
Genetic Diversity, Adaptive Potential, and Population Viability in Changing Environments

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Heterozygosity is the most widely used estimator of genetic diversity. The enduring appeal of heterozygosity may stem from a widely held belief that a correlation between heterozygosity and fitness reflects genomewide benefits of genetic diversity (reviewed by Dewoody & Dewoody, 2005). Heterozygosity can be thought of as the probability that an individual will have more than one allele at a particular genetic locus and is usually reported as expected heterozygosity, H_E (Box 13.1). Lasting popularity of H_E with conservation biologists is rather surprising because it has long been known that this estimator is relatively insensitive to population bottlenecks of short duration (Hedgcock & Sly, 1990). Other measures of within-population genetic diversity include individual multilocus heterozygosity, proportion of polymorphic loci, squared size difference between two alleles at a locus, and the effective number of alleles per locus (Box 13.1).

In addition to estimating genetic diversity within populations, conservation biologists are often interested in estimating the proportion of the total genetic diversity that is partitioned among geographically separated populations. This is useful when deciding where to locate protected areas and whether to translocate individuals between populations. The most popular method of estimating population structure is with Wright's hierarchical F statistics (Wright, 1965), which are derived from Wright's inbreeding coefficient (Box 13.1) and can

be calculated from the deficiency of heterozygotes at nuclear molecular markers. His most famous statistic, the fixation index F_{ST} , is the genetic variation within subpopulations relative to that within the total population. At equilibrium, F_{ST} is inversely related to the product of the migration rate among the subpopulations and their effective population size. Another statistic that is useful, especially when the pedigree is unknown, is Wright's within-individual inbreeding coefficient F_{IS} , which partitions the amount of heterozygosity in individuals relative to that within the subpopulation. His final statistic, F_{IT} , partitions the amount of heterozygosity in individuals relative to that within the total population. Excoffier and colleagues (1992) describe a computer program (Arlequin) that computes F_{IS} , F_{ST} , and F_{IT} from molecular markers and can test for structure among predefined groupings of the subpopulations.

Inbreeding depression has been broadly defined as a decline in fitness that arises from decreasing heterozygosity (or increasing homozygosity) across the genome (Reed, this volume). An increase in homozygosity caused by small population size can decrease population viability through three mechanisms. First, matings between close relatives becomes more common. This is problematic because close relatives are likely to have inherited the same recessive deleterious allele at a given locus, which increases the frequency of expression in the population. This is inbreeding in the strict sense,

BOX 13.1 Introduction to Neutral Molecular Markers for Ecologists

Routine nonlethal collection of small tissue samples for later genotyping with molecular markers is becoming an increasingly common component of ecological studies. Small tissue samples can be preserved for later extraction drying them, or by putting small pieces in 95% ethanol or commercial salt solutions. Commercial kits are now available that can reliably extract DNA from tissue, hair, blood, fin clips, or feces. Most molecular markers in common use today (Box Table 13.1) require only small amounts of crude DNA extract because they use PCR to amplify the target DNA region exponentially until there is enough to genotype. After PCR, one of three genotyping steps usually takes place: (1) the PCR fragment is sent to be sequenced by a local automated DNA sequencing facility, (2) the PCR fragment is subjected to electrophoresis in a capillary tube filled with acrylamide gel to determine its size, or (3) a PCR fragment containing an SNP is labeled with allele-specific primers or probes, which are genotyped automatically in a specialized machine.

Neutral Molecular Markers Glossary

allozymes	Different alleles at an enzyme locus produce proteins that differ in net charge, which affects their relative migration rate on a starch gel.
effective number of alleles per locus, A_E	The number of alleles it would take to obtain a given level of the expected heterozygosity if all alleles were equally frequent. $A_E = 1/(1 - H_E)$
expected heterozygosity, H_E	Often called genetic diversity, D . For a single locus it is estimated using the formula $H_E = 1 - \sum p_i^2$, where p_i is the frequency of the i th allele.
expressed sequence tag (EST) markers	Complementary DNA (cDNA) libraries are created when messenger RNAs (mRNAs) are harvested from a particular tissue and converted to DNA using the enzyme reverse transcriptase before being cloned into a plasmid vector. These “complementary” or cDNA libraries are meant to have a complete copy of every mRNA in that tissue. cDNA libraries contain different alleles for a particular gene either because the organism was heterozygous at that locus or because the library was made from multiple individuals. The forward or reverse sequences of each clone in a cDNA library are often publicly available in EST databases and can be used by ecologists to discover SNPs or microsatellites that are within or immediately adjacent to a particular protein-coding region.
F_{IS}	Wright’s (1965) within-individual inbreeding coefficient. This measures the amount of nonrandom mating within subpopulations that results in excessive homozygosity at the individual level.
F_{ST}	Wright’s (1965) fixation index. The average number of pairwise differences among subpopulations relative to the average number within populations. This is the most used of Wright’s F statistics that estimate the hierarchical partitioning of genetic variance within and among populations.

continued

inbreeding coefficient	The probability that two alleles for a particular gene are identical by descent as determined from a known pedigree containing a shared ancestor.
individual multilocus heterozygosity	The proportion of heterozygous loci within an individual (Coltman & Slate, 2003)
microsatellites	A genetic locus with alleles that consist of a variable number of copies (usually 10–30) of simple tandem repeat (STRs) of 2 to 4 bp of DNA sequence. Different alleles have different PCR fragment sizes and can be distinguished by their relative migration rates on an acrylamide gel.
neutral molecular marker	A base pair substitution, insertion, or deletion in a DNA region that is close to neutral with respect to the fitness of the organism in that it does not change its viability or fertility. For example, third codon positions in protein-coding genes are synonymous substitutions.
nonsynonymous substitution	A single base pair substitution in a protein-coding DNA region that does result in an amino acid substitution and therefore could experience a different selection coefficient.
observed heterozygosity, H_O	Number of individuals that are heterozygous at a locus over the total number sampled.
PCR	Polymerase chain reaction. This is a modern method of making millions of copies of a DNA sequence of 100 to 5,000 bp pairs that is surrounded at each end by a known “primer” sequence of 20 to 30 base pairs. This is done by putting some dilute DNA extract, the forward primer (usually labeled with a fluorescent dye), the reverse primer, extra bases of all four types, and heat-resistant DNA polymerase, into an eppendorf tube and then using a thermocycler machine to cycle the reaction between three temperatures that are typically: 95°C, 50°C, and 72°C. This has largely replaced molecular cloning as a method of obtaining sufficient quantities of a particular gene fragment to allow genotyping in conservation genetics laboratories.
proportion of polymorphic loci	Proportion of the loci that have more than one allele that has a frequency greater than 5%.
single nucleotide polymorphism (SNP)	DNA sequence variation at a single base pair in a nuclear or mtDNA fragment. The differences in sequence were traditionally detected by DNA sequencing, but high-throughput genotyping methods that use hybridization to oligonucleotide chips or primer extension reactions with labeled nucleotides are becoming more common.
squared size difference between two alleles at a locus	This is a useful measure of genetic diversity at a microsatellite locus where the absolute size differences among alleles is known precisely.
synonymous substitutions	A single base pair substitution in a protein-coding DNA region that does not change the amino acid.

