IDEA

Increasing resource specialization among competitors shifts control of diversity from local to spatial processes

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Abstract

We argue that an increase in the number of specialized consumers can shift the control of ecological dynamics from local to spatial processes. When there are only a few specialized types, local dynamics maintains most types within each patch. As the number of types increases, the probability of local extinction rises. Subsequent colonizations perturb local dynamics, setting off another round of extinctions and the potential for later recolonization. Global processes of colonization and extinction reduce local diversity and increase differentiation among patches. We draw an analogy between the specificity of host-parasite genetics and the specificity of consumer–resource pairs.

Keywords

Colonization, competition, dispersal, extinction, host-parasite, metapopulation.

Suppose that several species compete for a limited resource. The competitive vigor of each species depends on the abundance of a distinct resource on which that species is uniquely specialized. Define the dimensionality of the system as the number of different resources available for specialization. We argue that changes in dimensionality cause qualitatively different ecological dynamics and patterns of diversity.

Low-dimensional systems maintain most of the few possible species in each local patch, with dynamics controlled by local processes of competition. As the number of species rises, the average abundance per species declines (Tilman & Pacala 1993). Lower average abundance increases the probability that one or more species become extinct locally by deterministic nonequilibrium fluctuations and by demographic stochasticity (May 1974).

Local extinctions make individual patches prone to rapid growth of colonists. Suppose a particular species has recently become extinct in a particular patch. The unique resource consumed by that species will increase. Then a colonist of that species can invade and increase rapidly because its competitive ability will be boosted by an abundant supply of its special resource. The rapid increase of the colonist will drive down the abundance of competitors, making them prone to local extinction. Any extinctions will be followed by an increased supply of the matching resources. Another round of colonization, competition, and extinction is inevitable. Cycles of local colonizations and extinctions continue, each bout coupled with a turnover in local diversity.

Figure 1 summarizes the comparative predictions about dynamics and diversity. The dynamics of low-dimensional systems are governed by local processes of competition. Each additional specialist that can be stably maintained locally increases both local and global diversity. As dimensionality rises, the risk of local extinctions increases. At some point, spatial processes of colonization and extinction dominate and periodic, extreme competition from colonists drives down local diversity. Thus a rise in the number of specialist types decreases average local diversity but increases the diversity maintained in the metapopulation.

Frank (1989, 1993, 1997) developed a similar argument for host-parasite genetics. The host-parasite models can be considered part of a wider class of genetic models of specific recognition. In those models, the consumer genotype must avoid specific recognition and defence by the resource genotype. Successful attack leads to a benefit to the consumer at a cost to the resource. Empirical studies of plant-pathogen genetics, cytoplasmic male sterility, and other genetic systems of recognition and antagonism often reveal high dimensionality of specificity and a strong influence of colonization-extinction dynamics (Gouyon & Couvet 1985; Thompson & Burdon 1992; Frank 1997). But there is not enough comparative evidence to analyse the main prediction – that a shift in dynamics and diversity occurs as systems change from low to high dimension.

We suggest that the logic connecting dimensionality to dynamics applies both to host-parasite genetics and to a broad class of consumer–resource models of competition.

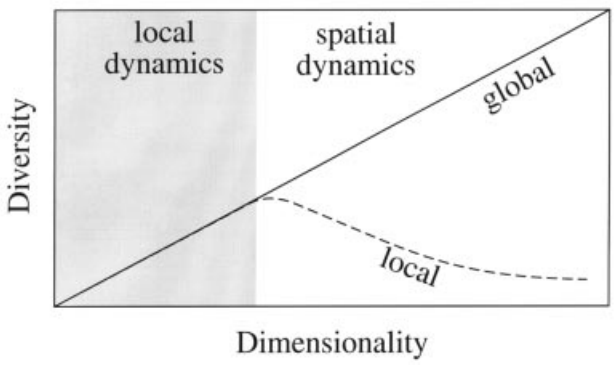

Figure 1 Increasing dimensionality causes a shift in the control of dynamics from local to spatial processes.

