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ORIGINAL PAPER

Patterns of genetic variation in anthropogenically impacted populations

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Abstract Genetic variation is considered critical for allowing natural populations to adapt to their changing environment, and yet the effects of human disturbance on genetic variation in the wild are poorly understood. Different types of human disturbances may genetically impact natural populations in a predictable manner and so the aim of this study was to provide an overview of these changes using a quantitative literature review approach. I examined both allozyme and microsatellite estimates of genetic variation from peer-reviewed journals, using the mean number of alleles per locus and expected heterozygosity as standardized metrics. Populations within each study were categorized according to the type of human disturbance experienced (''hunting/harvest'', ''habitat fragmentation'', or ''pollution''), and taxon-specific, as well as time- and context-dependent disturbance effects were considered. I found that human disturbances are associated with weak, but consistent changes in neutral genetic variation within natural populations. The direction of change was dependent on the type of human disturbance experienced, with some forms of anthropogenic challenges consistently decreasing genetic variation from background patterns (e.g., habitat fragmentation), whereas others had no effect (e.g., hunting/harvest) or even slightly increased genetic variation (e.g., pollution). These same measures appeared sensitive to both the time of origin and duration of the disturbance as well. This suggests that the presence or absence, strength, type, as well as the spatial and temporal scale of human disturbance experienced may warrant careful consideration when conservation management plans are formulated for natural populations, with particular attention paid to the effects of habitat fragmentation.

Keywords Conservation genetics · Genetic variation · Heterozygosity · Human disturbance · Mean number of alleles per locus

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Introduction

Genetic variation is the raw material on which selection acts and thus critical for evolutionary change. Genetic variation may be particularly important in the case of rapid environmental change, where evolution must also be rapid if a population is to persist (Burger and Lynch 1995; Lande and Shannon 1996). However, as dramatic environmental changes are often associated with human activities (e.g., De Pippo et al. 2006), it is here that genetic variation may be most important. Indeed, human impacts themselves are thought to decrease genetic variation (Caizergues et al. 2003; Kang et al. 2005), thus compromising necessary evolutionary change. The aim of this study is therefore to examine how human activities influence genetic variation in natural populations.

The ideal experiment to examine human impacts on genetic variation in nature is to screen a population before and after a disturbance. However, it is often not possible to carry out such experiments, therefore, as an alternative I have examined a large number of published studies to find a consensus on the effects of different types of human disturbance on genetic variation. This consideration is motivated in part by the conflicting results from different studies of genetic variation. In particular, some studies report reductions in genetic variation as a result of human

disturbance (Caizergues et al. 2003; Kang et al. 2005), whereas others find no such effect (Berckmoes et al. 2005; Goosens et al. 2005). Genetic variation will reflect a balance between selection, mutation, and drift, and so human activities that differentially impact these forces may have very different effects on genetic variation. Human impacts that reduce population size and increasingly isolate populations may increase genetic drift and thereby reduce genetic variation. Human impacts that change environmental conditions may increase selection and thereby also reduce genetic variation. Human impacts that increase mutation rates (e.g., Chernoble; Ellegren et al. 1997) may increase genetic variation. To examine these effects, I divide different types of human impacts in accordance with the primary deterministic factors that contribute to modern population extinction events (for review see Frankham 2003).

Hunting and harvesting reduce population size and at least sometimes cause significant declines in neutral genetic variation (Frankham 1996; Godt et al. 1996). In these cases, genetic variation may be lost through random genetic drift as the effective population size decreases (Lacy 1997). Further, inbreeding may increase the proportion of homozygous individuals within a population, which ultimately leads to a reduction in fitness (Crnokrak and Roff 1999). Trophy hunting in particular may also exert strong directional selection by targeting animals with the largest ornaments, which may then remove specific alleles or genotypes from a population (Fitzsimmons et al. 1995; Coltman et al. 2003). The prediction here would therefore be a decrease in genetic variation for hunted and harvested populations.

Habitat fragmentation, due to human settlements, fenced motorways, channels, and habitat clearing, results in the subdivision of populations into smaller, more discrete units, with limited dispersal among them. These changes can, in at least some cases, erode genetic variation due to increased inbreeding and genetic drift within fragments, and to reduced gene flow among fragmented units (Young et al. 1996; Frankham et al. 2002). The prediction here would therefore also be a decrease in genetic variation for fragmented populations.

Pollution may influence genetic variation, although the outcome is much less certain here than for the factors mentioned above (Bickham et al. 2000). On the one hand pollution might decrease genetic variation owing to genetic drift and inbreeding, particularly in cases of increased mortality that decrease population size (Posthuma and Van Straalen 1993; Belfiore and Anderson 2001). Genetic variation may also decrease owing to selection for pollution-tolerant genotypes (Keane et al. 2005). On the other hand, populations chronically exposed to chemical pollutants may experience an increase in genetic variation due to increased mutation rates (Yauk and Quinn 1996; Baker et al. 2001) or selection for heterozygotes (i.e., overdominant hypothesis; see Bickham et al. 2000). Because of this complexity, it remains uncertain as to the type of effects that pollution will have on average.

