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"Topics in Evolutionary Ecology"

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TOPICS IN EVOLUTIONARY ECOLOGY

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ABSTRACT. Various topics in evolutionary ecology are discussed, ranging from evolution as optimization, the joint evolution of avoidance and tolerance of toxins, dispersal and dormancy as adaptations in variable environments, seed dispersal models, and diffuse coevolution of chemical defenses and detoxification mechanisms.

1. Introduction

Mathematical models have played a central role in evolutionary theory at least since the pioneering work of Fisher, Wright and Haldane. Two approaches have been predominant: mechanistic and reductionistic ones that assume considerable detail about genetic mechanisms, and phenotypically-based ones that assume quantitative inheritance, or that suppress genetic detail entirely. Despite the successes of both approaches, they have in general failed to deal adequately with some of the central problems of evolutionary ecology – those in which strong nonlinear feedbacks result from intra-specific or inter-specific frequency dependence. Thus, the need for new ideas and new approaches is as pressing as ever.

These notes, which are based on a series of lectures given at the Université de Montréal by the first author, begin with a discussion of evolution as optimization, and the constraints that arise due to history, stochasticity, and frequency dependence. Life-history evolution in variable environments is treated through a consideration of dispersal and dormancy strategies, in which the importance of frequency dependence is illustrated. Finally, consideration is given to coevolution in host-parasite and plant-herbivore systems,

S. Lessard (ed.), Mathematical and Statistical Developments of Evolutionary Theory, 327–358.

with special attention to the coevolution of virulence and resistance in situations of tight and diffuse interactions.

2. Evolution as optimization

To a great extent, evolutionary ecology deals with the study of adaptations. The adaptationist approach views evolution as a problem solver, providing solutions that are in some sense "optimal" in a given environment. In short, evolution optimizes some nebulous quantity, the organism's "fitness". Herein lie the principal problems with the adaptationist approach: the identification of the end result (the definition of fitness) and the determination of the "purpose" of the genetic algorithm. As Gould (1977) argues, "...evolution has no purpose. Individuals struggle to increase the representation of their genes in future generations, and that is all." Lewontin (1977) clearly states that "Adaptation, for Darwin, was a process of becoming rather than a state of final optimality." Jacob (1977) expounds this view very eloquently when he points out that the process of evolution, being more similar to the work of a tinkerer rather than that of a master craftsman, is constrained by past history, chance, and the mechanics of the evolutionary process itself.

Sewall Wright, Ronald Fisher, and J.B.S. Haldane were among the most prominent figures in the development of the modern synthesis of population genetics and evolutionary theory. Among their contributions were the development of a mathematical framework for population genetics and the exploration of the population-level consequences of natural selection and other evolutionary processes. The paradigm developed through Fisher's Fundamental Theorem of Natural Selection and Wright's Adaptive Landscape – that through natural selection, fitness will gradually improve at a rate proportional to the remaining genic variance, and that the process can be viewed as hill-climbing – has become one of the most powerful in evolutionary theory, and provided a mathematical justification for the rise in status of optimization theory.

However, the usefulness of this paradigm always has been questioned. Recently, Provine (1986) has argued that the underlying concept of the multi-dimensional adaptive landscape is a useless and misleading metaphor, and indeed that Wright's own presentation of the notion has itself gone through substantive evolution. Levin (1978, 1983a), somewhat more positive on the subject, nonetheless has argued that it can mislead: "the conclusion that populations evolve towards maximization of mean fitness is easily vitiated, and the worst culprit is frequency dependence."

Despite the above objections, the paradigm that emerges from these approaches still provides one of the most important theoretical concepts in evolutionary theory. In some special simple cases it provides the correct picture (see Levin 1978; Ewens, 1979). In more complex situations, it provides a possible starting point for future extensions. For example, various authors have shown that variants on the metaphor are still valid for traits that are density-dependent, although multiple loci and frequency dependence introduce more fundamental problems. Although versions of the Fisher theorem can be developed under weak frequency dependence or weak epistasis (Ewens 1969a,b; Nagylaki 1976), in

general the undulating landscapes that arise under frequency dependence and coevolution mandate entirely new approaches.

We begin by discussing the problems that arise when landscapes are rugged and very high-dimensional, and then move on to the central problems of evolutionary ecology – frequency dependence and coevolution.

TOWARDS A GENERAL THEORY OF ADAPTIVE WALKS

The problems of high-dimensionality and ruggedness have been examined by Kauffman and Levin (1987), and their discussion forms our starting point. In particular, consideration of the hill-climbing metaphor as a heuristic solution algorithm is shown to be fraught with problems – false peaks, multiple pathways, etc. Computer simulations of simple cases reinforce our understanding of the importance of stochasticity and history.

The mutational process plays a crucial role in the generation of genetic variability. In this section we assume that we are dealing exclusively with point mutations that switch, insert, or delete single nucleotide bases. This is a reasonable starting point, because other types of mutations can be thought of as mechanisms that produce many mutations of the above type simultaneously. Kauffman and Levin (1987) construct a genotypic space by assuming that each genotype is surrounded by 1-mutant neighbors; that is, a single mutational alteration will transform the genotype under consideration into a neighboring type. We have then a space in which each point denotes a particular genotype and has as its immediate neighbors genotypes that differ by a single mutation. Note that the topology on this space is given by the mutational "move" generator that specifies the allowable transformations, i.e., that specifies which entities can mutate in one step to one another. Furthermore, observe that in this case the process is symmetric and reversible. This restriction can be relaxed.

Kauffman and Levin (1987) define a mapping from this genotypic space to the appropriate phenotypic space, and specify the fitness associated with each attribute (phenotype). The discrete distribution of fitness values across the genotypic lattice will be referred to as the fitness landscape. If, for example, we restrict ourselves to a haploid organism with DNA genotypes of length 100,000 nucleotides, then each position in the DNA sequence can be occupied by 4 alternative bases, and each genome has 300,000 1-mutant neighbors in the space of haploid genotypes. Hence, each genotype is surrounded by huge numbers of 1-mutant neighbors with (possibly) different fitness values. In this scenario, to the extent that the metaphor of the previous section is valid, adaptive evolution can be thought of as an uphill walk via 1-step fitter variants until a local or a global optimum is reached. This gradual adaptive climbing through mutation and selection provides the simplest trial and error optimization method, and is mimicked by the development of heuristic methods in combinatorial optimization (e.g., Lin and Kernighan 1973) and neural computing (Hopfield 1982).

What are the constraints that arise from such an approach to improvement? If the notion of neighborhood is extended to include k point changes (k = 1,2,3,...), how many local optima are there in the space with respect to k-mutants? If adaptive movement is allowed only through fitter neighbors, what is the expected number of improved variants

passed on the way to a local optimum? How long are adaptive walks that use this optimization algorithm? In how many ways can adaptive walks branch at each uphill step? How many local optima are there available for an arbitrary initial genotype? What role does the initial fitness play in the availability of local optima? What is the probability of attaining a global maximum? What is the correlation structure in a fitness landscape?

