# Genetic diversity and the survival of populations

Plant Biology

Booy, G.; Hendriks, R.J.J.; Smulders, M.J.M.; Groenendael, J.M.; Vosman, B.

https://doi.org/10.1055/s-2000-5958

# Genetic Diversity and the Survival of Populations

G. Booy[1], R. J. J. Hendriks[2], M. J. M. Smulders[1], J. M. Van Groenendael[2], and B. Vosman[1]

[1] Plant Research International, Wageningen, The Netherlands
[2] Department of Ecology, Nijmegen University, Nijmegen, The Netherlands

Received: July 5, 1999; Accepted: May 10, 2000

***Abstract:*** **In this comprehensive review, a range of factors is considered that may influence the significance of genetic diversity for the survival of a population. Genetic variation is essential for the adaptability of a population in which quantitatively inherited, fitness-related traits are crucial. Therefore, the relationship between genetic diversity and fitness should be studied in order to make predictions on the importance of genetic diversity for a specific population. The level of genetic diversity found in a population highly depends on the mating system, the evolutionary history of a species and the population history (the latter is usually unknown), and on the level of environmental heterogeneity. An accurate estimation of fitness remains complex, despite the availability of a range of direct and indirect fitness parameters. There is no general relationship between genetic diversity and various fitness components. However, if a lower level of heterozygosity represents an increased level of inbreeding, a reduction in fitness can be expected. Molecular markers can be used to study adaptability or fitness, provided that they represent a quantitative trait locus (QTL) or are themselves functional genes involved in these processes. Next to a genetic response of a population to environmental change, phenotypic plasticity in a genotype can affect fitness. The relative importance of plasticity to genetic diversity depends on the species and population under study and on the environmental conditions. The possibilities for application of current knowledge on genetic diversity and population survival for the management of natural populations are discussed.**

***Key words:*** **Adaptation, fitness, genetic diversity, life history characteristics, molecular markers, population history, population management, quantitative traits.**

## Introduction

### *Importance of diversity*

The importance of diversity in flora and fauna can be investigated at different taxonomical levels: a) the importance of species diversity for ecosystem functioning, b) the importance of genetic diversity to predict the vulnerability of a species to extinction, and c) the importance of genetic diversity for survival of populations within a species. The consequences of reduced species diversity on ecosystems is a current research topic. Several studies indicate that ecosystem processes are primarily dependent on differences in species composition and thus on functional diversity rather than on the species diversity (MacGillivray and Grime, 1995[163]; Tilman et al., 1997[255]; Wardle et al., 1997[267]; Hooper and Vitousek, 1997[121]). Grime (1997[100]) concluded from these studies that there is no convincing evidence that higher levels of biodiversity are crucial for ecosystem processes. However, he stressed that a point can be reached at which further loss of specific species will endanger the functioning of the ecosystem and the usefulness for the humans. Underlying the importance of species diversity for ecosystem functioning is the importance of genetic diversity at the population level, which is assumed to determine species survival and thereby species diversity. This is a matter of continuing debate. In this review, a survey of the importance at the population level is presented.

### *Outline of this review*

First, some background information concerning minimum viable population sizes, metapopulations, inbreeding and random genetic drift is presented, and this is followed by methods to measure genetic diversity in qualitative and quantitative traits. The influence of several life history characteristics (such as mating and reproduction system) and the population history (occurrence of bottlenecks and founder events) at the level of genetic diversity is discussed. The central question of the importance of genetic diversity for population survival is considered. The application of current knowledge for management of natural populations is presented, followed by a final discussion and future research needs.

### *Minimum viable population size*

A population must consist of a certain minimum number of individuals (the minimum viable population size, MVP) and needs a certain minimum area (minimum area requirement, MAR) in order to have a reasonable chance of survival for a certain period (Soulé, 1980[244]; Gilpin and Soulé, 1986[89]; Shaffer, 1987[235]; Menges, 1992[168]). Factors like demographic, environmental, and genetic stochasticity influence population dynamics and, thus potentially, extinction risks (Shaffer,

Plant biol. 2 (2000) 379 – 395

ISSN 1435-8603

1981[234], 1987[235]). The relative importance of the factors in determining extinction probabilities in natural populations is not clear (Lande, 1988[138]; Menges, 1992[168]; Alvarez-Buylla et al., 1996[9]). Demographic and genetic factors, however, cannot be considered as independent variables, as, e.g., effects of inbreeding can have a direct influence on birth and death rates and on fertility, and thus on the demography. Quantification of the relationship between genetic diversity and MVP is difficult as loss of genetic variation only indirectly leads to extinction (Nunney and Campbell, 1993[188]). However, recently it has been shown experimentally that inbreeding in natural butterfly populations can indeed result in extinction (Saccheri et al., 1998[226]).

The MVP depends on the ratio between effective ($N_e$) and actual (N) population size (Lande and Barrowclough, 1987[139]). At a given level of N, $N_e$ is negatively affected by unequal sex ratios, differences in reproductive capacity, close genetic relationships, an unbalanced age composition, and fluctuations in population size across generations (Falconer, 1981[73]; Hartl and Clark, 1997[109]). The ratio $N_e/N$ is generally between 0.25 and 1.0 (Nunney, 1993[187]), but can be drastically smaller (Briscoe et al., 1992[25]; Hedgecock, 1994[112]). Therefore, knowing the ratio between $N_e$ and N is of great importance, as large populations are not automatically protected from risks of genetic stochasticity. Certain factors, including overlapping generations (Nunney and Elam, 1994[189]) and population substructuring (Nei and Takahata, 1993[184]), may increase $N_e/N$ to levels higher than 1.0. It is in this way that conservation programmes seek to maintain genetic diversity despite small N.

### *Metapopulations*

A metapopulation consists of a series of local populations with a limited rate of gene flow and is characterized by more or less frequent local extinctions and recolonizations (Lande and Barrowclough, 1987[139]). A metapopulation structure generally has a positive effect on survival of the total population (Gilpin and Hanski, 1991[88]). Extinction of patches depends on genetic, demographic and environmental factors acting within those patches. Recolonization depends on the spatial structure of the metapopulation. Genetic differentiation between habitats can be very important for protection against extinction (Gilpin, 1987[87]; Nunney and Campbell, 1993[188]; Young et al., 1996[280]). However, in the case of a strong gene flow or frequent recolonization, the structure of the metapopulation will become relatively homogeneous and will not contain more variation than one large population. If local populations are very different in size and number of individuals, large patches with a relatively low genetic diversity can be a threat for the genetic diversity present in small patches. Another situtation in which a metapopulation structure has no surplus value for survival of the total population is if a major environmental factor (e.g., a severe winter) strikes all subpopulations at the same time.

### *Inbreeding, inbreeding depression, random genetic drift and selection*

Inbreeding is considered as the main genetic factor which threatens short-term survival of populations, while genetic drift is considered to be the main cause of the loss of genetic variation in the long term, thus threatening the adaptability of populations (Shaffer, 1987[235]). Inbreeding and genetic drift lead to homozygotation; loss of variation is caused by genetic drift or selection (Templeton and Read, 1994[254]). Population differentiation increases as populations may become fixed for other alleles or if selection pressures differ in the populations (Ellstrand and Elam, 1993[69]).

Two theories concerning inbreeding depression result in different predictions on the relative tolerance of populations to inbreeding (Charlesworth and Charlesworth, 1987[46]; Lacy, 1992[135]). In the mutation–selection balance theory, inbreeding depression is mainly the result of expression of deleterious recessive alleles (i.e., genetic load [Wallace, 1970[266]]). According to this theory, populations that have been small for a long period of time, may exhibit lower levels of inbreeding depression than large populations, due to purging of deleterious alleles. In the overdominance theory, inbreeding depression is due to a lack of superior heterozygotes. As a result, small populations may suffer greater inbreeding depression compared to large ones because of reduced effectiveness of selection relative to genetic drift. Although selfing plant species generally do suffer less from inbreeding depression than outcrossers (which is consistent with purging of deleterious alleles), theoretical studies (Holsinger, 1988[119]) and research in wild plant species show that the relation between inbreeding depression and the level of selfing varies considerably (Charlesworth and Charlesworth, 1987[46]; Charlesworth et al., 1990[48]; Holtsford and Ellstrand, 1990[120]; Barrett and Kohn, 1991[13]; Ågren and Schemske, 1993[3]; Charlesworth et al., 1994[47]). The degree of inbreeding depression may also depend on environmental factors (Barrett and Kohn, 1991[13]), and on the stage in the plant life cycle (Charlesworth and Charlesworth, 1987[46]; Barrett and Kohn, 1991[13]; Husband and Schemske, 1996[124]; Affre and Thompson, 1997[1]; Byers and Waller, 1999[41]). Inbreeding depression is probably the result of a combined effect of lethal recessive alleles, which can be purged from the population by inbreeding, and a possibly large number of other genes with small negative effects on fitness, which cannot be purged effectively, not even under extreme inbreeding conditions (Simmons and Crow, 1977[239]; Lande and Schemske, 1985[140]; Barrett and Kohn, 1991[13]; Husband and Schemske, 1996[124]; Byers and Waller, 1999[41]).

## Measuring Genetic Diversity

### *Variation in quantitative traits*

Quantitative traits play a central role in adaptive evolution (Lande and Barrowclough, 1987[139]; Milligan et al., 1994[170]; Lande and Shannon, 1996[141]). The phenotypic variation measured consists of a genetic component (i.e., additive genetic variance, which contributes to the selection response, and variance due to non-additive gene action such as dominance and epistasy), an environmental component and genotype–environment interactions. Information on the narrow sense heritability, being the ratio between additive genetic variance and total phenotypic variance, and on genetic correlations between traits is important to develop strategies for conservation of genetic variation underlying quantitative traits (Ritland, 1996[224]). Traditionally, the additive genetic variance is estimated by controlled crosses (Via and Lande, 1985[262]; Houle, 1992[122]). The necessity of large experimental designs in order to draw statistically reliable conclusions, and of cross-

ing experiments often preclude solid quantitative genetic analyses in threatened species, and explains why these techniques are only very rarely used in studies on genetic diversity for conservation purposes (Schemske et al., 1994[231]). However, crossing experiments can now be circumvented by analyzing quantitative inheritance in natural populations based on marker-estimated relatedness between individuals (Ritland, 1996[224]).

*Variation in isozyme and DNA markers*

For several decades, numerous population genetic studies used isozymes, and therefore the genetic basis of isozyme patterns is well established in many species. With the development of PCR for direct amplification of DNA fragments, the use of DNA markers in population studies has increased tremendously. Of all possibilities offered by molecular genetics, the sequence tagged microsatellites (STMS) and AFLP markers are most widely used. With the co-dominant STMS marker system, generally more polymorphic loci and more alleles per locus can be analyzed compared to isozymes. However, the development of microsatellite markers for any new species requires a large effort. It is often not known if variants are indeed different alleles of the same locus or alleles of different loci. In many studies one assumes the first situation without an actual check using segregation studies. Using dominant DNA markers (such as AFLPs) in classic population genetic models is a point of discussion (Travis et al., 1996[256]). These factors make the interpretation of results on the basis of DNA markers in population genetic research generally more difficult than when using isozymes.

Several measures can be used for genetic characterization of a population, see for some reviews Peet (1974[208]), Brown and Weir (1983[30]), Weir (1989[271]), Templeton (1994[253]), Hamrick and Godt (1996[107]), and González-Candelas and Palacios (1997[93]). The expected heterozygosity, $H_{ep}$, in a population represents the chance that two copies of a locus which are sampled at random from a gene pool have different allelic states (Templeton, 1994[253]). It is biologically the most meaningful measure of genetic diversity when consequences of mating systems are investigated (Templeton and Read, 1994[254]). As a population differentiation measure, $G_{ST}$, which represents that part of the total genetic diversity found among populations, is generally used (Nei, 1973[182]). $G_{ST}$ is equivalent to the $F_{ST}$ defined by Wright (1965[277]), which represents the ratio between the additive genetic variance among populations and the total additive genetic variance.

*Relationship between variation in quantitative traits and molecular variation*

Schaal et al. (1991[228]) suppose that isozyme variation generally reflects the level of total genomic variation. Hedrick et al. (1986[114]) and Storfer (1996[246]) argue that variation in quantitative traits, because they are generally polygenic, may be better correlated with the total genetic variation than variation in single locus DNA markers. It may be more appropriate to compare variation in a quantitative trait, which is often based on a limited number of major genes and many minor genes, with variation based on a combination of single locus DNA markers used in population studies or on multi-locus DNA markers, such as AFLPs. Although a matter of discussion, Lande and Barrowclough (1987[139]) and Lande and Shannon (1996[141]) believe that most phenotypic changes in evolution are the result of the accumulation of quantitative polygenic modifications of existing phenotypes rather than single gene modifications with major effects. As it is generally not known whether or not the DNA markers used thus far for measuring genetic diversity are located in coding regions, or linked to major genes, Karp et al. (1997[128]) emphasize the importance of gaining information about their selective neutrality. In this context, finding a relationship between quantitative variation and molecular markers also highly depends on the kind of quantitative characters analyzed, i.e., whether or not they are expected to be fitness-related and thus exposed to selection.

The number of publications describing empirical research on the relation between single locus isozyme or DNA markers and quantitatively inherited variation is very limited, and the results are variable (Kobylianski and Livshits, 1983[131]; Zink et al., 1985[281]; Strauss, 1991[247]; Yezerinac et al., 1992[279], and others). Interpretation of these results is difficult because the genetic component of the phenotypic variation has been determined in only a few studies (e.g., Briscoe et al., 1992[25]).