From Hartl and Clark (2007).

continued

BOX 13.1 Introduction to Neutral Molecular Markers for Ecologists (*cont.*)

BOX TABLE 13.1 Popular Neutral Molecular Markers (Box 13.1) Used to Estimate Genetic Diversity in Multicellular Animals, Their Characteristics, and Assumptions, and Best Methodology for Genotyping

Marker	Type	Neutral?	Genotyping	Allelic Diversity	Reproducible among Labs?	Comments
Allozyme (protein)	Biparental nuclear, codominant	Often	Starch gel electrophoresis	Low per locus	Somewhat, if relative allele mobility used	Historical data sets abundant
SNP (nDNA exons single-copy genes)	Biparental nuclear, codominant	Third codon position	Primer extension, SSCP or direct sequencing of PCR product	Low per site but one SNP per 1,000 bp	Highly if sequenced	Direct sequencing if many alleles
SNP (nDNA introns)	Biparental nuclear, codominant	Usually	Primer extension, SSCP; can sequence homozygotes	Moderate	Highly if sequenced	Length polymorphism common
SNP (ITS, NTS ribosomal genes)	Biparental nuclear, codominant	Usually	SSCP	Moderate and several copies	Highly if sequenced	Concerted evolution of gene copies
SNP (mtDNA)	Maternal haploid, codominant	Third codon position	Primer extension, SSCP or direct sequencing	Moderate	Highly if sequenced	Nuclear pseudogenes
Microsatellite = STR = SSR	Biparental nuclear, codominant	Usually	Capillary DNA automated sequencer	High per locus	Yes, if same machine with test samples	Often too many alleles, if sample size is small
AFLP	Biparental nuclear, dominant	Most	Fragment length analysis	Moderate but many loci per gel	Somewhat	Useful for little-studied genomes
RAPD	Biparental nuclear, dominant	Often	Fragment length analysis	± only, but many loci per gel	No	Historical interest
MHC	Biparental nuclear, codominant	Balancing selection	SNP, SSCP	High	Highly if sequenced	High gametic phase disequilibrium

See also *Avise, 2004*.

which has been defined as the proportion of alleles identical by descent (Brook, this volume). Second, genetic drift results in fixation of deleterious and slightly deleterious alleles. Accumulation of mutations through drift can cause extinction by mutation meltdown (Reed, this volume). Third, strong genetic drift causes rare alleles to be lost and thus reduces a population's ability to adapt to new environments. I will show that reduction in evolutionary potential (Soulé, 1987) can also cause extinction.

Most morphological, behavioral, and life history traits are quantitative and thus show continuous variation that cannot easily be separated into discrete phenotypic classes (Falconer & MacKay, 1996). Estimating the genetic diversity of quantitative, or complex, traits is challenging, because phenotypes in this case are determined by multiple genetic loci and environment. Furthermore, unlike single-locus Mendelian traits such as eye color, loci responsible for complex traits are only now being identified (Hill, 2005). The most common within-population measure of genetic diversity used for complex traits is still narrow-sense heritability (Charmantier & Garant, 2005), which is defined as the proportion of the phenotypic variance that is directly heritable (Box 13.1). There is also a between-population measure of genetic diversity for quantitative traits, Q_{ST} (Merilä & Crnokrak, 2001), which is analogous to F_{ST} .

NEUTRAL VERSUS NONNEUTRAL MARKERS

Neutral theory (Kimura, 1968; Kimura & Ohta, 1971) proposed that the majority of base substitutions that become fixed in populations are nearly neutral with respect to selection. Theory also predicts that fixation rate of neutral substitutions ν is equivalent the mutation rate per gene per successful gamete. Neutral theory has been supported by empirical population genetics studies, beginning in the 1960s with allozyme protein electrophoresis (Avice, this volume). Allozymes are variants for a particular enzyme that have an amino acid substitution that affects protein net electrical charge and enables alleles to be distinguished using their relative migration on starch gels (Avice, 2004a). It is now recognized that although substitution of alleles segregating at most allozyme loci are neutral with no detectable effect on fitness, substitutions at other

loci are not. Correlation between the frequency of particular allozyme alleles and particular environments have been observed and are likely the result of habitat-dependent selection (see, for example, Johannesson & Tataronov, 1997). As discussed later, modern PCR-based markers can also be nearly neutral or nonneutral, depending on linkage to other genes under differential selection in different environments.

IS GENETIC DIVERSITY RELEVANT IN A CONSERVATION CONTEXT?

One paradigm of modern conservation biology is that management of small populations entails estimates of molecular genetic diversity (Avice, 2004a). It is unclear, however, whether molecular markers yield an accurate estimate of population viability. Lynch (1996) reviewed the usefulness of molecular markers in conservation biology and questioned whether they provided much insight into (1) the loss of potential to adapt to future environmental change, (2) accumulation of deleterious mutations, and (3) the fate of individuals translocated from a large and genetically diverse population to a small and inbred one. In contrast, O'Brien and colleagues (1996) described two examples in which reduced variation at molecular markers in populations of large cats was correlated with elevated sperm abnormalities and reduced testosterone levels. Thus, some evidence suggests that reduced variation at molecular markers can signal a severe loss of mean population fitness. The question remains, however: Are molecular markers useful for estimating the potential of a population to adapt to future environmental change?

Stockwell and colleagues (2003) argue that conservation biologists thus far have been mostly concerned with maximizing genetic diversity rather than considering trade-offs arising from contemporary (or rapid) evolution. For example, they point out that strong selection for adaptation to current environments erodes genetic variation that might be needed to adapt to future environmental change. As a second example they remind us that translocation into an inbred population will reduce the level of inbreeding but will decrease its local adaptation.

In this chapter I discuss the effect of genetic diversity on the adaptive potential and viability of small populations experiencing environmental change.

I review the most widely used types of molecular markers and discuss methods of using such markers to estimate N_e . I specifically investigate (1) the extent to which genetic diversity at neutral molecular markers predicts population viability, (2) nascent technical and statistical solutions that address the problem of estimating evolutionary potential, and (3) the strength of the relationship between heritability and the adaptive potential of a complex trait in a series of populations connected by migration.

VARIATION AT MOLECULAR MARKERS

“Neutral” Loci

Heterozygosity and N_e

The level of inbreeding per generation and the loss of alleles from genetic drift both increase when the effective population size, N_e , is small (Brook, this volume). Indeed, if we ignore mutation and migration, then the rate of loss of heterozygosity for a single locus, at generation $t + 1$ as a function of the heterozygosity at generation t , is

$$H_{t+1} = H_t \left(1 - \frac{1}{2N_e} \right) \quad (13.1)$$

where N_e is the effective population size (Freeman & Herron, 2006). The decline in heterozygosity with N_e is somewhat more complex to model for a quantitative trait because the phenotype is determined by a few to hundreds of loci that combine additively, by epistasis, by dominance, and by the maternal and rearing environments (Falconer & Mackay, 1996). A past meta-analysis of 19 studies by Reed and Frankham (2001) found no correlation between heritabilities and genetic diversity at molecular markers. However, Reed (this volume) now argues that levels of genetic variation, whether measured as heritabilities, allelic diversity, or heterozygosities, correlate well with theoretical and empirical evolutionary potential.

Commonly Used “Neutral” Molecular Markers

Allozyme electrophoresis has been almost entirely replaced in conservation genetics by other

molecular markers that use PCR to amplify specific DNA fragments (Awise, this volume). The main advantages of PCR-based DNA markers are that they (1) allow genotyping of very small tissue samples, (2) can amplify DNA fragments from impure genomic DNA extracts from ethanol or salt-preserved tissues, (3) can use noninvasively obtained tissues such as hair or fecal samples, and (4) vary in their rates of molecular evolution, allowing the most appropriate one to be chosen. This has made possible studies that were never possible before. Tissues used for allozyme electrophoresis had to be fresh or stored at temperatures less than -80°C to avoid denaturation of the enzyme because it was needed to catalyze the reaction that stained the gel (Awise, 2004a).

Currently, the two most commonly used markers in conservation genetics studies of animal populations are SNPs, both in mitochondrial and single-copy nuclear genes, and length polymorphisms in microsatellites (Box Table 13.1, Box 13.1). Microsatellites, which are also known as *simple tandem repeats* (STRs) or *simple sequence repeats* (SSRs), are particularly useful for paternity analysis because they typically have a large number of alleles per locus. Both SNPs and microsatellites are popular because they are PCR-based and because, with care and automation, the genotyping results are reproducible among different laboratories (Box Table 13.1). However, several new methods of genotyping a large number of SNP loci for a large number of individuals have been developed that use multiplexing of allele-specific extension primers or probes (Morin et al., 2004). These allele-specific methods emit distinctive fluorescent signals that are interpreted by automated allele-scoring software and need only a little human correction. Therefore, studies using SNP markers are usually lower in cost and higher in throughput than microsatellite markers, even after the lower polymorphism of SNP markers is taken into account. Also, large numbers of SNP loci are becoming available for many model organisms because of the availability of short DNA sequences of expressed sequence tags (ESTs) in public databases (Box 13.1) and because of new, more efficient methods of resequencing previously published genomes (Morin et al., 2004). If a large number of markers is needed for a nonmodel organism, then AFLP is still widely used, even though heterozygotes usually cannot be distinguished from homozygotes and reproducibility is only moderate (Box Table 13.1).