Frank (1993, 1997) illustrated host-parasite dynamics with a simple model. Here we introduce a similar model to illustrate consumer–resource dynamics.

Each of m consumer species, with abundance N_i, is specialized on a resource with abundance R_i. The dynamics are given by

$$\Delta N_i/\Delta t = cbN_i(R_i - \Sigma R_k N_k/K)$$

$$\Delta R_j/\Delta t = a(S - R_j) - bR_jN_j,$$

where each individual of species i consumes bR_iN_i units of resource per time period and converts those resources into reproduction at a rate c. The supply of resource i increases at a rate $a(S - R_i)$, where S is a carrying capacity for resource abundance and a is a rate constant (Tilman 1982). Competition among consumers for another, limited resource sets the carrying capacity of all consumers at K.

The dynamical system is easier to analyse when written in nondimensional form (Segel 1972; Murray 1989) by using the following substitutions:

$$n_i = N_i/K,\ r_i = R_i/S,\ \alpha = a/bcS,\ \beta = K/cS,\ \Delta\tau = bcS\Delta t,$$

yielding

$$\Delta n_i/\Delta\tau = n_i(r_i - \Sigma r_k n_k)$$

$$\Delta r_j/\Delta\tau = \alpha(1 - r_j) - \beta r_j n_j$$

for $i, j, k = 1, \ldots, m$. The system is controlled by the four parameters α, β, m, and $\Delta\tau$. These difference equations become continuous (differential) as $\Delta\tau \to 0$.

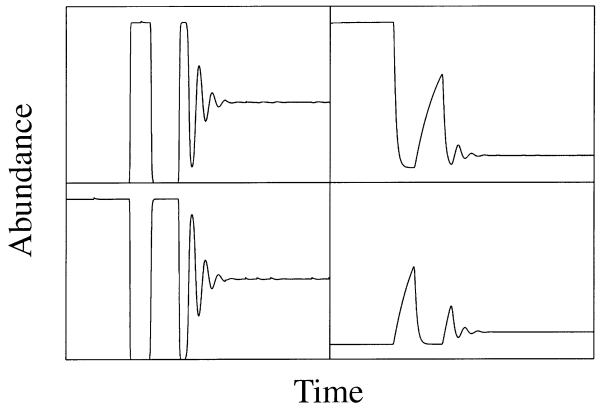

Figure 2 Time series of the dynamical system described in the text. The parameters are $\alpha = 0.005$, $\beta = 0.05$, $\Delta\tau = 0.1$, and $m = 2$. The system was run for an initial 15,000 iterations (not shown); the following 20,000 iterations are plotted. Each iteration is a nondimensional time step of length $\Delta\tau$. Extinction is simulated by setting to zero any abundance less than 0.01. Colonization is simulated by adding 0.01 to the abundance of each consumer in each iteration if a random number between 0 and 1 is less than 5×10^{-4}. Thus the average number of iterations between each colonization event is 2000.

Figures 2 and 3 illustrate the change in dynamics as the dimensionality, m, increases. In Fig. 2, the two consumers ($m = 2$) specialize on two different resources. The temporal dynamics for consumers are shown in the left column of panels. In each row, the right panel shows the matching dynamics of the resource on which the consumer specializes. After initial transients of local extinctions and recolonizations, the system in Fig. 2 settles to an equilibrium with both consumers maintained locally.

Figure 3 has identical parameters except that there are four consumer–resource pairs. This system could, in theory, settle to an equilibrium with all types equally abundant. But the transient dynamics caused by repeated extinctions and stochastic colonizations tend to keep the system fluctuating away from the basin of attraction to the equilibrium. Any consumer–resource pair sufficiently displaced from its equilibrium eventually sets off another round of transient dynamics. The probability that at least one pair is displaced increases as the dimensionality, m, rises. Increasing dimensionality shifts the control of the dynamics from local to spatial processes.

