the rust, in a process known as *gene-for-gene coevolution*.

Passion-vines (*Passifloraceae*) produce toxic compounds as a general defence against herbivores. *Heliconius* butterfly larvae have overcome these, and the passion-vines employ more specialised defence strategies, such as hooks to immobilise *Heliconius* larvae and structures that mimic *Heliconius* eggs, against them (Gilbert 1983). This seems to be another example of an arms race.

Swollen-thorn acacias have a mutualistic relationship with ants; they provide shelter to the ants within their thorns, and in return the ants deter herbivores from eating the acacia (Janzen 1966). Other acacias synthesis toxic cyanogenic glycosides to deter herbivores, but none does both, and so no arms race seems to have taken place in this case (Rehr *et al.* 1973).

Several vertically transmitted bacterial symbionts provide resistance in pea aphids (*Acyrthosiphon pisum*) against the parasitoid wasp *Aphidius ervi*, but are only seen at intermediate frequencies in natural populations. Any one pea aphid very rarely harbours more than one species of bacterial symbiont, so multiple strategies are very rarely employed (Oliver et al. 2003). Again, no arms race has occurred here.

Hedgehogs (small spiny mammals of the subfamily *Erinaceinae* and the order *Erinaceomorpha*) have two alternative defence strategies to their predators, to run or to roll up into a ball. These are true alternatives: it is not possible to employ both these strategies at once.

1.2. *Rare enemy effect and strategy-blocking*

Co-evolutionary arms races seem to occur in some cases but not in others. Dawkins (1982) introduced the concept of the *rare-enemy effect*, arguing that because there are costs involved in any adaptation, it is not advantageous to develop a defence against a rare enemy. This may explain the lack of an arms race in some cases. In the example of brood parasites that we shall consider later the enemy is not particularly rare, but we shall show that when there are two possible defence strategies that may be deployed by a host against a parasite, each of which is advantageous on its own, an extension of the rare-enemy effect may be used to understand when a combination of the two is advantageous. One strategy may prevent the appearance of the other, a phenomenon we shall call strategy-blocking.

1.3. *Brood parasite natural history*

A general treatment of this area is given in Davies (2000). Brood-parasitic birds lay their eggs in the nest of another bird, the host; if the parasitism

is successful the host raises the parasite offspring to independence. Many brood-parasitic chicks, when they hatch, eject all host eggs or chicks from the nest, so that they are raised alone. The following questions arise.

- Is it evolutionarily advantageous for a host to defend itself against a brood parasite?
- In particular, is it advantageous for the host to develop a strategy for ejecting brood-parasitic eggs from its nest?
- Is it advantageous for the host to develop a strategy for ejecting or deserting brood-parasitic chicks in its nest?

The archetypal Old-World brood parasite is the Eurasian cuckoo (*Cuculus canorus*), which parasitises several host species. Reed warblers (*Acrocephalus scirpaceus*) often recognise cuckoo eggs and reject them. In response to this, cuckoos lay eggs that mimic those of the reed warbler. On the other hand reed warblers never recognise cuckoo chicks, and will raise them as their own. The first stage of an arms race has taken place, but not the second. Dunnocks (*Prunella modularis*) do not even recognise cuckoo eggs, which are quite unlike dunnock eggs. Not even the first stage of the arms race has occurred. In Australia, superb fairy-wrens (*Malurus cyaneus*) fail to recognise the eggs of their brood parasites, Horsfield's bronze-cuckoo (*Chrysococcyx basalis*), but do sometimes desert their nest once the bronze-cuckoo chick has ejected all their offsring. They and reed warblers use alternative rejection strategies. There is no known example of a host species that rejects both the eggs and the chicks of its brood parasite. A very simple explanation of this fact could be that in no case has sufficient evolutionary time passed for both rejection behaviours to evolve. In this paper we shall ask whether a deeper reason exists.

