A Model of the Effects of Dispersal Distance on the Evolution of Virulence in Parasites

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Abstract

The effects of differing dispersal distances on the evolution of virulence in parasites was explored through the use of a configuration model. Both hosts and parasites were individually represented on a two-dimensional surface. Each parasite had a "gene" for virulence that determined the amount of energy the parasite removed from its host in a single time step. When a parasite reproduced, its offspring was placed near to, or far away from the parent depending on a dispersal distance parameter. The offspring's virulence was also mutated slightly at reproduction. Distance of dispersal had a small but statistically significant, positive effect on the evolution of the parasite's virulence. That is, those parasites that were forced to disperse their offspring further away, evolved a higher level of virulence than the parasites that dispersed their offspring locally.

Introduction

There is a growing interest in the application of evolutionary theory to disease control and treatment [Ewald, 1983, Williams and Nesse, 1991]. At the same time, computer modeling is opening up new areas of evolutionary theory by allowing us to ask questions that were previously extremely difficult to test in the lab [Koza, 1994, Lindgren and Nordahl, 1994, for example]. This paper represents the cross fertilization of these two research projects in a model of the evolution of virulence in parasites.

The Evolution of Virulence

Why is the parasite that causes the common cold less deadly than the protozoa that causes malaria (genus *Plasmodium*) or the bacteria that cause cholera (*Vibrio cholerae*)? Are these parasites becoming more or less virulent as they coevolve with humans, and why? These questions are neither academic nor intractable.

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The Theory I am using the term parasite in the broadest possible understanding. A parasite is any organism that gains its nutrients from other living organisms and usually has a deleterious effect on those organisms [Begon *et al.*, 1990]. It was long thought that a parasite's relationship to its host would evolve to become less deleterious, or virulent, over time. This was based on an essentially group selectionist, or "good of the species" argument. Parasites that did less damage to their hosts would thereby preserve their hosts for longer and thus live longer themselves as compared to parasites that were more damaging to their hosts. This kind of argument still persists today despite advances in the theory. Anderson and May have published extensive analyses of the coevolution of parasites and hosts.

> ...formal studies make it clear that the coevolutionary trajectory followed by any particular host-parasite association will ultimately depend on the way the virulence and the projection of transmission stages of the parasite are linked together: depending on the specifics of this linkage, the coevolutionary course can be toward essentially zero virulence, or to very high virulence, or to some intermediate grade. [Anderson and May, 1982]

In other words, there are significant pressures on parasites that might select for varying levels of virulence, not just for avirulence or commensalism. This can be seen formally in a simple equation for the reproductive rate given by May and Anderson (1990).

$$R_0 = \frac{\lambda(N)}{\alpha + b + v} \tag{1}$$

Here N is the current population size of the hosts and $\lambda(N)$ is the rate of successful transmission of the parasite offspring to other hosts as a function of the population density of the hosts. The denominator contains the variables that might reduce the pool of hosts available for infection. Here, α is the disease induced death rate of the hosts, b is the death rate for the hosts from causes other than the disease, and v is the recovery rate of the hosts. After a host has recovered, it is considered to be immune to the disease.

Given this relatively simple equation and the assumption that selection puts pressure on the parasites to maximize R_0, May and Anderson show that the virulence of the parasite, α, may be expected to evolve toward any number of results. For example, if transmission, λ, and recovery, v, are independent of virulence, α, then R_0 is maximized by avirulence, $\alpha \rightarrow 0$, a commensal relationship. However, such independence is not the norm. If we assume that the likelihood of transmission, λ, increases as a linear function of the virulence of the parasite, α, then R_0 is maximized by maximum virulence, $\alpha \rightarrow \infty$. This would evolve regardless of the extreme likelihood that the parasites would thereby drive their hosts to extinction and thus perish themselves. May and Anderson (1990) conclude,

> What are needed are factual data about the functional relationships among λ, v and α, derived from the life history details of specific host-parasite associations. In no single instance do we have enough information to determine the likely evolutionary trajectory dictated by maximizing R_0 in [equation 1].