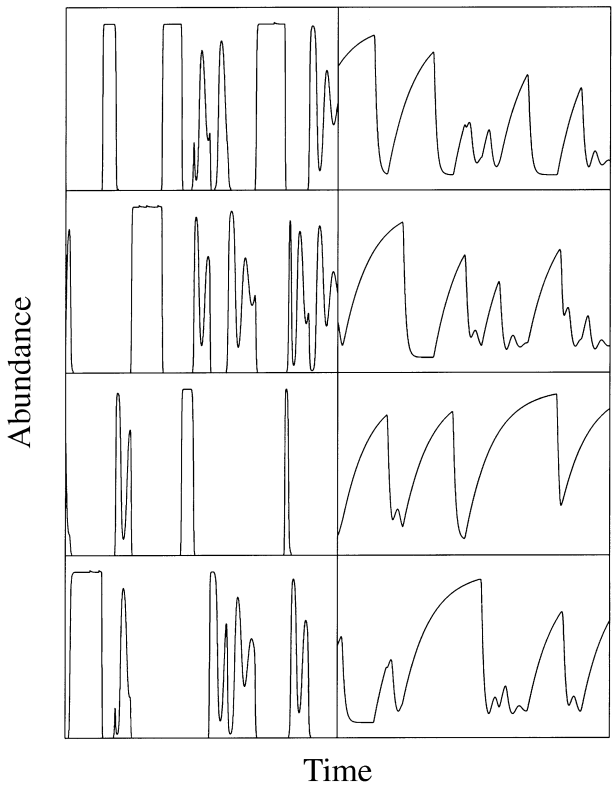

Figure 3 Time series for the system with $m = 4$ consumer–resource pairs. All other parameters and methods are the same as in Fig. 2.

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BIOSKETCH

Steven Frank studies specific recognition and polymorphism in host-parasite interactions, the evolution of social behavior, and conflict and cooperation in symbiotic relationships.

IDEA

Do Bertalanffy's growth curves result from optimal resource allocation?

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Abstract

Bertalanffy's equation is commonly used to model indeterminate growth. Bertalanffy claimed that this growth pattern results from growth potential decreasing with age. An alternative approach provided by life history theory predicts that indeterminate growth is optimal for organisms in a seasonal environment and results not from decreasing growth potential but from allocating increasingly less energy with age into growth, and more into reproduction. Bertalanffy's curves are the result of evolutionary optimization and should not be used in optimization models as an assumption, but they can be used as a tool to describe the indeterminate growth pattern phenomenologically.

Keywords

Bertalanffy's equation, growth curves, indeterminate growth, life history evolution, optimal resource allocation, optimization models, reproduction, seasonality, trade-off.

Many amphibians, reptiles, fishes, annelids, mollusks, crustaceans, and other animals continue to grow after maturation, slowing down their growth with age. Such an indeterminate growth pattern is often approximated by Bertalanffy's equation (Bertalanffy 1957; Beverton & Holt 1959; Sibly & Calow 1986; Charnov 1993; Weinberg & Helser 1996). Although growth curves generated this way fit the field data well, Kozlowski (1996), Day & Taylor (1997), and Kozlowski & Teriokhin (1998) warn that Bertalanffy's equation is misused in the theory of life history evolution.

In Bertalanffy's model, the rate of change in body weight is the difference between the rates of anabolism (tissue production) and catabolism (tissue dissipation), with anabolism proportional to the two-thirds power of body weight and catabolism directly proportional to body weight. According to Bertalanffy, the diminishing – with size – difference between anabolism and catabolism slows down growth with age; growth finally stops when

