PHILOSOPHICAL TRANSACTIONS OF THE ROYAL SOCIETY B BIOLOGICAL SCIENCES

The Interplay of Population Dynamics and the Evolutionary Process [and Discussion]

Joseph Travis and J. J. D. Greenwood

Phil. Trans. R. Soc. Lond. B 1990 **330**, 253-259
doi: 10.1098/rstb.1990.0196

The interplay of population dynamics and the evolutionary process

JOSEPH TRAVIS

Department of Biological Science, Florida State University, Tallahassee, Florida 32306-2043, U.S.A.

SUMMARY

Long-term maintenance of genetic diversity is affected by ecological forces that are driven in turn by current levels of genetic variation. The strength of population regulation and the consequent patterns of population fluctuations determine the likelihood of genetic changes considered pivotal for rapid speciation. However, genetic diversity in the susceptibility to regulatory forces can reduce the magnitude of such fluctuations and minimize the likelihood of genetic revolutions. A group of populations that experiences local extinctions and recolonizations may hold lower levels of genetic diversity than in the absence of such extinctions, but local adaption, which provides enhanced genetic diversity, can reduce the likelihood of local extinctions. Tightly regulated populations experience different selection pressures than poorly regulated populations, although tighter regulation itself can evolve. When genotypic variation affects the outcome of interspecific interactions on a local scale, this effect, coupled with appropriate spatial variation, can enhance the resilience of the interactive system.

INTRODUCTION

In this paper several areas of interplay among population dynamics, ecological diversity, and the evolutionary process are explored. The primary goal is to review recent work that illuminates the many facets of that interplay. The secondary goals are to offer a synthesis of some diverse results and to focus attention on problems for which we lack sufficient information to draw general conclusions. I have tried to ensure that the literature cited can lead the general reader to a wide range of examples but that it can also lead the afficionado of specific topics to more detailed treatments.

ECOLOGICAL PROCESSES AND GENETIC DIVERSITY

(*a*) *Population fluctuations and genetic diversity*

When a population is reduced to a few individuals there are several population genetic consequences even in the absence of natural selection. The random sampling effects on gene frequencies that cancel one another in large populations can alter gene frequencies from the ancestral state. Similar sampling effects generate non-random combinations of the segregating alleles from many loci. Small numbers of individuals restrict the possibilities for accruing new genetic variation through mutation and promote inbreeding through the high levels of relatedness among subsequent progeny. The 'genetic size' of a population depends upon which of the above consequences are of interest because the same census number of individuals can represent different degrees of genetic 'smallness' for different consequences (Maruyama & Kimura 1980; Ewens 1982; Crow & Denniston 1988).

The measurement of 'genetic size', the effective population size, is the numerical size of an idealized population (random mating, no selection, equal contributions of all individuals to the next generation, etc.) that is suffering the genetic consequences of interest at the same rate or level as the real population under study. It is a surrogate number that encompasses the census number and other factors such as gender ratios, equality of contribution of progeny, breeding system, or mode of transmission of genes (Crow & Denniston 1988). Because these 'other factors' may change with density in natural populations the numerical relation between the census size and the effective size can be a complex one (see Travis & Mueller 1989).

Real populations fluctuate in their census numbers, so their effective sizes and consequent genetic properties fluctuate also. Periodic fluctuations in numbers generate matching periodic fluctuations in expected levels of genetic variation for many types of fluctuations (cyclical, stochastic, density dependent; see also the references in Motro & Thompson (1982)).

In many cases the genetic effects of one or more constrictions in numerical size linger for many generations after large numbers have been re-established (Nei *et al.* 1975; Chakraborty & Nei 1977; Motro & Thompson 1982; Maruyama & Fuerst 1985). Repeated constrictions in size reduce genetic variation dramatically when the cycle length of such repetitions is short and population growth rates are slow (Motro & Thompson 1982; see also Watterson (1989)).

The preceding theoretical results have inspired several hypotheses about specific empirical situations. Past bottlenecks have been postulated *a posteriori* to account for the cheetah's extraordinarily small amount of genetic variation for its current numerical size (see, for example, O'Brien *et al.* (1985); also Wayne *et al.*

(1986)). A more intriguing, *a priori* general prediction is that species experiencing regular dramatic fluctuations in population size with short cycle lengths should harbour less genetic variation than comparable species that do not have such fluctuations (Smith *et al.* 1975). This prediction fails for both cycling rodents (Gaines 1985) and birds (Gyllensten 1985) and the apparent reason for its failure shows the complexity of the interactions between ecological and genetic processes.