The Observations In fact, parasite reproduction is generally correlated with disease severity [Ewald, 1983]. As Ewald puts it, the question then becomes, "What environmental conditions select for different levels of parasite reproduction and, hence, for different levels of severity?" Two examples help to illustrate the point. Various species of protozoa in the genus *Plasmodium* cause the disease malaria in human hosts. These parasites are transmitted to new hosts through the biting of mosquitoes that transfer small amounts of blood from human to human. The high levels of virulence in these parasites is hypothesized to be an adaptation to their mode of dispersal. By reproducing in great numbers the parasite achieves two effects. First, any small amount of blood taken from the host is likely to contain a few protozoans. Second, by causing a sever reaction, the host is immobilized and thus more easily bitten by mosquitoes. In contrast, the common cold is transmitted through social contacts and so a parasite that immobilized its host would thus be reducing its likelihood of transmission to new hosts. This may help to explain why the cold is a relatively mild affliction.

Ewald (1983) collected data on a large number of human diseases and found a correlation between disease severity and parasites transmitted through biting insects. The cholera epidemic in India in the 1950's and 1960's provides a tantalizing evidence of the relationship between mode of parasite transmission and virulence. The bacteria that cause cholera (*Vibrio cholerae*) are generally transmitted through drinking-water supplies. The bacteria cause diarrhea in their hosts. The diarrhea may contaminate the water supplies either through poor sewage disposal or through the washing of contaminated sheets. In any case, the parasite's chances of being transmitted are not decreased by immobilizing its host. In fact, its chances of getting into the water supply may even increase if care-takers wash contaminated bed-clothes in the water supply of a down-stream community.

> As water supplies were purified in cholera-endemic regions in India during the 1950's and 1960's, the milder agent of cholera, the El Tor type of *Vibrio cholerae*, displaced the more dangerous form, classical *V. cholerae*. [Ewald, 1993]

Unfortunately, the evidence for the evolution of virulence in relation to the parasite's mode of dispersal remains largely observational, and thus only correlational [Ewald, 1983]. Ewald (1983) goes on to predict that parasites that are highly mobile should cause severe diseases.

The correlation between virulence and parasite mobility fits with a body of evolutionary theory that analyzes the continuum from purely vertical transmission, from parent to offspring, to purely horizontal transmission, i.e., transmission between unrelated hosts. When a parasite is transmitted vertically, it maximizes its reproductive success by maximizing its host's reproductive success. Thus, the parasite should evolve to a commensal, if not symbiotic relationship with the host species. However, a parasite that is transmitted horizontally to unrelated hosts has no particular interest in preserving its current host. Thus horizontally transmitted parasites are expected to evolve high levels of virulence [Williams and Nesse, 1991, Herre, 1993, Nowak, 1991, Hurst, 1991].[1]

Configuration Models of Parasites

Differential and difference equations have been used for many years to model population dynamics [May, 1973]. These models lump individuals together into homogenous groups that can be represented by a single number, the number of individuals in that group. They may break a population down into a number groups depending on any number of characteristics, like age, sex, weight, etc. [Caswell, 1989]. However, in each subgroup, the individuals within the groups are still treated as identical. In this way, these models may describe the distribution of individuals in the population. Caswell and John (1992) label these distribution models. In contrast, a relatively new form of computer model, often called an individual-based model, explicitly represents each individual in the population as a separate data structure. The model keeps track of the configuration of all of the individuals in the population. Since the distribution model is also based on the individuals in the population, this new form of model may be called a configuration model [Caswell and John, 1992].

Although configuration models are relatively new to ecological modeling [Huston *et al.*, 1988], they have been essential to artificial life. Emergent dynamics are often observed through the local interaction of individuals. This is the basis of cellular automata models as well as many of the early models in artificial life [Langton, 1992, Fontana, 1992, Koza, 1992, Taylor *et al.*, 1989, Travers, 1989, Reynolds, 1987, for example]. Recently, attention

[1] It should be noted that definitions of virulence vary amongst these authors. For example, Herre (1993) operationalizes virulence as a reduction in lifetime reproductive success of the host, rather than disease induced death rate as per Ewald (1983) and May and Anderson (1990).

has been focused on the theory behind the construction of configuration models [Maley and Caswell, 1993, Baveco and Lingeman, 1992]. configuration models open up the possibility of explicitly representing the interactions between individuals. On occasion this has produced evidence that contradicts or extends the analysis of distribution models [Boerlijst and Hogeweg, 1992, Collins and Jefferson, 1992].

In the case of a host-parasitoid interaction, a configuration model can explicitly model the reproduction and transmission of parasites. This allows an intuitive and direct analogy between the dynamics of the model and the dynamics of the biological system. Baveco and Lingeman (1992) have constructed such a model to examine the population dynamics of a host- parasitoid system in a patchy environment.