Molecular population genetics generate methods for studying variation in (major) loci coding for quantitative traits. One approach is to find markers that are linked to those loci using a linkage map and QTL (i.e., quantitative trait loci) mapping. An alternative is to directly estimate variation in the genes involved. In the latter approach, functional diversity (i.e., variation in candidate genes which are expected to play a role in the fitness) is measured using molecular techniques. For conservation genetics, it would be very interesting if molecular variation could be linked to adaptive quantitative genetic variation using efficient screening methods. However, defining which traits are important is difficult, since they have to comprise not only traits which are decisive for reproduction and survival at the present time, but also those which may be exposed to selection in future. From the point of view of climatic and environmental changes, traits, such as drought, heat and metal tolerance, will play an important role in fitness in future and thus in survival of populations and species (e.g., Blum, 1996[21]; Linhart and Grant, 1996[158]; Monneveux and Belhassen, 1996[177]; Maywald and Weigel, 1997[167]; Winicov, 1998[276]). Genetic variation in genes which are involved in signal transduction in plants and animals may also be useful, as variation in such genes may reflect the ability of an organism to respond to changes in the environment.

## Mating and Reproduction System, Population History and the Level of Genetic Diversity

*Effects of mating and reproduction system on genetic diversity and population structure*

Quantitative estimates of mating system parameters in plant populations are necessary to explain the genetic structure of these populations and to predict evolutionary processes. The mating system of a plant species can vary in space and time (e.g., Rick, 1983[223]; Brown, 1990[28]). The fact that sexual reproduction in plants remains the major reproduction system, despite several possibilities for clonal reproduction, plays an important role in the discussion about the evolutionary importance of sexual reproduction in relation to adaptive capacity

(Hamilton, 1980[104], 1982[105]; Rice, 1983[222]; Case and Taper, 1986[44]; Ellstrand and Roose, 1987[70]).

In contrast to assumptions of most theoretical models, mating in cross-fertilizing plant species is generally not random (Levin and Kerster, 1974[157]; Levin, 1981[155], 1983[156]; Willson, 1983[273], 1984[274]). Also in animal populations, the mating pattern often deviates from random mating (Bateson, 1983[15]; Harvey and Ralls, 1986[110]; Gharrett and Smoker, 1993 a[83], 1993 b[84]; but see also Greenwood et al., 1978[98]; Van Noordwijk et al., 1985[258]). Animals can realize an optimal level of outbreeding by several strategies (Bateson, 1983[15]; Partridge, 1983[207]; Harvey and Ralls, 1986[110]). Variation in mating patterns has consequences for the genetic diversity within populations (Hamrick and Godt, 1990[106]; Schneller and Holderegger, 1996[233]; Dietl and Langhammer, 1997[64], and others). Especially in plants, deviation from random mating may produce a local genetic substructure in a population, enabling local differentiation within populations in response to selection and random drift (Willson, 1984[274]). The level of variation within mainly clonal plant and animal species can be as high as within sexually reproducing species (Parker, 1979 b[203]; Templeton, 1982[252]; Hamrick and Godt, 1990[106]). The level of clonal diversity can differ among populations (e.g., Ellstrand and Roose, 1987[70]; Godt and Hamrick, 1999[92]). Most clones are restricted to one or a few populations and widely distributed clones are rare (Parker, 1979 a[202]; Ellstrand and Roose, 1987[70]; Grashof-Bokdam et al., 1998[97]). The population genetic structure of such species is generally very complex. Clonality contributes to the maintenance of genetic diversity by extension of the generation time. The establishment of seedlings is important for the maintenance of genetic diversity, while the survival of adapted clones and the ability to rapidly colonize favourite habitats is ensured by vegetative reproduction (Watkinson and Powell, 1993[269]; Heimann and Cussans, 1996[115]; Arens et al., 1998[11]). Studies to find if there is a connection between the observed distribution of clones within populations and fitness components would give insight into which processes determine the number and relative size of different clones in mainly clonal plant species.

Selfing plant species have a lower total diversity with a higher degree of differentiation between populations than outcrossers (Hamrick and Godt, 1990[106]). Such species consist of populations with high as well as low diversity levels (Brown and Schoen, 1992[29]). The lower levels of genetic diversity in self-fertilizing plant species can be explained in several ways (Hamrick and Nason, 1996[108]). Many are annuals or short-lived perennials, whose populations may be subject to large fluctuations in size and thus are sensitive to extinction. Furthermore, it is unlikely that a new allele from a population will be introduced into another population, resulting in a loss of this allele when the original population goes extinct. New alleles will become present in homozygous form and therefore be exposed to selection, while new alleles in outcrossers can be introduced in other populations by gene flow and can remain present in heterozygous form.

### *Effects of population history on genetic diversity*

Severe bottlenecks and founder events in the past can have a large effect on the current level of genetic diversity (e.g., O'Brien, 1994[190]; Gallardo and Köhler, 1994[79]; Gallardo et al., 1995[80]; Kretzmann et al., 1997[134]; Sun, 1997[251]). Outcrossers which were once widely distributed and which possessed a high level of genetic diversity will be most sensitive to a reduction in genetic variation as a result of a restriction in their distribution or population size (Schaal et al., 1991[228]). An extreme reduction in population size will result in more matings between related individuals, and these populations will suffer from inbreeding depression (Charlesworth and Charlesworth, 1987[46]; Hedrick, 1987[113]; O'Brien et al., 1987[192]; Packer et al., 1991[199]; O'Brien, 1994[190]; Bryant et al., 1999[33]). The effect of population history on genetic diversity is clearly shown in studies of genetic variation in beavers (*Castor fiber;* Ellegren et al., 1993[68]) and northern elephant seals (*Mirounga angustirostris;* Bonnel and Selander, 1974[22]; Le Boeuf, 1974[142]; Hoelzel et al., 1993[116]; Halley and Hoelzel, 1996[103]), where, in spite of a considerable current population size, the genetic diversity is very low due to severe bottlenecks in the past. When a population remains small for a longer period after a genetic bottleneck, further shifts in allele frequencies and loss of alleles will take place by random genetic drift. Reduction in mean heterozygosity per locus depends on the size of the bottleneck, as well as on subsequent population growth rate (Nei et al., 1975[183]; Chesser and Ryman, 1986[49]), but is rather small when population size increases rapidly after a bottleneck, even with a low number of founders. In contrast, the mean number of alleles per locus is mainly influenced by the bottleneck itself and much less by the population growth rate after the bottleneck. This difference is mainly due to the fact that many rare alleles are lost by genetic drift (Nei et al., 1975[183]). Bottleneck experiments showed that strong phenotypic differentiation between bottleneck lines can occur (Brakefield and Saccheri, 1994[24]), depending on the size of the founder population (Bryant and Meffert, 1990[35]).

The mating system plays an important role in bottleneck effects (e.g., Godt and Hamrick, 1996[91]). A strong reduction in population size in mainly self-fertilizing species may not have an influence on heterozygosity, but can cause a major loss of allelic variation (Barrett and Kohn, 1991[13]). Furthermore, loss of variation is reduced in autopolyploids with polysomic inheritance (Haldane, 1930[102]; Mayo, 1971[166]) and in allopolyploids with high levels of fixed heterozygosity (Barrett and Kohn, 1991[13]). Despite well-developed theoretical models to predict the effects of bottlenecks on genetic diversity, empirical research, especially in wild populations, is very limited, caused by lack of knowledge of the evolutionary history of most plant species.

Species which show low levels of isozyme variation due to bottlenecks or founder events do not necessarily have little variation at other loci (Moran et al., 1981[178]; Ager et al., 1983[2] [but see also Mosseler, 1995[179]]; Giles, 1983[85]; Overton and Johnson, 1983[198]; Warwick et al., 1987[268]; Polans and Alard, 1989[211]; Cheverud et al., 1994[50]), as the level of genetic variation found in a population not only depends on the effective population size but also on the type of genetic variation and on natural selection acting on this variation (Lande, 1976[136], 1980[137]; Kimura, 1983[130]; Hedrick et al., 1986[114]; Lande and Barrowclough, 1987[139]; Lynch, 1988[160]). Selection can affect the genomic anatomy, ranging from small mutations with no obvious adaptive use to linkage arrangements of genes (Hurst, 1999[123]). When a population has lost much of its genetic variation during a prolonged bottleneck or due to several bottle-

necks, genetic variation in quantitative traits is believed to be restored by mutation on a time scale of hundreds to thousands of generations (Lande and Barrowclough, 1987[139]). For single locus variation the time to restore a high level of heterozygosity is much longer, up to $10^7$ generations for variation on single loci which are selectively neutral (Nei et al., 1975[183]). This difference can partly be due to difficulties in detecting these mutations in single alleles. Some parts of the genome can have higher mutation rates. Microsatellite loci have been estimated to have mutation rates of about $10^{-4}$ per generation (Weber and Wong, 1993[270]). Bottlenecks can result in increased phenotypic variation in the case of non-additive gene effects (Bryant et al., 1986[34]; Goodnight, 1987[94], 1988[95]; Willis and Orr, 1993[272]; Fowler and Whitlock, 1999[75]), producing a discrepancy with the level of heterozygosity in neutral molecular markers which decreases linearly with the inbreeding coefficient (Lynch, 1996[161]).

## The Importance of Genetic Diversity for Population Survival

### *Relationship between genetic diversity and fitness*

Estimation of individual fitness is a complex matter because it is composed of many polygenic traits. Often, it is based on measurements of several fitness components directly or by analysis of quantitative trait loci (QTLs) in which genes affecting fitness-related traits are mapped. Using QTLs, the genetic basis of fitness-related traits can be unravelled (e.g., Fry et al., 1998[78]; Alonso-Blanco et al., 1999[8]; Shook and Johnson, 1999[237]). Traditionally, for QTL mapping in natural populations, crosses have to be made between individuals which segregate for the traits of interest. Recently, it has proved to be possible to detect QTLs in natural populations, provided detailed life history data and good pedigree information are available (Slate et al., 1999[240]). In plants, fitness components used for fitness estimations include percentage seed germination, seed germination rate, growth rate, time to reproductive stage, number of flowers or inflorescences, pollen germination and pollen tube growth, number of fruits, fruit weight, number of seeds, seed weight and survival; components in animals are growth rate, time to fecundity, sperm amount and quality, and survival. In addition, the presence of resistance traits also has important fitness consequences in the case of a disease outbreak. Brown et al. (1993[31]) defines fitness in terms of energy, namely as the degree to which resources, next to those necessary for growth and maintenance, can be exploited from the environment and used for reproduction. If the mating system is known and if mutation, migration and genetic drift can be assumed to have neglible effects on genotype frequencies, the fitness of a genotype can be derived, indirectly, from the genotype frequencies at different stages of the life cycle, or at the same stage in subsequent generations (Allard et al., 1966[5]; Prout, 1969[213]; Clegg et al., 1978[57]). However, genetic models for estimations of genotype fitness do not fully explain the origin of differences in fitness between genotypes as they are based on a direct link between phenotype and its reproductive success, while the individual fitness is not only determined by the genotype or phenotype but also by an interaction between the genotype and its environment (see "The importance of genotype–environment interaction with regard to genetic diversity"), making fitness estimations and determination of genetic variation for fitness-related traits in natural populations even more complex.

Developmental stability represents the ability of an organism to compensate for small disturbances during its development, resulting in the production of a (more) genetically predestined phenotype (Lerner, 1954[153]; Møller and Swaddle, 1997[176]). Determination of the developmental stability also constitutes an indirect estimation of individual fitness (e.g., Leary et al., 1984[147]; Palmer and Strobeck, 1986[200]; Jones, 1987[127]). Moreover, changes in developmental stability can probably predict changes in fitness, assuming that changes in developmental stability are visible in the phenotype before a detectable change in fitness components occurs (Clarke, 1995[53]). Generally, developmental stability in animals is estimated by determination of fluctuating asymmetry, which represents non-directional differences between the left and right sides for bilateral characters. Fluctuating asymmetry results from the inability of the organism to develop according to some ideal developmental programme due to environmentally-induced random disturbances (Van Valen, 1962[260]). Compared to animals, the number of studies in plants on fluctuating asymmetry in relation to stress is very limited and is mainly restricted to recent years. The extent to which fluctuating asymmetry reflects developmental stability and its ability to predict fitness changes depends on the trait considered. Studies in a large number of taxa showed that fluctuating asymmetry can be an indicator for environmental as well as for "genetic stress" (as a result of an increase in homozygosity in a normally variable population) in animals (reviews from Leary and Allendorf, 1989[145]; Parsons, 1990[204]; Møller, 1997[174]) and in plants (Sherry and Lord, 1996[236]; Møller, 1997[174], 1998[175]; Midgley et al., 1998[169]; Wilsey et al., 1998[275]). Relations between developmental stability, fitness and stress have been found in several studies (review Clarke, 1995[53]). Loss of genetic variability in a normally variable population generally co-occurs with a decrease in stability (i.e., an increase in fluctuating asymmetry and phenotypic variance; Lerner, 1954[153]; Leary et al., 1983[146], 1984[147], 1985[148]; Quattro and Vrijenhoek, 1989[217]; Vrijenhoek, 1996[265]; but see also Beacham, 1991[16]; Britten, 1996[26]; Brookes et al., 1997[27]). Fluctuating asymmetry and phenotypic variance also increase with inbreeding (Biémont, 1983[20]; Clarke et al., 1986[54]; Leary and Allendorf, 1989[145]; Parsons, 1990[204]; Clarke, 1992[51]; Leamy, 1984[143], 1992[144]). Clarke (1995[53]) mentions some limitations for this indirect estimation of fitness: the traits under investigation must undergo development during exposure to stress, problems can arise in organisms with a long generation time or if the stress exposure is short or restricted to mature individuals, developmental stability not always appears to be correlated with fitness parameters (e.g., Graham, 1992[96]; Evans and Marshall, 1996[72]), and stress does not always result in changes in developmental stability.

