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Natural selection and population dynamics

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To what extent, and under which circumstances, are population dynamics influenced by concurrent natural selection? Density dependence and environmental stochasticity are generally expected to subsume any selective modulation of population growth rate, but theoretical considerations point to conditions under which selection can have an appreciable impact on population dynamics. By contrast, empirical research has barely scratched the surface of this fundamental question in population biology. Here, we present a diverse body of mostly empirical evidence that demonstrates how selection can influence population dynamics, including studies of small populations, metapopulations, cyclical populations and host–pathogen interactions. We also discuss the utility, in this context, of inferences from molecular genetic data, placing them within the broader framework of quantitative genetics and life-history evolution.

Introduction

That genetically based individual variation in life-history traits could influence population dynamics has interested population biologists since at least 1952, when Dennis Chitty proposed a mechanism involving natural selection to explain population cycles in field voles *Microtus agrestis* ([1], reviewed in [2]). Previously, in 1930, Fisher [3] concluded that, under the idealised conditions of a constant environment, natural selection increases the growth rate of a population, but that, in practice, environmental deterioration owing to physical and biotic changes, including increased population density, has the opposite effect, such that the intrinsic rate of increase fluctuates around zero. Thus, although natural selection often determines which individuals survive and reproduce, the actual number of individuals that survive is generally determined by one or more external limiting factors, such as food, space or predation [4]. For instance, even strong selection on any trait is unlikely to make a marked difference to population dynamics in a hole-nesting bird population that is limited by the availability of nest sites. However, there are other situations where a link between selection and population dynamics is more likely. For instance, in a butterfly metapopulation (see Glossary) persisting in a balance between local extinctions and colonisations in a fragmented habitat, selection on traits influencing the migration of independently dispersing individuals will have (meta)population dynamic consequences via its impact on the rate of fragment recolonisation.

Theoretical framework

Charlesworth presented a general quantitative framework relevant to studying the consequences of selection on population size, taking into account age-specific and density-dependent survival and fecundity [5]. In this framework, it becomes clear that the population dynamic response can be critically sensitive to selection on some specific life-history components, but not on others. This approach draws broadly from population ecology and evolutionary biology; historically, however, the interplay between selection and population dynamics has been approached from three complementary and overlapping perspectives. The first is that of population ecologists, who have recognised the need to incorporate individual variation in models of population regulation [1,2,6,7]. The second is that of population geneticists, who extended

Glossary

Additive genetic variance: the component of the phenotypic variance for a trait that is due to the additive effects of genes; it is the chief cause of resemblance between relatives and determines the response of the population to selection.
Association mapping: a general method of locating the chromosomal region of genes affecting a trait of interest by looking for associations between a large panel of genetic markers and phenotypic variation in the trait.
Candidate gene approach: a strategy to identify genes affecting a trait of interest by focusing on genes known to have a related function in other organisms.
Genetic drift: random fluctuations in allele frequency owing to finite sampling of gametes, generally resulting in reduced genetic variation and the eventual fixation of one allele within any given population.
Inbreeding depression: reduction in mean fitness or any of its components in the offspring arising from reproduction between relatives.
Linkage disequilibrium: the nonrandom association of alleles at two or more loci. It can arise by chance through genetic drift, or through selection for a particular multi-locus combination.
Metapopulation: a network of often small but reproductively more or less independent local populations (often called demes) connected by migration.
Mutational meltdown: a hypothetical process whereby the accumulation of mildly deleterious mutations through genetic drift leads to a declining population growth rate, causing further drift and mutation accumulation, ultimately sending the population to extinction.
Overdominance (or heterozygote advantage): the case in which the heterozygote genotype has a higher fitness than either the homozygote dominant or the homozygote recessive genotype.
Pleiotropy: when a gene affects more than one phenotypic trait.
Virulence: the capacity of a pathogen to cause disease, expressed as percentage mortality of infected hosts.

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the basic population genetics models to density-regulated populations, ostensibly to explore consequences for genotype frequencies but, in so doing, also revealing possible reciprocal effects of such evolutionary changes on population size [8]. The third approach is that of evolutionary biologists wishing to understand life-history dynamics in relation to environmental conditions, including population density [9], through the assessment of tradeoffs within the limits of the available additive genetic (co)variance [10]. There is a clear trend toward the increasing integration of these perspectives [11–13], reflected by a growing acceptance that the traditional dichotomy between ecological and evolutionary timescales is a false one.