Variation at “Adaptive” Molecular Markers

Major Histocompatibility Complex Markers

Neutral molecular markers may not resolve fine-scale population structure nor be able to assign individuals to closely spaced populations. This problem has encouraged a number of laboratories to develop “selected” or “adaptive” molecular markers. If local adaptation causes some alleles to become substantially more common at some sites than others, then that will enable the assignment of individuals to particular populations. For example, major histocompatibility complex (MHC) loci are currently a popular type of “selected” molecular marker for jawed vertebrates and have been used successfully to look at the genetic diversity of bottlenecked populations in the Felidae (Yuhki & O’Brien, 1990). Major histocompatibility complex genes are part of the immune response to foreign antigens, and their high mutation and recombination rates are thought to be an adaptive strategy for rapid evolution in response to disease pathogens. The antigen recognition site part of the protein is unusual in showing more non-synonymous substitutions among individuals than synonymous substitutions (Box 13.1), suggesting positive selection is taking place (Hughes & Nei, 1988, 1989). This positive Darwinian selection is hypothesized to promote higher charge profile diversity in the antigen-binding cleft of class I MHC molecules and thus high substitution rates (Hughes et al., 1990). Major histocompatibility complex loci were useful for fine-scale population differentiation in Sacramento River chinook salmon (Kim et al., 1999). They were also found to give higher F_{ST} values than microsatellite markers, allowing finer scale detection of among-population differentiation in sockeye salmon (Miller et al., 2001).

Markers that are Linked to Loci Under Selection

A low percentage of the loci for any type of molecular marker can be classified as “selected” or “adaptive” molecular markers because it is tightly linked physically to QTL that experience selection for different optima in different habitats. For example, 5% of the 306 AFLP loci genotyped in a marine snail population showed different frequencies in the upper and lower shore ecotypes that were

divergent in morphological and life history characteristics (Wilding et al., 2001). An average of 2% to 3% of the AFLP loci showed different frequencies for normal and dwarf ecotypes of whitefish living in the same lake (Campbell & Bernatchez, 2004). In both these cases, the neutral AFLP loci showed little or no significant genetic differentiation between the two ecotypes, likely because gene flow between them was too high. A promising new type of marker to use to study adaptive genetic divergence is SNPs in ESTs. Vasemägi and colleagues (2005) found correlations between 5 of 75 EST-associated SNPs, and the salinity and latitude of the habitat of Atlantic salmon populations.

ADAPTIVE POTENTIAL

The importance of maintaining the evolutionary potential of a population was first mentioned by Soulé (1987). Franklin (1980) used mutation rates for neutral quantitative traits in *Drosophila* to propose that populations had to have an effective size, N_e , of at least 50 in the short term to avoid inbreeding and of 500 in the long term to maintain their evolutionary potential. Lynch and Lande (1998) do not agree that $N_e = 500$ is large enough to maintain the evolutionary potential, and argue that effective population sizes of 1,000 to 5,000 are needed, especially for single-locus traits because of their lower mutation rates. This is a concern because populations targeted for conservation are likely to have effective population sizes much smaller than 5,000.

Complex Traits Determined by Quantitative Trait Loci

There has been considerable effort recently to locate the QTLs that determine complex morphological, life history, and behavior traits that are of most interest to conservation biologists. Only 10 to 100 neutral molecular markers are typically available for most nonmodel organisms. However, fine-scale genetic linkage maps with thousands of markers now exist for model organisms used in agriculture, and these can allow identification of the actual DNA sequence that comprises the QTL (Hill, 2005).

Detection of QTLs is often achieved by genotyping backcrosses or F_2 crosses between divergent populations with neutral molecular markers and looking for statistical associations with phenotypic

traits. With family sizes of 300, only QTLs that contribute 5% or more to the additive genetic variance can typically be detected (see, for example, Tao & Boulding, 2003). Two lines of corn that had been divergently selected for 100 generations were used to discover 440 SNP markers that enabled detection of QTLs that contributed less than 1% to the genetic variance for oil content (Hill, 2005). The results from the corn QTL analysis and results from a recent QTL analysis on body size in a line of poultry support the hypothesis that many quantitative traits are determined by many segregating genes (about 50 in corn and 13 in poultry), each with small effects that combine additively (Hill, 2005). However, in many other lines of poultry, body size is determined by mostly a few segregating loci, each with a large effect (Hill, 2005), perhaps because other loci that could potentially contribute have become homozygous. The number of segregating loci cannot be determined by just observing at the phenotypic distribution of the trait. Even only two loci may give a continuous phenotypic distribution after epistasis, dominance, maternal, and environmental effects are included (Lynch & Walsh, 1998).

Variation at Quantitative Trait Loci Currently “Neutral” and N_e

The amount of allelic variation at a neutral QTL in an isolated population depends on the equilibrium between mutation and genetic drift (Lynch & Walsh, 1998). The magnitude of mutation rate depends on the DNA region (Hartl & Clark, 2007) whereas the amount of genetic drift depends inversely on N_e , as shown in Eq. 13.1. The situation for a quantitative trait like body size is more complex than for a single-locus Mendelian trait because if the effects at different loci are additive, then they can cancel each other out. Imagine that each locus has only two possible alleles, one “plus” allele that increases body size and a second “minus” allele that decreases body size. Then at some QTLs, the plus allele will drift toward a higher frequency and at others it will drift toward a lower frequency so that small changes in allelic frequencies will cancel out, to some extent, and body size will remain constant. Evolution of quantitative traits is often modeled using the infinitesimal model, which assumes that the phenotype, z , depends on an infinitely large number of genes, each having an infinitely small

additive effect (Bulmer, 1985). The large number of loci means that the allelic frequencies at each QTL and the additive genetic variance at linkage equilibrium remain approximately constant over time and that the breeding values (Box 13.2) of each individual in the population are normally distributed (Bulmer, 1985).

Variation at Quantitative Trait Loci Currently under Measurable Selection

The amount of allelic variation at a QTL in an isolated population depends on the equilibrium between mutation, selection, and genetic drift (Lynch & Walsh, 1998). Strong selection for a prolonged period will reduce the additive genetic variances, and therefore the heritabilities for traits important in local adaptation will decrease (Falconer & MacKay, 1996). If directional selection is strong, as when there are human-induced effects on a population, then the breeding values will no longer be normally distributed and the loci may show positive gametic phase disequilibrium (Box 13.2) and may deviate from the traditional infinitesimal model.

After the population mean has reached a new optimum and is under pure stabilizing selection, then excessive genetic variance will actually decrease the short-term fitness of the population. Lande and Shannon (1996) point out that conservation biologists often assume that a higher additive genetic variance is always better, but that this is not true if the optimum is fixed, because the environment is constant. They argue that when there is strong stabilizing selection toward a fixed optimum, a larger genetic variance increases the average distance of the individual’s phenotype from the optimum and reduces the mean fitness of the population. In contrast, as I discuss later, higher heritabilities are beneficial if discrete or continuous environmental change is occurring (Lande & Shannon, 1996).

Geographic Variation at Quantitative Traits and Q_{ST}

The geographic distribution of a species at risk is often more fragmented than it was historically, and managers must often decide whether to translocate individuals among locations. Although translocation will reduce inbreeding, it might cause the population to be maladapted if, for example, species in

BOX 13.2 Basic Quantitative Genetics for Ecologists

Quantitative genetics is the study of the underlying genetics of continuous traits, meristic (or discrete) traits, and threshold traits, which includes most traits of interest to ecologists. Such complex traits are usually determined by multiple genetic loci and by the maternal and external environments. The theory of quantitative genetics was developed by animal and plant breeders who traditionally used parametric statistics to compare the phenotypic values of related to unrelated individuals for a particular trait. This allowed estimation of the heritability that, when multiplied by the intensity of artificial selection, successfully predicted the evolution of the phenotypic mean in the next generation (Falconer & Mackay, 1996). Modern applied quantitative genetics still relies on statistical analysis of the phenotypic measurements of a pedigreed population of animals rather than knowledge of what is occurring at the molecular level (Falconer & Mackay, 1996). Indeed it is only recently that quantitative trait loci (QTLs) that contribute to such complex traits have actually been identified (Hill, 2005). However, location of QTLs has been achieved by creating crosses that result in gametic phase disequilibrium between molecular markers and QTLs (Lynch & Walsh, 1998). Higher values of a particular trait will be statistically associated with a particular allele at a molecular marker locus (Lynch & Walsh, 1998). If the genome is saturated with enough molecular markers, then it is possible to clone and sequence the DNA region that contributes to the trait. This is being attempted for the California condor to locate the mutation for chondrodystrophy (Zoological Society of San Diego, 2008) so that likely carriers of this disease can be identified.