Consider the expected character of adaptive walks in (uncorrelated) spaces where the fitness value of each genotype is drawn at random from some fixed underlying distribution. Furthermore, replace the actual fitness values assigned to the genotypes in the space by their rank orders. The least fit has rank 1, the most fit has rank T. We assume that there are no tie values and that the fitness values are distributed uniformly on the real line. For concreteness consider (as in Kauffman and Levin 1987) a space of length-N peptides that can use only two amino acids. Hence we can represent each peptide as a binary string of length N. The space of peptides is the N-dimensional Boolean hypercube. There are 2^N strings, and we assign order fitnesses from 1 to 2^N at random without replacement to each of the points in the N-dimensional Boolean hypercube. The probability that a vertex is a local maximum is given by

$$P_m = 1/(N+1), \tag{1}$$

and the expected number of local optima with respect to 1-mutant neighbors, M_1, is

$$M_1 = \frac{2^N}{N+1}. \tag{2}$$

For 1- and 2-step mutant neighbors, the expected number of local optima, M_2, is

$$M_2 = \frac{2^{N+1}}{2+N(N+1)}, \tag{3}$$

and for k-step mutants, the expected number of local optima M_k is

$$2^{N+k} \Big/ \sum_{j=0}^{k} \binom{N}{j}. \tag{4}$$

Hence, in an uncorrelated fitness landscape, the number of local (1-step) optima is of the same order of magnitude as the number of possible peptides. If the peptides use B amino acids, then any peptide of length N has $D = (B-1)N$ 1-mutant neighbors, and M_1 is now given by

$$M_1 = \frac{B^N}{D+1} = \frac{B^N}{N(B-1)+1}. \tag{5}$$

As before, the number of local optima increases exponentially in N.

Kauffman and Levin (1987) show that the probability that an entity in this space is a local optimum is low if its rank is low, and rises rapidly when the rank increases. They further show that an upper bound for the average walk length R is $R = \log_2(D-1)$, and find that for greedy walks (those that always choose the best improvement) in an uncorrelated space the average walk length is less than 2. Gillespie has independently derived this result recently (Gillespie, personal communication), and Weinberger (1988) has confirmed it by developing more accurate estimations of walk length. In addition, using the fact that the number of alternative pathways towards increased fitness values decreases linearly with rank order, Kauffman and Levin (1987) calculate an upper bound for the expected number of local optima B accessible from the lowest rank entity:

$$B = D^{(\log_2 D-1)/2} \quad (6)$$

Hence, only a tiny fraction of all local optima are accessible from any entity on adaptive walks via 1-mutant fitter variants in uncorrelated landscapes. Walks in correlated landscapes in general will be longer.

For some implications of the results of this baseline case to the length of adaptive walks in the immune system, and to branching phylogenies in biological evolution, see Kauffman and Levin (1987) and Kauffman et al. (1988). Gillespie (1983, 1984) uses a variant of this model on the molecular clock hypothesis to show that burst-like evolution fits better with a selectionist theory that with a neutral theory.

Kauffman and Levin (1987) examine these results further by applying this evolutionary algorithm to optimization problems such as the traveling salesman problem (Lin and Kernighan 1973, Johnson and Papadimitrou 1985). They demonstrate the tendency of the scheme to get hung up on false peaks, and the importance of stochastic phenomena, especially early in evolution. They furthermore show that the most efficient optimization occurs for intermediate levels of mutation: low levels of mutation rapidly lock the system in to false peaks, whereas high levels do not take advantage of local information and progress already made. Furthermore, optimization is significantly enhanced when the process occasionally can go downhill and traverse valleys. This approach, which allows one to get free from false peaks, involves "simulated annealing" in heuristic combinatorial optimization, and can arise from a number of genetic mechanisms (shifting balance, genotypic variance, outcrossing, etc.).

EVOLUTION IN VARYING ENVIRONMENTS

The problems associated with the adaptationist approach include: the definition of the putative quantity to be maximized; the determination of what is heritable; the high dimensionality, which leads to large numbers of optima; pleiotropy, linkage and epistasis; temporal variation in fitness; frequency and density dependence; and coevolutionary interactions (among populations and with the environment). The last two classes of problems are perhaps the central ones in understanding natural communities and ecosystems. Essentially, whatever is being optimized is a tradeoff against different

environments, which the species defines and alters as it evolves. One of the most important sets of constraints, and one of the least explored in theory, arises from the tradeoffs involving different phenotypic aspects. These may involve pleiotropy or interactions among loci, but most often involve different genotypes being favored in different parts of a heterogeneous environment. Castillo-Chavez et al. (1989) explore the tradeoffs between the evolution of habitat selection and physiological adaptation in a heterogeneous environment. They start by developing a two-locus model that considers a panmictic population in which prereproductive individuals are mobile enough to move among patches. Alleles at one locus code for the absence or presence of physiological adaptation to detrimental patches, and alleles at the second locus code for the absence or presence of behavior that cause avoidance to detrimental patches. It is further assumed that the effects of alleles controlling physiology and behavior are additive and that fitnesses are frequency independent.

Table 1: Fitnesses of the various genotypes.

The fitness (w) of each genotype is dependent upon whether it is in the toxic (T) or non-toxic (UT) environment. The fitnesses (w) in the environment (L) are $w_{L(RR)}$, $w_{L(\pi)}$, and $(w_{L(RR)}+w_{L(\pi)})/2$ for individuals with RR, π, and Rr respectively, where L is either (T) or (UT). X and Y are defined in the text.

$$w_{(RRAA)} = Xw_{T(RR)} + (1-X)w_{UT(RR)}$$

$$w_{(RRAa)} = \left(\frac{X+Y}{2}\right)w_{T(RR)} + \left(1 - \frac{X+Y}{2}\right)w_{UT(RR)}$$

$$w_{(RRaa)} = Yw_{T(RR)} + (1-Y)w_{UT(RR)}$$

$$w_{(RrAA)} = X\left[\frac{w_{T(RR)}+w_{T(\pi)}}{2}\right] + (1-X)\left[\frac{w_{UT(RR)}+w_{UT(\pi)}}{2}\right]$$

$$w_{(RrAa)} = \left(\frac{X+Y}{2}\right)\left[\frac{w_{T(RR)}+w_{T(\pi)}}{2}\right] + \left(1 - \frac{X+Y}{2}\right)\left[\frac{w_{UT(RR)}+w_{UT(\pi)}}{2}\right]$$

$$w_{(Rraa)} = Y\left[\frac{w_{T(RR)}+w_{T(\pi)}}{2}\right] + (1-Y)\left[\frac{w_{UT(RR)}+w_{UT(\pi)}}{2}\right]$$

$$w_{(\pi AA)} = Xw_{T(\pi)} + (1-X)w_{UT(\pi)}$$

$$w_{(\pi Aa)} = \left(\frac{X+Y}{2}\right)w_{T(\pi)} + \left(1 - \frac{X+Y}{2}\right)w_{UT(\pi)}$$

$$w_{(\pi aa)} = Yw_{T(\pi)} + (1-Y)w_{UT(\pi)}$$

More specifically, two semi-dominant alleles at a single locus, allele R (resistant) and allele r (susceptible), determine the degree of physiological resistance. Repellency is governed by two semi-dominant alleles at the second locus: allele A codes for a high degree of avoidance, while allele a codes for lower avoidance. Ten genotypes are possible. To impose a price on avoidance, Castillo-Chavez et al. (1989) allow the fitnesses (w) to depend on whether the individual is found in a chemically treated (T) or untreated (UT) environment. Define the probabilities that a particular individual will be found in a given environment as:

$$\begin{aligned} &\text{Prob(individual AA is in T)} = X, \\ &\text{Prob(individual aa is in T)} = Y, \\ &\text{Prob(individual Aa is in T)} = (X+Y)/2. \end{aligned} \tag{7}$$

The Rr genotype's fitness in a particular environment is defined to be the arithmetic mean of the corresponding (identical at the complementary locus) homozygous genotypes in the same environment. The overall expected fitness for any genotype is the arithmetic mean of its expected fitnesses, W_T and W_{UT}, respectively in toxic and nontoxic environments, weighted by the probabilities specified by (7) (the overall fitnesses are summarized in Table 1, from Castillo-Chavez et al. 1989).