2. Modelling

2.1. *Monomorphic populations*

Our model is based on the archetypal Nicholson–Bailey (1935) model for a host-parasitoid system in discrete time:

$$P' = c(1 - f(P))H, \quad H' = RHf(P),$$

where P and H are the numbers in the parasitoid and host populations, R is the basic reproductive ratio of the host population, $f(P)$ is the fraction of hosts that escape parasitism, c is the mean number of parasitoids from each parasitised host that survive to breed, and there is no survival between

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generations. An acknowledged problem with this model is a lack of self-limitation, with consequent unlimited oscillatory growth of the populations, and we shall introduce self-limitation into the host population only, for simplicity. To adapt the model for brood parasites we also require survival between seasons. The equations become

$$P' = (1 - \mu)P + c(1 - f(P))H,$$
$$H' = H\Psi(H)(1 - \nu + Rf(P)).$$

where Ψ is the self-limitation function, e.g. $\Psi(H) = 1/(1 + H/k)$, and μ and ν are the annual probabilities of death of parasites and hosts in the absence of density-dependent effects. Note that the host steady state H^* in the absence of parasitism is given by $H^* = k(R - \nu)$, an increasing function of k, which may therefore be thought of as the richness of the environment.

Defence is not taken into account in the models above. Let a fraction $1 - g$ of parasitised hosts successfully defend themselves against parasitism (and hence produce hosts in the next season), leaving a fraction g that fail to do so (and hence produce parasites in the next season). Let this defence be *cost-free*. The equations become

$$P' = (1 - \mu)P + cg(1 - f(P))H,$$
$$H' = H\Psi(H)\left(1 - \nu + Rf(P) + R(1 - g)(1 - f(P))\right).$$

Defence costs are of two kinds. Parasite-independent costs are incurred whether or not the parasite is present. It costs to have an immune system, whether or not it is ever used to fight off a disease. If a host defends against brood parasites by ejecting eggs that it believes to be parasitic, it will occasionally make a false-positive identification error and eject one of its own eggs, even if no parasite is present. Parasite-dependent costs are only incurred when the parasite is present. One example is the cost of fighting off a microparasitic disease. In the brood parasite case, a strategy of rejecting the parasitic chick is costly if that chick has time to eject host brood before it is rejected.

Incorporating the cost of defence into the model for brood parasites, the equations become

$$P' = (1 - \mu)P + cg(1 - f(P))H,$$
$$H' = \Psi(H)Hw(P),$$

where the *relative fitness function* w is given by

$$w(P) = 1 - \nu + R\theta f(P) + (1 - g)R\phi(1 - f(P)).$$

Here ϕ and θ are pay-offs relative to non-defending hosts with and without parasitism, taking into account parasite-dependent and parasite-independent costs.

2.2. Dimorphic populations

It is straightforward to generalise the model to two parasite and two host types. The equations become

$$P_0' = (1 - \mu_0)P_0 + c_{00}g_{00}\bar{f}_{00}(P_0, P_1)H_0 + c_{01}g_{01}\bar{f}_{01}(P_0, P_1)H_1,$$

$$P_1' = (1 - \mu_1)P_1 + c_{10}g_{10}\bar{f}_{10}(P_0, P_1)H_0 + c_{11}g_{11}\bar{f}_{11}(P_0, P_1)H_1,$$

and

$$H_0' = \Psi_0(H_0, H_1)H_0 w_0(P_0, P_1),$$

$$H_1' = \Psi_1(H_0, H_1)H_1 w_1(P_0, P_1),$$

where $w_i(P_0, P_1)$ is the relative fitness of type i, and

$$\begin{aligned}
w_i(P_0, P_1) = {} & 1 - \nu_i + R\theta_i f_i(P_0, P_1) \\
& + (1 - g_{0i})R\phi_{0i}\bar{f}_{0i}(P_0, P_1) + (1 - g_{1i})R\phi_{1i}\bar{f}_{1i}(P_0, P_1).
\end{aligned}$$

Here

$$\bar{f}_{ji}(P_0, P_1) = \mathbb{P}\left\{H_i \ is \ \text{parasitised by} \ P_j\right\},$$

$$f_i(P_0, P_1) = \mathbb{P}\left\{H_i \ \text{is not parasitised by} \ P_0 \ \text{or} \ P_1\right\},$$

so

$$f_i(P_0, P_1) = 1 - \bar{f}_{0i}(P_0, P_1) - \bar{f}_{1i}(P_0, P_1).$$

The simplest model generalises $f(P) = e^{-aP}$ in Nicholson–Bailey:

$$\bar{f}_{ji}(P_0, P_1) = \frac{P_j}{P_0 + P_1}\left(1 - \exp(-a(P_0 + P_1))\right)$$

$$f_i(P_0, P_1) = \exp(-a(P_0 + P_1)).$$

The costs of counter-attack may be encoded in the c_{ij} parameters.