Baveco and Lingeman locate the hosts and parasites in a 5×5 grid of patches. Both hosts and parasites have a three stage life history; eggs, larvae, and adults. Hosts can only be parasitized in their larval stage. Parasite transmission in their system is density-independent. Similarly, host reproduction is density-independent. Furthermore, the life span of an individual is predetermined at birth. The individual will die after the determined number of time steps unless it is a larval host that becomes infected by the parasites and so dies before it can reproduce. Parasites disperse from a patch to a neighboring patch when no hosts remain in their patch.

Baveco and Lingeman found that if the reproduction rate of the parasites was greater than the reproduction rate of the hosts, the parasites quickly drove the hosts to extinction. On the other hand, if the reproduction rate of the parasites was less than the reproduction rate of the host, the parasite population never caught up to the host population. This later case is probably due to the fact that host reproduction was assumed to be density-independent. However, in the former case, the extinction of the hosts was found to be a robust effect for most of their parameter settings. Baveco and Lingeman tried increasing the dimensions of the environment from 5×5 up to 10×10. They tentatively conclude that an environment of 10×10 patches is about the minimum size necessary for the persistence of the host population.

Baveco and Lingeman were not trying to model the evolution of virulence. In fact, they were not trying to model evolution at all. They took a classical distribution model [Nicholson and Bailey, 1935] of an ecological interaction between hosts and parasites and examined the dynamics of the system when all the individuals were explicitly represented. However, a similar model might be constructed where the parasites might have a "gene" for virulence. Given the possibility of mutation at the time of reproduction, the parasites might evolve varying levels of virulence over time in response to the selective pressures in the model.

A Model of the Evolution of Virulence

Ewald (1983), May and Anderson (1982 and 1990) all predict that virulence will evolve in relation to the mode of transmission of the parasite. However, mode of transmission is not an easily quantifiable characteristic. The problem can be simplified by focusing on the distance over which the parasites disperses. Whether it is transmission through biting insects, contaminated water supplies, or through sneezing, the dispersal of the parasites can be quantified by the distance of dispersal from the infected host. The question can now be asked, what is the effect of the distance of dispersal on the evolution of virulence in parasites?

The Hypothesis

Hamilton (1992) suggested that we should expect parasites that disperse over short distances to evolve to be less virulent than parasites that disperse over long distances. This follows from the continuum from vertical to horizontal transmission. Parasites that reproduce locally will be likely to share their host with their offspring. Thus they damage their own reproductive fitness by damaging their host. On the other hand, a parasite that disperses over long distances is less likely to share its host with its offspring, and so can exploit its host with near impunity.

General Structure

The hypothesis as it stands requires a system of only four significant components: host organisms, parasite organisms, spatial structure in the environment so that the parasites can disperse to new hosts, and a parasite gene for level of virulence that can evolve over time. It is necessary to use a configuration model because the spatial relationships between organisms directly affects the dynamics of the model. Because the hypothesis does not involve the immune response of the host, host immune systems were excluded from the model.

I chose to use an energy flow model [Dewdney, 1989, Rizki and Conrad, 1986, Taylor *et al.*, 1989]. Each discrete time step, a quantified amount of energy flows from the environment into the host organisms. At the same time, parasites are removing energy from the hosts to support the loss of energy in the parasites due to their metabolism. Additional energy is used by both hosts and parasites when they reproduce and that energy is given to the newborn organisms.

Hosts and parasites interact in a two-dimensional environment, or grid. The edges of the grid are connected to form a toroidal surface. Each patch in the grid can hold a single host. Each host can hold a virtually unlimited number of parasites while it still lives.[2] Death occurs when an organism's energy level drops to zero.

Hosts are relatively uninteresting. They only have one attribute: their energy level. Hosts cannot move. They can reproduce when their energy reaches a certain threshold. However, the newborn only survives if there

[2] The maximum number of parasites allowed to infest a single host was set to 500. Since the actual number of parasites infesting a host tended to stay below 10, this should not have influenced the dynamics of the model.

is an empty patch in the grid adjacent[3] to the parent. Since the hosts have no "genes" they cannot evolve.

Parasites have two attributes: energy and virulence. The virulence attribute is the number of energy units the parasite takes from its host each time step. Like hosts, parasites reproduce when their energy level reaches a certain threshold. However, they have genes for virulence, so the virulence attribute may mutate (e.g., ± 6) in the newborn parasite. Furthermore, there is a parameter in the model, dispersal distance, that restricts where the newborn parasite may be randomly placed in the environment.