In cycling populations allele frequencies are altered during the low density phases because of genetic drift (Gaines & Whittam 1980; Bowen 1982; Gyllensten 1985). At high density phases there is increased gene flow among local subpopulations that reduces the levels of genetic differentiation among local populations and thereby increases dramatically the effective size of the entire ensemble (Bowen 1982; Plante *et al.* 1989). High emigration rates are regularly associated with the high density phase of cycling rodents (Krebs 1966; Gaines *et al.* 1979) and cycling grouse appear to migrate long distances (Gyllensten 1985). Thus changing movement patterns of individuals associated with local density fluctuations preclude lowering of the effective population size and concomitant genetic effects. This interpretation is supported by the observation that isolated vole populations on islands show a reduction in genetic variation (Kilpatrick & Crowell 1985).

The role of genetic interchange among subpopulations in overcoming the effects of local bottlenecks has been shown experimentally in plants (Polans & Allard 1989). A single generation of random mating restored the mean performance that had been lost due to inbreeding effects in a series of small isolates. If such interplay among local densities, migration rates and genetic structure were widespread then effects of periodic crashes in local populations on the genetic variation in the entire ensemble may be minimized. Present empirical data are insufficient for general conclusions. For example, insect emigration rates increase with increases in density (see, for example, Lawrence (1988) and references therein), but the extent to which such movement is translated into actual gene flow remains unclear.

There are circumstances in which fluctuations in population size can enhance the amount of additive genetic variation in a specific trait or set of traits. The additive variation is that portion of the genetic variation in a trait that is responsive to selection. When the population that enters a bottleneck has a high proportion of its total genetic variation for a trait contributed by epistatic effects (interactions among alleles at different loci) then the population can emerge from the bottleneck with more additive genetic variation than it had upon entering (Goodnight 1988). This occurs through the conversion of epistatic variance into additive variance. The most conducive situation for such conversion is an extended numerical constriction at a moderate effective size (Goodnight 1988). Traits likely to have the genetic prerequisites for this effect are those with either a long history of directional selection (reviewed in Travis & Mueller (1989)) or those strongly associated with mate recognition (Bryant & Meffert 1988). However, the absolute magnitude of epistatic variances in natural populations and the consequent magnitude of any potential enhancement of additive genetic variance is an unresolved empirical issue. The level of epistasis among individual loci appears small (Barker 1979; Hastings 1985) but interactions among groups of loci (blocks of genes on separate chromosomes, structural genes and their polygenic regulators, sex chromosomes and autosomes) often generate considerable epistatic variance (Kidwell 1969; Sved & Ayala 1970; Jones & Yamazaki 1974; Malpica & Vasallo 1980; Bao & Kallman 1982; Miyashita & Laurie-Ahlberg 1984; Kallman 1984).

Enhancement of genetic variation after a constriction in size plays a key role in two related areas. First, enhanced additive genetic variation allows further response to selection and enables a population to increase its overall level of adaptation to a specific selection pressure (Lande 1976, 1979). Second, the rearrangement of genetic variation after a constriction may allow a population to respond to selection dramatically and rapidly and thereby enhance the likelihood for rapid genetic differentiation among conspecific populations. This is the essential model for speciation via founder effects (Carson & Templeton 1984). Rapid differentiation via numerical constriction without such a conversion of genetic variation appears to be unlikely (Rouhani & Barton 1987; Charlesworth & Rouhani 1988) so this effect plays a central role in theories about the most profound stage in the evolutionary process.

(*b*) *The extinction and recolonization of subpopulations*

The numerical dynamics produced by the extinction and recolonization of independently fluctuating subpopulations is a subject of considerable debate with respect to its role in producing population stability (Kareiva, this symposium). There are two areas in which this ecological process has been a focus of population genetic debate. First, does such a process enhance the overall amount of genetic variation present in a species? Second, does this process affect the distribution of the extant genetic variation in a way that enhances the amount of differentiation among populations? The answers to these distinct questions bear on two larger contexts, the preservation of genetic variation in captive or managed populations and the creation of conditions conducive to a 'shifting balance' process of evolutionary change (Wade & McCauley 1988).

The first question has been addressed analytically by Ewens (1989) and via simulation by Maruyama & Kimura (1980), but with only limited success. If new colonists are drawn from only one randomly chosen subpopulation, if the extinction rate is not high, and if there is negligible gene flow among extant subpopulations, then a system with such an extinction–recolonization process will lose genetic variation more slowly than a comparable system without it

(Ewens 1989). Other models of the colonization process give different results (Maruyama & Kimura 1980; Ewens 1989) so no general answer is apparent.

The second question also has contingent answers (Slatkin 1977; Wade & McCauley 1988). When new populations are founded by colonists from many extant populations and if there is little gene flow among extant populations then this process impedes the stochastic differentiation of subpopulations. However, when colonists originate from a single subpopulation and the number of colonists is below the typical migration rate among extant subpopulations then this process enhances local differentiation.