Given our interest in evolutionary potential, we would most like to track changes in genetic variation at fitness related traits. This information, however, is largely lacking for natural populations. Instead, it is sometimes possible to use *neutral* genetic variation as a surrogate (Frankham et al. 2002). This can be tenuous when examining variation among populations (McKay and Latta 2002), but it is often defensible within populations (Gilligan et al. 2005). Indeed, neutral genetic variation largely appears associated with population fitness and extinction risk (Frankham 2003, 2005; Reed and Frankham 2003). I will therefore analyze patterns of neutral genetic variation in hope that it also informs the amount of variation for traits and genes under selection.

In the present study, I specifically test the null hypothesis that estimates of neutral genetic variation are not significantly different between populations in habitats not disturbed by humans versus those in habitat subject to the above types of human disturbance. My analyses are based on a compilation of studies examining allozyme and microsatellite variation across a wide range of species. Other studies have performed similar analyses (see Garner et al. 2005), but mine differs in (1) explicitly examining different types of human disturbance, (2) excluding cases of disturbances not directly related to human activity (i.e., stochastic factors) (3) including more studies (and from a wider range of taxa), and (4) examining effects of the age and duration of disturbance.

Methods

I searched the literature for allozyme and microsatellite data on genetic variation in disturbed or undisturbed populations in nature. This process took the form of keyword searches (genetic variation, heterozygosity, allelic diversity, natural population, and population size) in Pubmed, Web of Science, BIOSIS Previews, and BioOne databases. Note that no keyword suggestive of disturbance was included, thus avoiding a bias toward studies specifically examining this effect. Keyword searches were then supplemented by examining the literature cited section of papers thus revealed.

Studies were included in the database if they met specific criteria. First, at least one of two relevant measures of genetic variation had to be reported: mean number of alleles per locus or heterozygosity. The mean number of alleles per locus is representative of the potential genetic

polymorphism, dictating the true limit of the response to selection (Schoen and Brown 1993; Bataillon et al. 1996). Heterozygosity is often thought of as a measure of actual genetic diversity (Nei 1987). For each study, I averaged population-specific values to obtain an overall value within each study. Mean heterozygosities were arc-sine square root transformed and number of alleles were $\log_{10}$-transformed, which improved normality. Second, I avoided pseudoreplication by using only a single study for a given species, specifically the most recent study. Third, genetic variation had to be reported for at least five microsatellite or polymorphic allozyme loci. Fourth, at least ten individuals had to be sampled per population. Fifth, the populations examined had to be natural, rather than domestic, captive, or experimental.

Information recorded from each study included the species, the number of populations sampled, the average number of individuals per population, the type of marker used, the number of loci, the mean number of alleles per locus, and the mean observed and expected heterozygosity. When loci deviated from Hardy–Weinberg equilibrium, heterozygosity values were recalculated, where possible, after eliminating those loci. This was done because the causes of deviation from Hardy–Weinberg equilibrium could be many (null alleles, admixture, selection), and the specific cause is rarely known. Expected heterozygosities were reported in most studies (87% of all papers collected), and when they were not, I instead used observed heterozygosities, which should be similar at equilibrium (Hedrick 2000).

Human disturbance within each study was categorized as ''hunting/harvest'', ''habitat fragmentation'' (including habitat loss), or ''pollution''. Studies of populations experiencing natural disturbances, such as disease, predation, natural disasters, and fire, were excluded in an attempt to restrict the focus to anthropogenic factors. If a population suffered more than one type of disturbance (29% of studies), it was included in the analysis for only the primary disturbance type mentioned in the publication (thus preventing non-independent data points). Papers in which the primary disturbance type was either not explicitly stated or unclear were excluded. When no disturbance was noted in a study, the populations were considered ''undisturbed''. This was confirmed by reading relevant references also cited within these papers. When studies included both disturbed and undisturbed populations of the same species, both were included in the analysis (see also below). In the end, a total of 220 relevant publications were identified (Appendix).

In order to consider the long-term effects of human activity on genetic variation, disturbances were further classified as to their time of origin and duration. A disturbance was deemed ''historic'' if it had occurred and ended prior to 1900. A disturbance was considered ''recent'' if it occurred after 1950. Few disturbances began between 1900 and 1950 and were therefore not here considered. Further, a ''short-term'' disturbance is one that occurred after 1950 and is still present, whereas a ''long-term'' disturbance is one which began prior to 1950 and persists to the present. These distinctions could not be made for 26 studies, which were therefore excluded from this part of the analysis.

Statistical analyses

Formal meta-analytic approaches require that studies report measures of variability from which effect sizes can be calculated (Arnqvist and Wooster 1995; Gurevitch and Hedges 1999). This was not the case for many studies in the database, and so I instead relied on conventional statistical tests. These tests may have lower power than formal meta-analyses, but Type I error rates are at least similar when the pattern of sampling-error variances is not substantially different among categories (Gurevitch and Hedges 1999).