Quantitative Genetics Glossary

additive genetic variance animal model	V_a , proportion of phenotypic variance that is directly heritable and results from the sum of the allelic effects at all the loci that affect a particular trait.
breeding values	A maximum likelihood method of estimating heritabilities and genetic correlations that is particularly precise when there are measurements of traits for a population over several generations, an estimate of the pedigree from observation or paternity analysis with molecular markers. The heritable genetic value of an individual as judged by the average value of a particular trait in its offspring. When mated at random, its breeding value will be twice the average deviation of its progeny from the population mean.
dominance genetic variance	V_d , variance in the phenotype caused by inheritance of alleles at a particular heterozygous quantitative locus that cause a single copy of the plus allele to contribute to the phenotype as if the locus was homozygous for the plus allele.
environmental variance	V_e , variance in the phenotype caused by variation in the environment.
epistatic variance	V_i , variance in the phenotype caused by nonadditive interactions between particular alleles at different loci that contribute to a quantitative trait.
gametic phase disequilibrium (or linkage disequilibrium)	A nonrandom association of alleles at multiple loci created by physical linkage, nonrandom mating, or mixing of two populations under different selection regimes. Positive gametic phase disequilibrium occurs when particular gametes have more plus alleles at the multiple loci that contribute to a trait than would be expected given the average frequency of plus alleles in the population.

continued

BOX 13.2 Basic Quantitative Genetics for Ecologists (*cont.*)

genetic correlation	Degree to which two traits respond to selection independently; can be caused by two genes being determined by some of the same genetic loci or by gametic phase disequilibrium.
genetic variance	The sum of the additive genetic variance, the dominance variance, and the epistatic variance.
genetic variance/covariance matrix	Matrix with additive genetic variances on main diagonal and genetic covariances on the subdiagonals.
heritability	h^2 or H^2 , proportion of phenotypic variance that is heritable. Narrow-sense heritability (h^2) is ratio of additive variance to phenotypic variance. Broad-sense heritability (H^2) is ratio of total genetic variance to phenotypic variance. Both are estimated by a breeding design that compares similarity of a trait in relatives and nonrelatives that is due only to their genes. The most common way to estimate narrow-sense heritability is from a pedigree that includes half siblings.
linear selection differential	$S = \mu_s - \mu$, where μ is the original population mean and μ_s is the mean of those that survive to breed. S measures direct and indirect selection caused by selection on traits that are highly phenotypically correlated with the trait of interest. S is equivalent and can be converted to β , the univariate linear selection gradient (Freeman & Herron, 2006).
maternal effects	Variance in the phenotype caused by genetic and environmental variation in the maternal environment. Offspring are more similar to their mother than expected from the additive effect of the genes that they have inherited from her.
multivariate linear selection gradient	Statistical methodology that separates direct selection on a trait from indirect selection on the trait; traditionally obtained by using the standardized trait values as the independent variables in a multiple linear regression with relative fitness as the dependent variable (Lande & Arnold, 1983).
nonadditive genetic variance	The sum of the dominance variance and the epistatic variance.
phenotype	The value of the traits that you observe on the organism.
phenotypic correlation	Pearson's correlation between two different traits.
phenotypic variance	V_p , sample variance of trait from actual measurements on the organism.
Q_{ST}	Amount of among heritable population variation in a quantitative trait relative to the amount of additive genetic variation within populations.
response to directional linear selection	$R = \mu' - \mu$, where μ is the original population mean and μ' is the mean of the next generation. The response is the change in the mean value of the trait in the next generation relative to their parent's generation.

From Falconer and Mackay (1996).

different populations are locally adapted to different climate optima along a latitudinal gradient. One way to assess whether maladaptation of translocated individuals will be a problem is to compare the amount of genetic differentiation among populations shown by neutral molecular markers with that shown by quantitative traits.

Spitze (1993) defined Q_{ST} to be the among-population variation in a quantitative trait relative to the within-population variance. He pointed out that if Q_{ST} was larger than the F_{ST} value, then the among-population variance in the trait (in his case, body size in the water flea *Daphnia*) must be the result of local adaptation rather than neutral phenotypic evolution.

Palo and colleagues (2003) compared Q_{ST} from a maternal half-sib design with F_{ST} from eight microsatellite loci for six frog populations along a latitudinal gradient and found that Q_{ST} exceeded F_{ST} for all three life history traits, suggesting considerable local adaptation. However, they also found there was no correlation between pairwise estimates of Q_{ST} and F_{ST} for any populations. This lack of correlation supports the hypothesis that knowledge of F_{ST} from neutral molecular markers is not helpful in predicting geographic differentiation in the quantitative traits under strong selection (Stockwell et al., 2003), and therefore other methods such as estimation of Q_{ST} must be used.

Linkage between Neutral Molecular Markers and Quantitative Trait Loci

Estimation of Q_{ST} may be difficult or impossible for small populations of a species at risk because precise estimates require at least five families in a controlled breeding design per population and at least 20 populations (O'Hara & Merilä, 2005). One possible alternative is to use the $\cong 2\%$ to 5% of neutral molecular markers that are tightly and physically linked to QTLs and to calculate F_{ST} instead of Q_{ST} . Calculation of F_{ST} from only those molecular markers showing exceptionally high values was done for the marine snail (Wilding et al., 2001), the whitefish (Campbell & Bernatchez, 2004), and the salmon populations (Vasemägi & Primmer, 2005) discussed earlier. Thus, we may be able to estimate geographic differentiation at QTLs by looking at the F_{ST} values of physically linked molecular markers. It would be optimal to use high-throughput markers such as SNPs so that a large number of markers for

a large number of individuals from each population could be genotyped.

Heritability and Fisher's Equation

The breeder's equation states that the rate of evolution in response to new selective pressures is proportional to the heritability (Etterson, this volume; Falconer & Mackay, 1986):

$$R = b^2S \quad (13.2)$$

where b^2 is the narrow-sense heritability and S is the selection differential (Box 13.2). Therefore, to maintain the future ability to adapt to changes in the environment, managers of populations must maximize or at least maintain heritability. Unfortunately, narrow-sense heritabilities are difficult to assess quickly for animals because their estimation usually involves rearing offspring to adulthood in a paternal half-sib design to avoid maternal effects (see, for example, Boulding & Hay, 1993). Rearing the offspring in several environments is also important because the heritability of traits not closely tied to fitness, such as morphometric traits, has been shown to increase when environmental conditions are favorable (reviewed by Charmantier & Garant, 2005). Furthermore, estimation of heritabilities involves estimating variance components and, because of this, heritabilities typically have very large 95% confidence limits unless more than 500 half-sib families are used (Falconer & MacKay, 1996; Lynch & Walsh, 1998). This makes it difficult to detect whether a prolonged population bottleneck has resulted in a significant reduction in the additive genetic variance. For these reasons, the best method of determining whether a prolonged population bottleneck has resulted in a small effective population size might be with neutral molecular markers, because such markers are quicker and less invasive.

Genetic Variance/Covariance and Lande's Multivariate Equation

In many cases, local adaptation of populations along a gradient can involve several traits that are correlated phenotypically with one another (see, for example, Palo et al., 2003). If estimates of the multivariate linear selection gradient and genetic variance/covariance matrix are both available, then

one can predict the change in the mean of each trait using Lande's 1979 matrix equation for multitrait evolution:

$$\Delta z = G\beta \quad (13.3)$$

where Δz is a vector of the response to selection, G is the genetic variance/covariance matrix estimated by measuring traits on related and unrelated individuals, and β is a vector of multivariate linear selection gradient coefficients (Etterson, this volume; Lande & Arnold, 1983) (Box 13.2). This matrix equation was a major step forward because it incorporates both the direct and the correlated response to selection that previously had to be calculated separately (Falconer & Mackay, 1986). The effect on evolutionary potential of genetic constraints caused by genetic correlations among multiple traits has rarely been considered by conservation geneticists (Etterson, this volume; Etterson & Shaw, 2001) but could further increase the minimum population size required to maintain evolutionary potential.

POPULATION VIABILITY

Do Neutral Molecular Markers Estimate Population Viability?