According to Clarke (1993[52]), genomic co-adaptation also plays an important role in the genetic basis of developmental stability in natural populations. The balance between the importance of level of heterozygosity and co-adaptation probably depends on the mating system and the genetic structure of the system under study. Genomic co-adaptation refers to the selection process by which harmonious cooperating genes are accumulated in the gene pool of a population (Dobzhansky, 1951[67]). Disturbance of co-adapted gene complexes results

in lower developmental stability (Graham, 1992[96]; Clarke and McKenzie, 1987[55]; 1992[56]). One of the major problems in studying the genetic basis of developmental stability is the practical inability to manipulate the level of heterozygosity or the gene balance without simultaneously affecting the other; this is only possible in a very few model systems (Clarke, 1992[51]).

A large number of studies in natural populations show a relation between isozyme heterozygosity and various fitness components, among which are viability, growth rate, developmental stability and physiological variables like oxygen consumption (reviewed in Mitton and Grant, 1984[173]; Allendorf and Leary, 1986[6]; Mitton, 1994[172]). Especially in ecologically unstable environments, a higher level of heterozygosity corresponded to higher individual fitness (reviewed in Nevo, 1986[185]; Müller-Starck and Hattemer, 1989[181]; Prus-Glowacki, 1991[214]; Oleksyn et al., 1994[195]; Müller-Starck, 1995[180]; reviewed in Parsons, 1996 a[205], 1996 b[206]; Rankevich et al., 1996[220]; Nevo et al., 1997[186]). Ledig (1992[150]) concluded that heterozygosity can thus be promoted as a result of stress, but that this is also to be expected if homozygosity only represents inbreeding, resulting in a lower fitness of the homozygotes which will be even more disadvantaged under stress conditions. A large number of studies have failed to find a relation between isozyme heterozygosity and fitness. A problem with several studies on heterozygosity and fitness is that they were carried out at the population level without checking whether heterozygotic individuals were actually favoured. Meta-analyses of published correlation coefficients between multilocus heterozygosity and the two fitness parameters, growth rate and fluctuating asymmetry, carried out by Britten (1996[26]), showed on the whole a significant but generally weak positive correlation. The overall significance resulted from only a few major studies, mainly on bivalve molluscs and pine trees. The phenomenon is difficult to detect and interpret as the correlation, when found, typically accounts for a small proportion of the observed variance, necessitating large sample sizes (in the range of thousands) in order to distinguish signal from noise (David, 1998[60]).

Which mechanisms underlie the fitness increase of highly heterozygote individuals is the subject of discussion (Mitton and Grant, 1984[173]; Allendorf and Leary, 1986[6]; Ledig, 1986[149]; Strauss, 1986[248]; Clarke, 1993[52]; Smouse, 1986[242]; Zouros and Pogson, 1994[283]; David, 1997[59], 1998[60]; Deng and Fin, 1998[63]; Pamilo and Pálsson, 1998[201]). Three main hypotheses are put forward: 1) true or functional overdominance, 2) associative overdominance and 3) inbreeding or dominance. Other mechanisms which could be responsible are 4) balanced enzyme pathways, 5) null alleles or 6) chromosomal loss (Zouros and Mallet, 1989[282]).

In the case of true or functional overdominance, the correlation between individual isozyme heterozygosity and fitness-related traits is the result of intrinsic functional differences between enzyme variants at the loci scored (heterozygote superiority, Lerner, 1954[153]). This would mean that isozyme markers are far from "neutral" with respect to fitness. In the case of associative overdominance, positive correlations result from heterozygosity at loci which are in linkage disequilibrium with the studied loci (Ohta, 1971[194]). In contrast to true overdominance, the correlation with associative overdominance is not specific for the type of marker used. As a further difference, under true overdominance, background fitness is mere noise and heterozygosity–fitness correlations should appear stronger in homogeneous backgrounds, while effects under associative overdominance rely on variation in genetic background (David, 1998[60]). According to this, differences in the heterozygosity–fitness correlations between populations point to associative overdominance rather than to true overdominance. The inbreeding hypothesis assumes that isozyme loci are selectively neutral and serve only as markers for the overall level of genomic heterozygosity which covers deleterious recessives (Kimura, 1983[130]). In contrast to the (true or associative) overdominance model, the inbreeding model predicts no locus-specific effects, a negative relationship between heterozygosity and variance in fitness, and an increasingly low fit of the adaptive distance model (Smouse, 1986[242]) with an increasing number of loci (David, 1997[59]; David et al., 1997[61]). (According to Smouse [1986[242]], the adaptive distance of a heterozygote is zero, and that of a homozygote is the inverse of the frequency of its allele, so that the fitness of the more common homozygote is greater than that of a rarer one.) By comparing RFLP and isozyme heterozygosity with growth rate in the scallop *Placopecten magellanicus,* Pogson and Zouros (1994[210]) only found a positive correlation with isozyme markers, which contradicts the associative overdominance hypothesis. David (1998[60]), however, critisized the way that they came to this conclusion, as they considered the enzyme and RFLP loci as a group in comparing heterozygosity–fitness correlations, while a large locus-specific effect was associated with an RFLP locus. Furthermore, their conclusions were not statistically founded, and differences could be the result of sampling variance. Population-dependent heterozygosity–fitness correlations were found in the Atlantic cod *Gadus morhua* (Pogson and Fevolden, 1998[209]) and in *Salix eriocephala* (Aravanopoulos and Zsuffa, 1998[10]), indicating the action of the associative overdominance mechanism. Simulation studies from Pamilo and Pálsson (1998[201]) showed that large associative overdominance is expected in small populations. They concluded that most examples of positive heterozygosity–fitness correlations have been found in species that either have a heterozygote deficiency (a putative sign of inbreeding; see also Charlesworth, 1991[45]) or species that live in small and structured populations. In large, random mating populations linkage disequilibria are zero, and heterozygosity–fitness correlations are reduced to their direct contribution to the phenotype (Smouse, 1986[242]). Evidence for the inbreeding model was found, e.g., in the rare plant species *Gentiana pneumonanthe* (Oostermeijer et al., 1995[196]) and the marine bivalve *Spisula ovalis* (David et al., 1997[61]).

The role of genetic diversity in natural populations in relation to disease outbreak and fitness or survival chance of the host is important. Pathogens can play an important role in determining plant community structure and, thereby, affect the functioning of ecosystems (Dobson and Crawley, 1994[66]). The effects which parasites and pathogens can cause are more difficult to predict in natural populations than under cultivation. The main difference with agricultural systems is the scale in time and space in which the disease occurs in natural populations – the disease is characterized by an extreme patchiness (Burdon and Jarosz, 1990[39]). Preservation of genetic diversity for disease resistance genes can be of great importance for population survival (O'Brien and Evermann, 1988[191]; Foster

Hünneke, 1991[74]). This particularly appeared to be the case in studies on diversity in the major histocompatibility complex (MHC) in natural animal populations (O'Brien et al., 1985[193]; O'Brien, 1994[190]). Parasite-mediated selection against inbreeding was found in a natural population of Soay sheep in Scotland, thereby acting to maintain genetic variation in the host (Coltman et al., 1999[58]). The number of examples in natural plant populations is limited. Severe epidemics can arise when newly established host–pathogen combinations occur (Hamrick and Godt, 1996[107]) or after other drastic disturbances in the ecosystem (Dinus, 1974[65]), but also in less disturbed ecosystems (Browning, 1974[32]). Significant variation for resistance against specific pathogens has been found within and between natural plant populations (Burdon, 1985[37], 1987[38]). Reciprocal host–pathogen interactions result in diversification in host and pathogen until a certain balance is reached (Burdon and Jarosz, 1990[39]). Recent genetic studies, however, suggest that co-evolution between the plant and one of its enemies generally is diffuse, i.e., it depends on the presence of other enemies by which the pattern of selection can be altered (Rausher, 1996[221]). The frequency distribution in the host population of plants differing in resistance level depends on several factors, among which the history of disease pressure, environmental conditions, the genetic basis of resistance in the host, the resistance costs in relation to fitness, and the mating system of the host (Jayakar, 1970[126]; Leonard, 1977[150]; Leonard and Czochor, 1980[152]; Burdon, 1987[38]; Rausher, 1996[221]) are important. Long-term effects of the disease on the frequency of resistant and susceptible individuals are largely unknown. An important problem in the interpretation of long-term changes in the frequency of resistant individuals is to establish whether such patterns reflect changes in selection for resistance itself or in selection for associated traits which show a selection response due to linkage with resistance genes (Burdon, 1987[38]).

In summary, it is clear that, despite the availability of many direct and indirect fitness parameters, an accurate estimation of individual fitness is complex. From numerous studies it can be concluded that there is no general relationship between genetic diversity (generally measured as isozyme heterozygosity) and various fitness components. However, if a lower level of heterozygosity represents an increased level of inbreeding, a reduction of fitness can be expected.

*Relationship between genetic diversity and adaptability*

Adaptation implies the genetic or phenotypic response of a population or an individual to an environmental change in order to increase or maintain fitness. In this section, only the genetic response of a population will be considered, the phenotypic response (plasticity) will be discussed in "The importance of genotype-environment interaction with regard to genetic diversity". Geographic variation of genetic diversity between populations results from a balance between factors causing local genetic variation, such as mutation, genetic drift and adaptation due to natural selection on the one hand, and factors causing genetic homogeneity, such as gene flow on the other hand (Slatkin, 1987[241]). A prerequisite for adaptive differentiation between populations is the presence of genetic variation within a local population. This, however, implies the presence of suboptimally adapted genotypes, i.e., a genetic load. In the case of adaptive differentiation, gene flow can result in outbreeding depression, i.e., lower fitness as a result of hybridization due to a disturbance of co-adapted gene complexes (Carvalho, 1993[43]; Ellstrand and Elam, 1993[69]). Extensive gene flow can even prevent local adaptation, particularly of small populations. However, limited gene flow and small population size, as often found with marginal populations, can cause genetic drift resulting in genetic differentiation, possibly in combination with local adaptation (Van Rossum et al., 1997[259]).

The ability of a population to adapt to unpredictable environments is the basis for stability of each ecosystem. The fitness of a population is generally influenced by only a small percentage of the variation present within the population after exposure to a new stress factor (Parsons, 1996 a[205], 1996 b[206]). The adaptability of each individual to different environmental conditions is restricted by the fact that its genetic variation cannot be unlimited (Gregorius, 1989[99]). This means that a population can only achieve its adaptability by distribution of the variation across individuals (e.g., Den Boer, et al., 1993[62]). Besides that, heterozygosity can be higher in stressful environments (review Parsons, 1996 a[205], 1996 b[206]; Rankevich et al., 1996[220]; Nevo et al., 1997[186]; Prus-Glowacki et al., 1999[216]), the relation between heterozygosity and stress tolerance can be variable (e.g., Kopp et al., 1994[133]), and it is also possible that – in the case of functional importance of an isozyme genotype with regard to a stress factor or in the case of linkage with genes involved in stress tolerance – selection of resistant genotypes results in an increase in the frequency of specific adaptive alleles while the total heterozygosity for that isozyme locus decreases (Hattemer and Müller-Starck, 1989[111]; Prus-Glowacki and Godzik, 1991[215]; Kopp et al., 1992[132]) because alleles which are only present in susceptible genotypes are lost. On the basis of these results, Guttman (1994[101]) and Fox (1995[76]) concluded that the formation of a resistant but genetically uniform population can imply increased susceptibility for new disturbances in the environment. Hoffmann and Merilä (1999[117]) compared several hypotheses on the effect of unfavourable conditions on genetic variation of a trait, some of them predicting an increase of heritable variation, others a decrease or unpredictable effects. More case studies are necessary to determine whether there may be some general trends under particular conditions or whether the effects of unfavourable conditions may be diverse.

Quantitative traits are very important in adaptive evolution (e.g., Milligan et al., 1994[170]; Lande and Shannon, 1996[141]), but the importance of genetic variation in quantitative traits in relation to population survival and adaptation has not yet been evaluated. Lande (1988[138]) and Schemske et al. (1994[231]) posit that, in the short term, genetic variation generally is less important than other factors for population survival but, in the long term, genetic variation can play a crucial role because it enables a population to survive and to adapt in a changing environment (e.g., Lande and Shannon, 1996[141]). Molecular markers are often assumed to be useful in predicting the amount of quantitative inheritable variation. However, the amount of variation in quantitative traits and in marker loci can be very different (Milligan et al., 1994[170]). For this reason, the level of variation detected on marker loci will not necessarily be a direct reflection of the level of variation which determines the adaptability or individual fitness. These two types of variation differ in evolutionary dynamics and will re-

spond differently to fluctuations in population size. Furthermore, several studies have shown that genetic diversity, in terms of heterozygosity, and quantitatively inherited fitness traits are not always correlated (see "Relationship between genetic diversity and fitness"). The level of genetic variation in selectively neutral marker loci is mainly determined by mutation and genetic drift (Kimura, 1983[130]). The level of genetic variation in quantitative traits depends on a balance between mutation and selection, or between different selection pressures (Barton and Turelli, 1989[14]; Roff, 1998[225]). One could expect that selection would erode variation in fitness-related quantitative traits, resulting in little heritable variation for such traits in populations at equilibrium. However, genetic variation in fitness traits appears to be common in natural populations due to environmental heterogeneity (also see "The Importance of genotype–environment interaction with regard to genetic diversity"). Hurst (1999[123]) states that the effect of selection depends on the recombination rate, the population size and the number of excess progeny. He concluded from recent case studies that selection can affect minuscule genomic changes, such as point mutations (even when resulting in the same amino acid) to broader scale genomic patterns, such as linkage and chromosomal location. In the case of diversification by selection, the balance between selection and migration can result in considerable differences between populations. With migration being the same for all genes, selection differs in different parts of the genome. Therefore, neutral loci do not necessarily predict patterns of variation in traits which are subject to differential selection (Brown and Schoen, 1992[29]; Gharrett, 1994[82]; Karhu et al., 1996[129]; Yang et al., 1996[278]). Hence, molecular markers will provide little insight on loci which underlie adaptive variation unless a strong linkage exists between marker loci and loci coding for relevant quantitative traits (QTLs), or if markers are found for structural genes that underlie adaptive characters and show a high level of differentiation, i.e., molecular markers which directly reflect functional diversity (Karhu et al., 1996[129]; Lynch, 1996[161]). Such markers have started to attract more attention.