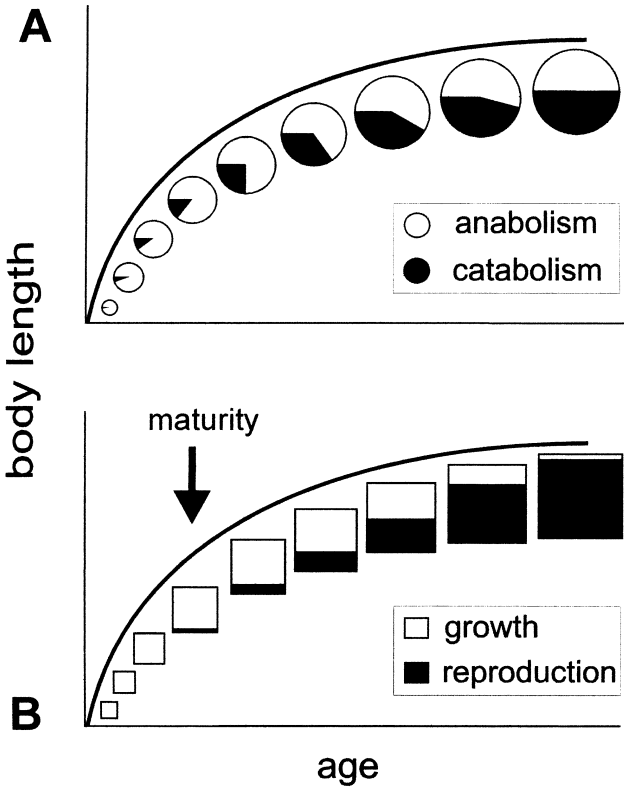

Figure 1 In Bertalanffy's model (A) the growth rate of an individual equals the difference between its anabolism and catabolism rates (white and black areas, respectively), both of which increase with size and age; catabolism rises faster than anabolism does, leading to a decreasing-with-age growth rate which finally stops when catabolism offsets anabolism (black area equals white area). In the optimal resource allocation model (B) the amount of available resources (square) increases as an organism grows; slowing growth with age results from the optimal strategy, allocating an increasingly bigger fraction of the resources into reproduction than into growth (black and white areas, respectively). The resulting growth curve resembles Bertalanffy's curve.

catabolism balances anabolism (Fig. 1A). The resulting Bertalanffy's growth curve for body length is often described as $l_t = l_\infty\{1-\exp[-k(t-t_0)]\}$, where l_t is the body length at age t, l_∞ is the asymptotic length, k is the growth constant, and t_0 is the hypothetical age at which length equals zero.

From an evolutionary point of view, Bertalanffy's model is problematic: how can natural selection favour organisms that keep growing beyond the size at which the difference between their anabolism and catabolism rates is at maximum, and continue to grow to the point where the difference equals zero? Production of offspring tissue, like the production of the animal's own tissue, requires a surplus of anabolic over catabolic processes. If organisms tend to maximize their reproductive success, how should they reach the point where reproduction is lower than the maximum possible or is even not possible at all?

Models of resource allocation result in growth curves resembling Bertalanffy's if net production (reflecting the difference between anabolism and catabolism) increases monotonically with size, but after reaching maturation an increasing fraction goes into reproduction (Fig. 1B). Such a possibility was suggested by Roff (1983), who assumed a constant gonadosomatic index (reproductive tissue weight/body weight ratio) after maturation. Although this assumption resembles what is found in nature, it should rather be a result of an optimization model. Kozl owski & Uchmañski (1987), Kozlowski (1996), and Kozl owski & Teriokhin (1998) showed that if long-lived organisms inhabiting highly seasonal environments (with periods of conditions suitable for growth or reproduction regularly interrupted by winters or droughts) stick to the optimal allocation rule, maximizing lifetime reproductive success, they should increase the proportion of production channeled to reproduction as they age. Sand snails studied by Noda *et al.* (1995) illustrate this problem. In the aseasonal tropical environment, *Umbonium vestarium* has almost determinate growth (9 mm at maturity, 12 mm maximum size), whereas in the temperate seasonal environment *U. costatum* grows indeterminately (11 mm at maturity, 22 mm maximum size) with the production fraction allocated into reproduction gradually increasing with age. According to the theoretical studies (Kozlowski & Uchmañski 1987; Kozlowski 1996; Kozlowski & Teriokhin 1998), the discontinuity of environmental conditions makes it optimal to switch back from reproduction to growth in the years following maturation, whereas diminishing returns (slower than linear increase of production with size) make it optimal to put a larger fraction of available resources into reproduction as individuals become older (bigger) (Fig. 1B). The resulting growth curves for linear measures can indeed be well fitted by Bertalanffy's equation if the production rate increases with body weight allometrically with the exponent $\leqslant 2/3$ (Kozlowski 1996; Kozlowski & Teriokhin 1998). If the exponent is greater than 2/3, curves with an inflection point give a better approximation.