For each time step, the model executes four steps. 1. The energy levels in the hosts are updated in parallel. 2. The energy levels in the parasites are updated in parallel. 3. Newborn parasites are placed in the environment. 4. Newborn hosts are placed in the environment.

Energy flows in the following way: First, each host gains a fixed amount of energy, *food*, each time step. Second, each host loses energy equal to the sum of the virulences of the parasites infesting it. Third, each parasite gains its *virulence* worth of energy units each time step, as long as its host is still alive, $host\ energy > 0$. Fourth, each parasite loses a fixed amount of energy, *cost of living* each time step. In addition to this basic energy flow, an organism loses a fixed amount of energy, *cost of reproduction*, each time it reproduces The newborn organism starts off with that amount of energy.

Parameters

Although the basic structure of the model has only four components, there are a host of details that must be specified in order to model the life histories and interactions of the organisms. These details can be divided into three categories: fundamental parameters, parameters that can be derived from previously defined parameters, and the initial conditions in the model at the beginning of a simulation. [In all cases the default settings will be put in brackets.]

Fundamental Parameters The fundamental parameters in the model are the bottom line. They are used to derive reasonable quantities for many other parameters in the model, and so define basic choices about the scale of the model. There are only four such fundamental parameters: *dispersal, reproduction rate, host cost of reproduction* , and *size* .

Dispersal distance is the independent variable in the experiment. The *dispersal distance* represents the maximum distance between a parasite and the placement of its offspring. *Dispersal distance* defines the size of the square, with the parental parasite's patch in the center, in which the offspring parasite is randomly placed. The probability of placement for a parasite was the same for every patch within that square.

[3] Every patch has eight adjacent patches corresponding to the eight points of the compass. Since the edges of the grid wrap around, the surface actually forms a torus with no edge effects.

Reproduction rate is the number of time steps it would take an uninfested host to build up enough energy to reproduce. In other words, it is a sort of generation time. *Reproduction rate* determines the granularity of time in the model. [2]

Host cost of reproduction is the number of energy units transferred from a host parent to its offspring at birth. This determines the basic energy scale or granularity in the model. The larger the number the finer the grain. [1000]

Finally, *size* determines the relative scale between the hosts and parasites. It is roughly the number of parasites that can fit in a host. The larger the number, the smaller the parasites. [5]

Derived Parameters Most of the parameters in the model are derived from *size, host cost of reproduction*, and *reproduction rate*. They generally determine the specifics of energy flow through the model.

Energy enters the cycle as *food* . *Food* is the amount of energy given to a host each time step. $food$ = *host cost of reproduction / reproduction rate*. [500]

Similarly, energy leaves the cycle in the *cost of living for parasites.* This is the amount of energy removed from every parasite each time step. *cost of living for parasites = food / size*. [100]

Hosts automatically reproduce when their energy level reaches the *host reproduction threshold* . At that point, the host loses the *host cost of reproduction* amount of energy. *Host reproduction threshold* = $2\times$ *host cost of reproduction*. [2000]

Parasites also lose energy when they reproduce equal to the *parasite cost of reproduction = host cost of reproduction / size*. [200]

Like hosts, parasites automatically reproduce when their energy level rises above the *parasite reproduction threshold = host reproduction threshold / size* . However, unlike hosts, parasites might be so virulent that their energy level jumps far above the *parasite reproduction threshold* . Parasites are forced to reproduce multiple times until the repeated deductions of the *parasite cost of reproduction* lowers the parasite's energy level below the *parasite reproduction threshold.* In this way, highly virulent parasites reproduce more quickly than less virulent parasites. [400]

The *mutation factor* scales the range of mutation of the virulence gene in the parasites. [8] It is not strictly a derived parameter since it does not formally depend on other parameters. However, it has been set such that the actual range of mutation ($\frac{food}{2\times mutation\ factor\times size}$ [= 6]) in the virulence gene is small compared to the amount of energy entering a host, *food.*

Finally, for ease of programming, there is a *carrying capacity of the hosts* parameter that puts a strict limit on the number of parasites that may infest the host. This was set high enough that in actuality it never affected the dynamics of the model. [500]

Initial Conditions The only pieces that remain to describe are the initial settings of the characteristics in the organisms at the start of a simulation. Hosts only