The adaptation process not only occurs at the population scale but also within populations where habitat differentiation can be found. The niche–variation hypothesis suggests that genetic diversity within local populations can be preserved if the genotypes differ in relative fitness in different microhabitats or niches (Van Valen, 1965[261]). The high frequency of genotype diversity in clonal plant populations, for instance, is often explained as a result of heterogeneity on a microscale, by which clones can coexist due to diversifying selection (i.e., the selection of different genotypes under different environmental conditions; Solbrig and Simpson, 1974[243]; Burdon, 1980[36], and others). Although in several studies no correlation has been found between habitat characteristics on the microscale and distribution of individual genotypes (e.g., Steiner and Levin, 1977[245]; Silander, 1979[238]), local genetic differentiation has been reported for a large number of plant species (e.g., Prentice et al., 1995[212]; Linhart and Grant, 1996[158]; Lönn et al., 1996[159]).

In conclusion, genetic variation is essential for the adaptability of a population in which quantitatively inherited, fitness-related traits play a central role. Molecular markers can be used to study adaptability or individual fitness provided that they represent a QTL or are themselves genes involved in adaptive responses.

### *The importance of genotype-environment interaction with regard to genetic diversity*

Phenotypic plasticity stands for the extent to which environmental variation can modify the expression of a genotype on the phenotypic level (Gause, 1947[81]; Bradshaw, 1965[23]). Using phenotypic plasticity, plants can respond, within certain limits, to changes in their environment without genetic changes. This phenomenon is very widespread in plants and often is most pronounced under stressful circumstances. Plasticity is likely the result of differences in allelic expression, i.e., differential expression of alleles of a certain gene in different environments (Via and Lande, 1985[262], 1987[263]), as well as of changes in interactions between loci in different environments (Lynch et al., 1988[162]; Scheiner and Lyman, 1989[230]). Plasticity is not a function of heterozygosity (Scheiner, 1993[239]). Genetic variation in phenotypic plasticity, i.e., variation in the magnitude of the response to the environment, is also known as genotype–environment interaction (Falconer, 1981[73]). Studies on the genetic analysis of fitness-related traits by determination of heritabilities (e.g., Bennington and McGraw, 1996[19]; Campbell, 1997[42]; Qvarnström, 1999[218]) or by QTL mapping (e.g. Mitchell-Olds, 1996[171]; Fry et al., 1998[78]; Shook and Johnson, 1999[237]) often show strong interactions. The genotype–environment interaction can be antagonistic pleiotrophic, in which selection favours alternative alleles in different environments (Gillespie and Turelli, 1989[86]), or conditional neutral, in which alleles can affect fitness in some environments but not in others (Fry et al., 1998[78]). The fact that genotypes do not have a consistent relative fitness in all environments is a possible explanation for the preservation of polygenic variation in natural populations (Gillespie and Turelli, 1989[86]). However, the number of studies on comparison of phenotype response curves with regard to fitness of genotypes over a whole range of relevant environmental variables is limited (Sultan and Bazzaz, 1993[250]; Bell and Sultan, 1999[18]). As the expression of genes affecting fitness-related traits generally strongly depends on the envrionment, results from genetic analyses of laboratory or field experiments cannot be extended to natural populations. For understanding the role of major genes in natural populations, wild populations in undisturbed environments have to be analyzed (Arnold, 1994[12]; Mitchell-Olds, 1996[171]). Ennos (1990[71]) proposed study of ecological systems instead of specific species in which genetic analyses within specific classes of ecological interactions may be used to determine in which way variation in ecologically important traits affects the viability or reproductive success of individuals. By this approach, insight can be obtained into the way in which genetic differences are translated into fitness differences in specific ecological situations.

Sarukhán et al. (1984[227]) and Sultan (1987[249]) underline the importance of environmental factors with respect to phenotypic variation within natural plant populations. According to them, differences in growth between individual plants are a reflection of environmental factors rather than genetic factors. However, there are cases known with genetic differentiation between habitats despite considerable gene flow, indicating the possibility of strong selection for stress resistance within

natural populations (e.g., Prentice et al., 1995[212]; Lönn et al., 1996[159]). Differences among genotypes in susceptibility to environmental changes can be the result of genetic variation with regard to plasticity of a stress response, as well as of genetic variation with regard to the response itself (Hoffmann and Parsons, 1991[118]). No unequivocal relation exists between individual plasticity and genetic variation of a population or a taxon (Schlichting, 1986[232]). It is not clear to what extent phenotypic plasticity can fully compensate for low levels of genetic variability in heterogeneous environments. On the other hand, genetic variation can contribute substantially to the success of a population if phenotypic plasticity within a species is absent or in the case of a large environmental variation (Foster Hünneke, 1991[74]). Several factors influence the level at which phenotypic plasticity and genetic variation contribute to the phenotypic variation within and between populations. These include stress frequency, stress duration, extent of local heterogeneity in stress levels, costs of plasticity and restrictions of plastic responses. Furthermore, genetic changes will occur if an individual genotype does not have the highest fitness under all environmental conditions, resulting in the selection of different genotypes under different circumstances (Hoffmann and Parsons, 1991[118]). Adaptation in colonizing plant species to the different and variable environments in which they have settled, can take place via phenotypic plasticity or via co-occurrence of different genotypes within a population which are each adapted to a slightly different environment (Allard and Bradshaw, 1964[4]; Bradshaw, 1965[23]; Jain, 1976[125]), with phenotypic plasticity being the main strategy for the colonizing species *Xanthium strumarium* (Moran et al., 1981[178]). Provenance tests in Scots pine *(Pinus sylvestris)* showed an extremely high level of phenotypic plasticity as populations originating from very different environments were able to grow and even to compete with local populations (Mátyás, 1989[164]). As phenotypically stable populations are expected to have a larger ability to adapt to environmental fluctuations, Mátyás (1996[165]) stresses the importance of gaining knowledge of genetic mechanisms underlying high levels of plasticity.

Natural selection affects the level of phenotypic plasticity by attuning this to the level of environmental variability to which a population is subjected. Generally, plants from favourable environmental conditions tend to have higher levels of plasticity compared to plants from unfavourable habitats (Hoffmann and Parsons, 1991[118]). Because most studies have concentrated on variation in plasticity on the interspecific level, more research is necessary to extrapolate these interspecific patterns of phenoptypic plasticity to the intraspecific level. Especially, effects of fluctuating environments or continuously stressful environments on the genetic variation for plasticity levels should be studied.

Summarizing, next to a genetic response of a population to an environmental change, the presence of phenotypic plasticity in a genotype can contribute to a fitness increase. The relative importance of plasticity to genetic diversity is dependent on the species and population under study and on the environmental conditions.

## Application of Current Knowledge for Management of Natural Populations

### *Recommendations for population management*

When focusing on the possibilities for application of current knowledge to the management of natural populations, one can ask two questions: 1) which recommendations for management can be given on the basis of current knowledge, and 2) for which practical genetics-related management problems do we still have to find answers? With regard to the first question, a number of aspects can be mentioned. First, when looking at the minimal viable population size (MVP), we have shown in this review that numerous factors are influential, making it almost impossible to formulate standard MVPs for (groups of) species. On the other hand, current theory can provide some rules of thumb for nature conservation management. For example, it is crucial for conservation managers to be aware of the possible discrepancy between actual and effective population size, even more so when different types of stochastic events (e.g., environmental stochasticity and genetic drift) are likely to co-occur. Furthermore, population size in some cases can be considered to be a species characteristic – some organisms always occur in small populations (Rabinowitz, 1981[219]). Therefore, monitoring changes in population size rather than absolute numbers is recommended in order to be able to estimate extinction risks to populations.

Another common problem in present-day population management (especially in highly populated industrialized countries) is the increased level of isolation of populations. As we have discussed, this can lead to a series of problems, including inbreeding and genetic drift. For some species, migration can be restored by strengthening source populations, creating corridors or increasing the number of suitable habitat patches within a certain area, and by active human interference, such as reintroduction of species into habitats where they have disappeared, introduction into restored habitats where they have not yet appeared or the restocking individuals into dwindling populations (Given, 1994[90]). On the other hand, hybridization due to restored gene flow can lead to outbreeding depression. It is not easy to predict whether the result of reconnecting populations will be beneficial or counterproductive. In a metapopulation that originates from habitat fragmentation, gene flow will be limited compared to the former unfragmented situation. In such a case, detrimental effects in the (small) subpopulations are likely. Gene flow then ought to be stimulated to the level of the pre-fragmented situation (Leung et al., 1993[154]). Inbreeding risks will mainly occur in decreasing species with formerly widespread occurrences. Outbreeding, for example, can be a problem in selfing plant species that occur naturally in small and isolated populations.

In conclusion: population history will have to be included when evaluating the necessity of restoration of gene flow. A final recommendation for population management that should be mentioned here is the special attention that should be given to populations in marginal habitats and at the borders of the geographic range of a species. These populations may contain specific genetic variation and therefore may be of specific value for maintenance of the long-term evolutionary perspective of a species.

*Practical management problems that still have to be answered*

Further indications of MVPs and maximal dispersal distances for species are needed. As discussed above, it is very difficult to estimate these parameters at the species level. A comparative analysis of a number of relevant parameters for a large number of species would probably make it possible to come to a level of generalization that is suitable for conservation management decision making. The parameters that should be included in such an analysis follow from current hypotheses on the importance of genetic diversity for population survival and include different life history characteristics and demographic factors. In this review a large number of such relevant parameters have been mentioned. To be able to include data on a large group of species, cooperation between different research groups is of vital importance.

Another subject that needs further clarification is the pros and cons of (re)introduction of species, including restocking of populations. As mentioned above, such actions can be useful to prevent the detrimental effects of diminished gene flow and small population size. However, before these measures can be applied in practice, it is necessary to have knowledge of a number of aspects: 1) for which species are introductions a useful complementary measure in addition to other conservation measures, such as habitat restoration and designation of reserves? 2) where do introductions have to take place? 3) what source material should be used? and 4) how many individuals have to be used? Especially the last two points have clear relationships with genetic considerations, as mentioned in this review. The success of reintroduction strategies can highly depend on the level of adaptation to ecological conditions (Burnham-Curtis et al., 1995[40]; Allendorf and Waples, 1996[7], and others). On the basis of current knowledge, rough answers to the above questions can already be given (Van Groenendael et al., 1998[257]). For a sound application of such measures more specific indications for groups of species are indispensable.

## Future Research Needs

The importance of loss of genetic variation for adaptability and long-term prospects is difficult to predict for any given population. First, the relation between genetic diversity and fitness has to be studied in order to be able to make any prediction on the importance of genetic diversity for that specific population. For this, reliable methods for a good estimation of total fitness are a prerequisite. As fitness not only depends on the genotype but also on an interaction between the genotype and its environment, insight has to be obtained into the way in which genetic differences are translated into fitness differences in a specific ecological situation. Second, monitoring the natural population is necessary to be able to predict the consequences of ecological factors on long-term population dynamics.

Studies of population dynamics and knowledge of the importance of genetic diversity in relation to adaptability are of fundamental importance for any prediction of future population dynamics. Therefore, case studies in natural populations which can assign avoidance of extinction to the role of genetic variation are necessary. It is important to include both demography and genetics in such studies because of the crucial role of the interaction between these two sides of the extinction problem in small and isolated populations (Ouborg and Van Treuren, 1997[197]). Useful demographic information with regard to mating systems, inbreeding depression, effective population size and metapopulation structure can be obtained from genetic marker data. Case studies in wild populations on the effects of bottlenecks on genetic diversity are scarce because of lack of knowledge of the evolutionary history of most species. As phenotypic plasticity can play an important role in population adaptation, knowledge on the genetic regulation of phenotypic plasticity is needed too.

Several scientists propose that preservation of the maximum quantity of genetic variation is the primary aim of conservation. They base this on the observed relation between lack of genetic variation and increase of extinction chance in small populations (e.g., Beardmore, 1983[17]; Gilpin and Soulé, 1986[89]; Vrijenhoek, 1994[264]; Frankham, 1995[77]). On the basis of the fact that the frequently used markers for studies on genetic diversity (isozymes, DNA) mostly concern a limited number of loci which are generally assumed to be selectively neutral, and that (from this review) these may not reflect well the genetic diversity in quantitative traits which are important for fitness and adaptability, this maximum quantity of genetic variation should comprise not only neutral markers, but also ecologically important (i.e., functional) traits. Besides conventional quantitative genetic research, diversity in quantitative traits can be determined using molecular markers via QTL mapping, or directly via estimating the variation on the loci involved in the functions. It will be interesting to see how such studies will add to our knowledge of genetic diversity based on "neutral" markers.

## Acknowledgements

We thank F. A. Van Eeuwijk for critically reading the manuscript.

## References

[1] Affre, L. and Thompson, J. D. (1997) Population genetic structure and levels of inbreeding depression in the Mediterranean island endemic *Cyclamen creticum* (Primulaceae). Biol. J. Linn. Soc. 60, 527 – 549.

[2] Ager, A., Guries, G., and Lee, C. H. (1983) Genetic gains from red pine seedling seed orchards. Durham, New Hampshire: Proc. 28th Northeastern Forest Tree Improvement Conference.

[3] Ågren, J. and Schemske, D. F. (1993) Outcrossing and inbreeding depression in two annual monoecious herbs *Begonia hirsuta* and *B. semiovata.* Evolution 47, 125 – 135.

[4] Allard, R. W. and Bradshaw, A. D. (1964) Implications of genotype-environmental interactions in applied plant breeding. Crop Sci. 4, 503 – 508.

[5] Allard, R. W., Harding, J., and Wehrhahn, C. (1966) The estimation and use of selective values in predicting population change. Heredity 21, 547 – 563.