Introduce the notation $w_{(RRAA)} = \alpha$, $w_{(RRaa)} = \beta$, $w_{(rrAA)} = \gamma$, $w_{(rraa)} = \delta$, for the fitnesses of the double homozygotes, and let $x_1(t)$, $x_2(t)$, $x_3(t)$, and $x_4(t)$ denote the frequencies of the chromosomal types RA, Ra, rA, and ra, respectively. Then the frequencies of these chromosomal types in successive generations are related by the iterative scheme (Felsenstein 1965):

$$x_i' = \frac{w_i}{\bar{w}(x)} x_i + e_i k D \frac{w_H}{\bar{w}(x)}, \quad i = 1,2,3,4, \tag{8}$$

where $e_1 = e_4 = -e_2 = -e_3 = -1$. The prime denotes the succeeding generation, D denotes the linkage disequilibrium coefficient, k the recombination fraction between the two loci and $w_H = (\alpha+\beta+\gamma+\delta)/4$ the double heterozygote fitness. w_i is the mean fitness associated with allele x_i, and $\bar{w}$ is the mean fitness in the population.

The most general outcome of this scheme is fixation for a single gametic type. For example, if in the presence of a toxin the type (ra) that can make no response is the least fit ($\delta < \alpha,\beta,\gamma$), then the plant species will evolve either a physiological or behavioral response, and may evolve both if the double homozygote possessing both (resistance and avoidance) is the most fit double homozygote ($\alpha > \beta,\gamma$). On the other hand, if in contrast, $\alpha < \beta,\gamma$, then a bistable situation may result in which either resistance or avoidance can evolve, depending on initial gene frequencies. In this case, which implies a burden to having both features when one will do, the most fit double homozygote will not necessarily prevail. The initial conditions, the recombination rate, and the values of α and γ can influence recombination. The complexity of the situation is illustrated in Figure 1 (from Castillo-Chavez et al. 1989).

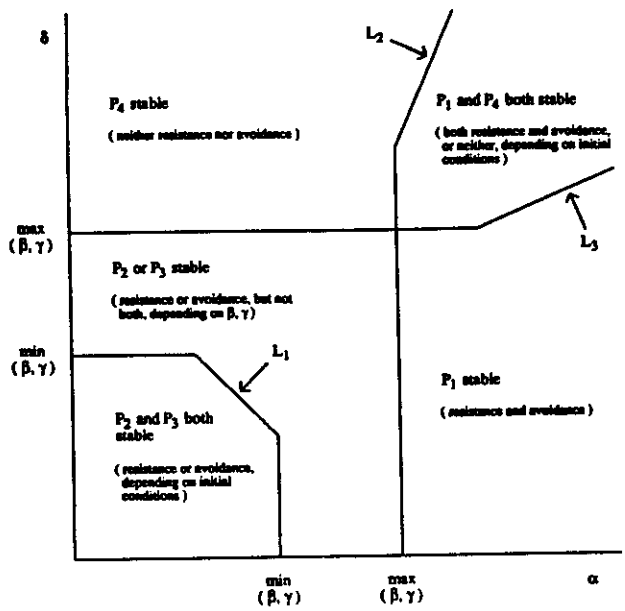

Figure 1. Stability regions for various equilibria.

$$L_1 : \alpha + \delta + \max(\beta,\gamma) = \frac{3+k}{1-k} \min(\beta,\gamma) ,$$

$$L_2 : \beta + \gamma + \delta = \frac{3+k}{1-k}\alpha , L_3 : \beta + \gamma + \alpha = \frac{3+k}{1-k}\delta .$$

The problem of joint selection for behavioral and physiological traits is exemplary of more general problems concerned with multiple genetic responses to single selective factors (e.g., Cohan 1984). Problems such as this frustrate the application of simple optimization approaches, because interest must be on the diversity of environments that might be confronted. Nonetheless, there are situations in which the objections to the adaptationist programme are more fundamental because of the importance of nonlinear feedbacks through frequency dependence and coevolution. We begin to treat these in the next section.

3. Dispersal

Among the central problems in ecology are the statistical description of movement and the understanding of population distributions in terms of individual behavior. Questions regarding issues as diverse as the evolution of life history traits or the spread of genetically

engineered organisms are crucially tied to our understanding of the dispersal patterns of plants and animals. One of the most fascinating challenges in evolutionary ecology is to determine the role that the spatial and temporal structure of the environment plays in the dispersal of individuals. These questions were raised years ago by Skellam (1951) and Hutchinson (1951). The evolutionary aspects of dispersal have received much less attention because of the complexities introduced by frequency dependence (Levin 1987).

In 1977, Hamilton and May asked: What are the advantages of dispersal for annual plants living in a renewable and stable environment? If there is a cost to dispersal, and if the habitat is uniformly good (or bad), then why disperse at all? The use of naive optimization arguments in such situations would dictate against dispersal, since there is cost without apparent gain. If, however, we consider the frequency dependence that is implicit when different genotypes are in competition, then the answer is quite different. Dispersers outcompete nondispersers. Furthermore, by applying the concept of evolutionarily stable strategies, Hamilton and May found that the best possible strategy within their model is to disperse with a probability equal to the reciprocal of 1 plus the probability of loss during dispersal. Hence, even if 90% of the dispersers are lost before reaching an appropriate site, the evolutionarily stable strategy (ESS) still is to disperse 52.6% of the seeds.

The Hamilton and May approach is elegant in its clear and simple demonstration of the basic need to consider frequency dependence. To examine the evolution of dispersal, however, one must consider a more general class of environments and strategies. Indeed, dispersal is just one possible evolutionary response to local unpredictability (broadly understood) and takes its place in a spectrum that includes dispersal, dormancy, diapause, iteroparity, and vegetative spread. In what follows we focus on two particular strategies: dispersal and dormancy.

Intuitively, dispersal and dormancy can be thought of as alternative strategies for individuals that have to deal with the spatial and temporal variability of the environment. In the previous section, we discussed an analogous problem: in the face of a toxic environment, two alternative strategies are physiological resistance and behavioral avoidance. It was seen that there are tradeoffs among these. Hence, similarly, one expects that the evolved level of one factor (dispersal or resistance) is a function of the other (dormancy or avoidance).

To gain understanding of the differences and similarities between dormancy and dispersal, and with the objective of determining the conditions needed for a strategy to dominate, Levin et al. (1984) developed a simple model of population growth in varying environments. In what follows, the effects of the spatial and temporal structure of the environment, as well as the relative cost of both strategies, are discussed in the context of the Levin, Cohen, Hastings model. Details can be found in Cohen and Levin (1987).

A seed population of annual plants in a patchy environment is considered. For patch j, the basic growth equation before germination, for the seed population, is given by:

$$S^j_{t+1} = S^j_t[GY^j_t(1-D)+(1-G)V] + \frac{ADG}{L}\sum_{i=1}^{L} Y^i_t S^i_t . \qquad (9)$$

We assume that an equation of this type applies for each genotype (= phenotype). In the above equation, G denotes the constant annual germination fraction and D denotes the constant dispersal fraction of seeds. Only the parameters G and D are genotype dependent; all others are assumed to be the same for all genotypes (although the approach could be extended to examination of the evolution of those parameters as well). A denotes the fraction of dispersing seeds that are successful at reaching a safe habitat, and V denotes the survival of those nongerminating seeds that remain dormant. Since the seeds are dispersed uniformly over all L patches, we take the summation over L. Y denotes a density-dependent yield function that is assumed to have the form $Y(Z) = K/(Z+b)$, where Z denotes the total density of all competing types in a given patch, and K is a random variable that denotes the total seed yield of the patch. For more general growth functions, see Levin et al. (1984). K is assumed to be independently distributed among patches; however, within a given patch, K has several possibilities: it may vary independently among years, or it may show a positive or a negative temporal correlation. D^* [G^*], the evolutionarily stable strategy for dispersal [germination], is defined by the condition that its genotype, once established, cannot be invaded by any rare mutant playing a different strategy. In what follows, the details of the genetic system are ignored and alternative strategies are assumed to involve competing asexual clones.