I first evaluated the relationship between the mean number of individuals sampled in a study and the mean number of alleles per locus (Von Segesser et al. 1999). These variables were weakly, but significantly correlated for microsatellites ($r^2 = 0.061$, $P < 0.0001$) and not significant for allozymes ($r^2 = 0.032$, $P = 0.10$). Sample size variation was therefore unlikely to affect interpretations based on alleles per locus. I nevertheless repeated all analyses (see below) after standardizing the number of alleles by the number of sampled individuals. Standardized and un-standardized estimates were significantly and positively correlated with each other (Pearson Product Moment: $r = 0.58$, $P < 0.0001$), and observed patterns were similar in all cases. Analyses of numbers of alleles were therefore based on unstandardized values.

Two types of analyses were performed. First, I compared genetic variation among studies, which itself involved several analyses. Second, I compared genetic variation among populations within studies. All statistical analyses were performed using SPSS v11.1 software, at the $\alpha = 0.05$ level of significance.

Genetic variation among studies was primarily analyzed with MANOVAs. The dependent variables were numbers of alleles and heterozygosity (referred to jointly as ''genetic variation''). The independent variables were disturbance and molecular marker type (both fixed). These analyses were supplemented by separate univariate ANOVAs for each marker type and genetic variance measure, followed by Fisher's LSD post hoc tests. This analysis was repeated for only the two best-represented groups in the database: mammals and plants, which ensured that

observed patterns were not dependant on particular disturbance types having a disproportionate number of data points from a particular taxon. In all instances, a full model was first run and non-significant interactions were then removed. Overall inferences about changes in genetic variation were based on the MANOVAs, whereas inferences about specific response variables were based on the ANOVAs. These analyses should be broadly similar given that the two genetic variation measures were strongly correlated with each other (Pearson Product Moment, $r = 0.905$, $P < 0.0001$). The data were treated in a similar manner when the temporal effects of human disturbance were considered (with time of origin or duration of a disturbance as fixed factors).

Opposing effects in different taxa, however, may cancel each other out in a metaanalysis (e.g., disturbance may lead to a decrease in genetic diversity in mammals, but an increase in birds, and thus no effect overall), and so a comparison among individual taxa is important. To test for taxonomic effects, species were grouped into mammals, birds, fish, herp-fauna (i.e., amphibians and reptiles), invertebrates, and plants. These analyses included only taxa with at least two disturbed and undisturbed species and pooled the various disturbance types (to ensure sufficient sample size). Similar to above, MANOVAs were employed, with the numbers of alleles and heterozygosity as dependent variables. In this case, however, the independent variables were disturbance, molecular marker type, and taxon (all fixed factors). These analyses were also supplemented by separate univariate ANOVAs for each marker type and genetic variation measure.

Variation within studies was analyzed by considering differences between disturbed and undisturbed populations within a given study ($N = 50$ studies). This analysis thus controls for differences in the methodology employed by each individual study (e.g., marker loci used, study species, and sample size). Further, 11 of these data sets actually included the same populations before and after human disturbance, thus controlling for site-specific differences. In particular, I used Wilcoxon Signed Rank t tests to assess the relationship between mean heterozygosity in disturbed versus undisturbed reference populations within the same study and species. Heterozygosity is measured on a scale ranging from 0 to 1 and thus lends itself to this type of analysis.

Results

In analyses of variation among studies, genetic variation was much higher for microsatellites than for allozymes (MANOVA: $F_{2,270} = 290.98$, $P < 0.0001$), and was also influenced by disturbance type (MANOVA: $F_{6,540} = 2.86$, $P = 0.009$); different types of human disturbance had different genetic effects. In general, genetic variation in undisturbed populations was significantly higher than that in fragmented populations, non-significantly higher than that in hunted/harvested populations, and non-significantly lower than that in polluted populations (Table 1). For allozyme markers in particular, disturbance had a significant effect on the mean number of alleles per locus ($F_{3,87} = 6.75$, $P < 0.0001$) but not heterozygosity ($F_{3,96} = 2.33$, $P = 0.08$); fragmented populations had fewer allozyme alleles than did polluted ($P = 0.001$), hunted/harvested ($P = 0.041$), or undisturbed ($P < 0.0001$) populations. The same was true for the number of alleles ($F_{3,192} = 3.23$, $P = 0.024$) and heterozygosity ($F_{3,197} = 2.24$, $P = 0.085$) estimated with microsatellite markers; fragmented populations had fewer microsatellite alleles than did polluted ($P = 0.041$) or undisturbed ($P = 0.011$) populations, although in this case, not hunted/harvested ($P = 0.459$) populations. Thus, habitat fragmentation clearly had the strongest effect, consistently decreasing genetic variation from background patterns.