Mortality and ageing affect growth curves qualitatively and quantitatively. When mortality increases with age, it is optimal to grow and reproduce for some years following maturation, and then to stop growing completely and only reproduce (Kozlowski & Teriokhin 1998). Nevertheless, Bertalanffy's equation matches the growth pattern at least at the beginning of the organis m's life, and discovering a deviation from Bertalanffy's curve caused by rare individuals surviving to old age when their

growth stops is unlikely for statistical reasons. Mortality rate in favourable and unfavourable seasons also affects the system quantitatively, defining the optimal age at maturity, the maximum size, and the fraction of size reached at maturity (Kozlowski & Teriokhin 1998).

Seasonality does not explain all cases of indeterminate growth: growth in many short-lived species also resembles Bertalanffy's curves. We can suggest by analogy that discontinuities of production and/or mortality also lie behind this phenomenon in such species. If offspring are produced in clutches, increased maintenance costs of gravid females may decrease production, and their mortality is also likely to increase. Theoretical studies should indicate whether these phenomena lead to the optimality of indeterminate growth. If this is the case, the focus should be on the constraints causing reproduction in clutches rather than series of single eggs. Design constraints may also be responsible for indeterminate growth; e.g. clams that brood their young in gill chambers, may face the space limitation during reproduction, which makes the Bertalanffy-like growth pattern optimal (Heino & Kaitala 1996).

Because of the coincidental agreement between the predictions of optimization models and Bertalanffy's growth curves it is tempting to apply them as an assumption in such models. This is not justified because it implies that the change in energy allocation that occurs at maturity does not influence the growth rate after maturation (Day & Taylor 1997). In such a case, altering age at maturity in the models does not affect the shape of the growth curve, clearly contradicting current knowledge about the trade-off between reproduction and growth (Roff 1992; Stearns 1992; Kozlowski 1992; Perrin & Sibly 1993; Jokela 1997). Timing of maturation and further allocation decisions define growth curves. Results and assumptions should not be confused.

The theory of life history evolution explains indeterminate growth without the logical flaw imbedded in Bertalanffy's model. Although Bertalanffy's growth curves should not be used as an assumption in modelling life history evolution, they can be applied as phenomenological descriptions if their parameters are not given any deeper meaning.

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BIOSKETCH

Marcin Czarnołęski graduated in biology from the Jagiellonian University, Kraków, Poland. He is currently doing his PhD research, guided by J. Kozlowski. He is working on the life history evolution; in particular, on optimal resource allocation models concerning growth and reproduction and on testing their predictions with the data on freshwater clams *Dreissena polymorpha*.

IDEA

Coping with multiple enemies – the evolution of resistance and host-parasitoid community structure

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Abstract

Recent work has shown that the evolution of *Drosophila melanogaster* resistance to attack by the parasitoid *Asobara tabida* is constrained by a trade-off with larval competitive ability. However, there are two very important questions that need to be answered. First, is this a general cost, or is it parasitoid specific? Second, does a selected increase in immune response against one parasitoid species result in a correlated change in resistance to other parasitoid species? The answers to both questions will influence the coevolutionary dynamics of these species, and also may have a previously unconsidered, yet important, influence on community structure.

Keywords

Coevolution, community structure, *Drosophila melanogaster*, evolution of defence, natural enemies, parasitoids, virulence.

When we look around us, it is evident that all organisms face attack from many different natural enemies. This tangled web of interactions between species leads us to ask two important questions. First, how does the evolution of defence against one natural enemy affect the victim's ability to defend against other enemies? Second, are the trade-offs associated with resistance to one natural enemy specific to that enemy, or general in expression? Thompson (1994) was one of the first to note the importance of questions such as these from a perspective of the evolution of local adaptations, but answers are also crucial if we wish to understand how evolutionary dynamics could influence community structure.