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2.3. *Some extensions of the model*

2.3.1. *Continuous trait values*

In some cases strategies are better described by continuous variables, e.g. to quantify how much resource is devoted to defence (for hosts) or attack (for parasites). Let the parasite trait value be $x \in [0, 1]$, and the host trait value $y \in [0, 1]$. Let $P(x)$ and $H(y)$ be densities of parasite and host populations in terms of their trait values. With some simplifications, this leads to the following system of integro-difference equations:

$$P(x)' = (1 - \mu)P(x) + c\frac{1 - f(\|P\|)}{\|P\|}P(x)\int_0^1 g(x, y)H(y)dy,$$

where $\|P\| = \int_0^1 P(x)dx$, $f(\|P\|) = \exp(-a\|P\|)$ (in the simplest model), with

$$H(y)' = \Psi(y, H)H(y)w(y, P),$$

and

$$w(y, P) = 1 - \nu + R\theta(y)f(\|P\|)$$
$$+ R\phi(y)\frac{1 - f(\|P\|)}{\|P\|}\int_0^1 (1 - g(x, y))P(x)dx;$$

$w(y, P)$ is the relative fitness of host y in an environment of parasites P.

Much of the bifurcation analysis that we describe later can be extended to this system, but we shall not do this here.

2.3.2. *Inclusion of mutation*

Let an offspring of a parasite of type ξ be of type x with probability density $M(x, \xi)$, where $\int_0^1 M(x, \xi)dx = 1$, M typically positive and symmetric, and similarly for host mutation. Define $\hat{P}$, offspring in the absence of mutation, by

$$\hat{P}(x) = c\frac{1 - f(\|P\|)}{\|P\|}P(x)\int_0^1 g(x, y)H(y)dy,$$

With mutation, the equation becomes

$$P(x)' = (1 - \mu)P(x) + \int_0^1 M(x, \xi)\hat{P}(\xi)d\xi.$$

Similarly for host mutation, with kernel $N(y, \eta)$:

$$\hat{H}(y) = R\theta(y)f(\|P\|)H(y)$$
$$+ R\phi(y)\frac{1 - f(\|P\|)}{\|P\|}H(y)\int_0^1 (1 - g(x, y))P(x)dx,$$

$$H(y)' = \Psi(y, H)\left((1 - \nu)H(y) + \int_0^1 N(y, \eta)\hat{H}(\eta)d\eta\right).$$

We again have a system of integro-difference equations, but now with double integrals. Again, many results may be extended to this case, but we shall not do this here.

3. Analysis

We return to the essentially ecological model for two parasite types and two host types, with some simplifications,

$$P_i' = (1 - \mu)P_i + cg_{i0}\bar{f}_{i0}(P_0, P_1)H_0 + cg_{i1}\bar{f}_{i1}(P_0, P_1)H_1,$$

$$H_i' = \Psi(H)H_i w_i(P_0, P_1),$$

with $H = H_0 + H_1$ (host types ecologically identical), and with

$$w_i(P_0, P_1) = 1 - \nu + R\theta_i f_i(P_0, P_1)$$
$$+ (1 - g_{0i})R\phi_{0i}\bar{f}_{0i}(P_0, P_1) + (1 - g_{1i})R\phi_{1i}\bar{f}_{1i}(P_0, P_1).$$

For the results in this article, all we need is to consider invasion eigenvalues. As an example, consider whether a mutant H_1 employing strategy 1 will invade a steady state $(P_0^*, 0, H_0^*, 0)$ consisting of hosts and parasites all employing strategy 0. Linearising about this steady state, H_1 will invade if

$$\lambda = \Psi(H_0^*)w_1(P_0^*) > \Psi(H_0^*)w_0(P_0^*) = 1,$$

or $w_1(P_0^*) > w_0(P_0^*)$. Very simply, *the fittest host wins*. If the growth of two types is only limited by a common parasite P, then they are in *apparent competition*: the one that can survive a higher parasite population survives, and drives the other to extinction (Holt, 1977). At first sight it seems that we shall never see host populations mixed for defensive strategy. However, we shall see later that this is not the case, and our model does not preclude co-existence of two host types.