The empirical data on this topic are few but the theoretical treatments offer unusually clear guidelines towards which data are most critical for producing different outcomes. The issues are reviewed thoughtfully by McCauley (1989) and include the origins of colonists, the rate of 'normal' interchange among extant populations, the typical numbers of colonists that found a new population, the extinction rate, and the assumption that extinction probabilities are independent of the size or age of the subpopulation. The increasing attention that ecologists are giving to such systems in the context of population regulation offers an excellent opportunity to acquire the demographic data that would enable us to understand whether extinction and recolonization contribute to the genetic structure of natural populations.

ECOLOGICAL DIVERSITY AND GENETIC PROCESSES

(*a*) *Overview*

In the previous sections, population dynamics were taken as 'given' and the genetic consequences of various patterns were reviewed. Diversity in dynamic patterns generates different levels and arrangements of genetic variation. However, natural populations do not fluctuate in a vacuum. Changes in numbers are produced by a variety of influences on mortality and natality rates and the phenotypic variants in a population may be affected differently by different limiting or regulating factors. In addition there are, invariably, fundamental changes in ecological relations that are associated with changes in population density. These two effects have consequences for both the action of natural selection and the patterns of population dynamics that result from evolutionary changes in vital rates. In the next two sections each type of effect is highlighted.

(*b*) *Multiple agents of population limitation and genetic heterogeneity in susceptibility to those agents*

Whether a particular population is regulated tightly or whether its growth is limited in more stochastic fashion there are usually several abiotic and biotic agents that perform the regulatory or limiting task function. Long-term studies of a variety of natural populations have revealed that only a subset of these several forces may act in any given year or be important for any given generation (Grant 1986; Marquis 1990). In this section the consequences of this observation for the intertwined problems of the net intensity of selection and the stability of population dynamics are examined. While the importance of questions about the stability of population dynamics will be self-evident to many readers, that of net selection intensities may not. Large intensities of selection are not compatible with long-term predictions from quantitative genetic models of evolution so the actual distribution of selection intensities in natural populations is an important, but unresolved, empirical issue (Endler 1986; Travis & Mueller 1989).

Studies of natural selection offer convincing evidence that there is invariably some level of genetic variation within a population in the vulnerability to any single agent of mortality or the susceptibility to any single factor that limits fertility (Manly 1985; Endler 1986; Travis 1989). In many cases variation is wide enough to produce large selection intensities. A variety of theoretical approaches can be used to show that such internal heterogeneity in vital rates (what the population geneticist would call variation in absolute fitness) can enhance the stability of the ensuing population dynamics (Wallace 1977; Lomnicki 1988; see Chesson (1989) for an analogous argument in community ecology). This effect has been explored extensively in host–parasite systems (May & Anderson 1983; Cohen & Newman 1989), inspired in part by the ubiquity of genetically based variation in host vulnerability (see, for example, Madhavi & Anderson 1985; Parker 1988; Alexander 1989). The effect is not as well appreciated in more general contexts.

Multiple agents of mortality, or more generally, multiple agents of population limitation, introduce complications. In some years or generations only one agent may be critical while in others a different agent or all of the agents may be critical. The qualitative consequences of this ecological diversity will vary with the correlation structure of vulnerability to different agents, but will also depend on the other sources of internal heterogeneity. Each situation is likely to be idiosyncratic, and I offer a detailed example to show how complexity can arise.

Larval anurans in temporary ponds face a number of mortality risks (complete descriptions of this ecological system can be found in Morin (1983); Wilbur (1984); Travis *et al.* (1987)). The major agents of mortality include predatory salamanders, predatory insects and drying of the pond. Each type of predator is limited by prey size such that rapidly growing tadpoles escape predation risk sooner than more slowly growing ones. The extensive variation in growth rate, in part genetically based, provides internal heterogeneity in vulnerability that affects population dynamics and generates directional selection for rapid growth. Hence vulnerability to each type of predator, salamander and insect, is positively correlated among genetic variants.

Patterns of microhabitat use as prey refugia complicate the situation. Salamanders search the water column for moving tadpoles and the prey have a refuge from them in vegetated or littered microhabitats (Morin 1986). The major insect predators, odonate naiads, occupy those very habitats and force tadpoles into the water column. The refuge from one predator is the locus of primary susceptibility to the other. Travis *et al.* (1985) manipulated the presence and absence of both predators in field enclosures with habitat structure and showed that when only one predator was present there was little differential mortality with respect to growth rate, presumably because of the availability of refuge. When both predators were present overall mortality rates increased, but not beyond the product of each predator's individual effect. However, the amount of differential mortality increased dramatically: relative fitness of slowly growing tadpoles when both predators were present was significantly below the expectation based on each predator's individual effect as an agent of selective mortality.