Numerical simulations show that D^* is an increasing function of the germination fraction, G, if the latter is held fixed, and is a decreasing function of V (the survival of nongerminating seeds). On the other hand, Ellner (1985) shows that in the absence of dispersal, G^* is given implicitly by

$$1/V = \text{Expectation}(S_t/S_{t+1}),$$

and the numerical simulations seem to agree with Ellner's result as D^* approaches zero.

In the more general case, simulations indicate that G^* is an increasing function of dispersal (D), and of the effectiveness of dispersal (A). Furthermore, A and D seem to affect optimal germination mostly through the factor $F = AD/(1-D)$, which represents the seed's effective dispersal fraction, and G^* approaches zero as V approaches unity.

When dispersal and dormancy both are subject to selection, the optimal strategy is obtained as the intersection of the curves $D^*(G)$ and $G^*(D)$. In the absence of temporal correlation in environmental variation, this optimum appears to be a stable equilibrium. If, however, the environment is cyclical, then this internal equilibrium is unstable and there are two competing boundary equilibria. Simulations show that coexistence among these boundary equilibria is possible, but that more generally one of the strategies outcompetes the other. More specifically, the conclusions of the numerical simulations as reported in Cohen and Levin (1987) are:

(1) The optimal dispersal decreases as the level of dormancy $H = (1-G)V$ increases.

(2) The optimal dormancy level decreases as the level of dispersal $F = AD/(1-D)$ increases.

(3) The ratio between dispersal and dormancy in the joint optimal strategy is affected by the ratio between the effectiveness of dispersal A and the survival of dormant seeds V. Therefore, the distribution of dispersal and dormancy among plant families or species

from the same environment should be negatively correlated. This agrees with observations (Ellner and Shmida, unpublished, Venable and Lawlor 1980).

(4) In environments that vary periodically, there is no single joint optimal strategy with intermediate levels of dispersal and dormancy. Cohen and Levin (1987) used simulations to investigate stability and found that the only stable equilibria were at the boundaries $G^* = 1$ or $D^* = 0$. It was found, however, that two such boundary strategies could coexist in a stable frequency dependent equilibrium, which would be the eventual evolutionary equilibrium reached among many competing mutants with a wide range of dispersal and dormancy levels. For further details see Cohen and Levin (1987). Further investigations, to be published (Cohen and Levin, submitted), have focused on the influences of temporal and spatial correlation patterns and environmental variability.

From the above summary we can see that the tradeoffs between dispersal and dormancy are somewhat analogous to the tradeoffs between resistance and behavior previously discussed. It is rare to find populations that select both strategies, since this will add the burden of having both features when one will do.

The most important conclusion of these investigations is the essential nature of the concept of ESS when frequency dependence is involved. We cannot compare one strategy against another unless we put them in competition, and attempts to approach such problems from the view point of optimization theory generally give incorrect answers.

RANDOM WALK MODELS OF DISPERSAL

Having made the evolutionary case for the existence of dispersal – to escape local environmental deterioration, to reduce sib competition, to average the negative consequences of unpredictability, and to explore new habitats – we make a detour to ask about the observed patterns of dispersal. How far and how rapidly do organisms disperse?

The classical models of movement (e.g., Skellam 1951, Okubo 1980) are based on random walk models. Random walk models are derived from the assumption that individuals move in a series of discrete steps, the direction of each step being determined by probabilities totally specified by positional information (but see Kareiva and Shigesada (1983) for a discussion of correlated walks). The application of such models to populations of organisms (or molecules) does not require that the basic assumptions be valid for the actual movements of individuals, but rather that other details of how the individual moves be irrelevant to the patterns of spread of populations.

The simplest one-dimensional random walk model can be motivated by the following experiment (see Okubo 1980, Levin 1986). Assume that an organism is located at the origin of the real line; that at discrete times $k\tau$, it jumps either forward (right) or backward (left) λ units; and that either event has probability $1/2$. If m and n are integers, then the probability that at time $n\tau$ the organism is at position $m\lambda$ after its latest jump is given by the general term of the Bernoulli distribution:

(10) $$\text{Prob} = \left(\frac{1}{2}\right)^n \frac{n!}{\left(\frac{n+m}{2}\right)!\left(\frac{n-m}{2}\right)!}.$$

As n increases, this converges to the Gaussian distribution given by

(11) $$Ce^{-m/2}$$

where

(12) $$C = \sqrt{\frac{2}{\pi n}}.$$

If we let $x = \lambda m$ and $t = \tau n$, then the Gaussian distribution is given by

(13) $$C \exp\left(-\frac{x^2}{4t} \cdot \frac{2\tau}{\lambda^2}\right).$$

This tends to

(14) $$\rho(x,t) = C \exp\left(-\frac{x^2}{4Dt}\right), \text{ where } C = \frac{1}{2\sqrt{\pi Dt}},$$

provided that λ and τ shrink to zero in a way that the limit

(15) $$\lim_{\lambda,\tau \to 0} \frac{\lambda^2}{\tau} = 2D$$

exists. D is known as the diffusion coefficient. For generalizations to higher dimensions see Okubo (1980) or Lin and Segel (1974). As Okubo (1980) points out, the diffusion approximation is valid only on scales that involve a great many individual steps.

Observe that the population is normally distributed for $t > 0$, and has variance $2Dt$, which increases linearly with time; and that this distribution satisfies the diffusion or heat equation. Note further that the diffusion equation more generally describes the spread of a diffusing population with any distribution (that is, not just the normal distribution that would result if all individuals began at the same point in space and time). Furthermore, it can be shown that the variance $V(t)$ has the general form:

(16) $$V = V_0 + 2Dt;$$

that is, the variance increases linearly with time from its initial value V_0.

Kareiva (1983), using data on the foraging movements of phytophagous insects, estimated D from the slope of the regression of V on t. He used his estimate of D to generate a series of probability distributions for the spread of insects, and to compare them

with actual observations. Agreement was excellent in many cases, but in some instances the habitat-dependent diffusion model,

$$\frac{\partial P}{\partial t} = \frac{\partial^2}{\partial x^2} \cdot (D(x)P) \tag{17}$$

provided a better fit. He concluded that the basic diffusion model was an excellent starting point, but that modifications of this basic formalism are necessary to take into consideration the substantial habitat variability that organisms often experience.

If growth and spread occur simultaneously, then the diffusion model gives way to

$$\frac{\partial P}{\partial t} = D\frac{\partial^2 P}{\partial x^2} + F(P,x,t)\ , \tag{18}$$

where $F(P,x,t)$ denotes local population growth. If $F(P,x,t) = rP(1-P)$, then we arrive at the simplest model introduced by Fisher (1937) to describe the rate of advance of advantageous alleles, given that selection is operating on two alleles at a single autosomal locus. A more general, cubic, form is necessary when there is partial or complete dominance, and can lead to fundamentally different results. P in this context denotes the frequency of the advantageous allele. The correct approach to the population genetics problem is to imbed this within a fuller treatment of genotype frequencies (see Aronson and Weinberger 1975, Hoppensteadt 1975, Hadeler and Rothe 1975). However, the basic insights that emerge from Fisher's model, at least regarding rates of spread, are essentially the same (see Hadeler 1976).