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4. Results

4.1. *Alternative strategies*

Let host and parasite strategies 0 and 1 be true alternatives, i.e. it is not physically possible to employ *both* 0 and 1. Let parasite strategy 0 be the counter-attacking strategy to host strategy 0, and parasite strategy 1 the counter-attacking strategy to host strategy 1. Let host strategy 1 be the defence to parasite strategy 0, and host strategy 0 the defence to parasite strategy 1.

We might expect strategies to cycle:

H_0 high $\implies P_0$ high $\implies H_1$ high $\implies P_1$ high.

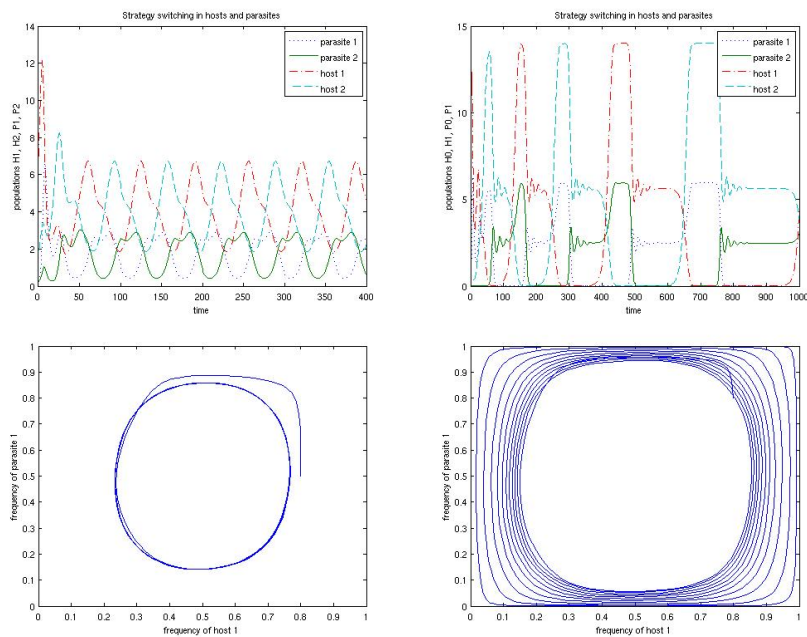Numerical results show that this is indeed the case.

Figure 1.   Cycling strategies. Depending on the parameters of the system it either tends to a periodically cycling solution (left panels) or to a singular solution that switches between the points at the corners of the frequency diagram (right panels).

4.2. *Arms races*

Let H_0 and P_0 be resident naive (no defence, no counter-attack) host and parasite types, and assume that a mutation occurs that produces a defending host type H_1. If the benefits of the defence outweigh the costs of deploying it, then (in the absence of stochastic extinction) we expect this mutation to invade the resident steady state and go to fixation. In mathematical terms, the mutant type H_1 invades the naive steady state $(P_0^*, 0, H_0^*, 0)$ and goes to fixation, resulting in a steady state $(P_0^*, 0, 0, H_1^*)$. Now assume that a mutation occurs in the parasite population, leading to a mutant counter-attacking parasite P_1^*. If the benefits of the counter-attack outweigh its costs, the mutant parasite type will invade the steady state and go to fixation, resulting in a steady state $(0, P_1^*, 0, H_1^*)$. Mathematically, invasion eigenvalues may be calculated which determine whether the invasions occur. With *cost-free* defence and counter-attack, invasion of both types occurs. The process may be repeated indefinitely, leading to a gene-for-gene arms race such as the one that seems to have occurred for flax and flax rust.
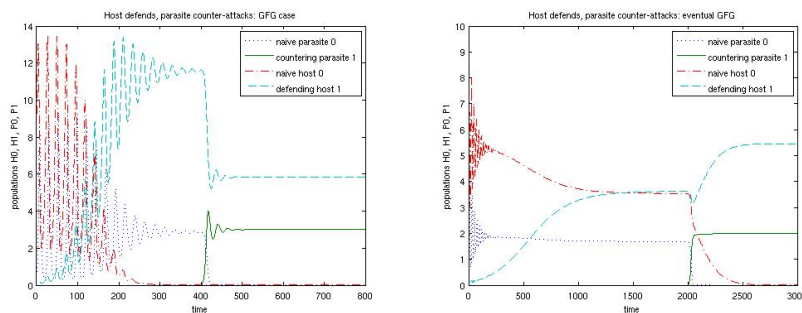