[6] Allendorf, F. W. and Leary, R. F. (1986) Heterozygosity and fitness in natural populations of animals. In Conservation biology. The science of scarcity and diversity (Soulé, M. E., ed.), Sunderland: Sinauer Associates Inc., pp. 57 – 76.

[7] Allendorf, F. W. and Waples, R. S. (1996) Conservation and genetics of salmonoid fishes. In Conservation genetics. Case histories from nature (Avise, J. C. and Hamrick, J. L., eds.), New York: Chapman and Hall, pp. 238 – 280.

[8] Alonso-Blanco, C., Blankestijn-De Vries, H., Hanhart, C. J., and Koornneef, M. (1999) Natural allelic variation at seed size loci in relation to other life history traits of *Arabidopsis thaliana.* Proc. Natl. Acad. Sci., USA 96, 4710 – 4717.

[9] Alvarez-Buylla, E. R., Garcia-Barrios, R., Lara-Moreno, C., and Martinez-Ramos, M. (1996) Demographic and genetic models in conservation biology: applications and perspectives for tropical rain forest tree species. Ann. Rev. Ecol. Syst. 27, 387 – 421.

[10] Aravanopoulos, F. A. and Zsuffa, L. (1998) Heterozygosity and biomass production in *Salix eriocephala.* Heredity 81, 396 – 403.

[11] Arens, P., Coops, H., Jansen, J., and Vosman, B. (1998) Molecular genetic analysis of black poplar (*Populus nigra* L.) along Dutch rivers. Mol. Ecol. 7, 11 – 18.

[12] Arnold, S. J. (1994) Multivariate inheritance and evolution: a review of concepts. In Quantitative genetic studies of behavioral evolution (Boake, C. R. B., ed.), Chicago: University of Chicago Press, pp. 17 – 48.

[13] Barrett, S. C. H. and Kohn, J. R. (1991) Genetic and evolutionary consequences of small population size in plants: implications for conservation. In Genetics and conservation of rare plants (Falk, D. A. and Holsinger, K. E., eds.), New York: Oxford University Press, pp. 3 – 30.

[14] Barton, N. H. and Turelli, M. (1989) Evolutionary quantitative genetics: how little do we know? Ann. Rev. Genet. 23, 337 – 370.

[15] Bateson, P. G. (1983) Optimal outbreeding. In Mate choice. Cambridge: Cambridge University Press, pp. 257 – 309.

[16] Beacham, T. D. (1991) Developmental stability, heterozygosity, and genetic analysis of morphological variation in pink salmon *(Oncorhynchus gorbuscha).* Can. J. Zool. 69, 274 – 278.

[17] Beardmore, J. A. (1983) Extinction, survival, and genetic variation. In Genetics and conservation: a reference for managing wild animal and plant populations (Schonewald-Cox, C. M., Chambers, S. M., MacBryde, B., and Thomas, W. L., eds.), Menlo Park, California: Benjamin/Cummings, pp. 125 – 151.

[18] Bell, D. L. and Sultan, S. E. (1999) Dynamic phenotypic plasticity for root growth in *Polygonum:* a comparative study. Am. J. Bot. 86, 807 – 819.

[19] Bennington, C. C. and McGraw, J. B. (1996) Environment-dependence of quantitative genetic parameters in *Impatiens pallida.* Evolution 50, 1083 – 1097.

[20] Biémont, C. (1983) Homeostasis, enzymatic heterozygosity, and inbreeding depression in natural populations of *Drosophila melanogaster.* Genetica 61, 179 – 189.

[21] Blum, A. (1996) Crop responses to drought and the interpretation of adaptation. Plant Growth Regulation 20, 135 – 148.

[22] Bonnell, M. L. and Selander, R. K. (1974) Elephant seals: genetic variation and near extinction. Science 134, 908 – 909.

[23] Bradshaw, A. D. (1965) Evolutionary significance of phenotypic plasticity in plants. Adv. Genet. 13, 115 – 155.

[24] Brakefield, P. M. and Saccheri, I. J. (1994) Guidelines in conservation genetics and the use of the population cage experiments with butterflies to investigate the effects of genetic drift and inbreeding. In Conservation genetics (Loeschcke, V., Tomiuk, J., and Jain, S. K., eds.), Basel: Birkhäuser Verlag, pp. 165 – 179.

[25] Briscoe, D. A., Malpica, J. M., Robertson, A., Smith, G. J., Frankham, R., Banks, R. G., and Barker, J. S. F. (1992) Rapid loss of genetic variation in large captive populations of *Drosophila* flies: implications for the genetic management of captive populations. Cons. Biol. 6, 416 – 425.

[26] Britten, H. B. (1996) Meta-analyses of the association between multilocus heterozygosity and fitness. Evolution 50, 2158 – 2164.

[27] Brookes, M. I., Graneau, Y. A., King, P., Rose, O. C., Thomas, C. D., and Mallet, J. L. B. (1997) Genetic analysis of founder bottlenecks in the rare British butterfly *Plebejus argus.* Cons. Biol. 11, 648 – 661.

[28] Brown, A. D. H. (1990) Genetic characterization of plant mating systems. In Plant population genetics, breeding, and genetic resources (Brown, A. D. H., Clegg, M. T., Kahler, A. L., and Weir, B. S., eds.), Sunderland: Sinauer Associates Inc., pp. 145 – 162.

[29] Brown, A. D. H. and Schoen, D. J. (1992) Plant population genetic structure and biological conservation. In Conservation of biodiversity for sustainable development (Sandlund, O. T., Hindar, K., and Brown, A. H. D., eds.), Oslo: Scandinavian University Press, pp. 88 – 104.

[30] Brown, A. H. D. and Weir, B. S. (1983) Measuring genetic variability in plant populations. In Development in plant genetics and breeding 1. Isozymes in plant genetics and breeding. Part A (Tanksley, S. D. and Orton, T. J., eds.), Amsterdam: Elsevier Science Publishers B. V., pp. 219 – 239.

[31] Brown, J. H., Marquet, P. A., and Taper, M. L. (1993) Evolution of body size: consequences of an energetic definition of fitness. Am. Nat. 142, 373 – 384.

[32] Browning, J. A. (1974) Relevance of knowledge about natural ecosystems to development of pest management programs for agroecosystems. Proc. Am. Phytopathol. Soc. 1, 191 – 199.

[33] Bryant, E. H., Backus, V. L., Clark, M. E., and Reed, D. H. (1999) Experimental tests of captive breeding for endangered species. Cons. Biol. 13, 1487 – 1496.

[34] Bryant, E. H., Combs, L. M., and McCommas, S. A. (1986) The effect of an experimental bottleneck upon quantitative genetic variation in the housefly. Genetics 114, 1191 – 1211.

[35] Bryant, E. H. and Meffert, L. M. (1990) Multivariate phenotypic differentiation among bottleneck lines of the housefly. Evolution 44, 660 – 668.

[36] Burdon, J. J. (1980) Intra-specific diversity in a natural population of *Trifolium repens.* J. Ecol. 68, 717 – 735.

[37] Burdon, J. J. (1985) Pathogens and the genetic structure of plant populations. In Studies on plant demography: a Festschrift for John L. Harper (White, J., ed.), London: Academic Press, pp. 313 – 325.

[38] Burdon, J. J. (1987) Diseases and plant population biology. Cambridge: Cambridge University Press.

[39] Burdon, J. J. and Jarosz, A. M. (1990) Disease in mixed cultivars, composites, and natural plant populations: some epidemiological and evolutionary consequences. In Plant population genetics, breeding, and genetic resources (Brown, A. D. H., Clegg, M. T., Kahler, A. L., and Weir, B. S., eds.), Sunderland: Sinauer Associates Inc., pp. 215 – 228.

[40] Burnham-Curtis, M. K, Krueger, C. C., Schreiner, D. R., Johnson, J. E., Stewart, T. J., Horrall, R. M., MacCallum, W. R., Kenyon, R., and Lange, R. E. (1995) Genetic strategies for lake trout rehabilitation: a synthesis. J. Great Lakes Res. 21 (Suppl. 1), 477 – 486.

[41] Byers, D. L. and Waller, D. M. (1999) Do plant populations purge their genetic load? Effects of population size and mating history on inbreeding depression. Ann. Rev. Ecol. Syst. 30, 479 – 513.

[42] Campbell, D. R. (1997) Genetic and environmental variation in life history traits of a monocarpic perennial: a decade-long field experiment. Evolution 51, 373 – 382.

[43] Carvalho, G. R. (1993) Evolutionary aspects of fish distribution: genetic variability and adaptation. J. Fish Biol. 43 (Suppl. A), 53 – 73.

[44] Case, T. J. and Taper, M. L. (1986) On the coexistence and coevolution of asexual and sexual competitors. Evolution 40, 366 – 387.

[45] Charlesworth, D. (1991) The apparent selection on neutral marker loci in partially inbreeding populations. Genet. Res. 57, 159 – 175.

[46] Charlesworth, D. and Charlesworth, B. (1987) Inbreeding depression and its evolutionary consequences. Ann. Rev. Ecol. Syst. 18, 237 – 268.

[47] Charlesworth, D., Lyons, E. E., and Litchfield, L. B. (1994) Inbreeding depression in two highly inbreeding populations of *Laevenworthia.* Proc. R. Soc. Lond. B 258, 209 – 214.

[48] Charlesworth, D., Morgan, M. T., and Charlesworth, B. (1990) Inbreeding depression, genetic load and the evolution of outcrossing rates in a multi-locus system with no linkage. Evolution 44, 1469 – 1489.
[49] Chesser, R. K. and Ryman, N. (1986) Inbreeding strategy in subdivided populations. Evolution 40, 616 – 624.
[50] Cheverud, J., Routman, E., Jaquish, C., Tardif, S., Peterson, G., Belfiore, N., and Forman, L. (1994) Quantitative and molecular genetic variation in captive cotton-top tamarins *(Saguinus oedipus).* Cons. Biol. 8, 95 – 105.
[51] Clarke, G. M. (1992) Fluctuating asymmetry: a technique for measuring developmental stress of genetic and environmental origin. Acta Zool. Fenn. 191, 31 – 35.
[52] Clarke, G. M. (1993) The genetic basis of developmental stability. I. Relationships between stability, heterozygosity and genomic coadaptation. Genetica 89, 15 – 23.
[53] Clarke, G. M. (1995) Relationships between developmental stability and fitness: application for conservation biology. Cons. Biol. 9, 18 – 24.
[54] Clarke, G. M., Brand, G. W., and Whitten, M. J. (1986) Fluctuating asymmetry: a technique for measuring developmental stress caused by inbreeding. Austr. J. Biol. Sci. 39, 145 – 153.
[55] Clarke, G. M. and McKenzie, J. A. (1987) Developmental stability of insecticide resistant phenotypes in blowfly; a result of canalizing natural selection. Nature 325, 345 – 346.
[56] Clarke, G. M. and McKenzie, J. A. (1992) Coadaptation, developmental stability and fitness of insecticide resistant genotypes in the Australian sheep blowfly, *Lucilia cuprina:* a review. Acta Zool. Fenn. 191, 107 – 110.
[57] Clegg, M. T., Kahler, A. L., and Allard, R. W. (1978) Estimation of life cycle components of selection in an experimental plant population. Genetics 89, 765 – 792.
[58] Coltman, D. W., Pilkington, J. G., Smith, J. A., and Pemberton, J. M. (1999) Parasite-mediated selection against inbred Soay sheep in a free-living, island population. Evolution 53, 1259 – 1267.
[59] David, P. (1997) Modeling the genetic basis of heterosis: tests of alternative hypotheses. Evolution 51, 1049 – 1057.
[60] David, P. (1998) Heterozygosity-fitness correlations: new perspectives on old problems. Heredity 80, 531 – 537.
[61] David, P., Delay, B., and Jarne, P. (1997) Heterozygosity and growth in the marine bivalve *Spisula ovalis:* testing altenative hypotheses. Genet. Res. Camb. 70, 215 – 223.
[62] Den Boer, P. J., Szyszko, J., and Vermeulen, R. (1993) Spreading the risk of extinction by genetic diversity in populations of the carabid beetle *Pterostichus oblongopunctatus* F. (Coleoptera, Carbidae). Neth. J. Zool. 43, 242 – 259.
[63] Deng, H.-W. and Fin, Y.-X. (1998) Conditions for positive and negative correlations between fitness and heterozygosity in equilibrium populations. Genetics 148, 1333 – 1340.
[64] Dietl, G. and Langhammer, M. (1997) Conservation of rare breeds of animals – objectives and possibilities. Arch. Tierz., Dummerstorf 40, 135 – 141.
[65] Dinus, R. J. (1974) Knowledge about natural ecosystems as a guide to disease control in managed forests. Proc. Am. Phytopathol. Soc. 1, 184 – 190.
[66] Dobson, A. and Crawley, M. (1994) Pathogens and the structure of plant communities. Trends Ecol. Evol. 9, 393 – 398.
[67] Dobzhansky, T. (1951) Genetics and the origin of species. Third edition. New York: Colombia University Press.
[68] Ellegren, H., Hartman, G., Johansson, M., and Andersson, L. (1993) Major histocompatibility complex monomorphism and low levels of DNA fingerprinting variability in a reintroduced and rapidly expanding population of beavers. Proc. Natl. Acad. Sci. USA 90, 8150 – 8153.
[69] Ellstrand, N. C. and Elam, D. R. (1993) Population genetic consequences of small population size: implications for plant conservation. Ann. Rev. Ecol. Syst. 24, 217 – 242.
[70] Ellstrand, N. C. and Roose, M. L. (1987) Patterns of genotypic diversity in clonal plant species. Am. J. Bot. 74, 123 – 131.
[71] Ennos, R. A. (1990) Detection and measurement of selection: genetic and ecological approaches. In Plant population genetics, breeding, and genetic resources (Brown, A. D. H., Clegg, M. T., Kahler, A. L., and Weir, B. S., eds.), Sunderland: Sinauer Associates Inc., pp. 200 – 214.
[72] Evans, A. S. and Marshall, M. (1996) Developmental instability in *Brassica campestris (Cruciferae):* fluctuating asymmetry of foliar and floral traits. J. Evol. Biol. 9, 717 – 736.
[73] Falconer, D. S. (1981) Introduction to quantitative genetics. Second edition. New York: Longman.
[74] Foster Hünneke, L. (1991) Ecological implications of genetic variation in plant populations. In Genetics and conservation of rare plants (Falk, D. A. and Holsinger, K. E., eds.), New York: Oxford University Press, pp. 31 – 44.
[75] Fowler, K. and Whitlock, M. C. (1999) The distribution of phenotypic variance with inbreeding. Evolution 53, 1143 – 1156.
[76] Fox, G. A. (1995) Tinkering with the tinkerer: pollution versus evolution. Environ. Health Perspect. 103 (Suppl. 4), 93 – 100.
[77] Frankham, R. (1995) Inbreeding and extinction: a treshold effect. Cons. Biol. 9, 792 – 799.
[78] Fry, J. D., Nuzhdin, S. V., Pasyukova, E. G., and Mackay, T. F. C. (1998) QTL mapping of genotype-environment interaction for fitness in *Drosophila melanogaster.* Genet. Res., Camb. 71, 133 – 141.
[79] Gallardo, M. H. and Köhler, N. (1994) Demographic changes and genetic losses in populations of a subterranean rodent *(Ctenomys maulinus brunneus)* affected by a natural catastrophe. Z. Säugetierkunde 59, 358 – 365.
[80] Gallardo, M. H., Köhler, N., and Araneda, C. (1995) Bottleneck effects in local populations of fossorial *Ctenomys (Rodentia, Ctenomyidae)* affected by vulcanism. Heredity 74, 638 – 646.
[81] Gause, G. F. (1947) Problems of evolution. Trans. Conn. Acad. Sci. 37, 17 – 68.
[82] Gharrett, A. J. (1994) Genetic dynamics of a small population system: a model applicable to interactions between hatchery and wild fish. Aquacult. Fish. Managem. 25, 79 – 92.
[83] Gharrett, A. J. and Smoker, W. W. (1993 a) A perspective on the relative importance of genetic infrastructure in salmon populations to ocean ranching in Alaska. Fish. Res. 18, 45 – 58.
[84] Gharrett, A. J. and Smoker, W. W. (1993 b) Genetic components in life history traits contribute to population structure. In Genetic conservation of salmonid fishes (Cloud, J. G. and Thorgaard, G. H., eds.), New York: Plenum Press, pp. 197 – 202.
[85] Giles, B. E. (1983) A comparison of quantitative and biochemical variation in the wild barley *Hordeum murinum.* Evolution 38, 34 – 41.
[86] Gillespie, J. H. and Turelli, M. (1989) Genotype-environment interactions and the maintenance of polygenic variation. Genetics 121, 129 – 138.
[87] Gilpin, M. E. (1987) Spatial structure and population vulnerability. In Viable populations for conservation (Soulé, M. E., ed.), Cambridge: Cambridge University Press, pp. 125 – 139.
[88] Gilpin, M. E. and Hanski, I. (1991) Metapopulation dynamics: empirical and theoretical investigations. London: Academic Press, Harcourt Brace Jovanovich Publishers, p. 336.
[89] Gilpin, M. E. and Soulé, M. E. (1986) Minimum viable populations: processes of species extinction. In Conservation biology: the science of scarcity and diversity (Soulé, M. E., ed.), Sunderland: Sinauer Associates Inc., pp. 19 – 34.
[90] Given, D. R. (1994) Principles and practice of plant conservation. London: Chapman and Hall.
[91] Godt, M. J. W. and Hamrick, J. L. (1996) Allozyme diversity in the endangered shrub *Lindera melissifolia* (Lauraceae) and its widespread congener *Lindera benzoin.* Can. J. For. Res. 26, 2080 – 2087.