The above trends were maintained when accounting for possible effects of taxon. First, when species were grouped into distinct taxa, genetic variation was typically (but not always) lower in disturbed versus undisturbed populations (Fig. 1A,B,C,D). Although marker type (MANOVA: $F_{2,261} = 145.17$, $P < 0.0001$) and taxon (MANOVA: $F_{10,522} = 3.47$, $P < 0.0001$) had a significant effect on genetic variation, surprisingly disturbance did not (MANOVA: $F_{2,261} = 1.88$, $P = 0.155$). Disturbance effects increased (and were significant), however, after removal of pollution studies (MANOVA: marker type, $F_{2,245} = 131.71$, $P < 0.0001$; taxon, $F_{10,490} = 2.86$, $P = 0.002$; disturbed versus undisturbed, $F_{2,245} = 3.33$, $P = 0.043$), reinforcing the idea that pollution had qualitatively different effects than other types of disturbance here considered. Following this modification, the mean number of alleles per locus (allozyme: $F_{1,72} = 9.41$, $P < 0.0001$; microsatellite: $F_{1,185} = 3.62$, $P = 0.029$), but not heterozygosity (allozyme: $F_{1,81} = 1.98$, $P = 0.15$; microsatellite: $F_{1,189} = 2.52$, $P = 0.084$), was significantly lower in disturbed populations across all taxa. Second, trends in genetic variation among the disturbance types were similar (pollution > undisturbed > hunting/harvest > fragmented) and significant (MANOVA: $F_{6,354} = 2.12$, $P = 0.05$), albeit marginally, when comparing genetic variation estimates strictly within plants and mammals (Table 2). Fragmented plant populations (disturbance type: $F_{3,43} = 4.042$, $P = 0.031$) had significantly fewer allozyme alleles per locus than undisturbed ($P = 0.015$) or polluted ($P = 0.027$) populations, whereas fragmented mammalian populations (disturbance type: $F_{1,4} = 14.59$, $P = 0.032$) had significantly fewer allozyme

Table 1 Mean allozyme and microsatellite genetic variation estimates in ''undisturbed'' and ''disturbed'' populations (disturbance categories: Hunting/Harvest, Habitat Fragmentation, and Pollution), characterized as the mean number of alleles per locus (A) and expected heterozygosity (H_e)

		Undisturbed	Hunting/Harvest	Fragmentation	Pollution
Allozyme[b,c]	A	2.13 ± 0.09[a] (46)	2.076 ± 0.34 (7)	1.56 ± 0.088* (30)	2.19 ± 0.17 (13)
	H_e	0.19 ± 0.016 (48)	0.16 ± 0.028 (8)	0.14 ± 0.02* (33)	0.22 ± 0.036 (14)
Microsatellite[b,c]	A	8.84 ± 0.57 (80)	6.89 ± 0.46 (49)	6.83 ± 0.52** (60)	13.12 ± 4.03 (6)
	H_e	0.65 ± 0.018 (82)	0.60 ± 0.02 (50)	0.59 ± 0.023 (62)	0.70 ± 0.088 (7)

[a] Values are means ± 1 SEM (N)

[b] MANOVA tests were carried out for both estimators of genetic diversity (A and H_e together), using disturbance and molecular marker type as fixed factors. Univariate ANOVA tests were also conducted to identify case specific differences

[c] An asterisk ''*'' indicates a significant difference from all other disturbance types, $P < 0.05$, whereas a double asterisk ''**'' indicates a significant difference from polluted ($P = 0.041$) and undisturbed populations ($P = 0.011$) only