GEOGRAPHIC VARIATION IN IMMUNE RESPONSE

Perhaps one of the best model systems available to understand questions such as these is provided by *Drosophila melanogaster* Meigen and its parasitoids. In insects, the principal defence against larval endoparasitoids is an immune response known as encapsulation (Godfray 1994; Quicke 1997). Specialized haemocytes identify foreign bodies as non self and cause other haemocytes to aggregate and form a melanized capsule (Strand & Pech 1995).

There is considerable geographical variation in the ability of European *D. melanogaster* to encapsulate two of its most important parasitoids, *Asobara tabida* Nees (Hymenoptera: Braconidae) and *Leptopilina boulardi* Barbotin *et al.* (Hymenoptera: Eucoilidae) (Kraaijeveld & van Alphen 1995). *Drosophila melanogaster* populations in southern Europe can encapsulate up to 60% of *A. tabida*, whereas less than 30% of *L. boulardi* are encapsulated, establishing that resistance rates are particular to the attacking parasitoid species. There was also a difference in the geographical patterns seen – immune response to *A. tabida* appeared to be almost clinal, with increased encapsulation ability found in central European and Mediterranean populations. In contrast, encapsulation ability against *L. boulardi* did not show any such pattern. Vass *et al.* (1993) demonstrated that there is a similar reaction of *D. melanogaster* larvae to attack by both species, but suggested that different genes may control these responses. The genes responsible for variation in encapsulation response against both species are located on chromosome 2 (Carton & Nappi 1997; Orr & Irving 1997).

TRADE-OFFS

Kraaijeveld & Godfray (1997) searched for trade-offs associated with an increased ability to encapsulate *Asobara tabida*. The only cost found was that resistant larvae were poorer at competing for food. Balancing selection between rates of parasitoid attack and resource availability is thought to explain the presence of heritable variation in resistance, as up to 100% of *D. melanogaster* larvae are attacked in some populations (Carton *et al.* 1986) and larval competition for food is frequently severe (Atkinson 1979).

CORRELATED RESPONSES AND COMMUNITY STRUCTURE

Although we have gone some way towards understanding what constrains the evolution of defence against *A. tabida*, we must return to our original questions. First, are defences against multiple enemies correlated? Across populations there is no relationship between levels of successful immune responses against *A. tabida* and *L. boulardi* (Kraaijeveld & van Alphen 1995). These

parasitoids use distinct counter-defence mechanisms to avoid encapsulation (Quicke 1997). If the evolution of defence against different parasitoid species is uncorrelated, then independent coevolutionary cycles between host resistance and parasitoid virulence will occur. However, if defences against different parasitoid species are indeed correlated, then diffuse coevolutionary patterns of increasing resistance and parasitoid virulence could result in constraints on the structure of host-parasitoid communities. For example, if there has been an increase in encapsulation ability against parasitoid species A, and this also increases the host's ability to respond to parasitoid species B, then the latter species is less likely to be able to invade that community. This is the critical point. Community ecologists have very rarely considered the impact of evolution occurring at ecological time scales on the structuring of communities. Therefore, we need to test the resistance of *D. melanogaster* lines selected for defence against *A. tabida*, and other lines selected against a different parasitoid species (such as *L. boulardi*) against several parasitoid species, including the reciprocal trial. This will tell us if (i) an increased response against one species results in correlated responses to other parasitoid species, and (ii) if there are asymmetries in this response. In essence, whether defence is parasitoid specific or general to many determines the size of the physiological refuge available to the host. The influence of non-physiological refuge size on parasitoid diversity has been considered by Hochberg & Hawkins (1994), who argued that if refuges are homogeneous then a greater diversity of polyphagous parasitoids would be found. When refuges are heterogeneous, then monophagy would be more common, and parasitoid diversity would decline. The specificity of the encapsulation response would have a similar influence on parasitoid diversity.