[92] Godt, M. J. W. and Hamrick, J. L. (1999) Population genetic analysis of *Elliottia racemosa (Ericaceae),* a rare Georgia shrub. Mol. Ecol. 8, 75 – 82.
[93] González-Candelas, F. and Palacios, C. (1997) Analyzing molecular data for studies of genetic diversity. In Molecular genetic techniques for plant genetic resources (Ayad, W. G., Hodgkin, T., Jaradat, A., and Rao, V. R., eds.), Report of an IPGRI Workshop, 9 – 11 October, 1995, Rome, Italy: International Plant Genetic Resources Institute, Rome, pp. 55 – 80.
[94] Goodnight, C. J. (1987) On the effect of founder events on epistatic genetic variance. Evolution 41, 80 – 91.
[95] Goodnight, C. J. (1988) Epistasis and the effect of founder events on the additive genetic variance. Evolution 42, 441 – 454.
[96] Graham, J. H. (1992) Genomic coadaptation and developmental stability in hybrid zones. Acta Zool. Fenn. 191, 121 – 132.
[97] Grashof-Bokdam, C. J., Jansen, J., and Smulders, M. J. M. (1998) Dispersal patterns of *Lonicera periclymenum* determined by genetic analysis. Mol. Ecol. 7, 165 – 174.
[98] Greenwood, P. J., Harvey, P. H., and Perrins, C. M. (1978) Inbreeding and dispersal in the great tit. Nature 271, 52 – 54.
[99] Gregorius, H.-R. (1989) The importance of genetic multiplicity for tolerance of atmospheric pollution. In Genetic effects of air pollutants in forest tree populations (Scholz, F., Gregorius, H.-R., and Rudin, D., eds.), Berlin: Springer-Verlag, pp. 163 – 172.
[100] Grime, J. P. (1997) Biodiversity and ecosystem function: the debate deepens. Science 277, 1260 – 1261.
[101] Guttman, S. I. (1994) Population genetic structure and ecotoxicology. Environm. Health Persp. 102, 97 – 100.
[102] Haldane, J. B. S. (1930) Theoretical genetics of autopolyploids. J. Genet. 22, 359 – 373.
[103] Halley, J. and Hoelzel, A. R. (1996) Simulation models of bottleneck events in natural populations. In Molecular genetic approaches in conservation (Smith, T. B. and Wayne, R. K., eds.), New York: Oxford University Press, pp. 347 – 363.
[104] Hamilton, W. D. (1980) Sex versus non-sex versus parasite. Oikos 35, 282 – 290.
[105] Hamilton, W. D. (1982) Pathogens as causes of genetic diversity in their host populations. In Population biology of infectious diseases (Anderson, R. M. and May, R. M., eds.), Berlin: Springer-Verlag, pp. 269 – 296.
[106] Hamrick, J. L. and Godt, M. J. W. (1990) Allozyme diversity in plant species. In Plant population genetics, breeding, and genetic resources (Brown, A. D. H., Clegg, M. T., Kahler, A. L., and Weir, B. S., eds.), Sunderland: Sinauer Associates Inc., pp. 43 – 63.
[107] Hamrick, J. L. and Godt, M. J. W. (1996) Conservation genetics of endemic plant species. In Conservation genetics. Case histories from nature (Avise, J. C. and Hamrick, J. L., eds.), New York: Chapman and Hall, pp. 281 – 304.
[108] Hamrick, J. L. and Nason, J. D. (1996) Consequences of dispersal in plants. In Population dynamics in space and time (Rhodes, O. E., Chesser, R. H., and Smith, M. H., eds.), Chicago: University of Chicago Press, pp. 203 – 236.
[109] Hartl, D. L. and Clark, A. G. (1997) Principles of Population Genetics. Third Edition. Sunderland: Sinauer Ass., p. 542.
[110] Harvey, P. H. and Ralls, K. (1986) Do animals avoid incest? Nature 320, 575 – 576.
[111] Hattemer, H. H. and Müller-Starck, G. (1989) Forest decline as an adaptational process. Allg. Forst-Jagdztg. 160, 222 – 229.
[112] Hedgecock, D. (1994) Does variance in reproductive success limit effective population sizes of marine organisms? In Genetics and evolution of aquatic organisms (Beaumont, A. R., ed.), London: Chapman and Hall, pp. 122 – 134.
[113] Hedrick, P. W. (1987) Genetic load and the mating system in homosporous ferns. Evolution 41, 1282 – 1289.
[114] Hedrick, P. W., Brussard, P. F., Allendorf, F. W., Beardmore, J. A., and Orzack, S. (1986) Protein variation, fitness, and captive propagation. Zoo Biol. 5, 91 – 99.
[115] Heimann, B. and Cussans, G. W. (1996) The importance of seeds and sexual reproduction in the population biology of *Cirsium arvense* – a literature review. Weed Res. 36, 493 – 503.
[116] Hoelzel, A. R., Halley, J., O'Brien, S. J., Campagna, C., Arnbom, T., Le Boeuf, B., Ralls, K., and Dover, G. A. (1993) Elephant seal genetic variation and the use of simulation models to investigate historical population bottlenecks. J. Hered. 84, 443 – 449.
[117] Hoffmann, A. A. and Merilä, J. (1999) Heritable variation and evolution under favourable and unfavourable conditions. Trends Ecol. Evol. 14, 96 – 101.
[118] Hoffmann, A. A. and Parsons, P. A. (1991) Evolutionary genetics and environmental stress. Oxford: Oxford University Press.
[119] Holsinger, K. E. (1988) Inbreeding depression doesn't matter: the genetic basis of mating system evolution. Evolution 42, 1235 – 1244.
[120] Holtsford, T. P. and Ellstrand, N. C. (1990) Inbreeding depression in *Clarkia tembloriensis* (Onagraceae) populations with different natural outcrossing rates. Evolution 44, 2031 – 2046.
[121] Hooper, D. and Vitousek, P. M. (1997) The effects of plant composition and diversity on ecosystem processes. Science 277, 1302 – 1305.
[122] Houle, D. (1992) Comparing evolvability and variability of quantitative traits. Genetics 130, 195 – 204.
[123] Hurst, L. D. (1999) The evolution of genomic anatomy. Trends Ecol. Evol. 14, 108 – 112.
[124] Husband, B. and Schemske, D. W. (1996) Evolution of the magnitude and timing of inbreeding depression in plants. Evolution 50, 54 – 70.
[125] Jain, S. K. (1976) Patterns of survival and microevolution in plant populations. In Population genetics and ecology (Karlin, S. and Nevo, E., eds.), New York: Academic Press, pp. 49 – 89.
[126] Jayakar, S. D. (1970) A mathematical model for interaction of gene frequencies in a parasite and its host. Theor. Pop. Biol. 1, 140 – 164.
[127] Jones, J. S. (1987) An asymmetrical view of fitness. Nature 325, 298 – 299.
[128] Karp, A., Edwards, K. J., Bruford, M., Funk, S., Vosman, B., Morgante, M., Seberg, O., Kremer, A., Boursot, P., Arctander, P., Tautz, D., and Hewitt, G. M. (1997) Molecular technologies for biodiversity evaluation: opportunities and challenges. Nature Biotech. 15, 625 – 628.
[129] Karhu, A., Hurme, P., Karjalainen, M., Karvonen, P., Kärkkäinen, K., Neale, D., and Savolainen, O. (1996) Do molecular markers reflect patterns of differentiation in adaptive traits of conifers? Theor. Appl. Genet. 93, 215 – 221.
[130] Kimura, M. (1983) The Neutral Theory of Molecular Evolution. Cambridge: Cambridge University Press.
[131] Kobyliansky, E. and Livshits, G. (1983) Relationship between levels of biochemical heterozygosity and morphological variability in human populations. Ann. Hum. Genet. 47, 215 – 223.
[132] Kopp, R. L., Guttman, S. I., and Wissing, T. E. (1992) Genetic indicators of environmental stress in central mudminnow *(Umbra limi)* populations exposed to acid precipitation in the Adirondack mountains. Environ. Toxicol. Chem. 11, 665 – 676.
[133] Kopp, R. L., Wissing, T. E., and Guttman, S. I. (1994) Genetic indicators of environmental tolerance among fish populations exposed to acid deposition. Biochem. Syst. Ecol. 22, 459 – 475.
[134] Kretzmann, M. B., Gilmartin, W. G., Meyer, A., Zegers, G. P., Fain, S. R., Taylor, B. F., and Costa, D. P. (1997) Low genetic variability in the Hawaiian monk seal. Cons. Biol. 11, 482 – 490.
[135] Lacy, R. C. (1992) The effects of inbreeding on isolated populations: are minimum viable population sizes predictable? In Conservation biology. The theory and practice of nature conservation, preservation and management (Fiedler, P. L. and Jain, S. K., eds.), New York: Chapman and Hall Inc., pp. 277 – 296.
[136] Lande, R. (1976) The maintenance of genetic variability by mutation in a polygenic character with linked loci. Genet. Res. 26, 221 – 235.