Figure 2.   A stage in an arms race: a naive host type is replaced by a defending one, and the parasite counter-attacks.

4.3. *Bifurcation diagrams with costly defence*

If defence is costly, things are more complicated. Typical fitness functions are as shown in figure 3 (if the defence has a cost even in the absence of parasitism).

A bifurcation analysis may be carried out with richness of the environment k as the bifurcation parameter. This may be done rigorously, but here
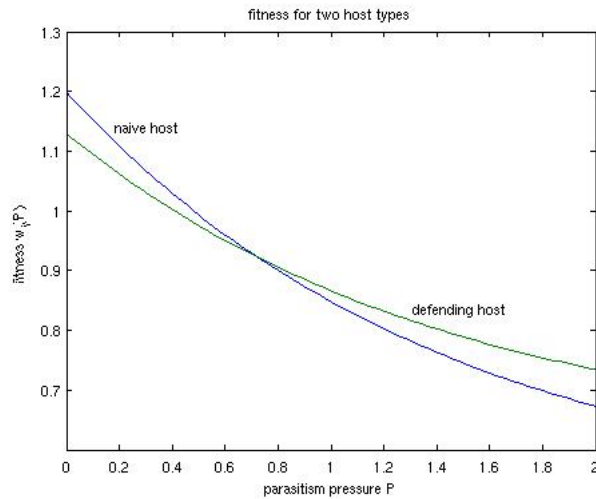
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Figure 3.   Typical fitness curves for a naive and a defending host, as functions of the parasitism pressure (population) P. Since the defence has a cost in the absence of parasitism, the naive host is fitter for low values of P.

we give an algorithm for constructing the qualitative features of the bifurcation diagram, as the richness of the environment k increases from zero, when there is a single parasite type and two host types, one that employs a defence against the parasite and one that does not, as in figure 3. The extension to more than two host types is straightforward. Note that as k increases then H^* increases and so P^* increases, so the idea is to construct the bifurcation diagram from the host fitness diagram. Note also, however, that H^* and P^* are not in general *strictly* increasing with k.

- As k increases from zero then so does the steady state host population size H^*, but for low values of k it is not sufficient to support the parasite. The fittest host type employs the naive (no-defence) strategy, and this is the type that persists.
- A bifurcation point occurs (at about $k = 25$ in figure 4) beyond which the parasite population is supported by the host, and both populations strictly increase with k. The fittest host type still employs the naive strategy.
- As k increases further, H^* and P^* increase until we reach a new bifurcation point (at around $k = 50$ in figure 4) where the naive and the defending host are equally fit. Beyond this point either (i) P^*

remains constant while the naive host is gradually replaced by the defending host (as in figure 4), or (ii) the naive host is immediately replaced by the defending host. The first of these alternatives is typical for brood-parasite systems.

- Once the naive host has been replaced by the defending host, the parasite population resumes its increase as the defending host population increases.
- Note that as $k \to \infty$ we tend to the point on the fitness diagram where host fitness drops to 1; however rich the environment it is not possible to progress beyond this point.
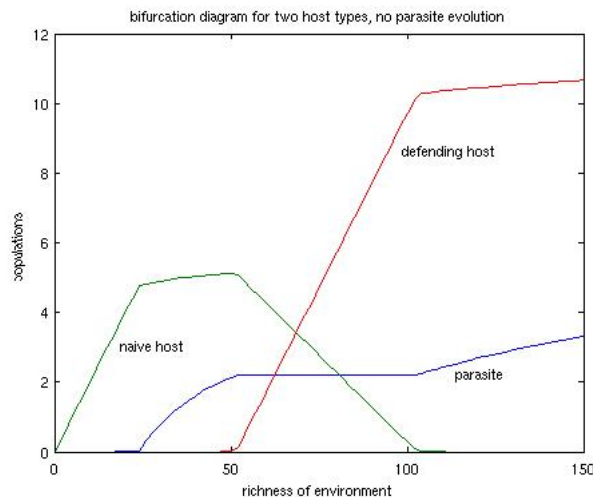

Figure 4. Bifurcation diagram for the fitness curves of figure 3, with bifurcation parameter k. Note the gradual replacement of naive host by defending host while the parasite population remains constant (so that each host type has equal fitness), between about $k = 50$ and $k = 100$.