Second, by performing the selection procedure against another species of parasitoid, we would also see if the constraints acting on defence against different parasitoid species are similar. If they are not, then selection pressures may result in variation in the rate of evolution of resistance to different parasitoid species. There are also other potential effects of having dissimilar trade-offs associated with defensive ability. Depending on the nature of these trade-offs, the size of the suite of parasitoids that the hosts can successfully resist may change. If resistance to many parasitoid species is associated with a range of expensive trade-offs then this will reduce the relative benefits of resistance, increasing the potential number of parasitoid species that can successfully attack the host population. However, if the costs of resistance against the parasitoid suite are not cumulative, then this would lead to a reduction in the number of attacking parasitoids, as the evolution of wider resistance would be less constrained.

CONCLUSION

Traditionally, there have been few attempts by evolutionary ecologists to empirically measure what constrains the evolution of resistance to natural enemies (Gemmill & Read 1998), and none that we are aware of have tried to link any correlated responses to community structure. Perhaps most exciting is the possibility of indirect effects on community structure mediated by asymmetries in correlated immune responses to different parasitoid species. Empirical investigations have only begun to hint at how evolution occurring on ecological time scales may influence the community dynamics of interacting species, and this is especially true of host–parasitoid associations.

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BIOSKETCH

M.D.E. Fellowes' main research interests revolve around the evolution of resistance to parasitoid attack and the interactions between evolution and community ecology, as well as broad interests in evolutionary ecology.

IDEA

Drugs and parasites: global experiments in life history evolution?

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Abstract
The use of chemotherapy to control parasites is likely to select for changes in parasite life histories. Recent commentators have assumed that parasites should respond to drugs affecting adult mortality by decreasing their age at maturity. Here we argue that in nematodes with larvae in host tissues and adults in the gastrointestinal tract, the opposite will occur; worms should postpone maturity and become bigger and more pathogenic.

Keywords
Anthelmintics, life history evolution, nematodes, pathogenicity, tissue migration, virulence.

Chemotherapy is used world-wide to combat a large number of parasites of medical and veterinary importance. Modern drugs are often highly effective and therefore impose potent selection on parasite populations. Morphological and biochemical mechanisms that enable parasites to tolerate chemotherapy thus spread rapidly (Conder & Campbell 1995). However, traits other than those conventionally associated with "drug resistance" are also likely to be under strong selection. Almost by definition, chemotherapy will alter size-and age-specific schedules of mortality and fecundity. These are key determinants of life history evolution (Stearns 1992), and affect the optimal allocation of resources between growth and reproduction. Current patterns of drug use almost certainly represent experiments in parasite life history evolution on a scale as large as they are poorly controlled, understood or even studied.

What kind of life history changes can we expect? As with free-living organisms (Roff 1992; Stearns 1992), there are unlikely to be simple generalizations. Parasites have an enormous variety of life histories, and chemotherapy involves a wide range of chemical compounds. Nevertheless, recent commentators have suggested that when drugs reduce adult life expectancy, natural selection will favour parasites that mature earlier (Medley 1994; Poulin 1998). In groups such as mammalian nematodes, developmental period, body size, and fecundity are strongly and positively correlated (Skorping *et al.* 1991; Morand 1996). This group includes some of the major targets of the pharmaceutical industry, such as the gastrointestinal nematodes of sheep, cattle, and humans. If drug-imposed selection does indeed favour earlier age to maturity, the outlook is rather optimistic: drug pressure should favour smaller and less fecund nematodes. This prediction, however, is apparently based on theoretical models (Stearns 1992) whose crucial assumptions may be violated by many, if not most, parasitic nematodes. Most anthelmintics used are most realistically viewed as being stage specific and not necessarily age specific (Table 1). A consequence of this is the potential evolution of nematodes more damaging to their hosts.