[137] Lande, R. (1980) Genetic variation and phenotypic evolution during allopatric speciation. Am. Nat. 116, 463 – 479.
[138] Lande, R. (1988) Genetics and demography in biological conservation. Science 241, 1455 – 1460.
[139] Lande, R. and Barrowclough, G. F. (1987) Effective population size, genetic variation, and their use in population management. In Viable populations for conservation (Soulé, M. E., ed.), Cambridge: Cambridge University Press, pp. 87 – 123.
[140] Lande, R. and Schemske, D. W. (1985) The evolution of self fertilization and inbreeding depression in plants. I. Genetic models. Evolution 39, 24 – 40.
[141] Lande, R. and Shannon, S. (1996) The role of genetic variation in adaptation and population persistence in a changing environment. Evolution 50, 434 – 437.
[142] Le Boeuf, B. J. (1974) Male-male competition and reproductive success in elephant seals. Am. Zool. 14, 163 – 176.
[143] Leamy, L. (1984) Morphometric studies in inbred and hybrid house mice. V. Directional and fluctuating asymmetry. Am. Nat. 123, 579 – 593.
[144] Leamy, L. (1992) Morphometric studies in inbred and hybrid house mice. VII. Heterosis in fluctuating asymmetry at different ages. Acta Zool. Fenn. 191, 111 – 120.
[145] Leary, R. F. and Allendorf, F. W. (1989) Fluctuating asymmetry as an indicator of stress: implications for conservation biology. Trends Ecol. Evol. 4, 214 – 217.
[146] Leary, R. F., Allendorf, F. W., and Knudsen, K. L. (1983) Developmental stability and enzyme heterozygosity in rainbow trout. Nature 301, 71 – 72.
[147] Leary, R. F., Allendorf, F. W., and Knudsen, K. L. (1984) Superior developmental stability of heterozygotes at enzyme loci in salmonid fishes. Am. Nat. 124, 540 – 551.
[148] Leary, R. F., Allendorf, F. W., and Knudsen, K. L. (1985) Inheritance of meristic variation and the evolution of developmental stability in rainbow trout. Evolution 39, 308 – 314.
[149] Ledig, F. T. (1986) Heterozygosity, heterosis, and fitness in outbreeding plants. In Conservation biology. The science of scarcity and diversity (Soulé, M. E., ed.), Sunderland: Sinauer Associates Inc., pp. 77 – 104.
[150] Ledig, F. T. (1992) Human impacts on genetic diversity in forest ecosystems. Oikos 63, 87 – 108.
[151] Leonard, K. J. (1977) Selection pressures and plant pathogens. Ann. N.Y. Acad. Sci. 287, 207 – 222.
[152] Leonard, K. J. and Czochor, R. J. (1980) Theory of genetic interactions among populations of plants and their pathogens. Ann. Rev. Phytopathol. 18, 237 – 258.
[153] Lerner, I. M. (1954) Genetic homeostasis. New York: John Wiley and Sons.
[154] Leung, L. K. P., Dickman, C. R., and Moore, L. A. (1993) Genetic variation in fragmented populations of an Australian rainforest rodent, *Melomys cervinipes*. Pacific Cons. Biol. 1, 58 – 65.
[155] Levin, D. A. (1981) Dispersal versus gene flow in plants. Ann. Mo. Bot. Gard. 68, 233 – 253.
[156] Levin, D. A. (1983) Plant parentage: an alternative view of the breeding structure of populations. In Population biology: retrospect and prospect (King, C. E. and Dawson, P. S., eds.), New York: Columbia University Press, pp. 171 – 188.
[157] Levin, D. A. and Kerster, H. W. (1974) Gene flow in seed plants. Evol. Biol. 7, 139 – 220.
[158] Linhart, Y. B. and Grant, M. C. (1996) Evolutionary significance of local genetic differentiation in plants. Ann. Rev. Ecol. Syst. 27, 237 – 277.
[159] Lönn, M., Prentice, H. C., and Bengtsson, K. (1996) Genetic structure, allozyme-habitat associations and reproductive fitness in *Gypsophila fastigiata* (Caryophyllaceae). Oecologia 106, 308 – 316.
[160] Lynch, M. (1988) The rate of polygenic mutation. Genet. Res. 51, 137 – 148.
[161] Lynch, M. (1996) A quantitative-genetic perspective on conservation issues. In Conservation genetics. Case histories from nature (Avise, J. C. and Hamrick, J. L., eds.), New York: Chapman and Hall, pp. 471 – 501.
[162] Lynch, C. B., Sulzbach, D. S., and Connolly, M. S. (1988) Quantitative-genetic analysis of temperature regulation in *Mus domesticus*. IV. Pleiotropy and genotype-by-environment interactions. Am. Nat. 132, 521 – 537.
[163] MacGillivray, C. W. and Grime, J. P. (1995) Testing predictions of the resistance and resilience of vegetation subjected to extreme events. Funct. Ecol. 9, 640 – 649.
[164] Mátyás, Cs. (1989) Genetic and ecological constraints of adaptation. Voronezh, USSR: Proc. IUFRO Int. Symp. on Forest Genetics, Breeding and Physiology, pp. 79 – 90.
[165] Mátyás, Cs. (1996) Climatic adaptation of trees: rediscovering provenance tests. Euphytica 92, 45 – 54.
[166] Mayo, O. (1971) Rates of change in gene frequency in tetrasomic organisms. Genetica 42, 329 – 337.
[167] Maywald, F. and Weigel, H. J. (1997) Biochemistry and molecular biology of heavy metal accumulation in higher plants. Landbauforsch. Volkenrode 47, 103 – 126.
[168] Menges, E. S. (1992) Stochastic modeling of extinction in plant populations. In Conservation biology. The theory and practice of nature conservation, preservation and management (Fiedler, P. L and Jain, S. K., eds.), New York: Chapman and Hall Inc., pp. 253 – 275.
[169] Midgley, G. F., Wand, S. J. E., and Musil, C. F. (1998) Repeated exposure to enhanced UV-B radiation in successive generations increases developmental instability (leaf fluctuating asymmetry) in a desert annual. Plant Cell Environm. 21, 437 – 442.
[170] Milligan, B. G., Leebens-Mack, J., and Strand, A. E. (1994) Conservation genetics: beyond the maintenance of marker diversity. Mol. Ecol. 3, 423 – 435.
[171] Mitchell-Olds, T. (1996) Genetic constraints on life-history evolution: quantitative-trait loci influencing growth and flowering in *Arabidopsis thaliana*. Evolution 50, 140 – 145.
[172] Mitton, J. B. (1994) Molecular approaches to population biology. Ann. Rev. Ecol. Syst. 25, 45 – 69.
[173] Mitton, J. B. and Grant, M. C. (1984) Associations among protein heterozygosity, growth rate, and developmental homeostasis. Ann. Rev. Ecol. Syst. 15, 479 – 499.
[174] Møller, A. P. (1997) Developmental stability and fitness: a review. Am. Nat. 149, 916 – 932.
[175] Møller, A. P. (1998) Developmental stability of plants and radiation from Chernobyl. Oikos 81, 444 – 448.
[176] Møller, A. P. and Swaddle, J. P. (1997) Developmental stability and evolution. Oxford: Oxford University Press.
[177] Monneveux, P. and Belhassen, E. (1996) The diversity of drought adaptation in the wide. Plant Growth Regulation 20, 85 – 92.
[178] Moran, G. F., Marshall, D. R., and Muller, W. J. (1981) Phenotypic variation and plasticity in the colonizing species *Xanthium strumarium* L. (Noogoora Burr.). Aust. J. Biol. Sci. 34, 639 – 648.
[179] Mosseler, A. (1995) Red pine: a model for the loss of genetic diversity in trees. In Population genetics and genetic conservation of forest trees (Baradat, Ph., Adams, W. T., and Müller-Starck, G., eds.), Amsterdam: SPB Academic Publishing, pp. 359 – 370.
[180] Müller-Starck, G. (1995) Genetic variation under extreme environmental conditions. In Population genetics and genetic conservation of forest trees (Baradat, Ph., Adams, W. T., and Müller-Starck, G., eds.), Amsterdam: SPB Academic Publishing, pp. 201 – 210.
[181] Müller-Starck, G. and Hattemer, H. H. (1989) Genetic effects of environmental stress on old stands and young growth of beech *(Fagus sylvatica)*. Forstarchiv 60, 17 – 22.
[182] Nei, M. (1973) Analysis of gene diversity in subdivided populations. Proc. Natl. Acad. Sci. USA 70, 3321 – 3323.

[183] Nei, M., Maruyama, T., and Chakraborty, R. (1975) The bottleneck effect and genetic variability in populations. Evolution 29, 1 – 10.
[184] Nei, M. and Takahata, N. (1993) Effective population size, genetic diversity, and coalescence time in subdivided populations. J. Mol. Evol. 37, 240 – 244.
[185] Nevo, E. (1986) Pollution and genetic evolution in marine organisms: theory and practice. In Environmental quality and ecosystem stability, Vol. III A/B. (Dubinsky, Z. and Steinberger, Y., eds.), Ramat-Gan: Bar-Ilan University Press, pp. 841 – 848.
[186] Nevo, E., Appelbaum-Elkaher, I., Garty, J., and Beiles, A. (1997) Natural selection causes microscale allozyme diversity in wild barley and a lichen at "Evolution Canyon", Mt. Carmel, Israel. Heredity 78, 373 – 382.
[187] Nunney, L. (1993) The influence of mating system and overlapping generations on effective population size. Evolution 47, 1329 – 1341.
[188] Nunney, L. and Campbell, K. A. (1993) Assessing minimum viable population size: demography meets population genetics. Trends Ecol. Evol. 8, 234 – 239.
[189] Nunney, L. and Elam, D. R. (1994) Estimating the effective population size of conserved populations. Cons. Biol. 8, 175 – 184.
[190] O'Brien, S. J. (1994) A role for molecular genetics in biological conservation. Proc. Natl. Acad. Sci. USA 21, 5748 – 5755.
[191] O'Brien, S. J. and Evermann, J. F. (1988) Interactive influence of infectious disease and genetic diversity in natural populations. Trends Ecol. Evol. 3, 254 – 259.
[192] O'Brien, S. J., Joslin, P., Smith III, G. L., Wolfe, R., Schaffer, N., Heath, E., Ott-Joslin, J., Rawal, P. P., Bhattacharjee, K. K., and Martenson, J. S. (1987) Evidence for African origins of founders of the Asiatic lion species survival plan. Zoo Biol. 6, 99 – 116.
[193] O'Brien, S. J., Roelke, M. E., Marker, L., Newman, A., Winkler, C. A., Meltzer, D., Colly, L., Evermann, J. F., Bush, M., and Wildt, D. E. (1985) Genetic basis for species vulnerability in the cheetah (mortality and feline infectious peritonitis). Science 227, 1428 – 1434.
[194] Ohta, R. (1971) Associative overdominance caused by linked detrimental mutations. Genet. Res. 18, 277 – 286.
[195] Oleksyn, J., Prus-Glowacki, W., Giertych, M., and Reich, P. B. (1994) Relation between genetic diversity and pollution impact in a 1912 experiment with East European *Pinus sylvestris* provenances. Can. J. For. Res. 24, 2390 – 2394.
[196] Oostermeijer, J. G. B., Van Eijck, M. W., Van Leeuwen, N. C., and Den Nijs, J. C. M. (1995) Analysis of the relationship between allozyme heterozygosity and fitness in the rare *Gentiana pneumonanthe* L. J. Evol. Biol. 8, 739 – 759.
[197] Ouborg, N. J. and Van Treuren, R. (1997) Inbreeding depression, environmental stochasticity and population extinction in plants. In The role of genetics in conserving small populations (Tew, T. E., Crawford, T. J., Spencer, J. W., Stevens, D. P., Usher, M. B., and Warren, J., eds.), Petersborough: Joint Nature Conservation Committee, pp. 143 – 154.
[198] Overton, R. P. and Johnson, L. C. (1983) Red pine - potential for genetic improvement and observations on cone and seed production. Durham, New Hampshire: Proc. 28th Northeastern Forest Tree Improvement Conference.
[199] Packer, C., Pusey, A. E., Rowley, H., Gilbert, D. A., Martenson, J., and O'Brien, S. J. (1991) Case study of a population bottleneck: lions of the Ngorongoro Crater. Cons. Biol. 5, 219 – 230.
[200] Palmer, A. R. and Strobeck, C. (1986) Fluctuating asymmetry: measurement, analysis, patterns. Ann. Rev. Ecol. Syst. 17, 392 – 421.
[201] Pamilo, P. and Pálsson, S. (1998) Associative overdominance, heterozygosity and fitness. Heredity 81, 381 – 389.
[202] Parker, E. D., Jr. (1979 a) Ecological implications of clonal diversity in parthenogenetic morphospecies. Amer. Zool. 19, 753 – 762.
[203] Parker, E. D., Jr. (1979 b) Phenotypic consequences of parthenogenesis in *Cnemidophorus lizards*. I. Variability in parthenogenetic and sexual populations. Evolution 33, 1150 – 1166.
[204] Parsons, P. A. (1990) Fluctuating asymmetry: an epigenetic measure of stress. Biol. Rev. Camb. Philosoph. Soc. 65, 131 – 145.
[205] Parsons, P. A. (1996 a) Stress, resources, energy balances, and evolutionary change. Evol. Biol. 29, 39 – 72.
[206] Parsons, P. A. (1996 b) Conservation genetics: adaptation to stress and the preservation of genetic diversity. Biol. J. Linn. Soc. 58, 471 – 482.
[207] Partridge, L. (1983) Non-random mating and offspring fitness. In Mate Choice (Bateson, P. G., ed.), Cambridge: Cambridge University Press, pp. 227 – 255.
[208] Peet, R. (1974) The measurement of species diversity. Ann. Rev. Ecol. Syst. 5, 285 – 307.
[209] Pogson, G. H. and Fevolden, S. E. (1998) DNA heterozygosity and growth rate in the Atlantic cod *Gadus murhua* (L). Evolution 52, 915 – 920.
[210] Pogson, G. H. and Zouros, E. (1994) Allozyme and RFLP heterozygosities as correlates of growth rate in the scallop *Placopecten magellanicus:* a test of the associative overdominance hypothesis. Genetics 137, 221 – 231.
[211] Polans, N. O. and Allard, R. W. (1989) An experimental evaluation of the recovery potential of ryegrass populations from genetic stress resulting from restriction of population size. Evolution 43, 1320 – 1323.
[212] Prentice, H. C., Lönn, M., Lefkovitch, L. P., and Runyeon, H. (1995) Associations between allele frequencies in *Festuca ovina* and habitat variation in the alvar grasslands on the Baltic island of Öland. J. Ecol. 83, 391 – 402.
[213] Prout, T. (1969) The estimation of fitness from population data. Genetics 63, 949 – 967.
[214] Prus-Glowacki, W. (1991) Biochemical polymorphism. In Developments in plant genetics and breeding. 3. Genetics of Scots pine (Giertych, M. and Mátyás, Cs., eds.), Amsterdam: Elsevier Publishers, pp. 73 – 86.
[215] Prus-Glowacki, W. and Godzik, St. (1991) Changes induced by zinc smelter pollution in the genetic structure of pine (*Pinus sylvestris* L.) seedling populations. Silv. Genet. 40, 184 – 188.
[216] Prus-Glowacki, W., Wojnicka-Poltorak, A., Oleksyn, J., and Reich, P. B. (1999) Industrial pollutants tend to increase genetic diversity: evidence from field-grown European Scots pine populations. Water Air Soil Poll. 116, 395 – 402.
[217] Quattro, J. M. and Vrijenhoek, R. C. (1989) Fitness differences among remnant populations of the Sonoran topminnow, *Poeciliopsis occidentalis.* Science 245, 976 – 978.
[218] Qvarnström, A. (1999) Genotype-by-environment interactions in the determination of the size of a secondary sexual character in the collared flycatcher *(Ficedula albicollis).* Evolution 53, 1564 – 1572.
[219] Rabinowitz, D. (1981) Seven forms of rarity. In The biological aspects of rare plant conservation (Synge, H., ed.), New York: John Wiley and Sons, pp. 205 – 217.
[220] Rankevich, D., Lavie, B., Nevo, E., Beiles, A., and Arad, Z. (1996) Genetic and physiological adaptations of the prosobranch landsnail *Pomatia olivieri* to microclimatic stresses on Mount Carmel, Israel. Isr. J. Zool. 42, 425 – 441.
[221] Rausher, M. D. (1996) Genetic analysis of coevolution between plants and their natural enemies. Trends in Genetics 12, 212 – 217.
[222] Rice, W. R. (1983) Parent-offspring pathogen transmission: a selective agent promoting sexual reproduction. Am. Nat. 121, 187 – 203.
[223] Rick, C. M. (1983) Evolution of mating systems: evidence from allozyme variation. In Genetics: new frontiers. Proc. XV Int. Congress of Genetics, New Dehli, India, December 12-21, 1983. New Dehli: Oxford and IBH Publishing, pp. 215 – 221.