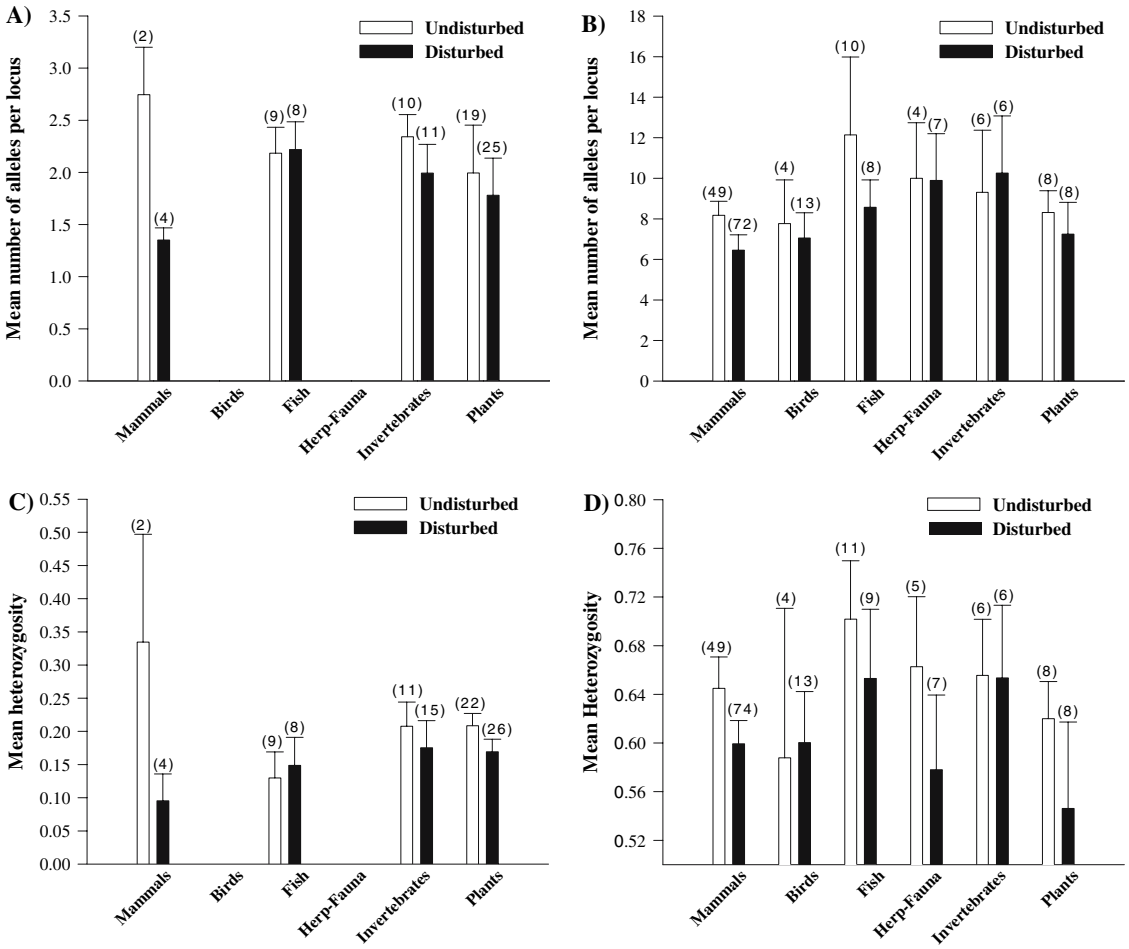

Fig. 1 Number of alleles per locus (**A**, **B**) and heterozygosity (**C**, **D**) across a wide range of animal taxa as a function of human disturbance, investigated using both allozyme (**A**, **C**) and microsatellite markers (**B**, **D**). *Numbers in parentheses* represent sample sizes (N). All values are means ± SEM

alleles than undisturbed populations ($P = 0.032$). Thus, the observed differences in genetic variation among disturbance categories did not appear to be dictated by a single taxonomic group.

Possible long-term effects of human disturbance on genetic variation were also assessed, but only for microsatellite markers (Fig. 2A,B) due to small sample sizes for allozymes. A subtle trend for a decrease in genetic varia-

Table 2 Mean allozyme and microsatellite genetic variation estimates in ''undisturbed'' and ''disturbed'' populations of mammals and plants (disturbance categories: Hunting/Harvest, Habitat Fragmentation, and Pollution), characterized as the mean number of alleles per locus (A) and expected heterozygosity (H_e)

		Undisturbed	Hunting/Harvest	Fragmentation	Pollution
Mammals: Allozyme[b,c]	A	2.75 ± 0.46[*,a] (2)	NA	1.43 ± 0.13 (3)	NA
	H_e	0.34 ± 0.16 (2)	NA	0.11 ± 0.051 (3)	NA
Mammals: Microsatellite[b,c]	A	8.18 ± 0.69 (49)	6.59 ± 0.55 (34)	6.17 ± 0.54 (38)	NA
	H_e	0.65 ± 0.026 (49)	0.60 ± 0.024 (35)	0.59 ± 0.029 (39)	NA
Plants: Allozyme[b,c]	A	1.99 ± 0.11[*] (20)	2.23 ± 0.49 (2)	1.68 ± 0.13 (18)	2.17 ± 0.21[*] (5)
	H_e	0.21 ± 0.018 (22)	0.18 ± 0.03 (2)	0.16 ± 0.022 (20)	0.21 ± 0.035 (5)
Plants: Microsatellite[b,c]	A	8.31 ± 1.074 (8)	8.27 ± 2.24 (5)	5.53 ± 2.00 (3)	NA
	H_e	0.62 ± 0.031 (8)	0.57 ± 0.061 (5)	0.51 ± 0.18 (3)	NA

[a] Values are means ± 1 SEM (N)

[b] MANOVA tests were carried out for both estimators of genetic variation (A and H_e together), using disturbance type, molecular marker type, and taxon as fixed factors. Only categories represented by at least two samples were included in the analysis

[c] An asterisk ''*'' indicates a significant difference from the fragmented group, $P < 0.05$

tion with increasing time since disturbance was evident (Fig. 2A) but non-significant (MANOVA: $F_{4,376} = 2.22$, $P = 0.066$). It was significant, however, when the number of alleles ($F_{2,192} = 4.36$, $P = 0.014$) and heterozygosity ($F_{2,197} = 3.45$, $P = 0.034$) were considered separately; undisturbed populations had significantly more alleles ($P = 0.007$) and higher heterozygosity ($P = 0.017$) than populations that had experienced disturbances prior to the 1900s, which was not the case for more recent disturbances (mean number of alleles per locus, $P = 0.07$; heterozygosity, $P = 0.099$). Human disturbances of increasing duration (Fig. 2B) also decreased genetic variation overall ($F_{4,334} = 2.38$, $P = 0.045$). It was only significant, however, for the mean number of alleles ($F_{2,171} = 3.77$, $P = 0.025$) and not heterozygosity ($F_{2,174} = 1.62$, $P = 0.2$) when considered separately. Populations experiencing long-term disturbances had significantly fewer alleles than undisturbed ($P = 0.007$) populations, and populations subject to short-term disturbances ($P = 0.046$).