Viability after a recent and prolonged population bottleneck

Lynch (1996) reviewed the literature on the relative mutation rates for molecular markers ($10^{-8} - 10^{-5}$ per locus per year) and quantitative traits ($10^{-3} - 10^{-2}$ per trait per generation) and concluded that the latter would recover their levels of additive genetic variance after a population bottleneck more quickly because they have higher mutation rates. However, this difference in mutation rates is not relevant when only very recent population bottlenecks are of interest, as is typically the case for endangered populations. If the bottleneck was recent, then not enough time will have passed for mutation to regenerate the genetic variance at either QTLs or the marker loci. Furthermore, many modern molecular markers, such as adenosine and cystine dinucleotide microsatellite markers have per-gamete mutation rates averaging 10^{-4} to 10^{-3} per year, depending on their length (Whittaker et al., 2003). This rate is more similar

to the mutation rates that Lynch (1996) cited for quantitative traits.

Equation 13.1 showed that the rate of loss of heterozygosity at neutral molecular markers was inversely related to effective population size. Equation 13.4 shows that the same is true for the rate of loss of variation at neutral quantitative traits. At equilibrium between mutation and genetic drift, the broad-sense heritability h_b^2 for a neutral trait is (Falconer & Mackay, 1996, p. 351)

$$h_b^2 = \frac{2N_e V_m}{2N_e V_m + V_E} \quad (13.4)$$

where V_m is all the mutational genetic variance and V_E is the environmental variance. If $V_m = 10^{-3}V_E$, which has been estimated from mutation rates for neutral characters in *Drosophila*, then at mutation-drift equilibrium the equation predicts that if $N_e = 100$ then $h^2 = 0.17$ and if $N_e = 10,000$ then $h^2 = 0.95$ (Falconer & Mackay, 1996, p. 351). However, most heritabilities for large populations are considerably lower than 0.95, suggesting that natural selection must be important in reducing them. Unfortunately, the effects of selection on additive genetic variance are only understood for major components of fitness in which most mutations are deleterious (Falconer & Mackay, 1996).

If there is no selection on a trait and the nonadditive genetic variance is assumed to be zero, then the reduction of heritability after t generations of inbreeding is

$$h_t^2 = \frac{h_{t-1}^2(1 - F_t)}{1 - h_{t-1}^2 F_t} \quad (13.5)$$

where h_t^2 is the heritability of the population and F_t is the inbreeding coefficient (Box 13.1) at time t , and h_{t-1}^2 is the heritability at time $t - 1$ (after Eq. 15.1 in Falconer & Mackay, 1986). The increase in the inbreeding coefficient per generation is a function of the effective population size, N_e (Eq. 3.6 in Falconer & Mackay, 1986):

$$F_t = \frac{1}{2N_e} + \left(1 - \frac{1}{2N_e}\right) F_{t-1} \quad (13.6)$$

If we assume that in the base population at time $t - 1$ that $F_{t-1} = 0$, then the second term in Eq. 13.6

disappears. Substituting the simplified version of Eq. 13.6 into Eq. 13.5 gives us the reduction in the heritability of the population after one generation at a small effective size:

$$h_t^2 = \frac{h_{t-1}^2 \left(1 - \frac{1}{2N_e}\right)}{1 - h_{t-1}^2 \frac{1}{2N_e}} \quad (13.7)$$

However, such a reduction would be impossible to detect after only one generation. For example, if we assume that $N_e = 5$ and that initially $h^2 = 0.50$, then Eq. 13.7 says that after one generation, the heritability will have been reduced to 0.43, which is 86% of its initial value. Similarly Eq. 13.1 shows that after one generation at $N_e = 5$ the heterozygosity at a single neutral molecular marker locus will be reduced to 90% of its initial value. It would be impossible to detect statistically such a small reduction in the heritability using the methodology typical for wild animal populations. It would also be impossible to detect the reduction in heterozygosity at any known molecular marker locus after one generation if the census population size was also only five animals.

Estimation of N_e with Molecular Markers

Considerable recent effort has been expended toward using molecular markers to estimate N_e , and two methods are currently popular. The first method uses the observation that populations that have recently experienced a very small effective population size often show a reduction in the number of alleles before they show a reduction in their observed heterozygosity, because rare alleles are lost first (see, for example, Hedgecock & Sly, 1990). Cornuet & Lucas (1996) devised a method using allelic frequencies from nuclear genes to determine whether a significant number of loci exhibit a larger heterozygosity than that expected from the observed number of alleles when it is assumed that the locus is at mutation–drift equilibrium (their program, BOTTLENECK 1.2.02, is freeware available at <http://www.montpellier.inra.fr/URLB/bottleneck/bottleneck.html>). Luikart and Cornuet (1998) took this approach with 56 allozyme and 37 microsatellite data sets from real populations, and under the assumptions of the stepwise mutation model, found a significant heterozygote excess in the bottlenecked populations about half the time. Beebee & Rowe

(2001) likewise used BOTTLENECK on data comprising eight microsatellite loci genotyped for 20 to 40 tadpoles from each of 50 endangered toad populations to confirm that they could identify populations for which the census data documented a severe population bottleneck (down to tens of toads) within the past 20 to 30 years. Luikart and Cornuet (1998) postulate that such bottlenecks should be detectable for 0.2 to $4 N_e$ generations after the bottleneck, where their N_e is the bottleneck population size.

A second method for precisely estimating N_e uses temporal changes in the allelic frequencies at molecular marker loci and assumes that selection, mutation, and migration can be ignored (Waples, 1989; Williamson & Slatkin, 1999). Anderson (2005) describes an efficient Monte Carlo method for estimating N_e from temporally spaced samples using a coalescent-based likelihood and even provides a computer program to do the calculations. Wang and Whitlock (2003) describe a new methodology for estimating effective population size (N_e) and migration rates (m) simultaneously from changes in the allelic frequencies of neutral molecular markers between two or more samples that are separated by periods of time that are short enough so that mutation can be ignored, but they do not provide a computer program.

It is reassuring that molecular markers such as microsatellites can detect a recent population bottleneck if the population has been recently reduced to a few tens of individuals, but the question is whether a decline of that magnitude will result in a reduction in the additive genetic variance.

It would seem likely that prolonged bottlenecks less than $N_e = 50$ would be detectable in wild populations using molecular markers. If bottlenecks are detected, then heritabilities could be estimated using modern statistical methods to determine whether they had declined (Falconer & Mackay, 1986; Lynch & Walsh, 1998). However, not everyone agrees that even severe bottlenecks can be detected. After their meta-analysis, Coltman and Slate (2003) conclude that, because of the low correlation between multilocus heterozygosity and phenotypic variability, at least 600 individuals might have to be genotyped for microsatellite markers before there is sufficient statistical power to detect inbreeding depression. Their conclusions might have differed, however, if they had analyzed correlations between multilocus heterozygosity and the additive genetic variance instead of correlations between

multilocus heterozygosity and the phenotypic variance.

Does Evolutionary Potential Decrease after Bottlenecks?

There is some evidence that reduced variation at molecular markers is correlated with a reduced response to selection, at least over the long term. Unfortunately, most available data are from founder–flush experiments where the bottleneck typically only lasts for one generation and is followed by exponential population growth up to a large carrying capacity. Briggs and Goldman (2004) found that genetic variation in AFLP markers and long-term selection response was reduced in laboratory plant populations (*Brassica rapa*) that had experienced a recent bottleneck of two individuals relative to control populations. In contrast, they found that heritability and short-term selection response actually increased for two of the three bottlenecked replicates for the first three generations after the bottleneck. They used the CoNe software (Anderson, 2005) to estimate N_e from the AFLP markers and got underestimates ($N_e = 9–19$) of the true N_e , which was known from the experimental design to be 25.

Founder–flush bottleneck experiments using laboratory populations of houseflies have also found that genetic variation at molecular markers declined, but heritabilities can actually increase after a single population bottleneck. Flies that had experienced a single population bottleneck of 1, 4, or 16 mated pairs were compared with a larger control population (Bryant et al., 1986). For five of the eight traits, the highest heritability was for four pairs, and this was interpreted as conversion of some of the nonadditive genetic variance into additive genetic variation (Bryant et al., 1986). The average rate of fixation of several allozyme loci for these lines were 7%, 13%, and 38%, respectively, and the viability of the one- or four-pair lines was significantly lower than that of the 16-pair lines (Bryant et al., 1986). Allozyme analysis of these bottlenecked populations documented that only 39.1%, 75.6%, and 80.1% of the alleles remained for the one-, four-, and 16-pair treatments, respectively, after five separate bottleneck founder–flush episodes. In a second experiment the additive genetic variance increased in the one-pair treatment after the first bottleneck but returned to the same level as the control after the fifth bottleneck, suggesting that by that point all

the nonadditive genetic variance had been converted to additive genetic variance (Bryant & Meffert, 1995).