With respect to selection the effect of the positive correlation in vulnerability acts to increase the intensity of directional selection when both predators are present. When only one predator is present spatial refugia reduce the intensity of selection below what one would expect based on innate vulnerability. The intensity of selection will vary markedly from generation to generation although its direction will not. This result suggests that isolated observations of very strong selection intensities in nature may represent extreme values of a distribution of selection intensities caused by ecological variability. With respect to population dynamics the positive correlations caused by innate heterogeneity ought to be destabilizing but the negative correlations induced by spatial heterogeneity would be stabilizing. There is considerable variation in the importance of each type of predator from year to year, and thus the net effect cannot be determined from merely the signs of the correlations.

There are other patterns of covariance in vulnerability that have very different consequences. A negative covariance was described by Weis & Abrahamson (1986) in their studies of a gall-making insect. Larger galls are more vulnerable to bird predation but less so to parasitoid attack; smaller galls leave the enclosed insect more vulnerable to parasitoids but less susceptible to bird predation. In this situation the net selection would be optimizing if both agents of mortality acted simultaneously in every generation (and concomitant enhancement of stability in dynamics relative to no heterogeneity). If the importance of each enemy varies among years then the net result is a regime of fluctuating directional selection that will have the same long-term effect as optimizing selection (see Travis 1989).

In many cases genetic variation in the vulnerability to one ecological factor is uncorrelated with vulnerability to another. These cases include variation among plant genotypes in resistance to different species of herbivore (Simms & Rausher 1989; Marquis 1990), variation among herbivores in their success on different species of plant hosts (Rausher 1984; Via 1984), and variation in performance at different densities and competitive situations in plants (Khan *et al.* 1976; Shaw 1986; Miller & Schemske 1990) and insects (Bradshaw & Holzapfel 1989). In these cases there is always a significant selection pressure, but that pressure will differ qualitatively as the ensemble of critical factors varies from generation to generation. A pattern of uncorrelated resistance to multiple agents enhances stability of numerical dynamics above that expected from no heterogeneity but the net effect relative to other patterns of correlation would seem to depend on the exact distribution of the yearly importance of each agent.

(*b*) *Density-dependent selection and response*

No concept has captured the shared imagination of ecologists and geneticists as much as the contrasting patterns of evolution in populations with chronically high or low densities. There has been a considerable amount of verbal and mathematical theory on the subject (reviewed lucidly by Mueller (1988)), but the existing empirical data are highly variable and often paradoxical. In this section I highlight some of these contradictions and suggest that paradoxical results have their genesis in the complexity of the relation between population densities, specific ecological agents of mortality, and the expression of genetic variation. The interrelations between population density and specific regulating forces will not surprise ecologists. Conversely, the changing genetic expression with density will not surprise geneticists.

The crucial basis for density-dependent selection is that the appropriate measure of fitness changes from the intrinsic rate of increase of a genotype (the measure in unregulated, density-independent contexts) to a function of the carrying capacity of a genotype (assuming weak selection at the genetic level (see Charlesworth (1980) and references therein)). The demographic results of evolution at single loci in these contrasting conditions are a maximization of population growth rate under density-independent conditions or the maximization of the number of individuals in the age class in which numerical regulation occurs under density-dependent conditions. There may or may not be significant elevation of the overall carrying capacity under density-dependent evolution; theory points only to the numbers in the critical age class. The age structure of density-dependence will determine the demographic outcome of evolution.

The importance of age-structure is shown in a number of paradoxical results from studies of the evolution of vital rates and life histories under the contrasting regimes. A common expectation derived from verbal models of life-history evolution is that evolution in a tightly regulated population will delay the onset of reproduction and lower adult fecundity. This expectation is supported by mathematical theory but so is the counter expectation of earlier reproduction and high fecundity; the differences arise from different assumptions of how increased densities affect the age-structured vital rates (Charlesworth 1980; Iwasa & Teramoto 1980). Several empirical laboratory studies of different species and different husbandry regimes have produced nearly the full spectrum of mathematically derived predictions that, in many cases, contradict the simpler verbally derived predictions

(Luckinbill 1978; Taylor & Condra 1980; Mueller & Ayala 1981; Bergmans 1984; Bierbaum *et al.* 1989).

More variability in outcome is evident when one examines the specific phenotypic traits that evolve under these contrasting conditions. Evolution acts on the genetic covariance between trait variation and fitness variation (Crow & Nagylaki 1977) and it is reasonable to expect that different traits will covary with a rate of increase than with a carrying capacity. This expectation is the basis for the predictions made by many ecologists (see, for example, Pianka (1970)). Density-dependent covariances that allow evolution involve two components, the covariance of the phenotypic trait variation with fitness and the requisite expression of genetic variation for that phenotypic trait at that specific density. These patterns of covariance differ from species to species in natural populations and show the complexity of trait evolution under varying density régimes.