Parameter	Description	Default
energy	Energy units accumulated by an organism	variable
virulence	Amount of energy a parasite removes from its host	variable
dispersal distance	Radius of dispersal for parasite offspring	1-15
reproduction rate	Uninfested host generation time	2
host cost of reproduction	Energy penalty for host reproduction	1000
size	Size scale between hosts and parasites	5
food	Energy given to a host each time step	500
cost of living for parasites	Energy taken from a parasite each time step	100
host reproduction threshold	Amount of energy triggering host reproduction	2000
parasite cost of reproduction	Energy penalty for parasite reproduction	200
parasite reproduction threshold	Amount of energy triggering parasite reproduction	400
mutation factor	Scales the range of mutation in virulence	8
carrying capacity of the hosts	Maximum number of parasites in a host	500

Table 1: The variables, fundamental parameters, and derived parameters that define the model.

possess a single characteristic, energy, while parasites possess both energy and virulence characteristics. Hosts start with an energy that is determined randomly in the range of 0 to *host reproduction threshold.* However, any newborn host starts out life with *host cost of reproduction* worth of energy units. Similarly, the parasite's initial energy was set randomly between 0 and the *parasite reproduction threshold,* and a newborn parasite starts life with *parasite cost of reproduction* energy units. The parasites initially placed in the model have virulences that are determined randomly in the range from the *cost of living for parasites* to 3× *cost of living for parasites.* [100 - 300] [4]

Quirks and Caveats

This model of a host-parasitoid system is extremely simplified. The principle of parsimony suggests that the bare minimum of detail should be included so as to test the hypothesis. Beyond this, there were a number of decisions that had to be made more or less arbitrarily. Usually ease of model construction prevailed over the choices.

Major Dynamics Left Out The most glaring exemption is the lack of resistance genes in the hosts. This removes the entire dynamic of coevolution. It also removes the possibility for sexual reproduction in hosts. Hamilton, Axelrod, and Tanese (1990) argue that sexual reproduction may have evolved as an adaptive response to parasitism.

We lack data on the relationship between parasite reproduction and virulence in general [May and Anderson, 1990, Ewald, 1983], or λ and α in equation 1. Therefore I have let the relationship be linear as a first approximation. Specifically, a parasite will produce $\lceil \frac{energy - parasite\ reproduction\ threshold}{parasite\ cost\ of\ reproduction} \rceil$ number of offspring in one time step. Furthermore, the model does not contain any sense of host recovery or immunity (v in equation 1).

On a more physical level, there is no dissipation of energy during transfer from environment to host, from host to parasite, and from parent to offspring. Specifically, parasites gain exactly the same amount of energy as their host loses in a single time step.

No distinction was made in the model between microparasites and macroparasites. Nor is there any representation of life histories of the parasites and host beyond the dispersal distance. Finally, there is no interspecific competition between different parasites species within the hosts. The model only simulates intraspecific competition amongst the parasites.

Additional Details of the Model When a host dies all of the parasites infesting it die. However, when a host reproduces, half of the parasites infesting the parent are transmitted to its offspring. This allows parasites to be transmitted vertically.

There are a number of quirks surrounding reproduction. If a host tries to reproduce when there are no empty adjacent patches, the host will lose the *host cost of reproduction* amount of energy, but no new host will be created, and so the parental host will retain all of its parasites. Similarly, there are a few rare cases where two parasites will try to place offspring in the same position of a single host, thus causing a "collision" since the model operates in parallel. In this case, the more virulent offspring will win out and the other parasite offspring is killed. In an even more unlikely case, a parasite will try to place an offspring into a host that already has the *carrying capacity of the hosts* number of parasites infesting that host. Again, the parasite's offspring is killed under these conditions.

The Experiment

The model was constructed in the language *C** and was run on a Thinking Machines CM5 parallel computer. Once the model had been constructed, the experiment was straight-forward. What is the relationship between distance of dispersal and virulence in parasites? The independent variable was *dispersal distance* and the dependent variable was the *virulence* in the population of parasites after a specified amount of time.

[4] If a parasite has a *virulence* less than the *cost of living for parasites* then it will inevitably die before reproducing.

Method

Each run of the model began with a host in every patch of the 30×30 environment. One of the hosts was also infected by a single parasite. The model began by simulating the invasion of the host population by the parasite. Initial runs indicated that the oscillations of the host and parasite population sizes were so violent as to crash in many cases when the *size* parameter was too large, so *size* was set at five for all of the runs.

The virulences of the parasites were sampled after a specified number of time steps. In order to determine an appropriate number of time steps, the model was run a few times under different dispersal conditions for extremely long periods of time (e.g., 10,000 time steps) and the average virulence was observed over time. Virulence seemed to level out after about 4,000 time steps in most cases, so the sampling time was set to be 5,000 time steps. At the end of this period of time, the virulence for each parasite in the population was saved in a file for later analysis.

