This model assumes environmental transmission, but where the parasite actively seeks out hosts. Contact is thus controlled by the parasite (so quantifies both the contact and infection processes), and contact is assumed to remove parasites from the environment. There are two hosts. I assume that the resident parasite strain exploits a single host and ask whether a mutant that exploits both hosts can invade. I assume that the cost of this generalism is that the shedding rate from both hosts is reduced by a factor *a*.

I am going to explore the consequence of varying the carrying capacity and host mortality rate on the evolution of generalism. As such, I will let and be the carrying capacities of the first and second host, respectively; similarly, will be the mortality rates for each host and and will be the shedding rates from each host. For simplicity, I will assume that all other parameters (traits) are equal between the hosts and parasites.

The full system is then

For the invasion analysis, we assume that the resident parasite comes to its ecological equilibrium with the hosts. Since it does not parasitize the second host, will go to its carrying capacity. The endemic equilibrium host densities are

Evaluating the stability of this equilibrium is a pain, but for it to have any hope of being stable (which invasion analysis usually assumes the equilibrium is), the extinction equilibrium must be unstable. This equilibrium is unstable if

This defines for this parasite (note that is the fraction of the host population that remains susceptible at equilibrium). An equivalent representation of this condition is

where is the probability that a spore in the environment infects a host and is the expected number of spores produced per infected host.

To determine whether a mutant parasite can invade the system, we are essentially asking whether the mutant parasite-free equilibrium of the full system is unstable. To evaluate at this, we can take advantage of the fact that the Jacobian matrix at this equilibrium is upper block-triangular (that is, it can be written as)

The eigenvalues of this matrix (which determine the stability of the equilibrium) are given by the eigenvalues of the matrices and . The eigenvalues of determine the stability of the resident-only system – by assumption, the resident-only system is stable. Thus, we only need to look at the eigenvalues of :

Applying the next-generation matrix theorem, we can determine that will have a positive eigenvalue (and thus, the mutant-free equilibrium will be unstable) if

From this, it is clear that if the invading parasite is capable of persisting on the second host alone, it will be able to displace the resident (because is the condition for instability of the parasite extinction equilibrium in the system). As before, an equivalent representation of this condition is

where is the probability that a spore in the environment infects host 1 and is the expected number of spores produced per infected host 1; the second set of terms have an analogous interpretation for host 2. Thus the invasion criterion is simply that, between the two hosts, each spore in the environment is expected to produce more than one new spore in the environment.

If we plug in the resident parasite endemic equilibrium into Eq. 1 (which is a bit simpler and easier to work with), it can be rewritten as

Note that the term on the left must be less than one, since by definition as the cost of generalism. Thus, we require that for invasion to have anychance of succeeding.

At this point, we can plug in values for the different parameters and investigate under what combinations of cost and host traits a generalist can invade. But we can also get a bit more insight by considering how changing the values of host and parasite traits affect the magnitude of the invasion fitness (the expression on the left side of the invasion criterion). That is, we can look at the derivatives of the invasion fitness with respect to host and parasite traits. For example, the derivative of the invasion fitness (let’s call it *r* for simplicity) with respect to *a* is just

which is always positive ( > 1). What this means is that, as the cost of generalism goes down (*a* increases), the invasion fitness increases, meaning that invasion is more likely. This result is, of course, intuitive.

If you increase the carrying capacity of the second host, you also get a very simple effect:

Since we require , this is always positive, implying that generalism is more likely when the alternative host is very abundant. A further implication that you can draw from this is that an abundant host is very unlikely to be unexploited, which is intuitive.

If you increase the mortality rate of infected individuals of the first host, on the other hand, you make it harder for a generalist to invade (this term is always negative because the first term ).

Similarly, if you increase the mortality rate of infected individuals of the second host, it is harder for a generalist to invade.

If you increase the mortality rate of parasites in the environment (), you make it harder for a generalist to invade. In other words, specialism is more likely in harsher environments.

|  |  |
| --- | --- |
| Parameter | Effect of increasing the parameter value on invasion fitness |
| Cost of generalism *a* | Increases |
| Parasite shedding | Increases |
| Second host carrying capacity | Increases |
| Host mortality rates | Decreases |
| Parasite environmental mortality rate | Decreases |

None of these results are surprising, which is itself not surprising – invasion analyses are typically only surprising when there are trade-offs between parameters of the model. For this system, there actually are some potential trade-offs between parameters that are not being considered at the moment.