Fisher's fundamental insight, based on such models, lies in his estimate of the asymptotic speed of advance of a wave front. Fisher's conjecture – that an advancing wave would relax asymptotically to a front with this characteristic speed – was formalized by Kolmogorov et al. (1937), who considered the general equation

$$\frac{\partial P}{\partial t} = D\frac{\partial^2 P}{\partial x^2} + f(P)\ , \tag{19}$$

where

$$f(0) = f(1) = 0\ ,\ f > 0 \quad \text{on} \quad (0,1) \tag{20}$$

and

$$f'(0) > f'(P) \quad \text{on} \quad [0,1]\ . \tag{21}$$

By looking for traveling waves (non-negative solutions of the form)

$$P = H(x-ct)\ ,\ c > 0\ , \tag{22}$$

Kolmogorov et al. (1937) proved the existence of monotone wave solutions for all wave speeds greater than or equal to the critical speed

$$c^* = 2\sqrt{D \cdot f'(0)} \quad . \tag{23}$$

There are no such solutions for $c < c^*$; furthermore, if P is initially given by a Heaviside distribution, then the wave corresponding to $c = c^*$ is attracting (see Hadeler 1976). For a complete mathematical treatment, the reader is referred to Bramson's (1983) monograph and to Fife (1984).

Skellam (1951) applied models of this type to the study of species invading new habitats, and Aronson and Weinberger (1975) have used systems of equations of this type in population genetics. Kendall (1965), Hadeler (1984), and other investigators have extended them to the study of the spread of epidemics. Recent applications are provided by Lubina and Levin (1988) and Andow et al. (1989). In many cases, the agreement between theory and experiment is excellent; in others, the assumption that movement is the result of numerous small steps clearly leads to the wrong answers, and more general redistribution kernels are necessary (see for example Mollison 1977).

ADVECTION-DIFFUSION MODELS OF DISPERSAL

The consideration of population rates of spread is predicated on assumptions concerning the movements of individuals. The spread of plant populations occurs via seeds and pollen. We therefore conclude the section on dispersal with a brief presentation of an advection-diffusion description for the wind dispersal of seeds and pollen (Okubo and Levin 1989). The shape of the *dispersal curve*, that is, the curve relating the number of dispersed seeds to distance from source, varies depending upon the speed of descent (the "settling" velocity), the height of release, wind speed and turbulence, and specific morphological adaptations for dispersal (Augspurger and Franson 1987). Typically, it falls off with large distances; but because of the effects of wind, it achieves its apex at some distance away from a point source. On the other hand, for a distributed source, we have a different situation, as the peak usually occurs at or close to the boundary of the source region.

To understand what factors control the forms of such dispersal curves, Okubo and Levin (1989) consider diffusive and advective forces with regard to properties of the propagules and height of release. As a first approximation, they do not take into account the influence of the parent plant on microscale air movements (Niklas 1984), and do not allow seeds to move once they strike the ground.

Dispersal curves with phenomenological derivations have been used widely; examples include the inverse power law (Gregory 1968), and the negative exponential (Frampton et al. 1942, Kiyosowa and Shiyomi 1972). These curves do not deal with transients, being confined to the asymptotic distribution of seeds, spores, or pollen from point releases, or the time-averaged solutions for continuous point sources. More importantly, they involve curve-fitting, and do not allow predictions to be made based on physical parameters such

as wind velocity, turbulence, seed weight or height of release. More details can be found in Gregory and Read (1949) and Minogue (1986).

The inverse power law is given by

$$y = as^{-b}, \tag{24}$$

where s denotes the distance from source, y the probability distribution associated with dispersal, and a and b are constants. It transforms to a straight line on a log-log plot, making parameter estimation simpler; b is dimensionless, and hence it provides an advantage when one is dealing with studies on different scales.

The log-linear (negative exponential) model has the shape

$$y = ae^{-bs}, \tag{25}$$

which transforms to linear on a semi-log plot. Note that for this model y remains finite as s tends to zero. Each of these models has advantages (see Gregory 1968, McCartney and Bainbridge 1984, Fitt and McCartney 1986). However (Okubo and Levin 1989), they do not allow extrapolation from one solution to another based on independently measured physical parameters, and provide no understanding of the underlying mechanisms.

GAUSSIAN PLUME MODELS

Gaussian plume models have been used primarily for the description of the dispersion of air pollutants from smokestacks, but they also have been applied to spore dispersal (see for example Gregory et al. 1961, Fitt and McCartney 1986).

The Gaussian plume method (Csanady 1973; Hanna et al. 1982) uses Sutton's (1947) steady-state solution for a special type of the diffusion equation. The assumptions are: reflection at the surface of the earth, constant wind speed u in the x-direction at source height, and a continuous point source at height H above the ground. In addition, diffusion in the x direction is neglected relative to advection. Furthermore, it is assumed that particles are deposited at the surface of the earth at horizontal position (x,y) at the rate

$$D = S(x,y,0)V_d, \tag{26}$$

where V_d is the deposition velocity (Chamberlain 1975).

Using the reflection boundary conditions, one obtains the solution

$$S(x,y,z) = n(x)\frac{\exp(-y^2/2\sigma_y^2)}{2\pi\bar{u}\sigma_z\sigma_y}\left[\exp\left(\frac{-(H-z)^2}{2\sigma_z^2}\right)\right], \tag{27}$$

where $n = n(x)$ is the effective *source* strength at distance x, and the standard deviations σ_z, σ_y are function of x. (See Pasquill and Smith 1983, p. 333.) Dependence of n on x allows for losses due to deposition (Horst 1977). This model assumes that we are dealing with very light particles, and hence it does not take gravity

into consideration. For heavy particles, the *tilted plume* model is obtained by replacing the effective height H of the plume by $H-xW_s/u$, where W_s is the settling velocity of seeds. This extends the plume model to the situation when particulates have a non-trivial settling velocity (see e.g. Csanady 1973).

Under simplifying assumptions (see Okubo and Levin 1989), it is found that the rate of deposition at the ground is given by

$$D = S(x,y,0)W_s , \tag{28}$$

where $W_s = V_d$. From this, Okubo and Levin (1989) determine an expression for the concentration of seeds at the ground level:

$$D = Q(x,y) = \frac{n(x)W_s}{2\pi\bar{u}\sigma_y\sigma_z} \exp\left\{\frac{y^2}{2\sigma_y^2} + \frac{(H-W_s x/\bar{u})^2}{2\sigma_z^2}\right\} . \tag{29}$$

The crosswind-integrated deposition rate (CWID) is obtained by integrating across the direction of the wind:

$$\text{CWID} = \int_{-\infty}^{\infty} Q(x,y)dy = Q(x) = \frac{nW_s}{\sqrt{2\pi}\,\bar{u}\,\sigma_z} \exp\left\{\frac{-(H-W_s x/\bar{u})^2}{2\sigma_z^2}\right\} . \tag{30}$$

Ignore the decay in $n(x)$ [set $n(x)$ = constant], and set

$$\sigma_z^2 = 2Ax/\bar{u} , \tag{31}$$

where A is the vertical diffusivity. This is motivated by the fact that, under pure diffusion, variance increases at the rate $2At$, and by the fact that the time to reach position x is x/u. The distribution is skewed; the maximum is less than or equal to the mean, and is given by

$$x_m = \frac{\bar{u}H}{W_s}\left[\{1+(A/HW_s)^2\}^{\frac{1}{2}} - (A/HW_s)\right] , \tag{32}$$

which agrees with the mean in the absence of vertical diffusivity ($A = 0$).