A recent meta-analysis suggests that severe inbreeding may cause a linear decrease in the levels of genetic variation, as predicted in Eq. 13.5, for quantitative traits that are not closely associated with fitness. Van Buskirk and Willi (2006) reviewed 22 published studies, mostly on insects, in which they compared the level of genetic variation in experimentally inbred populations (measured by V_A or h^2) with that in outbred control populations. They found the expected linear decrease in variation with inbreeding coefficient (Eq. 13.5) only for morphological or behavioral traits such as bristle number, wing length, and mating speed. For life history traits, the heritability actually increased after a bottleneck up to an inbreeding coefficient of 0.4, because of increases in the dominance and epistatic variance (Box 13.2). Despite this observation, they questioned whether bottleneck-induced variation actually increased the viability of the populations because of the inbreeding depression that often accompanies it.

Gilligan and colleagues (2005) estimated genetic variation for two neutral traits—abdominal and sternopleural bristle numbers—and allozyme heterozygosity in 23 populations of *Drosophila melanogaster* that they had maintained at effective population sizes of 25, 50, 100, 250, or 500 for 50 generations. They found that quantitative genetic variation was being lost at a similar rate to variation at molecular markers. However, both rates were significantly slower than those predicted by neutral theory. They attributed their success at detecting this significant relationship to a large range of inbreeding values present in the different treatments of their experiment.

Reed and associates (2003a) compared the number of offspring produced by outbred and by fully inbred population of *D. melanogaster* maintained in either single-stress, variable-stress, or benign conditions for seven generations before being transferred to an environment with novel stresses for seven generations. They used the addition of either copper sulfate or methanol to the food medium as the single or variable stress and then used the absence of sugar in the medium as the novel stress. They found that outbred populations adapted to the absence of sugar significantly better than inbred populations, and that populations that had been maintained in a stressful environment were better at adapting to

a novel stressful environment. They attributed the slower adaptation by flies from a benign environment where selection was relaxed to two possible factors: the loss of costly stress-resistant traits or mutation accumulation.

Estimating Heritabilities in the Field Using Molecular Pedigrees

To avoid complications arising from estimating evolutionary potential using molecular markers, many researchers prefer to estimate heritability directly. However, heritability estimation for a population of a species at risk is challenging because the number of individuals that are available is limited and because breeding is not controlled. Without controlled breeding it is not possible to apply a traditional half-sibling breeding design for estimating genetic parameters (Lynch & Walsh, 1998). One solution to this is to collect trait measurements over several generations and to use an animal model (Box 13.2) to estimate the heritabilities (Kruuk, 2004). Estimation of heritabilities and additive genetic variances in nonpedigreed wild populations is becoming more feasible because of the development of highly polymorphic molecular markers that estimate more accurately the relationships among the individuals on which the quantitative traits are measured (Garant & Kruuk, 2005; Wilson & Ferguson, 2002). Microsatellite markers have been used successfully to estimate genetic parameters in wild populations of deer (Kruuk et al., 2000) and trout (Wilson et al., 2003b). However, estimation of parental genotypes with molecular markers is not trivial, even if at least five offspring from each full-sib family are genotyped for 5 to 10 microsatellite loci (Lemay and Boulding, unpublished data; Smith et al., 2001). Furthermore, heritabilities estimated in natural populations, where the breeding design cannot be controlled, will be inflated if related individuals share a more similar environment than unrelated individuals (Lynch & Walsh, 1998). On the positive side, the environmental variance exhibited in the field will be at a natural high level rather than at the reduced level typical of a controlled laboratory environment. On the other hand, laboratory estimates of heritabilities and genetic correlations allow efficient breeding designs with known parents and can be improved by rearing offspring in more than one environment, which allows estimation of genotype-by-environment interactions (see, for example, Boulding & Hay, 1993;

Mahaney et al., 1999). A multivariate maximum likelihood analysis of three different cholesterol subfractions on 942 pedigreed baboons fed one of two diets found that the genetic correlations between three subfractions differed between diets because of epistatic interactions (Mahaney et al., 1999).

ADAPTATION TO CHANGING ENVIRONMENTS

After a change in the environment, populations will be initially maladapted, their fertility and viability will decrease, and their population growth rates may become negative (Lynch & Lande, 1993). Unless the populations evolve fast enough to adapt to the changing environment, they may go extinct. Fast rates of evolution are only possible if the populations are large enough so that they remain genetically diverse.

Discrete Environmental Change

One Population: r , K , and h^2

The question of whether populations with higher heritabilities are more likely to survive a discrete, or step-like, change in the environment was first addressed by Gomulkiewicz and Holt (1995). They presented an analytical model of the evolution of a quantitative trait after a discrete change in the environment caused the population to decline. In their model, the population did not go extinct if the trait evolved rapidly enough to adapt to the new optimum before the population dropped below an unspecified critical population size. They showed that heritability reduced the amount of time that a population spent below this critical, but undefined, population size and concluded that the effect of heritability was only important for large populations experiencing small discrete changes in the environment.

Boulding and Hay (2001) disagreed with this conclusion and used the results from an individual-based finite-locus model to argue that heritability was important in preventing extinction after environmental change. They showed that populations that had higher heritabilities recovered faster from a discrete environmental change than those with lower heritabilities, particularly when the initial population size was large (Fig. 13.1), and

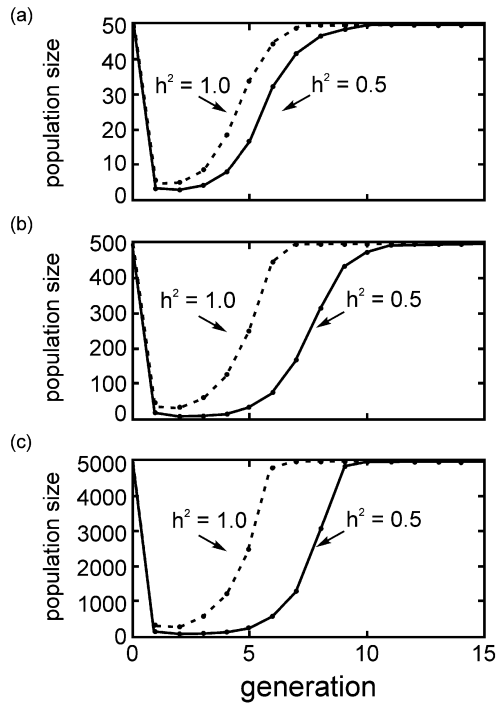


FIGURE 13.1 (A–C) Effect of heritability (h^2) on the number of generations required for an isolated population to recover to carrying capacity after a discrete shift in its optimal phenotype. Initial population size is 50 (A), 500 (B), and 5,000 (C). The percentage of populations that avoided extinction was dependent on initial population size when $N_i = 50$: 16% for $h^2 = 0.5$, 65% for $h^2 = 1.0$; when $N_i = 500$: 89% for $h^2 = 0.5$, 100% for $h^2 = 1.0$; when $N_i = 5,000$: 100% for $h^2 = 0.5$, 100% for $h^2 = 1.0$. Increasing the heritability and the initial population size increased population viability by increasing the minimum population size reached before the population began to increase again. (After Figure 1 in Boulding and Hay [2001].)

were consequently less likely to go extinct. Indeed, Figure 13.2 shows that for large populations ($N = 10,000$), increasing the heritability from almost zero to one increased the size of the shift in the optimum that could be tolerated by three phenotypic standard deviations (PSDs). They also found that the size of the shift in the optimum to which the organisms could adapt increased linearly with the \log_{10} of the fecundity ($r^2 = 0.97$, Fig. 13.3). Thus, species with high fecundities should be able to adapt to larger shifts in the optima than species

with low fecundities. The results of their model support the hypothesis that higher heritabilities, larger population size, and higher population growth rates increase population viability (Reed, this volume).