A more rigorous test of the effects of human disturbance on genetic variation was performed by correlating heterozygosity estimates from both disturbed and undisturbed reference populations of the same species, reported within the same study (Fig. 3). Variation within studies included analyses for 8 mammals, 3 birds, 12 fishes, 3 herp-fauna, 10 invertebrates, and 14 plants. As might be expected, genetic variation in disturbed and undisturbed populations was strongly correlated across studies (Pearson Product Moment: $r = 0.93$, $P < 0.0001$), but no consistent trend for differences (i.e., disturbed versus undisturbed) was evident when all disturbance types were considered together (Wilcoxon Signed Rank t test: $P = 0.31$). However, nine of the 12 studies showing qualitatively higher values in disturbed populations were for instances of pollution, and so polluted populations on their own had significantly higher genetic variation than their undisturbed counterparts (Wilcoxon Signed Rank t test: $P = 0.045$). When pollution data were removed from the analysis, disturbed and undisturbed heterozygosity estimates were significantly different among the remaining categories (Wilcoxon Signed Rank t test: $P = 0.004$), although, in this case, indicating a consistent negative impact of human disturbance on genetic variation. I observed the same pattern when the mean number of alleles per locus was analyzed in this manner (data not shown).

Discussion

My goal was to evaluate the genetic impacts of different types of human disturbance. I found that the direction of responses, in terms of changes in neutral genetic variation from undisturbed background patterns, were dependent on the type of disturbance experienced. In general, fragmentation reduced genetic variation, hunting/harvesting had no appreciable effect, and pollution may actually increase genetic variation, although this last effect was not significant when tested directly. These results were largely consistent across different taxa (Fig. 1, Table 2), and were robust to differences in molecular marker types (allozymes or microsatellites) and genetic variation estimators (numbers of alleles or heterozygosity). Interestingly, however, the mean number of alleles per locus was more likely to show significant differences than was heterozygosity. This result fits with work showing that allelic diversity is affected more by demographic disturbances than are other estimates of neutral genetic variation (Hartl and Pucek 1994). Further, the observed patterns remained when the number of alleles was expressed as a ratio of sample size, indicating that my results were not driven simply by differences in sampling effort.

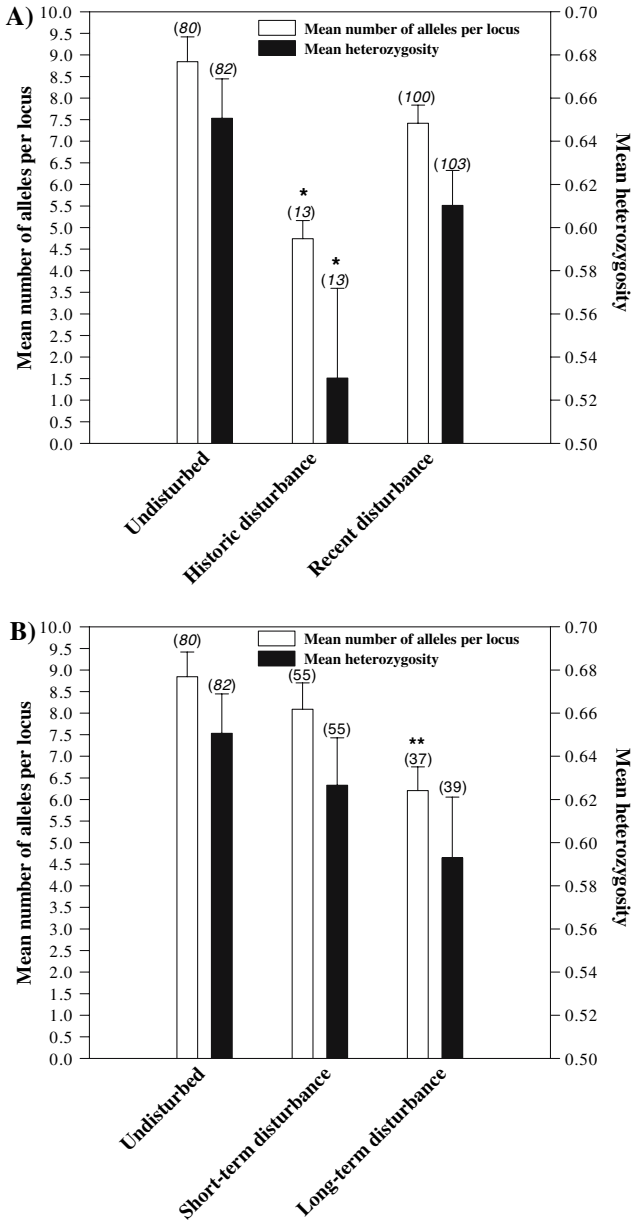

Fig. 2 Genetic variation (± SEM) in populations subject to historical or recent (**A**) in addition to short-term or long-term human disturbances (**B**) relative to undisturbed populations, considering microsatellite marker data only. MANOVA tests were carried out for both estimators of genetic variation (mean number of alleles per locus and heterozygosity together), using time of origin or duration of disturbance as fixed factors. Univariate ANOVA tests were also conducted to identify case specific differences. An *asterisk* ''*'' indicates a significant difference from the undisturbed group only ($P < 0.05$), whereas a *double asterisk* ''**'' indicates a significant difference from both the undisturbed and short-term disturbance group ($P < 0.05$). *Numbers in parentheses* represent sample sizes (N)