Define

$$2AH = W^* \text{ (vertical mixing velocity)} , \tag{33}$$

and rewrite (32) as

$$\lambda\frac{x_m}{H} = \frac{\bar{u}}{W_s} , \tag{34}$$

where

$$\lambda = (1+(W^*/2W_s)^2)^{\frac{1}{2}} + W^*/2W_s . \tag{35}$$

For small values of W^*/W_s (heavy seeds)

$$\lambda \approx 1 + W^*/2W_s \approx 1 , \tag{36}$$

and so $x_m \sim Hu/W_s$; whereas, for large values of $W^*/2W_s$ (light seeds),

$$\lambda \approx W^*/W_s \gg 1 , \tag{37}$$

and so $x_m \sim Hu/W^*$.

In Okubo and Levin (1989), the above model is extended to incorporate more precisely the dynamics of advective and diffusive movements in both the horizontal and vertical directions, and correct boundary conditions at the earth's surface. Horizontal advection is determined by mean wind speeds, while the vertical advective force is gravitational. These and other assumptions are made to determine the equations governing the dispersal of seeds or pollen from an isolated plant or tree. The major change in the calculation of the mode is that formula (35) is replaced by

$$\lambda = 1 + W^*/W_s . \tag{35a}$$

Actual dispersion relationships obtained with data in 15 studies are compared with model predictions (Okubo and Levin 1989).

These models, borrowed from the atmospheric diffusion literature, allow establishment of a framework for organizing data concerning the relationship of dispersion distances to environmental and species-specific parameters, and to such other parameters as height of release. This presents us with an improved situation because the conventional models are phenomenological, and hence do not provide a basis for extrapolation from one environment to another. Further details and a more elaborate discussion can be found in Okubo and Levin (1989).

4. Tight and diffuse coevolution

As mentioned previously, the study of coevolutionary interactions is one of the central problems in evolutionary biology. In this section, following now conventional usage, we make a clear distinction between *tight* coevolution, involving a few closely linked species, and *diffuse* coevolution, in which the influences are spread over many species.

Many models of tight coevolutionary interactions fall within the framework of explicit genetic models (e.g., Levin and Udovic 1977). This approach is useful when the bases for inheritance are well understood, and when the number of loci involved is small. However, for parasite-host systems, most such classical models ignore the central

ecological and epidemiological interactions that are faced by intimately interacting species. Incorporating the nature of these interactions into models is critical if we are to understand phenomena such as the evolution of virulence and disease, because classical models do not take account of the truly tight interdependence of the host and parasite.

The study of diffuse coevolutionary interactions, involving a multitude of species, demands a different perspective and a different approach. To this end, we discuss some preliminary work on the evolution of chemical defenses.

TIGHT COEVOLUTION: MODELS OF HOST-PARASITE COEVOLUTION

One of the best examples of the successful application of explicit genetic models for tight coevolutionary interactions involves the gene-for-gene systems of cereal plants and flax and their fungal pathogens (rusts). In these systems, specific genes for host resistance may be attached to specific genes for parasite virulence; and hence there is strong selection for specific characters (e.g., Feeny 1975, Janzen 1980).

The study of the cereal-rust interactions builds on the experimental work of Flor (1955, 1956) and the theoretical work of Mode (1958, 1960, 1961). It is a common characteristic of these models to omit the epidemiological details and formulate the probability of association between parasites and hosts in terms of a mass-action law. For a review of the literature on cereal-rust interactions, see Levin (1983b).

To be more specific, we briefly describe the treatment of this problem by Lewis (1981a, 1981b). In Lewis (1981a), the host is assumed to be a diallelic diploid and the pathogen, a diallelic haploid. Pathogen fitnesses, for each host-parasite association, are described in the table below. The fitness w of the host in each pair is 1 minus the pathogen fitness.

Table 2

Host Genotype

Pathogen Genotype	AA	Aa	aa
B	α	β	γ
b	γ	β	α

It is assumed that the frequencies of the particular associations are proportional to the products of the corresponding associated types. If, in addition, x denotes the fixed probability that a host is parasitized, then we arrive at the following model (due to Lewis):

$$\rho' = \rho \frac{\rho W_{AA}+(1-\rho)W_{Aa}}{\rho(\rho W_{AA}+(1-\rho)W_{Aa})+(1-\rho)(\rho W_{Aa}+(1-\rho)W_{aa})} \tag{38}$$

for the host. Here

$$W_{AA} = 1 - x + x[q(1-\alpha)+(1-q)(1-\gamma)] = 1 - x[q\alpha+(1-q)\gamma]$$

(39) $$W_{Aa} = 1 - x + x[q(1-\beta)+(1-q)(1-\beta)] = 1 - x[\beta]$$

$$W_{aa} = 1 - x + x[q(1-\gamma)+(1-q)(1-\alpha)] = 1 - x[q\gamma+(1-q)\alpha]$$

and

(40) $$q' = q\frac{V_B}{qV_B+(1-q)V_b}$$

for the pathogen, in which

(41) $$V_B = p^2\alpha + 2p(1-p)\beta + (1-p)^2\gamma .$$

and

(42) $$V_b = p^2\gamma + 2p(1-p)\beta + (1-p)^2\alpha .$$

Here, p and q are, respectively, the allelic frequencies of allele A in the host and allele B in the pathogen in the present generation, while p' and q' denote the frequencies of A and B in the next generation.

From the symmetry of the system, it follows that is has an internal polymorphic equilibrium at $p = q = 0.5$. However, this equilibrium is stable if and only if

(43) $$\beta < \frac{\alpha+\gamma}{2}\sqrt{1-2\frac{\alpha-\gamma^2}{(\alpha+\gamma)^2}} < \frac{\alpha+\gamma}{2}$$

From the viewpoint of the host population, this condition is stronger than marginal overdominance, which is always a necessary condition for the stability of polymorphic equilibria in the absence of frequency dependence (see Levin and Udovic 1977). In addition, oscillatory solutions have been found when β is increased above the threshold specified above, in agreement with fluctuations observed in many simplified agricultural systems.

It follows that when resistance is dominant and virulence recessive, as in many cereal-rust systems, stable polymorphisms cannot be established through such models, since marginal overdominance is impossible. How then is stability realized in host-parasite systems? The most likely explanation is through explicit or implicit frequency dependence, which stabilizes such interactions (see Gillespie 1975).

Frequency dependence of some form is inescapable when one is interested in the evolution of virulence. As Anderson and May (1982b) point out, most standard textbooks take the dogmatic approach that parasite evolution is towards less and less virulent pathogen strains, with commensalism the inevitable end point. The situation, however, is

not this simple. As Levin (1983a) states: "Evolution in parasite populations represents an interplay between conflicting factors: within an individual host, the race is to the swift and evolution will favor those with the highest rate of reproduction, which is likely to mean those with higher virulence. But the parasite population is a shifting mosaic of demes associated with individual hosts, and the capacity for profligate growth may doom one's host to a shorter life expectancy and reduce the contribution to the larger (mega-) population. Depending on the balance between these factors, some evolution towards attenuation might be expected among parasites, but this attenuation may be checked far short of commensalism (Levin and Pimentel 1981, Anderson and May 1982a, Bremermann and Pickering 1982)."