The issue of timescale is nonetheless relevant while considering the role of natural selection in population dynamics. It is evident that life histories affect population dynamics; life histories evolve and, in this sense, selection affects population dynamics. But we are more concerned here with population dynamics being affected by concurrent selection. To demonstrate that such selection affects population dynamics requires, minimally, the simultaneous measurement of: (i) density in spatially or temporally replicated samples; (ii) any ecological factors affecting population regulation and density; and (iii) selection on genetic variation for demographically significant traits. The simultaneous measurement of (i)–(iii) is a formidable logistic challenge in all but the simplest systems, particularly as many components of a life-history potentially influence population growth rate. Elasticity analysis [14] is an effective tool for evaluating the relative importance of different phenotypic traits on population growth rate through their effects on survival and reproduction. Some generalisations that are relevant to particular taxa should emerge from such analyses, bearing in mind that dissimilar ecological circumstances can alter the influence of particular traits even within a single species [15].

Conservation biology and the extinction vortex

The potential consequences of genetic factors on population growth rate and extinction risk were brought to prominence during the 1980s through the influential conservation biology texts by Otto Frankel and Michael Soulé [16–18]. At that time, the focus of conservation biology was on the persistence of small populations and, therefore, the effects of inbreeding depression and reduced adaptive potential through genetic drift ('genetic erosion') were widely discussed. These considerations were stimulated by the idea of an 'extinction vortex' generated by a positive feedback between declining population growth rate and genetic erosion [17]. Lande [19] reminded us that these genetic factors cannot be treated in isolation of the ecological context (i.e. population regulation), and he concluded that genetic factors are generally of secondary importance in setting minimum viable population sizes. This conclusion appeared to challenge the significance of genetic factors in biological conservation (reviewed in [20]).

Inbreeding depression in natural populations

Until recently, inbreeding depression has received greater emphasis than has adaptive genetic variation as a candidate for genetic impacts on demography. This makes sense, to the extent that fitness is generally expected to show an immediate decline with inbreeding through the effects of deleterious homozygotes, whereas the fitness consequences of life-history variants are likely to be more dependent on the ecological context and, therefore, more difficult to detect. A few longitudinal field studies of island populations involving near-complete pedigrees have proved invaluable for studying interactions between inbreeding and environmental stress. The first of these, by Keller *et al.* in 1994 [21], found that less-inbred song sparrows *Melospiza melodia* had higher probability of surviving a severe storm. In the same year, Jimenez *et al.* demonstrated that the cost of inbreeding (reduced survival) in mice is likely to be greater in natural than in benign captive environments [22]. Keller and Waller's compilation of cases of inbreeding depression in natural populations includes a diverse set of animals and plants [23]. Components of fitness that are likely to exhibit strong inbreeding depression include fertility, fecundity, juvenile and larval viability and competitive ability [24]. Generally, the severity of inbreeding effects is expected to increase sharply under conditions of environmental stress, as illustrated by parasite infection in Soay sheep *Ovis aries* [25] and high temperatures in *Drosophila melanogaster* [26].

The first demonstration of inbreeding increasing the risk of population extinction in a natural system came from a large-scale study more focused on the ecology than on the genetics of the large metapopulation of the Glanville fritillary butterfly *Melitaea cinxia* [27]. Further evidence comes from a simple but effective field experiment in the outcrossing plant *Clarkia pulchella* [28]. What is striking about this study is that a modest 10% inbreeding differential among populations led to a 60% increase in extinction risk. In other studies, the demographic effects of inbreeding have been revealed through recoveries of previously inbred populations owing to natural or experimental genetic rescue (reviewed in [20]).

Population regulation: hard versus soft selection

The question about population regulation occupied the minds of population ecologists for decades and led to occasionally heated debates [29]. For most ecologists, the matter is now straightforward: some density dependence in population dynamics is necessary to enable the long-term persistence of populations, although the strength and temporal and spatial scales in the occurrence of density dependence can vary greatly [30,31]. Generally, most, although not all, vertebrate populations are relatively tightly regulated, whereas many insect populations fluctuate wildly and give the impression of weak regulation [31]. During the 1990s, ecologists realised that many species inhabiting fragmented habitats occur in metapopulations with weakly regulated and ephemeral local populations, in which case much of the regulation occurs at the level of the entire metapopulation rather than at the level of local populations [32].