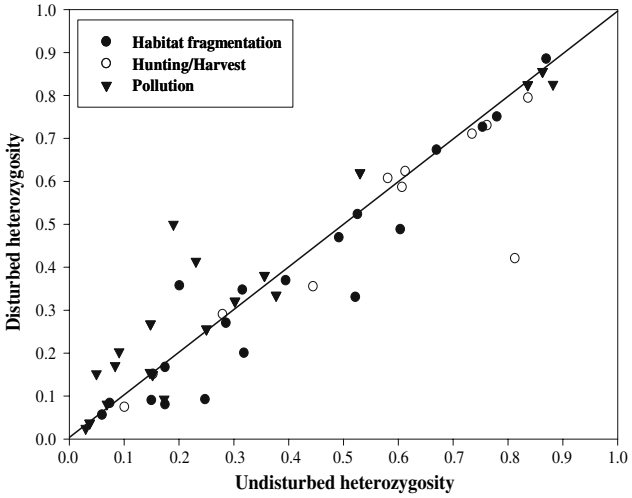

Fig. 3 The relationship between disturbed and undisturbed heterozygosity estimates reported within the same study (Pearson Product Moment Correlation: $r = 0.93$, $P < 0.0001$), considering all categories of disturbance ($N = 50$). The *line in bold* represents a line of unity, which is the point at which heterozygosity estimates in disturbed and undisturbed populations are equal. *Data points below the line* of unity indicate a negative impact of disturbance, whereas *points falling above the line* are positively impacted by human disturbance

Could my findings be the result of a publication bias? Such a bias could occur if studies reporting significant results are more likely to be published (Arnqvist and Wooster 1995; Gurevitch and Hedges 1999). This would be a problem in my study if there was a bias toward publication of disturbed populations that show reductions in genetic variation. Some such bias is possible but seems unlikely to explain all the main trends. First, patterns of genetic change were largely consistent across taxa, molecular marker type, and genetic variation estimators. Second, genetic changes owing to human disturbances are likely underrepresented in this study, as species or populations driven to extinction by human activities were not considered. Third, many of the studies included in the database collected data for purposes mostly unrelated to assessing the impacts of human disturbances on genetic variation (e.g., social structure, breeding biology, or isolation by distance). Fourth, the pollution data actually seem to suggest an increase in genetic variation, indicating that the decrease in fragmentation studies is unlikely to be just the result of a bias.

Do my results reflect *human* effects? I specifically examined disturbances attributable to humans, and so my results clearly apply to that context. It is also possible, however, that natural disturbances could have similar effects. Indeed, previous studies did not separate these effects (Garner et al. 2005). My main goal, however, is to compare different types of human disturbance, and so here inferences do not depend on an understanding of the effects of natural disturbances.

Disturbance types

Fragmentation clearly decreases genetic variation. One possible driver of this effect is reductions in population size (Young et al. 1996). Another is reduced gene flow as a result of habitat fragmentation (Frankham et al. 2002; Toro and Caballero 2005). Habitat fragmentation may reduce population size the most out of all disturbance types considered in this study, thus producing statistically significant reductions in genetic variation. Unfortunately, few studies provided estimates of census or effective population size, preventing a proper test of the idea that population size is heavily influencing the outcome. Alternatively, population size may decrease substantially with all disturbance types, and so the pronounced negative effect on genetic variation in fragmented populations may be due to reduced dispersal. Although previous work has shown a significant and positive relationship between population size and genetic diversity (Frankham 1996), further studies, comparing undisturbed and fragmented populations while controlling for population size, would indicate whether factors above and beyond population size are responsible for a lowering of genetic variation. Nonetheless, habitat fragmentation clearly has a significant impact on genetic variation in natural populations, and so conservation case studies involving fragmentation should be given priority.

Hunting/harvesting appeared to have little effect on genetic variation. This is surprising given the rapid reductions in population size generally associated with hunting and harvesting practices. Thus, I would expect a decrease in genetic variation owing to effects associated with bottlenecks (i.e., genetic drift and inbreeding), and yet I do not find this in my study. However, it should also be noted that this relationship is not always as straightforward as assumed, with past work identifying relatively abundant species having limited variability and other endangered populations maintaining high variability (for review see Frankham 1995; Amos and Harwood 1998). Thus, other factors may be involved, such as selection acting on specific genotypes, which are indirectly targeted by hunters (Fitzsimmons et al. 1995; Coltman et al. 2003; Hartl et al. 2003). One possible explanation for our results, however, is that hunting/harvest reduces population size to a lesser extent than other types of disturbance (i.e., fragmentation), and so the effects are weaker or more inconsistent (and thus non-significant).

Pollution appeared as though it might have a positive impact on genetic variation. I make this inference because every genetic variation measure was qualitatively greater for populations subject to pollution than for those in undisturbed conditions, although only some of these were significant owing to small sample sizes (Table 1). Moreover, comparisons within studies suggested a similar effect (Fig. 3), and negative genetic impacts of human disturbance were only evident when pollution data were removed from several analyses. Whether or not pollution *increases* genetic variation, it clearly has a qualitatively different effect than fragmentation, as evidenced by the significantly greater number of alleles and higher heterozygosity in polluted populations (Table 1). Thus, I suggest that pollution can have both positive and negative effects through different mechanisms. On the one hand, pollution may decrease population size (Posthuma and Van Straalen 1993) or increase selection for homozygous genotypes (Keane et al. 2005), which would decrease genetic variation. Indeed, some studies have clearly found reductions in genetic variation because of pollution (e.g., Ma et al. 2000; Belfiore and Anderson 2001). On the other hand, pollution could increase mutation rates at marker loci (Yauk and Quinn 1996; Baker et al. 2001) or increase selection for heterozygotes (Falconer and MacKay 1996). The net effect of pollution on genetic variation should therefore reflect a balance between these various forces.