Larval anurans in temporary ponds again offer a simple illustration. At very low tadpole densities predatory salamanders have no appreciable impact because tadpole growth rates are high and encounter rates are low due to both contact probabilities and refugia (Wilbur 1984; Travis *et al.* 1985). Greater numerical impact of salamanders and their role as a selective force for rapid growth rate occur only at higher tadpole densities. At extremely high densities all tadpoles grow so slowly, due to effective food limitation, that prey can be driven extinct (Wilbur 1984). The density of tadpoles can also influence the expression of genetic variation in larval growth rate (figure 1); in the spadefoot toad more genetic variation is expressed at low densities than at high ones. In other species there is no such effect of density (Travis 1983). The net result is that effective selection for growth rate (that producing a genetic response) will occur only within certain regions of ecological parameter space with respect to population density and those regions differ even among species using the same habitat.

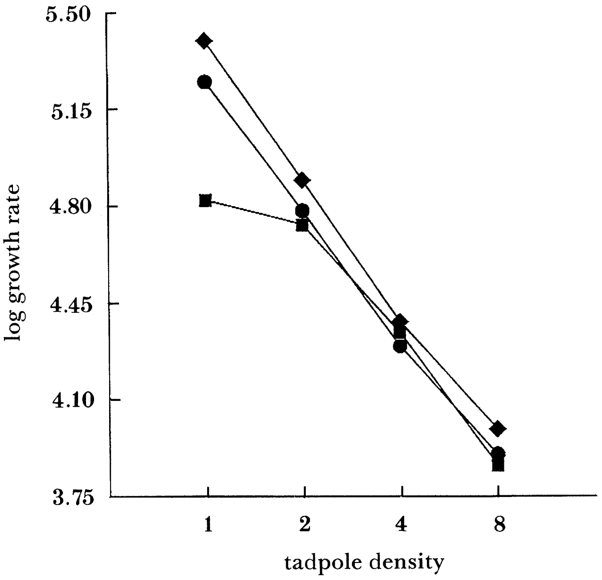

Figure 1. Larval growth rate on the logarithmic scale plotted against tadpole density (numbers of tadpoles per 500 ml water) for the eastern spadefoot toad, *Scaphiopus holbrooki*, of North America. Each symbol denotes the average value of eight replicates (density of 1) or four replicates (other densities). Different symbols denote different full sib families. Sibship differences exist at the lowest density but not at the others. This laboratory experiment is similar to those reported by Travis (1983).

These complexities are not restricted to viability selection in larval anurans. Conner (1989) pointed out that studies of sexual selection show all possible patterns of change in selection intensity with changes in population density. Traits that confer increased survival under high density conditions are irrelevant at low densities (Mueller & Sweet 1986). These patterns show that the frequency distribution of the critical ecological conditions must be known even to calculate the net direction and strength of selection on such traits (Gupta & Lewontin 1982). Density-dependent expression of trait variation is well known in laboratory populations of *Drosophila* (Clare & Luckinbill 1985; Luckinbill & Clare 1986; Bierbaum *et al.* 1989) and in some cases genetic variation exists in the sensitivity of trait expression to density effects (Khan *et al.* 1976; Marks 1982; cf. Travis 1983). Given these ecological and genetic complexities it may only be possible to make accurate predictions about density-dependent evolution from detailed models tailored to the ecology of specific organisms (Mueller (1988) shows this approach).

I owe a debt of gratitude to Professor M. P. Hassell and Professor R. M. May for provoking my thoughts on several of these topics. I thank Dr T. Miller, Dr D. Simberloff, Dr D. Strong and Dr A. Winn for discussions during the writing of this paper. My work has been supported by the National Science Foundation and during the writing of this paper I have been supported by BSR 88-18001.

REFERENCES

Alexander, H. M. 1989 An experimental field study of anther-smut disease of *Silene alba* caused by *Ustilago violacea*: genotypic variation and disease incidence. *Evolution* **43**, 835–847.

Barker, J. S. F. 1979 Interlocus interactions: a review of experimental evidence. *Theor. Popul. Biol.* **16**, 323–346.

Bao, I. Y. & Kallman, K. D. 1982 Genetic control of the hypothalamo-pituitary axis and the effect of hybridization on sexual maturation (*Xiphophorus*, Pisces, Poeciliidae). *J. exp. Zool.* **220**, 297–309.

Bergmans, M. 1984 Life history adaptation to demographic regime in labortory-cultured *Tisbe furcata* (Copepoda, Harpacticoida). *Evolution* **38**, 292–299.

Bierbaum, T. J., Mueller, L. D. & Ayala, F. J. 1989 Density-dependent evolution of life-history traits in *Drosophila melanogaster*. *Evolution* **43**, 382–392.

Bowen, B. S. 1982 Temporal dynamics of microgeographic structure of genetic variation in *Microtus californicus*. *J. Mammal.* **63**, 625–638.

Bradshaw, W. E. & Holzapfel, C. M. 1989 Life-historical consequences of density-dependent selection in the pitcher-plant mosquito, *Wyeomyia smithii*. *Am. Nat.* **133**, 869–887.