Nematode life histories are characterized by distinct developmental stages and often ontogenetic habitat shifts within the mammalian body (Read & Skorping 1995). For example, parasites like *Ascaris lumbricoides* in humans and *Strongylus vulgaris* in horses have long migrations in various tissues of the host before they reproduce in the gastrointestinal tract. Other species may have less dramatic but still very characteristic habitat changes during development – most gastrointestinal nematodes in cattle and sheep burrow into the intestinal wall and stay there during the larval phase, before returning to the lumen as adults. It is these differences in larval and adult habitat, not age *per se*, that produce differences in drug vulnerability in most nematodes.

Parasite	Anthelmintic	Efficiency	
		Adult	Tissue migrating juveniles
Ascaris spp.	Benzimidazols	High	Moderate/low
	Ivermectin	High	Low
Parascaris equorum	Thiabendazole	Moderate	Low
	Ivermectin	High	Low
Toxocara spp.	Benzimidazols	High	Low
	Ivermectin	High	Low
	Doramectin	High	Low
Strongylus equinus	Ivermectin	High	Low
Hookworms	Mebendazol	High	Moderate
	Ivermectin	Moderate	Low

Table 1 Some nematodes of humans and domestic animals and examples of commonly used stage-specific drugs. Data from Campbell (1990), Cook (1990), and Conder & Campbell (1995)

There are several reasons why successive stages in a parasite life cycle have different tolerance to drugs. When juvenile and adult stages are located in different organs they may experience different concentrations of the drug. Parasite stages may also differ in metabolic activity, and thereby have different vulnerability (Sangster 1996). Many drugs act by temporarily paralysing parasites – this can be fatal in the gut where they will be expelled, but not necessarily in the tissues where they may recover and resume activity (Mansour 1979).

If parasite stage and habitat are better predictors of future possibilities of survival than parasite age, the consequences of drug pressure on life history evolution for many nematode species may well be the opposite of that predicted by Medley (1994) and Poulin (1998). When exposed to a drug that increases mortality in the adult stage, a parasite should respond by prolonging the larval stage, i.e. by *postponing* maturity. Formal models of this argument in the context of free-living organisms are summarized by Roff (1992). Tissue-migrating worms such as *Ascaris, Strongylus,* and hookworms are relatively safe from the drugs in the larval stage (Table 1). Prolonged larval developmental would allow them to achieve higher daily fecundity in the face of drug-reduced adult life expectancy. This is because prolonged pre-reproductive growth results in greater size and hence fecundity at maturity. This effect is enhanced in the tissues where, independent of any effects of chemotherapy and for reasons still unknown, growth rates greater than those in the gastrointestinal tract can be achieved (Read & Skorping 1995).

Once they have begun reproducing, drug pressure eases the selective importance of the costs of reproduction in terms of future fitness, so that increased reproductive effort should be favoured where drug use is common (Stearns 1992). Longer tissue phases and higher reproductive outputs are probably positively correlated with pathogenicity. Prolonging the larval phase likely increases the lesions, inflammatory responses, and other pathological complications associated with migrating nematode larvae (Anderson 1992). And increased reproduction per unit time must involve greater rates of resource extraction from hosts and hence harm to hosts (e.g. May & Anderson 1983). Continuous use of drugs that disproportionately affect intestinal stages of tissue migrating nematodes could therefore select for more pathogenic strains.

So far as we are aware, there is no evidence (either way) of genetic changes in nematodes towards a longer tissue phase and higher fecundity in response to drug pressure. Such changes would be hard to detect because of a naturally occurring intraspecific variability in both prepatency and fecundity (Anderson 1992). They may also be confounded by any costs of biochemical resistance, which may increase in response to drug pressure in parallel with life histories evolution. Usually parasitologists are not studying the effects of drug selection until resistance is widespread. Nevertheless, the selection pressure for developing resistance may be less in tissue-migrating parasites than in nonmigrating worms. In horses, for example, there is widespread drug resistance in the small nonmigrating strongyles, whereas there are few reports of resistance among the large, migrating worms (Conder & Campbell 1995). This is what we would expect if tissue-migrating nematodes are better able to respond to drug exposure by adaptive changes in their life history traits.

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BIOSKETCH

Arne Skorping's research interests include evolution and population dynamics of parasites and effects of parasites on host life histories.