One Population: Migration

Migration is important to population viability because it can increase the heritability of traits that are under different local selection pressures in adjacent populations. A large difference between the genotypic means of the immigrants and that of the local population will generate strong, positive gametic phase disequilibrium, which will inflate the additive genetic variance and increase the response to selection until it is gradually broken down by recombination during reproduction (Tufto, 2001). Therefore, if migrants are prevented from reaching a population because of habitat fragmentation, there may be a large decrease in the heritability even if the population is moderately large.

In quantitative genetic models without demography, gene flow prevents local adaptation unless stabilizing selection is strong (Boulding, 1990; Bulmer, 1985). Similarly, in quantitative genetic models with demography, recurrent gene flow from a population adapted to an environment where the optimum is very different will result in local maladaptation and thereby increases the probability of extinction (Boulding & Hay, 2001; Tufto, 2001). In contrast, Holt and Gomulkiewicz (2004) present a density-dependent, individual-based quantitative model that shows that a somewhat higher number of migrants from a source population can facilitate slow adaptation by a low-density sink population to a novel environment over long periods of time. However, they found that migrants were no longer beneficial after local adaptation had taken place.

Linear Series of Populations Connected by Migration

The evolutionary potential of a population affects its ability to adapt to the abiotic and biotic effects of climate change. Populations at either end of a linear environmental gradient have an increased risk of extinction because unbalanced gene flow from the center of a cline results in poor local adaptation to abiotic factors such as temperature. Recent analytical quantitative genetic models have shown that gene flow can prevent populations on the

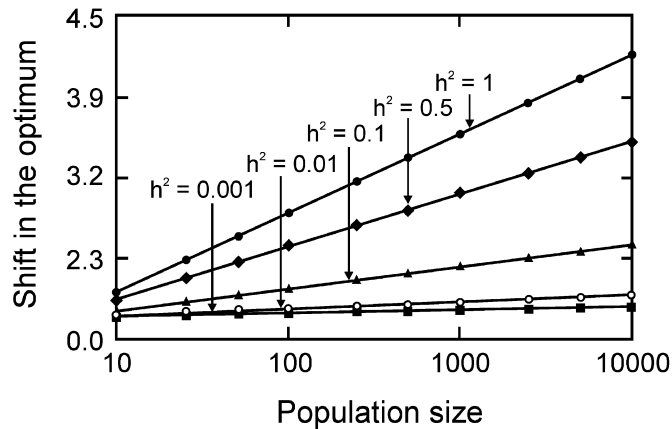


FIGURE 13.2 Effect of heritability on the maximum shift in the optimum that 95% of the populations tolerated for different initial population sizes. Increasing the heritability substantially increased the amount of discrete environmental change that a single, isolated population could withstand. (After Figure 2a in Boulding and Hay [2001].)

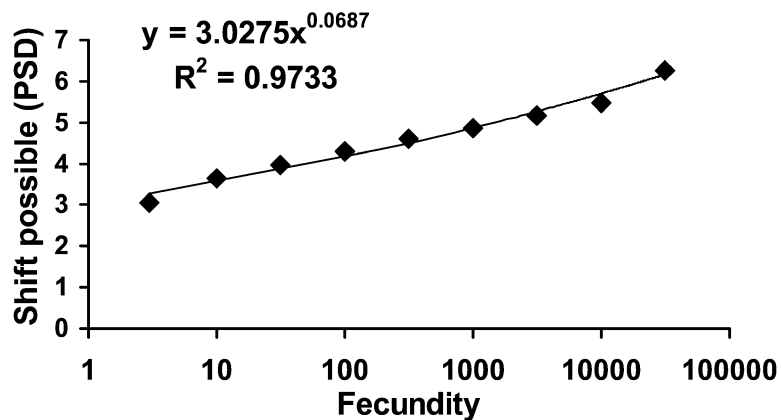


FIGURE 13.3 Effect of fecundity on the maximum shift in the optimum that 95% of the populations tolerated. The amount of discrete environmental change that an isolated population could withstand increased linearly with the \log_{10} of the fecundity. PSD, phenotypic standard deviation. (Data from Boulding and Hay [2001].)

periphery of a species' range from adapting to their local environment (García-Ramos & Kirkpatrick, 1997), causing them to become demographic sinks (Kirkpatrick & Barton, 1997).

Climate change can also change selection from the biotic environment. Global warming after the last ice age resulted in species that were good dispersers moving poleward at faster rates than those

that were poor dispersers (Gates, 1993). This suggests that predators may move poleward ahead of their usual prey species and begin to prey on indigenous prey. Subtropical invertebrate predators often have feeding appendages that are more specialized than those of comparable temperate predators likely because predator-prey coevolution is more escalated in the tropics (Vermeij, 1977, 1987).

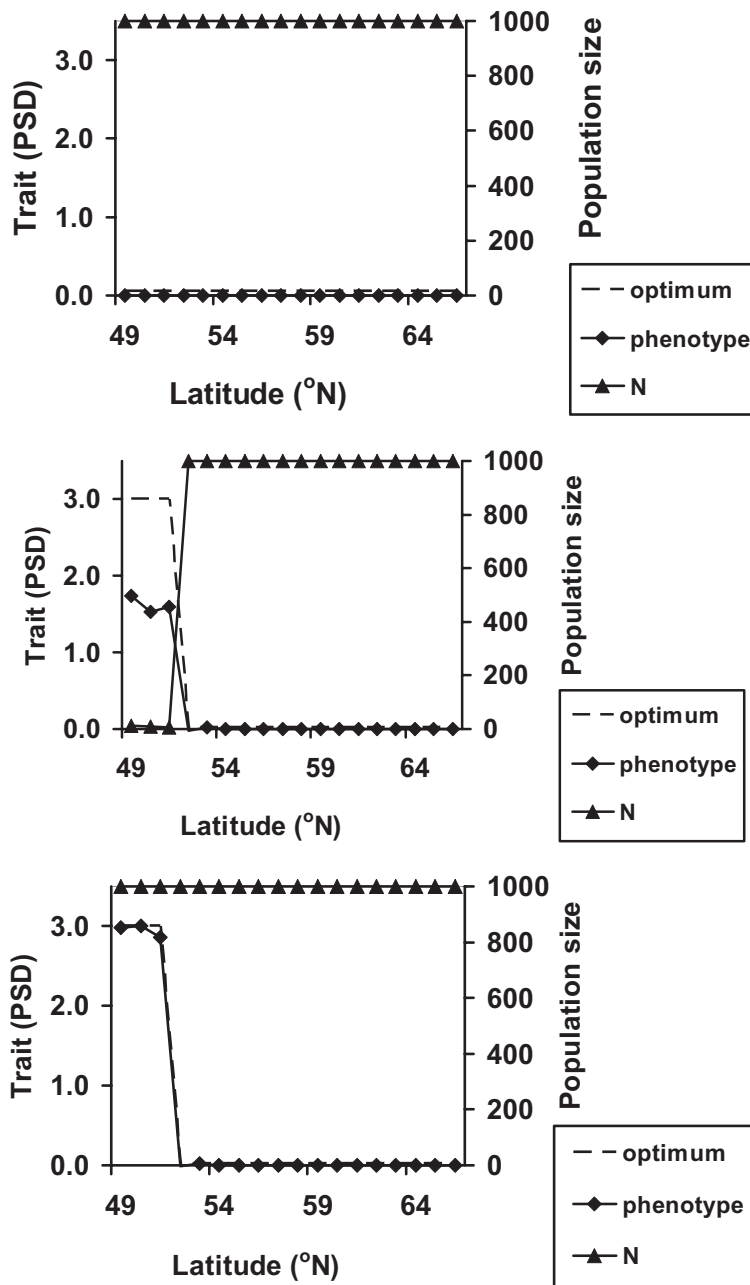


FIGURE 13.4 Evolutionary response to a discrete change in the optimal phenotype. (A) Before shift in optimal phenotype (all cline simulations). (B) Population sizes and evolution of the mean phenotype in the cline three generations after optimum in three southern populations was shifted 3 PSDs. Note that the populations in three end populations have dropped well below carrying capacity. Selection intensity = 0.1, heritability = 0.5, migration rate from adjacent uninvaded population = 0.025. (C) Mean phenotype and population size along the cline 17 generations after the shift in the optimum by 3 PSDs in the southern three populations. Note that the populations at 49°N and 50°N have completely adapted to the new optimum, but the population at 51°N still has not. Heritability = 0.5, migration rate from adjacent uninvaded population = 0.025. PSD, phenotypic standard deviation.