The most famous example of loss of reduced virulence occurred in the (European) rabbit-myxoma system in Australia. The myxoma virus was introduced to control the rabbit population, which had denuded the landscape (Fenner and Ratcliffe 1965); hence, loss of virulence may lead to loss of control. To examine this system, Levin (1983a, see also Levin and Pimentel 1981) built upon classical epidemiological models to arrive at the model:

$$
\begin{aligned}
\frac{dS}{dt} &= (r_o S + r_1 I_1 + r_2 I_2 + r_3 I_3) - bS - \beta_1 S I_1 - \beta_2 S I_2 + v_1 I_1 + v_2 I_2 \\
\frac{dI_1}{dt} &= \beta_1 S I_1 - (b+\alpha_1) I_1 - v_1 I_1 + w_2 I_3 - \gamma_2 \beta_2 I_1 I_2 \\
\frac{dI_2}{dt} &= \beta_2 S I_2 - (b+\alpha_2) I_2 - v_2 I_2 + w_1 I_3 - \gamma_1 \beta_1 I_1 I_2 \\
\frac{dI_3}{dt} &= (\gamma_1 \beta_1 + \gamma_2 \beta_2) I_1 I_2 - (w_1 + w_2) I_3 - (b+\alpha_3) I_3 .
\end{aligned}
\tag{44}
$$

Here, S denotes susceptible hosts; I_1, I_2 denotes hosts infected with strain 1 and 2 respectively; and I_3 denotes hosts infected with both strains. The parameters r_i represent the birth rates; v_i, w_i represent the recovery rates; b and $b + \alpha_i$ represent the death rates: β_i denote the transmission rates; and γ_i denote the secondary infection rates. Such a framework allows explicit consideration of evolutionarily stable strategies, while recognizing the importance of the host-parasite interaction.

Possible outcomes of this model include: competitive exclusion of either viral type, their stable coexistence, or unbounded behavior. A positive polymorphic equilibrium satisfies the following conditions

$$I_2 = \frac{\beta_1}{(\gamma_2\beta_2 - w_2 Q)}\left(S - \frac{b+\alpha_1+v_1}{\beta_1}\right)$$

(45)
$$I_1 = \frac{\beta_2}{(w_1 Q - \gamma_1\beta_1)}\left(\frac{b+\alpha_2+v_2}{\beta_2} - S\right)$$

$$I_3 = QI_1I_2 = \frac{\gamma_1\beta_1+\gamma_2\beta_2}{w_1+w_2+b+\gamma_3} I_1I_2 .$$

Note that in order for I_1 and I_2 to be positive, we need that:

(46)
$$\frac{b+\gamma_2+v_2}{\beta_2} > S > \frac{b+\gamma_1+v_1}{\beta_1}$$

and

(47)
$$\gamma_2\beta_2 w_1 > \gamma_1\beta_2(w_2+b+\gamma_3) .$$

The most important questions involve an explanation of which viral strains survive and why, and of how the virus can be coupled with other control measures to lead to effective control of the virus. We note that the results of the above modelling exercise only scratch the surface of the complicated questions regarding the evolution of virulence in the parasite and of resistance in the host. For the myxoma-rabbit system, other factors have to be considered: seasonality and multiple modes of transmission as well as the role played by this pathogen in regulating the host population.

Dwyer et al. (submitted) emphasize the importance of directing attention to an analysis of the myxoma-*Oryctolagus* interaction. The system is of fundamental theoretical and applied importance. If myxomatosis evolves to the point that control is lost, the rabbit population again may become a serious pest. Because the underlying processes occur on a variety of temporal and spatial scales, mathematical models are critical in dissecting the complex system, and in identifying underlying mechanisms. Dwyer et al. (submitted) develop a simulation model that incorporates many of the aspects left out by simple models. Preliminary investigations seem to show that the spatial structure of the population plays a very important role in the observed coexistence of intermediate types.

What can be said concerning the evolution of other viral diseases? Influenza, to be discussed in the next paragraphs, provides one particularly interesting example, because changes in a few surface antigens led to the proliferation of a variety of strains, and to the potential for reappearance of strains previously lost. Thus, periodic or other recurrent behavior is to be expected in influenza, and such behavior indeed is observed (see Liu and Levin 1989, Hethcote and Levin 1989). For AIDS, that modern scourge, the hope that might be raised by contemplation of the myxoma story is short-lived. By the standards of myxoma, AIDS is already attenuated, in that infected individuals live a very long time.

Thus, selection for reduced virulence is not a potent force at all, and the rapidly changing AIDS virus is more likely to evolve in the direction of increased virulence.

In the influenza-man system, attention is focused on the potential for cross-immunity (a measure of reduced susceptibility to related strains of type A influenza) to facilitate oscillations and coexistence of strains (Castillo-Chavez et al. 1988, 1989). Recent work shows the existence of long-lasting cross-immunity between related strains (i.e., variants of the same subtype) in human influenza (Couch and Kasel 1983). Cross-immunity implies that the presence of one strain of the virus can reduce the pool of susceptible individuals for co-circulating strains, introducing a form of exploitation competition (Catillo-Chavez et al. 1988, 1989).

Castillo-Chavez et al. (1989) present models to elucidate the recently observed co-circulation of related strains, by extending the classical epidemiological approaches to allow for immunological interactions between strains (cross-immunity). For a homogeneous population, they introduce the diagram

$$
\begin{array}{ccccc}
X & \Rightarrow & Y_1 & \Rightarrow & Z_1 \\
\Downarrow & & & & \Downarrow \\
Y_2 & & & & V_2 \\
\Downarrow & & & & \Downarrow \\
Z_2 & \Rightarrow & V_1 & \Rightarrow & W
\end{array} \tag{48}
$$

In the above system, the population has been divided into 8 classes: X (fraction susceptible), Y_i (fraction infected by strain i), Z_i (fraction recovered from the other strain), V_i (fraction infected by strain i after recovery from the other strain), and W (recovered from both strains). Castillo-Chavez et al. (1988, 1989) assume that the population is homogeneously mixing, and that the usual bilinear incidence function describes transmission. They then formulate the following two-stain epidemiological model:

$$X'(t) = [\beta_1(Y_1+V_1)+\beta_2(Y_2+V_2)-\mu]X + \mu\,, \tag{49}$$

$$Y_i'(t) = \beta_i(Y_i+V_i)X - (\gamma_i+\mu)Y_i\,, \tag{50}$$

$$Z_i'(t) = \gamma_i Y_i - [\sigma_j\beta_j(Y_j+V_j)+\mu]Z_i\,, \tag{51}$$

$$V_i'(t) = \sigma_i\beta_i(Y_i+V_i)Z_j - (\gamma_i+\mu)V_i\,, \tag{52}$$

(53) $$W'(t) = \gamma_1 V_1 + \gamma_2 V_2 - \mu W ,$$

where

(54) $$i = j , j = 2 \quad \text{or} \quad i = 2 , j = 1 .$$

In addition, β_i denotes the transmission coefficient of strain i. σ_i denotes the *susceptibility factor* (where $j = 3 - i$); that is, σ_i is a measure of the relative susceptibility of types Z_i and X in terms of their acquisition of strain j. Usually, but not always, σ_i is between 0 and 1. Furthermore, γ_i denotes the recovery rate form strain i, and μ denotes the (constant) natural mortality rate. Thus, the model is flexible enough to cover the range of possibilities, from closely related strains to distinct subtypes.

Mathematical analysis and numerical simulations indicate that the above system cannot produce sustained oscillations. However, slowly-damped and hence biologically important oscillations are generated as a result of the cross-immunity.