We can therefore describe the bifurcation diagrams for the parameters of our brood-parasite systems. For reed-warbler–cuckoo parameters the fitness diagram is as in figure 5, we expect no defence at low cuckoo densities, egg-rejection at high cuckoo densities, or a mixture of the two, but no other defence strategies. However rich the environment is, the cuckoo chick can never be other than a *rare enemy*, not worth defending against.

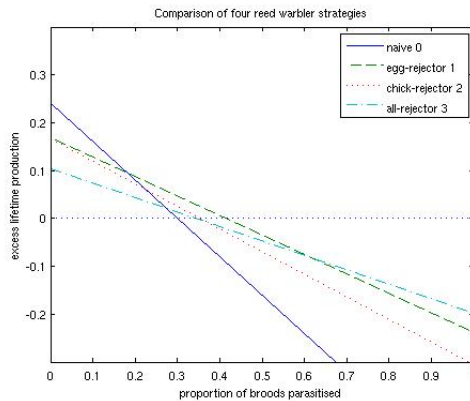For fairy-wren parameters the fitness diagram is as in figure 6, and we

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Figure 5.   The figure is in terms of excess lifetime production v rather than fitness w; these are related by $v = (w - 1)/\nu$, so we cannot progress beyond $v = 0$. The only host types we shall see are therefore naive or egg-rejectors, or a mixture of the two. Note that chick-rejection and all-rejection, although they are advantageous compared to the naive strategy at some parasitism rates, are blocked by the egg-rejection strategy.
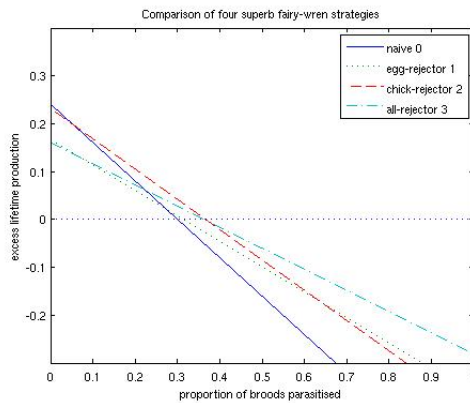

Figure 6.   Again we cannot progress beyond $v = 0$. Therefore, for these parameters, the only host types we shall see are naive or chick-rejectors, or a mixture of the two. Note that here egg-rejection and all-rejection, although they are advantageous compared to the naive strategy at some parasitism rates, are blocked by the chick-rejection strategy.

expect no defence at low cuckoo densities, chick-rejection at high cuckoo densities, or a mixture of the two, but no other defence strategies. However rich the environment is, the cuckoo egg can never be other than a *rare enemy*, not worth defending against.

The essential difference between the two sets of parameters that leads to this difference in outcome is the high fitness of fairy-wren chick rejectors at low P. This results from the much higher probability that fairy-wrens can raise a successful brood in the same season after deserting a nest, because of the longer Australian breeding season and the shorter fairy-wren hatching time.

5. Conclusions

- If defence is cost-free, even rare enemies are worth defending against, and an arms race should be expected.
- If defence has parasite-independent costs, then we expect no defence against rare parasites, and defence against common parasites, with a mixture of defensive strategies at intermediate levels.
- One strategy may prevent another otherwise advantageous strategy from appearing, a phenomenon known as *strategy-blocking*.
- If host basic reproductive ratio is small, then however rich the environment there will never be enough parasites to make costly defence worthwhile.
- There is no fundamental reason for hosts not to reject both eggs and chicks, despite this never having been observed in nature. We might expect this to happen for high R/ν and high k.

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