The independent variable, *dispersal distance*, ranged from 1 to 15. If the *dispersal distance* was 1, parasites could only transmit their offspring to the nearest neighbor hosts. In contrast, with a *dispersal distance* of 15, a parasite could transmit its offspring anywhere in the environment. The model was run under a *dispersal distance* of 1, 2, 4, 8, and 15. For each of these possible settings the model was run five times using different random number generator seeds. In total, data was collected for 25 runs of the model.

Results

All of the runs of the models produced oscillations in the host and parasite populations that were stable enough that neither population crashed in the sampled interval. However, the resulting sizes of the parasite populations varied from about 125 to 250 at the 5,000 time step mark.

An analysis of the variance in the resulting populations indicated that dispersal had a statistically significant effect on the evolved virulence of the parasites ($p < 0.0001$), as did the seed for the random number generator ($p < 0.05$). The line of best fit for the $virulence = 594.23 + 0.91 dispersal - 0.11 seed$.

Discussion

Why is the parasite that causes the common cold less deadly than the protozoa that causes malaria (genus *Plasmodium*) or the bacteria that cause cholera (*Vibrio cholerae*)? Perhaps the differences in virulence depend in part on the differences in the dispersal mechanisms of the parasites. The model gives some evidence that the distance over which a parasite transmits its offspring has a significant effect on the parasite's evolution of virulence. However, according to the model, this effect is relatively small. This is probably due to the fact that selection pressure against a high virulence is relatively small compared to the advantage of rapid reproduction in the short dispersal distance conditions. High virulence boosts a parasite's reproductive success primarily by allowing the parasite to reproduce quickly. Only on a secondary level does it reduces the resources in the area upon which the parasite's offspring must depend. Even if a parasite kills off its host, the parasite's offspring could have been transmitted to any of that host's eight neighbors. Thus, a high virulence only removes a fraction of the resources available to its offspring.

It is likely that the details of the life history of a real biological parasite would have a much stronger effect on its evolution of virulence than just its dispersal distance. For example, many parasites of humans are transmitted through the close physical contact of medical workers with their patients. The medical workers carry the parasites from patient to patient. Even though dispersal distance is relatively short in this case, it is to the parasite's reproductive advantage to have a severe effect on its human host, so that the host is taken to the hospital.

One oddity of the model ought to be addressed. It was observed that a high value for *size* (e.g., 100) caused the populations to crash for most settings of the *dispersal distance* parameter. That is, when many parasites could infect a host, the populations tended to crash. As *size* was adjusted downwards, the populations began to enter stable oscillations. This is probably due to the resultant infection rate in the population of hosts. Once a host has been infected, its days are numbered. The parasites begin draining off energy making it less likely that the host will be able to reproduce. Even if it does manage to reproduce, half of its parasites are transmitted to the offspring. Thus, if there are many parasites in a host, the only introduction of uninfected hosts must come from the reproduction of other uninfected hosts. Meanwhile, parasite virulence levels evolve to the point of draining all of the host's food in one time step. A large *size* parameter means that parasites can reproduce relatively easily. Thus a single infected host can be the staging ground for many new infections in a single time step. In this way, a parasite population can sweep through the host population until all the hosts are infected. Given even moderate levels of virulence, this spells disaster for both species.

In contrast, when the *size* parameter is small, only a few parasites can infect a single host. The result is that there is some chance that no parasite will be transmitted to an infected host's offspring. More importantly, a single infected host can only be used as a staging ground for the transmission of a few new parasites each time step. Thus, infection rates in the host population are lower. This maintains a resource of uninfected hosts producing uninfected offspring. The high levels if infection in the host population is probably the effect of two dynamics. First, it is relatively easy for a parasite to transmit its young to a new host. In fact, the survival of a parasite during transmission is almost 100 percent. Second, the hosts lack an immune system which might also serve to reduce infection rates in the host population.

The model is highly simplified. In the biological world, a parasite's mode of transmission is not fixed, and may evolve along with virulence. It is also clear that there are many complex interactions between real parasites and the host's immune system, as well as between different parasite species, not to mention other factors [Begon *et al.*, 1990]. These effects are likely to be dramatic compared to the effect of dispersal distance on virulence. However, it was the hope that by excluding these factors, and thus considering all things to be equal, it may be possible to begin to pick apart the complexities of the evolution of virulence in parasites.

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