Range extensions by subtropical predator species are interesting because they may cause extinction of temperate prey species, which could otherwise tolerate the stress of warmer temperatures.

Local adaptation to a discrete change in the environment, such as one caused by an invasion by a new predator, will be less likely if there are large numbers of maladapted immigrants from adjacent uninvaded populations. Boulding and colleagues (2007) describe an extension of an individual-based finite-locus model of the phenotypic evolution of a quantitative trait (Boulding & Hay, 2001) to a linear series of populations connected by migration. Figure 13.4 shows the evolutionary response to a discrete change in the optimal phenotype that occurs only in the three most southern populations. In generation 1 (Fig. 13.4A), the optimum in the three most southern of 18 populations is shifted by 3 PSDs, but the width of the fitness function is kept constant at 0.75 PSD. In this run, the population size three generations after the shift in the optimum declined from the carrying capacity of 1,000 to 12, nine, and five individuals at 49°N, 50°N, and 51°N latitude, respectively (Fig. 13.4B). The population at 49°N was buffered from gene flow from sites where the optimum was unchanged and recovered up to the carrying capacity by generation 9. However, the population at 50°N did not recover until generation 11, and the population at 51°N went temporarily extinct and did not recover until generation 15. For this run with a migration rate of $m = 0.025$ (adjacent populations exchanged 2.5% of their individuals), the three invaded populations eventually showed complete adaptation to their new optimum. For a higher migration rate of 0.25 (adjacent populations exchanged 25% of their individuals), adaptation was complete in the populations at 49°N and 50°N, but was not complete in the population at 51°N (91%), which directly received immigrants from its uninvaded neighboring population at 52°N.

Figure 13.5 shows that the initial heritability also had an effect on population viability on the most southern of the three populations. When the heritability was intermediate ($h^2 = 0.5$), as shown in the previous example, the rate of adaptation to the new optimum was relatively rapid (Fig. 13.5). When heritability was the maximum value possible ($h^2 = 1.0$), adaptation to the new optimum occurred more quickly so that the minimum population size was larger and the population returned to the carrying capacity sooner (Fig. 13.5). In contrast, when the

heritability was low ($h^2 = 0.1$), the population went extinct and did not recover within 180 generations (Fig. 13.5). Thus, higher heritabilities reduce the chance that a population would go extinct as a result of a discrete change in the local optimum that occurs in only part of a species' geographic range.

Continuous Environmental Change

Heritability and Continuous Linear Rates of Change

All analytical models published so far demonstrate that population persistence is more likely when the heritability of the quantitative trait is high. Pease and colleagues (1989) considered migration and adaptation in response to a moving linear environmental gradient and found that higher heritabilities increased the probability that the species would persist in the face of continuous environmental change. Lynch and Lande (1993) found that populations experiencing continuous environmental change were more likely to maintain positive rates of population growth if the heritability was high. They proposed that the critical rate of environmental change for a low-fecundity population that requires 50% of the individuals to survive and reproduce would be only $1.6 N_e \times 10^{-3}$ PSD per generation. Bürger and Lynch (1995) present an analytical model that explicitly includes demography, and a simulation model of adaptation to continuous environmental change as well. They found that populations in environments with larger carrying capacities could tolerate higher rates of environmental change. This was because when N_e was larger, more additive genetic variance was maintained in the population at equilibrium. They concluded that the maximum rate of continuous environmental change that can be tolerated could be as little as 0.1 PSD per generation when the carrying capacity was $K = 32$, but increased to 0.7 PSD per generation when $K = 512$.

Demography versus Genetics

Lande (1988) has argued that demographic processes are more important than genetic factors in small populations; however, genetic processes become important when there is strong selection for new optima as a result of climate change. Brooks (this volume), as well as the new data from our individual-based model (Boulding et al.

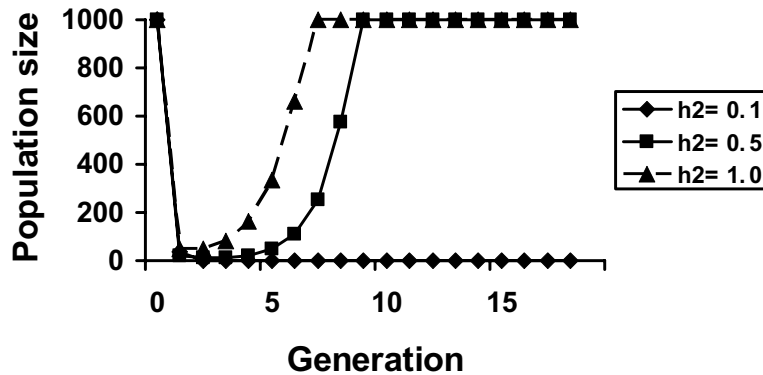


FIGURE 13.5 Effect of heritability on population dynamics of the population at 49°N, which is experiencing directional selection toward a larger optimum because of a predator invasion in the southern three populations of the cline. Migration rate from adjacent population = 0.025. Higher heritabilities increased population viability.

2007), shows that both demographic and genetic parameters facilitate adaptation by populations to changes in the environment. Higher heritabilities increase the likelihood that a population will adapt to discrete changes in the environments. Given sufficient time for the population to reach equilibrium between mutation, migration, and drift, the heritability of neutral traits will be larger if the effective population size is larger (Eq. 13.4), and the effective population size will be larger if the carrying capacity is larger (Freeman & Heron, 2006). However, under nonequilibrium conditions, heritability can vary independently of population size. For discrete environmental change, a large initial population size gives the population more time to adapt before it goes extinct. Furthermore, when stabilizing selection was strong and the shift in the optimum large, adaptation in our model often happened within 10 generations, which made the initial heritability more important than the mutation rate (Boulding & Hay, 2001).

GENERAL CONCLUSIONS

I conclude that neutral molecular markers can often be used to identify populations that have recently experienced a prolonged population bottleneck. Populations with a reduced number of alleles at neutral molecular markers will be those that have had a small effective population size for several generations. The literature suggests that bottlenecked

populations are also likely to have low heritabilities for quantitative traits at least for those traits that are closely related to fitness.

I presented new results from a previously described individual-based model of a single quantitative trait (Boulding & Hay, 2001; Boulding et al. 2007) that demonstrate that the viability of a population after a discrete change in its environment depended on its heritability, as well as the number and source of the migrants from adjacent populations. The model showed that a decrease in the heritability resulted in reduced evolutionary potential of populations along the cline to adapt to environmental change. Populations that were not able to adapt to changes in their environment had a higher probability of going extinct.

FUTURE DIRECTIONS

My review of the literature leads me to conclude that the most urgent need is for better statistical methodology for detecting reductions in heritable genetic variation using neutral molecular markers. This would be facilitated by more laboratory experiments (see, for example, Gilligan et al., 2005) that simulate small population size and simultaneously look at the genetic diversity of neutral molecular markers and the heritabilities of quantitative traits that are neutral as well as those closely related to fitness. Because Q_{ST} is so difficult to estimate, it would also be useful to have more population studies that

estimate F_{ST} with a large set of selected markers linked to QTLs, and then compare those values with the amount of genetic differentiation at neutral markers. Most ecological studies that have been published so far have compared F_{ST} at less than 10 selected markers with F_{ST} of a much larger number of neutral markers. This is important because estimation of F_{ST} with selected markers might be an alternative to estimation of Q_{ST} when less than 20 populations are available. It would also be useful to have theoretical models of genetics and demography for multiple quantitative traits rather than just for single quantitative traits. These are needed to assess the role of genetic architecture in constraining the potential of populations to adapt to environmental change. For example, antagonistic genetic correlations among traits could reduce the rate at which prairie plants adapt to global warming (Etterson, this volume; Etterson & Shaw, 2001).

SUGGESTIONS FOR FURTHER READING

For a gentle but complete introduction to molecular markers, see Avise's book (2004a). For a solid understanding of introductory quantitative genetics, see the classic by Falconer and Mackay (1996). Hartl and Clark (2007) give a detailed but clear introduction to population genetics, ecological quantitative genetics, and even genomics in their

new edition. Holt and Gomulkiewicz (2004) give a nonmathematical review of quantitative genetic models that include demography and review their own work, which emphasizes evolution over long timescales.

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