The type of population regulation relates to the concepts of hard and soft selection in population genetics (Box 1). In the metapopulation context, soft selection implies local population regulation, whereas hard selection is associated with global regulation: each local population contributes to the next generation in proportion to the mean fitness of individuals, as determined by their genotypes [33]. Entirely global regulation is not possible in real metapopulations [34], but regulation can be largely global, leading to hard selection. We could therefore expect to find examples of selection influencing population dynamics especially in species that persist as metapopulations, and indeed such examples have been documented (Box 2).

Soft selection and population cycles

Do we have evidence for soft selection affecting population dynamics? Although it has been shown that, in principle, density-dependent selection can generate regular oscillations in population size and allele frequency [8], there was until recently no direct empirical support from any natural system. The classic papers by Chitty ([1] and others) envisioned natural selection explaining regular population cycles of boreal and arctic voles, but the

Box 1. Hard and soft selection

The tradition in population genetics is to assume that, for any given environment in the absence of information to the contrary, fitnesses are independent of population density and of the frequencies of other genotypes. In reality, this is unlikely to be so in strongly regulated local populations. To take the consequences of local competition into account and, in so doing, explain how populations can sustain high genetic loads (i.e. the burden of suboptimal genotypes) without going extinct, Bruce Wallace introduced the concept of hard and soft selection [66,67]. Soft selection refers to situations where the strengths of selection coefficients s are density and frequency dependent; hard selection refers to cases where the values of s are independent of both. Additionally, s can also be density or frequency dependent.

The relevance of these concepts to the influence of natural selection on population regulation is that hard selection causes mortality independently of the factors related to local density (or genotype frequencies), be they selective or not, and might therefore have a direct effect on population size. The other density-dependent agents of population regulation might also compensate for the effects of such hard selection, depending on when during the life cycle it occurs, and on its magnitude [8]. For example, the same level of hard selection imposed by an extreme temperature shock is more likely to impact population growth rate when applied to adults (representing the survivors of density-dependent factors) than to juveniles. Density-dependent or soft selection, however, removes individuals that would die or fail to reproduce anyway, for reasons unrelated to their genotype, ideally with no effect on population size (Figure Ia). However, conditions do exist under which soft selection can affect population size, for instance through competition between genotypes whose reproductive rates are differentially sensitive to their own density [8,36] or to that of a predator [37]. Soft and hard selection can also be viewed in terms of local (within-population) and global (between-population) selection in the context of a metapopulation, with corresponding effects on the effective size of local populations (Figure Ib). Empirical data on the relative strengths of soft versus hard selection are limited [68]. One should also recognise that hard and soft selection represent extremes of a continuum, and, in practice, that natural selection is a mix of the two.