**Allometric and temperature scaling of host and parasite traits**

In particular, metabolic scaling theory predicts that host body size and temperature will simultaneously affect both host carrying capacity and host mortality rate. Savage et al. (2004) predict the following scaling relationships:

where is the Boltzmann factor, which describes how temperature affects reaction kinetics (e.g., metabolic rate), *W* is body mass*,* and are proportionality constants. *E* is the average activation energy of rate-limiting biochemical metabolic reactions and *k* is Boltzmann’s constant. It is worth noting at this point that increasing mass will decrease the carrying capacity and mortality rate, whereas increasing temperature decreases carrying capacity and increases mortality rate.

Shedding rate also scales with host body size. This is because larger hosts support larger abundances of parasites. Metabolic scaling theory (Poulin & George-Nascimento 2007, Hechinger 2013) predicts that parasite abundance should scale with host body mass to the 3/4 power for endoparasites and with host body mass to the 5/12 power for ectoparasites. (However, in an empirical dataset, Poulin & George-Nascimento calculated that, across 34 species of fish hosts, parasite abundance scaled linearly with host body mass.) Thus, assuming that shedding rate is a linear function of parasite abundance, then

If we plug these allometric functions into our expressions for invasion fitness (e.g., Eq. 1), we can again use the derivatives of the invasion fitness equation with respect to host mass and temperature to see how those affect the likelihood of invasion. For notational simplicity, we will let , where *f* is a scalar and *W* is the mass of the primary host.

*Endoparasites*

To determine how mass affects invasion,

Because every term of this expression is positive, this implies that increasing the masses of the hosts will always make invasion easier. In other words, **it is easier for generalists to invade if the available hosts are large-bodied.** A corollary of this prediction is that **large-bodied hosts are more likely to be infected by generalist than specialist parasites.**

To determine how *f* affects invasion,

which again is guaranteed to be positive. This implies that **it is easier for** a **generalist to invade if it infects hosts of a similar size.**

To determine how temperature affects invasion,

which is guaranteed to be negative, implying that **increasing temperature makes it harder for a generalist to invade.**

*Endoparasites versus ectoparasites*

We could repeat all of this analysis for an ectoparasite, with its different scaling of shedding rate with host body size. On the other hand, if we examine the invasion fitness expression in Eq. 2,

notice that for an endoparasite the expected spore production per infection () is:

whereas for an ectoparasite, it is:

Because shedding rate does not appear in the infections probabilities and , endo- and ectoparasites do not differ here. As such, we can immediately see that **a generalist endoparasite is more likely to be able to invade than a generalist ectoparasite** because they attain a higher abundance and thus shed more.

*Ectoparasites*

For ectoparasites, the effect of host body size (whether through *W* or *f*) is more complicated:

For both of these derivatives, the sign is determined by . Plugging in the scaling functions for and , this expression will be negative (making both derivatives positive) so long as

That is, unless the host is very large, it will be easier for a generalist ectoparasite to invade as host body size increases. Put another way, **there should be few generalist parasites of either very small bodied or very large bodied hosts**. If the primary host is very large, then it will be easier for a generalist to invade if the secondary host is much smaller.

The effect of temperature will be the same for both endo- and ectoparasites: generalists will have an easier time invading when temperatures are colder.

*Numerics*

If we want to be more quantitative, Allen e t al. 2002 report the following empirical relationship (Fig. 2) between temperature *T* (in Kelvin), population density *N*, and mass *W*:

Savage et al. (2004) also presents data on the relationship between temperature, mass, and instantaneous mortality rate for 175 fishes. They report the following relationship (Fig. 3b):

and report the value of *E* for fish to be , with

We can rearrange to find size- and temperature-dependent expressions for carrying capacity and mortality rate:

Some other notes:

For the resident parasite to be able to persist, the

Defining and , then the derivative of the persistence condition is equal to . But

and

Since and , this means that the two terms move the derivative are changing in opposite directions. In particular, it can be shown that if , then increasing *W* will decrease , thereby making it harder for the parasite to persist as the primary host gets larger. Biologically, what this implies is that the probability of contacting a host ( decreases faster than the spore production per infected host () increases. This makes evaluating the influence of host body mass on the evolution of generalism complicated because increasing body mass makes it harder for the specialist to persist. This is one of the reasons why it makes sense to also consider the host mass-dependence of shedding rate as well.