Bryant, E. H. & Meffert, L. M. 1988 Effect of an individual bottleneck on morphological integration in the housefly. *Evolution* **42**, 698–707.

Carson, H. L. & Templeton, A. R. 1984 Genetic revolutions in relation to speciation phenomena: the founding of new populations. *A. Rev. Ecol. Syst.* **15**, 97–132.

Chakraborty, R. & Nei, M. 1977 Bottleneck effects on average heterozygosity and genetic distance with the stepwise mutation model. *Evolution* **31**, 347–356.

Charlesworth, B. 1980 *Evolution in age-structured populations.* Cambridge University Press.

Charlesworth, B. & Rouhani, S. 1988 The probability of peak shifts in a founder population. II. An additive polygenic trait. *Evolution* **42**, 1129–1145.

Chesson, P. L. 1989 A general model of the role of environmental variability in communities of competing species. *Lect. Math. Life Sci.* **20**, 97–123.

Clare, M. J. & Luckinbill, L. S. 1985 The effects of gene-environment interaction on the expression of longevity. *Heredity* **55**, 19–29.

Cohen, J. E. & Newman C. M. 1989 Host-parasite relations and random zero-sum games: the stabilizing effect of strategy diversification. *Am. Nat.* **133**, 533–552.

Conner, J. 1989 Density-dependent sexual selection in the fungus beetle, *Bolitotherus cornutus*. *Evolution* **43**, 1378–1386.

Crow, J. F. & Denniston, C. 1988 Inbreeding and variance effective population numbers. *Evolution* **42**, 482–495.

Crow, J. F. & Nagylaki, T. 1976 The rate of change of a character correlated with fitness. *Am. Nat.* **110**, 207–213.

Endler, J. A. 1986 *Natural selection in the wild.* New Jersey: Princeton University Press.

Ewens, W. J. 1982 On the concept of the effective population size. *Theor. Popul. Biol.* **21**, 373–378.

Ewens, W. J. 1989 The effective population size in the presence of catastrophes. In *Mathematical evolutionary theory* (ed. M. W. Feldman), pp. 9–25. New Jersey: Princeton University Press.

Gaines, M. S. 1985 Genetics. In *Biology of New World* Microtus (ed. R. H. Tamarin), pp. 845–883. Pennsylvania: American Society of Mammalogists.

Gaines, M. S., Vivas, A. M. & Baker, C. L. 1979 An experimental analysis of dispersal in fluctuating vole populations: Demographic parameters. *Ecology* **60**, 814–828.

Gaines, M. S. & Whittam, T. S. 1980 Genetic changes in fluctuating vole populations: selective vs. nonselective forces. *Genetics* **96**, 767–778.

Goodnight, C. J. 1988 Epistasis and the effect of founder events on the additive genetic variance. *Evolution* **42**, 441–454.

Grant, P. R. 1986 *Ecology and evolution of Darwin's finches.* New Jersey: Princeton University Press.

Gupta, A. P. & Lewontin, R. C. 1982 A study of reaction norms in natural populations of *Drosophila pseudoobscura*. *Evolution* **36**, 934–948.

Gyllensten, U. 1985 Temporal allozyme frequency changes in density fluctuating populations of willow grouse (*Lagopus lagopus* L.) *Evolution* **39**, 115–121.

Hastings, A. 1985 Multilocus population genetics with weak epistasis. I. Equilibrium properties of two-locus two allele models. *Genetics* **109**, 799–812.

Iwasa, Y. & Teramoto, E. 1980 A criterion of life history evolution based on density-dependent selection. *J. Theor. Biol.* **13**, 1–68.

Jones, J. S. & Yamazaki, T. 1974 Genetic background and the fitness of allozymes. *Genetics* **78**, 1185–1189.

Kallman, K. D. 1984 A new look at sex determination in poeciliid fishes. In *Evolutionary genetics of fishes* (ed. B. J. Turner), pp. 95–171. New York: Plenum Press.

Khan, M. A., Antonovics, J. & Bradshaw, A. D. 1976 Adaptation to heterogeneous environments. III. The inheritance of response to spacing in flax and linseed (*Linum usitatissimum*). *Aust. J. Agric. Res.* **27**, 649–659.

Kidwell, J. F. 1969 A chromosomal analysis of egg production and abdominal chaeta number in *Drosophila melanogaster*. *Can J. Genet. Cytol.* **11**, 547–557.

Kilpatrick, W. W. & Crowell, K. L. 1985 Genetic variation of the rock vole, *Microtus chrotorrhinus*. *J. Mammal.* **66**, 94–101.

Krebs, C. J. 1966 Demographic changes in fluctuating populations of *Microtus californicus*. *Ecol. Monogr.* **36**, 239–273.