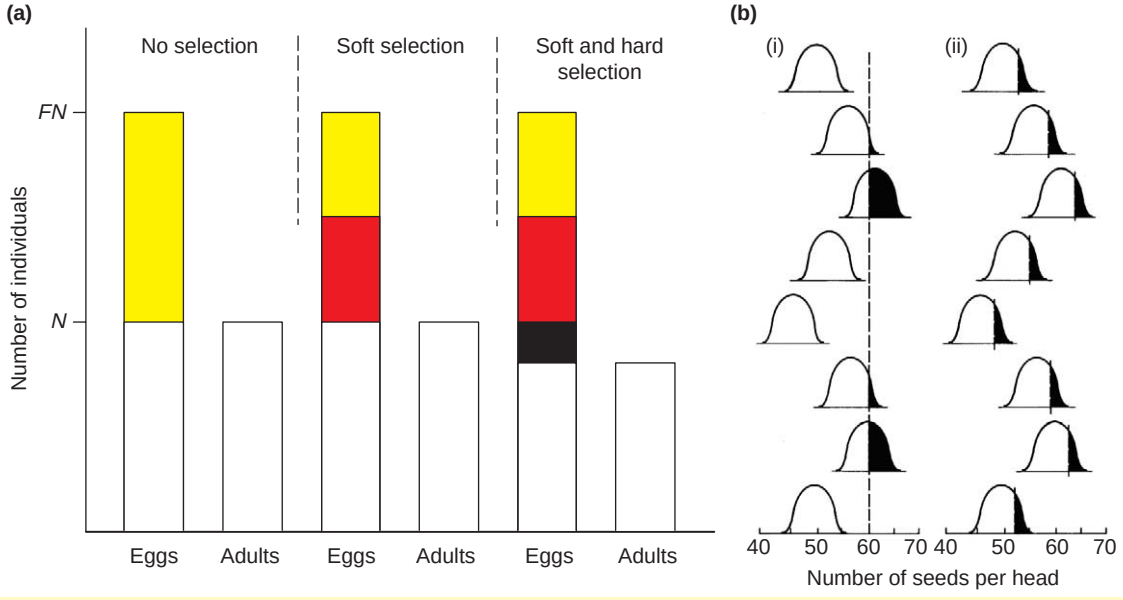

Figure I. Representations of hard and soft selection. **(a)** The concept of hard and soft selection (with respect to population density only, for simplicity not showing frequency dependence). A population of N adults with average fecundity of $2F$ per female produces FN eggs. As maximum adult population size is limited to N by some external factor, such as availability of space or food, $2F–N$ eggs must die each generation. This mortality can be random with respect to genotype and, therefore, nonselective (in yellow), or selective (soft selection, in red), but cannot depress population size below N. Hard selection (in black) introduces an additional, density-independent source of mortality, which reduces population size below N. **(b)** An early representation of hard versus soft selection in the context of a metapopulation. In **(i)**, the small-grain breeder saves all heads bearing 60 or more seeds for planting and future selection; some experimental plots are entirely discarded under this scheme. In **(ii)**, the breeder first samples a few heads from plants of each experimental plot, determines the statistical distribution of seeds per head for each plot, and then harvests what is estimated to be the best 5% of all heads of each plot for planting and further selection. Under this scheme, a few heads are saved from each plot regardless of its average number of seeds per head. Soft selection resembles the second scheme, hard selection the first. Black shading indicates those individuals that were selected to reproduce, no shading those that were not selected. Modified, with permission, from [69] (a) and [67] (b).

Box 2. Hard selection in a butterfly metapopulation

Studies of the large metapopulation of the Glanville fritillary butterfly *Melitaea cinxia* in Åland, southwest Finland, have detected hard (between-deme) selection (Box 1) in relation to several components of fitness. In this butterfly metapopulation, which is characterised by high rates of local extinction compensated for by the recolonisation of currently empty habitat patches, extinction risk is strongly influenced by population size *per se,* immigration from neighbouring populations (the rescue effect), climatically driven spatially correlated density fluctuations, and other ecological and environmental processes [32,70]. In addition, elevated inbreeding in small populations further increases the extinction risk [27]. An important message from this study is that, even in situations where environmental and demographic stochasticities dominate, selection against inbred genotypes can increase extinction risk.

Migration leading to gene flow in metapopulations is the process that enables populations consisting of superior genotypes to increase their share in the metapopulation as a whole. This leads to a mixture of local- and metapopulation-level selection, a syndrome dubbed the 'metapopulation effect' [71]. In the Glanville fritillary, migration leads to the assortment of individuals with genetically determined more and less dispersive phenotypes among new versus old populations [72]. Recent work on this species has shown that there are significant differences among individuals in terms of their flight metabolic rate, flight performance and fecundity associated with a single gene polymorphism (the glycolytic enzyme phosphoglucose isomerase *Pgi* [73]). Furthermore, population growth rate is also affected by the allelic composition of *Pgi*, although in a manner that is specific to the degree of isolation and area of habitat patches [65], owing to complex interactions between landscape structure and the performance of individuals with different phenotypes. This is a clear example of molecular-level variation influencing population dynamics. Another example in the same butterfly metapopulation arises from heritable variation in female host-plant oviposition preference. In brief, the correspondence between the host-plant preference of migrating females and the host-plant composition of potential target meadows has a strong and significant effect on colonisation rate [74], while the extinction–colonisation dynamics appear, in turn, to influence the evolution of host-plant preference in the metapopulation [75].

consensus now is that selection is not involved in vole dynamics [35]. Fifty years on, two independent studies, on species other than voles, [36,37], revealed a key role for genetic variation and soft selection in population density cycles. Sinervo *et al.* [36] showed that two-year adult density cycles in natural populations of the side-blotched lizard *Uta stansburiana* are due to alternating density- and frequency-dependent selection for two contrasting life-history strategies. At low density, *r*-type orange-throated females lay many small eggs, outcompeting *K*-type yellow-throated females, which lay relatively fewer but larger eggs, leading to high adult density and high orange morph frequency in the following year. At high density, although relatively rare, the larger, more competitive offspring produced by yellow-throated females gain the upper hand; adult population density subsequently declines and yellow morph frequency increases. Just as delayed density dependence is the key to population cycles in the absence of evolutionary dynamics, the time lag between soft (density- and frequency-dependent) selection on juveniles and the change in the intrinsic growth rate is what produces population oscillations in this system.