[224] Ritland, K. (1996) A marker-based method for inferences about quantitative inheritance in natural populations. Evolution 50, 1062 – 1073.
[225] Roff, D. A. (1998) Evolution of treshold traits: the balance between directional selection, drift and mutation. Heredity 80, 25 – 32.
[226] Saccheri, I., Kuusaari, M., Kankare, M., Vikman, P., Fortelius, W., and Hanski, I. (1998) Inbreeding and extinction in a butterfly metapopulation. Nature 392, 491 – 494.
[227] Sarukhán, J., Martinez-Ramos, M., and Pinero, D. (1984) The analysis of demographic variability at the individual level and its population consequences. In Perspectives on plant population ecology (Dirzo, R. and Sarukhán, J., eds.), Sunderland: Sinauer Associates Inc., pp. 83 – 106.
[228] Schaal, B. A., Leverich, W. J., and Rogstad, S. H. (1991) A comparison of methods for assessing genetic variation in plant conservation biology. In Genetics and conservation of rare plants (Falk, D. A. and Holsinger, K. E., eds.), New York: Oxford University Press, pp. 123 – 134.
[229] Scheiner, S. M. (1993) Genetics and evolution of phenotypic plasticity. Ann. Rev. Ecol. Syst. 23, 1 – 14.
[230] Scheiner, S. M. and Lyman, R. F. (1989) The genetics of phenotypic plasticity and genetic variation in populations of the grass *Danthonia spicata.* Evolution 38, 845 – 855.
[231] Schemske, D. W., Husband, B. C., Ruckelshaus, M. H., Goodwillie, C., Parker, I. M., and Bishop, J. G. (1994) Evaluating approaches to the conservation of rare and endangered plants. Ecology 75, 584 – 606.
[232] Schlichting, C. D. (1986) The evolution of phenotypic plasticity in plants. Ann. Rev. Ecol. Syst. 17, 667 – 693.
[233] Schneller, J. J. and Holderegger, R. (1996) Genetic variation in small, isolated fern populations. J. Veget. Sci. 7, 113 – 120.
[234] Shaffer, M. L. (1981) Minimum population sizes for species conservation. BioScience 31, 131 – 134.
[235] Shaffer, M. L. (1987) Minimum viable populations: coping with uncertainty. In Viable populations for conservation (Soulé, M. E., ed.), Cambridge: Cambridge University Press, pp. 69 – 86.
[236] Sherry, R. A. and Lord, E. M. (1996) Developmental stability in leaves of *Clarkia tembloriensis (Onagraceae)* as related to population outcrossing rates and heterozygosity. Evolution 50, 80 – 91.
[237] Shook, D. R. and Johnson, T. E. (1999) Quantitative trait loci affecting survival and fertility-related traits in *Caenorhabditis elegans* show genotype-environment interactions, pleiotropy and epistasis. Genetics 153, 1233 – 1243.
[238] Silander, J. A. (1979) Microevolution and clone structure in *Spartina patens.* Science 203, 658 – 660.
[239] Simmons, M. J. and Crow, J. F. (1977) Mutations affecting fitness in *Drosophila* populations. Ann. Rev. Genet. 11, 49 – 78.
[240] Slate, J., Pemberton, J. M., and Visscher, P. M. (1999) Power to detect QTL in a free-living polygynous population. Heredity 83, 327 – 336.
[241] Slatkin, M. (1987) Gene flow and the geographic structure of natural populations. Science 236, 787 – 792.
[242] Smouse, P. E. (1986) The fitness consequences of multiple-locus heterozygosity under the multiplicative overdominance and inbreeding depression models. Evolution 40, 946 – 957.
[243] Solbrig, O. T. and Simpson, B. B. (1974) Components of regulation of a population of dandelions in Michigan. J. Ecol. 62, 473 – 486.
[244] Soulé, M. E. (1980) Thresholds for survival: maintaining fitness and evolutionary potential. In Conservation biology. An evolutionary-ecological perspective (Soulé, M. E. and Wilcox, B. A., eds.), Sunderland: Sinauer Ass., pp. 151 – 169.
[245] Steiner, E. and Levin, D. A. (1977) Allozyme, SI gene, cytological and morphological polymorphisms in a population of *Oenothera biennis.* Evolution 31, 127 – 133.
[246] Storfer, A. (1996) Quantitative genetics: a promising approach for the assessment of genetic variation in endangered species. Trends Ecol. Evol. 11, 343 – 348.
[247] Strauss, R. E. (1991) Correlations between heterozygosity and phenotypic variability in *Cottus* (Teleostei: Cottidae): character components. Evolution 45, 1950 – 1956.
[248] Strauss, S. H. (1986) Heterosis at allozyme loci under inbreeding and crossbreeding in *Pinus attenuata.* Genetics 113, 115 – 134.
[249] Sultan, S. E. (1987) Evolutionary implications of phenotypic plasticity in plants. Evol. Biol. 21, 127 – 178.
[250] Sultan, S. E. and Bazzaz, F. A. (1993) Phenotypic plasticity in *Polygonum persicaria.* II. Norms of reaction to soil moisture and the maintenance of genetic diversity. Evolution 47, 1032 – 1049.
[251] Sun, M. (1997) Genetic diversity in three colonizing orchids with contrasting mating systems. Am. J. Bot. 84, 224 – 232.
[252] Templeton, A. R. (1982) The prophecies of parthenogenesis. In Evolution and genetics of life histories (Dingle, H. and Hegmann, J. P., eds.), New York: Springer-Verlag, pp. 75 – 101.
[253] Templeton, A. R. (1994) Biodiversity at the molecular genetic level: experiences from disparate macroorganisms. Phil. Trans. R. Soc. Lond. B 345, 59 – 64.
[254] Templeton, A. R. and Read, B. (1994) Inbreeding: one word, several meanings, much confusion. In Conservation genetics (Loeschcke, V., Tomiuk, J., and Jain, S. K., eds.), Basel: Birkhäuser Verlag, pp. 91 – 105.
[255] Tilman, D., Knops, J., Wedin, D., Reich, P., Ritchie, M., and Siemann, E. (1997) The influence of functional diversity and composition on ecosystem processes. Science 277, 1300 – 1302.
[256] Travis, S. E., Maschinski, J., and Keim, P. (1996) An analysis of genetic variation in *Astragalus cremnophylax* var. *cremnophylax,* a critically endangered plant, using AFLP markers. Mol. Ecol. 5, 735 – 745.
[257] Van Groenendael, J. M., Ouborg, N. J., and Hendriks, R. J. J. (1998) Criteria for the introduction of plant species. Acta Bot. Neerl. 47, 3 – 13.
[258] Van Noordwijk, A. J., Van Tienderen, P. H., De Jong, G., and Van Balen, J. H. (1985) Genealogical evidence for random mating in a natural population of the great tit (*Parus major* L.). Naturwissenschaften 72, 104.
[259] Van Rossum, F., Vekemans, X., Meerts, P., Gratia, E., and Lefèbvre, C. (1997) Allozyme variation in relation to ecotypic differentiation and population size in marginal populations of *Silene nutans.* Heredity 78, 552 – 560.
[260] Van Valen, L. (1962) A study of fluctuating asymmetry. Evolution 16, 125 – 142.
[261] Van Valen, L. (1965) Morphological variation and width of ecological niche. Am. Nat. 99, 377 – 390.
[262] Via, S. and Lande, R. (1985) Genotype-environment interaction and the evolution of phenotypic plasticity. Evolution 39, 505 – 522.
[263] Via, S. and Lande, R. (1987) Evolution of genetic variability in a spatially heterogeneous environment: effects of genotype-environment interaction. Genet. Res. 49, 147 – 156.
[264] Vrijenhoek, R. C. (1994) Genetic diversity and fitness in small populations. In Conservation genetics (Loeschcke, V., Tomiuk, J., and Jain, S. K., eds.), Basel: Birkhäuser Verlag, pp. 37 – 53.
[265] Vrijenhoek, R. C. (1996) Conservation genetics of North America desert fishes. In Conservation genetics. Case histories from nature (Avise, J. C. and Hamrick, J. L., eds.), New York: Chapman and Hall, pp. 367 – 397.
[266] Wallace, B. (1970) Genetic load: its biological and conceptual aspects. Engelwood Cliffs: Prentice-Hall.
[267] Wardle, D. A., Zackrisson, O., Hörnberg, G., and Gallet, C. (1997) The influence of island area on ecosystem properties. Science 277, 1296 – 1299.

[268] Warwick, S. I., Thompson, B. K., and Black, L. D. (1987) Life history and allozyme variation in populations of the weed species *Setaria faberi*. Can. J. Bot. 65, 1396 – 1402.
[269] Watkinson, A. R. and Powell, J. C. (1993) Seedling recruitment and the maintenance of clonal diversity in plant populations – a computer simulation of *Ranunculus repens*. J. Ecol. 81, 707 – 717.
[270] Weber, J. L. and Wong, C. (1993) Mutation of humans short tandem repeats. Human Mol. Genet. 2, 1123 – 1128.
[271] Weir, B. S. (1989) Sampling properties of gene diversity. In Plant population genetics, breeding, and genetic resources (Brown, A. D. H., Clegg, M. T., Kahler, A. L., and Weir, B. S., eds.), Sunderland: Sinauer Associates Inc., pp. 23 – 42.
[272] Willis, J. H. and Orr, H. A. (1993) Increased heritable variation following population bottlenecks: the role of dominance. Evolution 47, 949 – 957.
[273] Willson, M. F. (1983) Plant Reproductive Ecology. New York: Wiley-Interscience.
[274] Willson, M. F. (1984) Mating patterns in plants. In Perspectives on plant population ecology. (Dirzo, R. and Sarukhán, J., eds.), Sunderland: Sinauer Associates Inc., pp. 261 – 277.
[275] Wilsey, B. J., Haukioja, E., Koricheva, J., and Sulkinoja, M. (1998) Leaf fluctuating asymmetry increases with hybridization and elevation in tree-line birches. Ecology 79, 2092 – 2099.
[276] Winicov, I. (1998) New molecular approaches to improving salt tolerance in crop plants. Ann. Bot. 82, 703 – 710.
[277] Wright, S. (1965) The interpretation of population structure by F-statistics with special regard to systems of mating. Evolution 19, 395 – 420.
[278] Yang, R.-C., Yeh, F. C., and Yanchuk, A. D. (1996) A comparison of isozyme and quantitative genetic variation in *Pinus contorta* ssp. *latifolia* by $F_{ST}$. Genetics 142, 1045 – 1052.
[279] Yezerinac, S. M., Lougheed, S. C., and Handford, P. (1992) Morphological variability and enzyme heterozygosity: individual and population level correlations. Evolution 46, 1959 – 1964.
[280] Young, A., Boyle, T., and Brown, T. (1996) The population genetic consequences of habitat fragmentation for plants. Trends Ecol. Evol. 11, 413 – 418.
[281] Zink, R. M., Smith, M. F., and Patten, J. L. (1985) Association between heterozygosity and morphological variance. J. Hered. 76, 415 – 420.
[282] Zouros, E. and Mallet, A. L. (1989) Genetic explanations of the growth/heterozygosity correlation. In Reproduction, genetics and distributions of marine organisms: XXIII European Marine Biology Symposium (Ryland, J. S. and Tyler, P. A., eds.), Fredensborg, Denmark: Olsen and Olsen, pp. 317 – 324.
[283] Zouros, E. and Pogson, G. H. (1994) The present status of the relationship between heterozygosity and heterosis. In Genetics and evolution of aquatic organisms (Beaumont, A. R., ed.), London: Chapman and Hall, pp. 135 – 147.

G. Booy

Plant Research International
P. O. Box 16
6700 AA Wageningen
The Netherlands

E-mail: G.Booy@plant.wag-ur.nl

Section Editor: R. Aerts