Lande, R. 1976 Natural selection and random genetic drift in phenotypic evolution. *Evolution* **30**, 314–334.

Lande, R. 1979 Quantitative genetic analyses of multivariate evolution, applied to brain:body size allometry. *Evolution* **33**, 402–416.

Lawrence, W. S. 1988 Movement ecology of the red milkweed beetle in relation to population size and structure. *J. Anim. Ecol.* **57**, 21–35.

Lomnicki, A. 1988 *Population ecology of individuals.* New Jersey: Princeton University Press.

Luckinbill, L. S. 1978 r- and K-selection in experimental populations of *Escherichia coli*. *Science, Wash.* **202**, 1201–1203.

Luckinbill, L. S. & Clare, M. J. 1986 A density threshold for the expression of longevity in *Drosophila melanogaster*. *Heredity* **56**, 529–535.

McCauley, D. E. 1989 Extinction, colonization, and population structure: a study of a milkweed beetle. *Am. Nat.* **134**, 365–376.

Madhavi, R. & Anderson, R. M. 1985 Variability in the susceptibility of the fish host, *Poecilia reticulata*, to infection with *Gyrodactylus bullatarudis* (Monogenea). *Parasitology* **91**, 531–544.

Malpica, J. M. & Vasallo, J. M. 1980 A test for the selective origin of environmentally correlated allozyme patterns. *Nature, Lond.* **286**, 407–408.

Manly, B. F. J. 1985 *The statistics of natural selection.* New York: Chapman and Hall.

Marks, R. W. 1982 Genetic variability for density sensitivity of three components of fitness in *Drosophila melanogaster*. *Genetics* **101**, 301–316.

Marquis, R. J. 1990 Genotypic variation in leaf damage in *Piper arieianum* (Piperaceae) by a multispecies assemblage of herbivores. *Evolution* **44**, 104–120.

Maruyama, T. & Fuerst, P. A. 1985 Population bottlenecks and nonequilibrium models in population genetics. III. Genic homozygosity in populations which experience periodic bottlenecks. *Genetics* **111**, 691–703.

Maruyama, T. & Kimura, M. 1980 Genetic variability and effective population size when local extinctions and recolonization of subpopulations are frequent. *Proc. natn Acad. Sci.* **77**, 6710–6714.

May, R. M. & Anderson, R. M. 1983 Parasite-host coevolution. In *Coevolution* (ed. D. J. Futuyma & M. Slatkin), pp. 186–206. Massachussetts: Sinauer.

Miller, T. E. & Schemske, D. W. 1990 An experimental study of competitive performance in *Brassica rapa* (Cruciferae). *Am. J. Bot.* **77**. (In the press.)

Miyashita, N. & Laurie-Ahlberg, C. C. 1984 Genetical analysis of chromosomal interaction effects on the activities of the glucose 6-phosphate and 6-phosphogluconate dehydrogenases in *Drosophila melanogaster*. *Genetics* **106**, 655–668.

Morin, P. J. 1983 Predation, competition, and the composition of larval anuran guilds. *Ecol. Monogr.* **53**, 119–138.

Morin, P. J. 1986 Interactions between intraspecific competition and predation in an amphibian predator–prey system. *Ecology* **67**, 713–720.

Motro, U. & Thomson, G. 1982 On heterozygosity and the effective size of populations subject to size changes. *Evolution* **36**, 1059–1066.

Mueller, L. D. 1988 Density-dependent population growth and natural selection in food-limited environments: the *Drosophila* model. *Am. Nat.* **132**, 786–809.

Mueller, L. D. & Ayala, F. J. 1981 Trade-off between r-selection and K-selection in *Drosophila* populations. *Proc. natn Acad. Sci.* U.S.A. **78**, 1303–1305.

Mueller, L. D. & Sweet, V. F. 1986 Density-dependent natural selection in *Drosophila*: evolution of pupation height. *Evolution* **40**, 1354–1356.

Nei, M., Maruyama, T. & Chakraborty, R. 1975 The bottleneck effect and genetic variability in populations. *Evolution* **29**, 1–10.

O'Brien, S. J., Roelke, M. E., Marker, L., Newman, A., Winkler, C. A., Meltzer, D., Colly, L., Evermann, J. F., Bush, M. & Wildt, D. 1985 A genetic basis for species vulnerability in the cheetah. *Science, Wash.* **227**, 1428–1434.

Parker, M. A. 1988 Polymorphism in disease resistance in the annual legume *Amphicarpaea bracteata*. *Heredity* **60**, 27–31.

Pianka, E. R. 1970 On r- and K-selection. *Am. Nat.* **104**, 592–596.

Plante, Y., Boag, P. T. & White, B. N. 1989 Microgeographic variation in mitochodrial DNA of meadow voles (*Microtus pennsylvanicus*) in relation to population density. *Evolution* **43**, 1522–1537.