What would happen to the population cycles in the absence of life-history variation? In an elegant experiment, Yoshida *et al.* [37] addressed this question by manipulating the diversity of algal clones (life-history variation) in a predator–prey (rotifer–alga) system. Their results show that, in the absence of genetic variation, the two species oscillate as predicted by classic predator–prey models [38], whereas genetic diversity in the prey population fundamentally alters the dynamics, resulting in much longer cycles for both species that are completely out of phase with each other. Again, the explanation involves a delayed density response, in this case of the grazing predator with respect to abundance and nutritional quality of the algal population, which undergoes rapid cyclical evolution in relation to grazing pressure. This example illustrates how, through interspecific competition, selection in one species can affect population dynamics in another (Box 3).

Genetic mechanisms and constraints

The response to selection on any specific trait can only be fully understood in the context of the entire life history and the underlying genetic architecture. Selection coefficients can differ between age classes and the response of one trait could be influenced by selection on another with which it is genetically correlated, owing to pleiotropy or linkage disequilibrium. Therefore, hypotheses linking population dynamics to selection in natural populations need to consider explicitly the mechanistic basis of the selection response. Quantitative genetic theory of life-history evolution [10] provides a statistical framework for studying the evolutionary dynamics of complex phenotypes determined by many genes. The essential genetic parameters required for this approach are those describing the genetic variance–covariance matrix, estimated from phenotypic measurements on relatives and artificially selected lines [39].

As we discuss here, research projects are increasingly using molecular genetic techniques to characterise genetic

Box 3. Evolution affecting abundance in host–pathogen systems

The general question about how evolutionary dynamics within a community of two or more interacting species might influence abundances has been studied theoretically for a variety of standard ecological interactions, including interspecific competition, predator–prey, host–pathogen, and plant–herbivore–carnivore interactions [76,77].

The host–pathogen interaction is of particular interest because there is strong evidence that pathogens (bacteria, viruses, fungi, protozoa and parasites) can have a major regulatory influence on their host populations through high mortality [78]. Biological control of the European rabbit *Oryctolagus cuniculus* in Australia and Europe, through infection with myxoma virus, is a clear example of how the interplay between the evolutionary and population dynamics of pathogen and host can affect the abundance of both species [79]. The highly virulent strain of myxoma virus first introduced from South America during the early 1950s decimated rabbit populations in many parts of Australia and Europe, but was rapidly outcompeted by mutants of intermediate virulence, which gave more time for transmission by mosquito or flea vectors. On the host side, rabbit populations responded to the intense selection by evolving a degree of resistance to the pathogen. The result was partial recovery of rabbit populations, although the disease still has a considerable influence on their numbers [80].

and developmental mechanisms, often at the expense of quantitative genetic approaches. The latter approach is generally more efficient for studying polygenic variation and genetic correlations; the former for variation governed by few genes of large effect. The molecular approach also provides an alternative for characterizing genetic parameters in species that are difficult to breed in captivity. Where possible, the combined use of both approaches is likely to yield the most useful description of genetic architecture.

The contribution of molecular genetics

The rise of molecular genetics over the past 20 years has influenced substantially the way in which population ecology is conducted but, so far, has had only limited impact on our understanding of population regulation. The emphasis in molecular ecology has been on the application of neutral markers to describe population structure at different scales, information that can be used to make inferences about some demographic processes (e.g. gene flow [40] or population expansion and contraction [41]), but which on its own cannot contribute to a mechanistic understanding of population dynamics. Only recently have studies of genetic population structure begun to place greater emphasis on the need to incorporate actual data on density and other demographic parameters into analyses seeking to describe how particular patterns of genetic variation arise.

Over the same period, conservation genetics has produced information about inbreeding depression [23], theoretical elaborations on changes in additive genetic variance with inbreeding [42], theoretical models of minimum viable population size [43,44], the concept of 'mutational meltdown' [45], some applications of adaptive dynamics [13], and more data on population structure – all necessary and useful, but rarely addressing directly the question about selection and population dynamics. The disciplines of molecular genetics and conservation genetics have now matured, conceptually and technically, to the point where molecular genetic tools can make an important contribution to the selection–population dynamics question.