When a heterogeneous host population is considered (age-structured population), then the model above is replaced by:

(55) $$\frac{\partial x(a,t)}{\partial a} + \frac{\partial x(a,t)}{\partial t} = -(\lambda_1(t)b(a)+\lambda_2(t)b(a)+\mu(a))x(a,t) ,$$

(56) $$\frac{\partial y_i(a,t)}{\partial a} + \frac{\partial y_i(a,t)}{\partial t} = \lambda_i(t)b(a)x(a,t) - (\gamma_i+\mu(a,t)) , \quad i = 1,2$$

(57) $$\frac{\partial z_i(a,t)}{\partial a} + \frac{\partial z_i(a,t)}{\partial t} = \gamma_i y_i(a,t) - \sigma_j\lambda_j(t)b(a)z_i(a,t) - \mu(a)z_i(a,t) , \quad i = 1,2$$

(58) $$\frac{\partial v_i(a,t)}{\partial a} + \frac{\partial v_i(a,t)}{\partial t} = \sigma_i\lambda_i(t)b(a)z_j(a,t) - (\gamma_i+\mu(a))v_i(a,t) , \quad i = 1,2$$

(59) $$\frac{\partial w(a,t)}{\partial a} + \frac{\partial w(a,t)}{\partial t} = (\gamma_1+\gamma_2-\mu(a))w(a,t) ,$$

(60) $$\lambda_i(t) = \beta_i \int_0^\infty b(a')[y_i(a',t)+v_i(a',t)]da' ,$$

(61) $$x(0,t) = \rho , \; y_i(0,t) = 0 , \; z_i(0,t) = 0 , \; v_i(0,t) = 0 , \; w(0,t) = 0 ,$$

$$x(a,0) = x_0(a)\,,\ y_i(a,0) = y_{0i}(a)\,,\ z_i(a,0) = z_{0i}(a)\,,$$
$$v_i(a,0) = v_{0i}(a)\,,\ w(0,t) = w_0(a)\,. \tag{62}$$

Furthermore,

$$\rho = \left[\int_0^\infty e^{-M(a)}\,da\right]^{-1} \text{ where } M(a) = \int_0^a \mu(a)d\alpha\,. \tag{63}$$

Here $x(a,t)$, $y_i(a,t)$, $z_i(a,t)$, $v_i(a,t)$, and $w(a,t)$ denote the densities of the individuals in each class previously defined, and a is an independent variable that denotes the age of an individual. $b(a)$ represents the age-specific contact rate, λ_i denotes the instantaneous force of infection, β_i denotes the transmission scaling factor, $m(a)$ is the age-specific mortality rate, and σ_i denotes the (constant) recovery rate. In this case Castillo-Chavez et al. (1989) and Andreasen (1989) have suggested that sustained oscillations are possible, due to the interaction between cross-immunity and age structure.

DIFFUSE COEVOLUTION

The problem of diffuse coevolution is probably ecologically more important than tight coevolution, but is much less understood and rarely modeled. As Levin (1983b) remarks: "...many problems of interest in the evolution of ecological communities are much more diffuse, involving many species with varying degrees of relationship to one another. Problems of this sort arise in the consideration of the chemical defenses of plants in response to insects and other pests (Feeny 1982), for often these do not have the finely tuned species-for-species relationship already discussed for the cereals and their rusts. Similar problems occur in predator-prey systems, which are by nature less specific than the host-parasite relationships; in competition theory; and regarding the evolution of the vertebrate immune system". In this section, we describe some early and tentative attempts to approach such problems.

In what follows we provide a very brief introduction to preliminary investigations by Levin, Segel, and Adler (unpubl.), who have begun to develop a framework within which to examine the patterns of diffuse coevolution in plant-herbivore communities. They point out that "the lack of evidence for the tight coevolution between pairs of species may inappropriately direct attention away from the obvious coevolution of defensive chemicals and mechanisms for detoxifying them." Given the impracticability of a reductionistic approach that includes the detailed genetics of every species, they focus on macroscopic variables such as the number and frequency distribution of different kinds of chemicals, and on other community-level descriptors. Assume that the plants possess toxins that, if unneutralized, prevent herbivores from consuming them. First, consider an oversimplified situation in which there is a pool of N chemical defenses such that each plant has exactly n. In addition, each herbivore is able to detoxify m of these toxins. Furthermore, assume that the particular group of defensive chemicals or detoxifying agents is drawn at random by the plant or the herbivore from the pool; it is further assumed that the plant

repels or resists the herbivore if and only if it has at least one defensive chemical that the herbivore cannot counteract.

If $L(m,n)$ denotes the probability that a plant with n defensive chemicals will "lose" in an encounter with a herbivore capable of detoxifying m substances, then clearly

$$L(m,n) = 0 \text{ if } m < n. \tag{64}$$

Since, for $m \geq n$, $L(m,n)$ denotes the probability that m elements chosen at random from a set of N elements lie within a particular subset of size n, then

$$L(m,n) = \frac{m(m-1)\ldots(m-n+1)}{N(N-1)\ldots(N-n+1)} = \binom{N-n}{m-n} \Big/ \binom{N}{m}, \text{ if } m \geq n. \tag{65}$$

The above expressions allow calculation of the probabilities of various results of an individual encounter between a plant and a herbivore. Levin, Segel, and Adler (ms.) investigate the implications of these assumptions in the development or evclution of a community. The first question that they ask is: What will be the fate of a rare mutant or migrant that appears in the community? In order to answer this question, one must assign benefits and costs to the winners and losers during a given encounter. Furthermore, costs have to be assigned for a given level of chemical defense or detoxifying ability.

The simplest assumptions are that the new migrant (or mutant) cannot interbreed with the resident types, and that the number of encounters per unit time remains constant. More specifically, the total cost per plant per unit time associated with herbivory is

$$k\,L(m,n) + cn, \tag{66}$$

where the constant k is the product of the number of encounters per unit time and the cost per loss. The constant c gives the cost per unit time of keeping a single chemical defense. From here we easily conclude that the invasion of an (m,n) community by a plant with $n+1$ chemical defenses is possible provided that

$$\theta_p = \frac{c}{k} < L(m,n) - L(m,n+1). \tag{67}$$

If we replace the linear cost function cn by a function $f(n)$ of n, then we obtain:

$$\frac{\mu(n)}{k} < L(m,n) - L(m,n+1), \tag{68}$$

where $\mu(n) = f(n+1) - f(n)$ is the marginal cost of adding an additional defensive chemical to the n that already are present.

The net gain associated with herbivory per herbivore per unit time is, in the simplest case,

$$e\,L(m,n) - bm. \tag{69}$$

An (m,n) community can be invaded by a rare herbivore with m+1 detoxifying agents if and only if

$$\theta_h = \frac{b}{e} < L(m+1,n) - L(m,n) . \tag{70}$$

The linear cost function bm similarly can be generalized. Note that similar considerations show that inequalities (67) and (70) are also the conditions respectively that an (m,n+1) community cannot be invaded by a type-n plant and an (m+1,n) community cannot be invaded by a type-m herbivore. From this, Levin, Segel, and Adler (ms), endeavor to build a tapestry of increasing realism, initially expanding their investigations to the situation in which a distribution of phenotypes (containing different numbers of chemicals) exist within the community, and from there to consideration of the spectrum of available chemicals, and the distribution of phenotypes in this aspect space. At this point, the treatment makes contact with the earlier work of Levin and Segel (1984) on pattern diversity in aspect space.

These investigations of diffuse coevolution are very preliminary. The objective – to develop macroscopic descriptors at the community and ecosystem level – are essential to the development of interfaces between population biology and ecosystem science. Imaginative approaches to such problems represent one of the unmet challenges of evolutionary theory.

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