Polans, N. O. & Allard, R. W. 1989 An experimental evaluation of the recovery potential of ryegrass populations from genetic stress resulting from restriction of population size. *Evolution* **43**, 1320–1324.

Rausher, M. D. 1984 Tradeoffs in performance on different hosts: evidence from within- and between-site variation in the beetle *Deloyala gutata*. *Evolution* **38**, 582–595.

Rouhani, S. & Barton, N. H. 1987 The probability of peak shifts in a founder population. *J. Theor. Biol.* **126**, 51–62.

Shaw, R. 1986 Response to density in a wild population of the perennial herb *Salvia lyrata*: variation among families. *Evolution* **40**, 492–505.

Simms, E. S. & Rausher, M. D. 1989 The evolution of resistance to herbivory in *Ipomoea purpurea*. II. Natural selection by insects and costs of resistance. *Evolution* **43**, 573–585.

Slatkin, M. 1977 Gene flow and genetic drift in a species subject to frequent local extinctions. *Theor. Popul. Biol.* **12**, 253–262.

Smith, M. H., Garten, C. T. & Ramsey, P. R. 1975 Genic heterozygosity and population dynamics in small mammals. In *Isozymes. IV. Genetics and evolution* (ed. C. L. Markert), pp. 85–102. New York: Academic Press.

Sved, J. A. & Ayala, F. J. 1970 A population cage test for heterosis in *Drosophila pseudoobscura*. *Genetics* **66**, 97–113.

Taylor, C. E. & Condra, C. 1980 r- and K-selection in *Drosophila pseudoobscura*. *Evolution* **34**, 1183–1193.

Travis, J. 1983 Variation in development patterns of larval anurans in temporary ponds. I. Persistent variation within a *Hyla gratiosa* population. *Evolution* **37**, 496–512.

Travis, J. 1989 The role of optimizing selection in natural populations. *A. Rev. Ecol. Syst.* **20**, 279–296.

Travis, J., Keen, W. H. & Juilianna, J. 1985 The effects of multiple factors on viability selection in *Hyla gratiosa* tadpoles. *Evolution* **39**, 1087–1099.

Travis, J., Emerson, S. B. & Blouin, M. 1987 A quantitative genetic analysis of larvel life history traits in *Hyla crucifer*. *Evolution* **41**, 145–156.

Travis, J. & Mueller, L. D. 1989 Blending ecology and genetics: Progress toward a unified population biology. In *Perspectives in ecological theory* (ed. J. Roughgarden, R. M May & S. A. Levin), pp. 101–124. New Jersey: Princeton University Press.

Via, S. 1984 The quantitative genetics of polyphagy in an insect herbivore. II. Genetic correlations in larval performance within and among host plants. *Evolution* **38**, 896–905.

Wade, M. J. & McCauley, D. E. 1988 Extinction and recolonization: their effects on the genetic differentiation of local populations. *Evolution* **42**, 995–1005.

Wallace, B. 1977 Automatic culling and population fitness. *Evol. Biol.* **10**, 265–276.

Watterson, G. A. 1989 The neutral alleles model with bottlenecks. In *Mathematical evolutionary theory* (ed. M. W. Feldman), pp. 26–40. New Jersey: Princeton University Press.

Wayne, R. K., Modi, W. S. & O'Brien, S. J. 1986 Morphological variability and asymmetry in the cheetah (*Acinonyx jubatus*), a genetically uniform species. *Evolution* **40**, 78–85.

Weis, A. E. & Abrahamson, W. G. 1986 Evolution of host-plant manipulation by gall makers: ecological and genetic factors in the *Solidago–Eurosta* system. *Am. Nat.* **127**, 681–695.

Wilbur, H. M. 1984 Complex life cycles and community organization in amphibians. In *A new ecology: novel approaches to interactive systems* (ed. P. W. Price, C. N. Slobodchikoff & W. S. Gaud), pp. 196–224. New York: John Wiley & Sons.

Discussion

J. J. D. Greenwood (*British Trust for Ornithology, Herts, U.K.*). The possibility that for small mammals the fluctuations in effective numbers may be even greater than those in actual numbers should not be taken as a broader generalization. In the snail *Cepaea nemoralis*, for example, there is evidence that movement is greater at lower population densities than at higher densities, which will tend to lessen the variation in effective numbers (Greenwood 1974, 1976). One can see why emigration rates may adaptively increase at high densities in cyclical populations. But in species in which populations vary spatially in accord with the local availability of resources one might expect individuals to move around more in places where densities are low, to harvest the scarce resources in those places. We need to investigate several representative cases before coming to any general conclusions.

References

Greenwood, J. J. D. 1974 Effective population numbers in the snail *Cepaea nemoralis*. *Evolution* **28**, 513–526.

Greenwood, J. J. D. 1976 Effective population number in *Cepaea*: a modification. *Evolution* **30**, 186.