A relevant application of neutral markers is as a genealogical trace in the construction and validation of pedigrees or sibships [46]. Pedigrees coupled with life-history data are the ultimate resource for revealing variation in components of fitness, but are exceedingly difficult and labour intensive to follow in natural populations. Marker-assisted reconstruction will hopefully expand the sample of wild pedigrees, currently restricted to island populations of birds and ungulates, although samples involving overlapping generations remain problematic. Even in the absence of pedigrees, marker-inferred relatedness can be used to quantify genetic variation underlying traits of interest [47].

Spatiotemporal analysis of adaptive genetic variation

Several studies have demonstrated ongoing natural selection through changes in phenotype (genotype) frequencies over time and/or space, but few have investigated reciprocal effects of density and selection. Classic examples include clines (e.g. *Drosophila* alcohol dehydrogenase [48]; melanism in the peppered moth *Biston betularia* [49]; and insecticide resistance in the mosquito *Culex pipiens* [50]) and hybrid zones (e.g. of the grasshopper *Podisma pedestris*, fire-bellied toads *Bombina* spp., and races of the butterfly *Heliconius erato* [51]). Selection is often expected to have no or minimal effect on density owing to density dependence, but explicit demonstrations of this assumption in relation to natural environments are few [25,52].

Perhaps the greatest potential use of molecular genetics in this context is as a tool to score phenotypes that are usually difficult to detect directly in natural populations. This is only possible once genotype–phenotype correlations have been established, which is a nontrivial task. In addition to providing means to measure cryptic phenotypes, the advantage of PCR-based molecular assays (e.g. microsatellites or SNPs [53]) is that they can be applied efficiently to very large samples, often in a manner that is noninvasive to the population as a whole, thereby increasing the statistical power to detect individual-level genetic effects on demography across space and time. This general approach has so far been largely restricted to heterozygosity–performance correlations [25,54,55]. The implication of several studies [23] is that inbreeding depression is an important component of fitness variation in many natural populations, but it remains unclear to what extent such correlations reflect genome-wide expression of deleterious recessives, rampant overdominance or publication bias [56,57].

The search for ecologically important molecular genetic variation is not new [58] but it is fair to say that it is only now beginning to take off (e.g. [59,60]). Key demographic life-history traits for this effort will vary among species, but are likely to include components of mating success, fertility, fecundity, competitive ability, defence, disease resistance, stress tolerance and migration. A variety of strategies are being developed to apply association mapping to non-model organisms [61], and the candidate gene approach provides an increasingly viable alternative [62]. Large-scale assays of variable gene expression [63] provide another tool for characterizing the strength and scale of local adaptation, which could have population dynamic consequences. Combining cDNA microarrays with suppression subtractive hybridisation (SSH) [64] further extends our ability to identify differentially expressed genes underlying demographically relevant traits.

Conclusions

Many ecologists primarily interested in population dynamics have been sceptical of the relevance of genetics and natural selection in their research. Historically, this view stems from the belief that selection is invariably density dependent or soft, with negligible reciprocal effects on density itself. However, soft selection can generate population cycles [36,37], and conditions for hard selection might be relatively common, especially in fragmented landscapes [27,28,65]. Our own experience with the well-studied Glanville fritillary metapopulation, combined with several other lines of evidence, suggests

that, until one looks carefully, it is unwise to assume that genetic variation and selection are irrelevant to population dynamics. The detection of such effects in other systems is likely to require large-scale longitudinal field studies with a focus on variation in demographically important life-history traits. The effect of environmental context and population density on selection coefficients will only be revealed through a greater appreciation of the spatial and temporal scale of environmental heterogeneity as experienced by individuals with well-characterised life histories. For populations and communities of species pushed beyond their past zones of relative comfort through anthropogenic environmental change, hard selection will increasingly become the hard reality.

Beyond its use for inferring population structure and demographic history, the value of molecular genetics in population ecology will be judged by the extent to which it provides fresh insight into the components of functional variation among individuals and facilitates the measurement of such variation in natural populations. Gene hunting might be an expensive luxury for ecologists but, incorporated into a framework of well-understood population ecology and life-history quantitative genetics, the potential dividends are great. The inverse of this argument is that, in our drive to characterise genetic and developmental architectures, we should not lose sight of the fact that fitness depends on the usually complex ecological context.

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