That being said, conservation biologists may need to consider genetic threats from pollution carefully, separating them from other forms of human disturbance. Given the general belief that the maintenance of genetic variation is healthy in natural populations, in the short term, polluted populations may appear to be doing well genetically. Long-term effects of pollution, however, which may include adverse effects on the physiology of an organism and its environment as well as a possible increase in mutational load, are all detrimental to a population's viability.

Time of origin and duration of disturbance

The level of genetic variation maintained within a population may also be dependant on both the time of origin and duration of a particular human disturbance (Frankham 2003, 2005). Although rare alleles are likely the first to be lost, a long-term disturbance, acting over many generations, will cause the loss of more common alleles and a steeper decline in genetic variation (Lande 1988). In fact, a prolonged disturbance would likely leave a more distinct genetic ''footprint'' within a population than a transient challenge. My findings support this idea, with short-term disturbances having a lesser effect on genetic variation than long-term ones. Further, populations that had experienced historic disturbances were associated with a lower level of genetic variation than those disturbed only recently, suggesting that within-population genetic variation may be sensitive to the temporal scale of human-related activities. Although, increased conservation efforts in recent years could also explain the trend for higher genetic variation in populations disturbed only within the last 50 years.

Future considerations

The loss of genetic variation may not only affect organisms at the population level but lead to the loss of entire species given enough time, thus, the maintenance of genetic variation is of *critical* importance. But why is it important to understand genetic effects in natural populations specifically attributable to human activity? First, in order to mitigate against loss of genetic variation, it is essential we understand the source or cause. Second, by identifying specific human activities related to detrimental genetic effects we can either eliminate the source of the impact altogether or seek viable, less intrusive alternatives. Finally, a more comprehensive knowledge of past or current genetic impacts on natural populations may increase our predictive power and ability to control future impacts. This information would be of particular use to incorporate into existing models and simulation programs directed at threatened or endangered populations, where direct sampling is limited or often impossible. Although this issue merits further consideration, my study has provided essential baseline information which will facilitate future comparisons, and presents the most comprehensive assessment of genetic variation in human impacted populations to date.

The weak patterns of neutral genetic change observed in this study, despite large sample sizes in general, do raise one concern. Genetic variation is overwhelmingly monitored by neutral molecular variation in natural populations (Frankham et al. 2002) and so it was used in this study. However, there is a growing debate about whether molecular measures of genetic variation reflect adaptive differences among populations, or even the ability to respond to future environmental changes (Reed and Frankham 2001). Most environmental changes associated with human activities will affect different morphological or life-history traits of particular species, thus quantitative genetic variation may serve as a more sensitive bioindicator. In fact, a recent simulation study found that some human impacts on genetic variation could not be detected with neutral molecular markers, but only become apparent when changes in quantitative genetic variation were assessed (Carvajal-Rodríguez et al. 2005). Thus, although logistically difficult, a comprehensive assessment of quantitative genetic variation in natural populations may be the only means of estimating the ''true'' magnitude of human-related genetic effects.

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Appendix

Appendix Table A1 References for genetic variation data reviewed by DiBattista (2007)

Abbreviated reference	Complete reference
Allen et al., 1995	Allen, P. J., Amos, W., Pomeroy, P. P., and Twiss, S. D. (1995). *Molecular Ecology* **4:** 653–662.
Andersen et al., 1998	Andersen, L. W., Born, E. W., Gjertz, I., Wiig, Ø., Holm, L. E., and Bendixen, C. (1998). *Molecular Ecology* **7:** 1323–1336.
Andersen et al., 2001	Andersen, L. W., Ruzzante, D. E., Walton, M., Berggren, P., Bjørge, A., and Lockyer, C. (2001). *Conservation Genetics* **2:** 309–324.
Arigoni and Largiadèr, 2000	Arigoni, S. and Largiadèr, C. R. (2000). *Molecular Ecology* **9:** 2155–2169.
Arnaud et al., 2003	Arnaud, J., Madec, L., Guiller, A., and Deunff, J. (2003). *Heredity* **90:** 451–458.
Barcia et al., 2005	Barcia, A. R., López, G. E., Hernández, D., and García-Machado, E. (2005). *Molecular Ecology* **14:** 2933–2942.
Batista and Sosa, 2002	Batista, F. and Sosa, P. A. (2002). *Annals of Botany* **90:** 725–733.
Beaumont et al., 2001	Beaumont, M., Barratt, E. M., Gottelli, D., Kitchenere, A. C., Daniels, M. J., Pritchard, J. K., and Bruford, M. W. (2001). *Molecular Ecology* **10:** 319–336.
Becher and Griffiths, 1998	Becher, S. A. and Griffiths, R. (1998). *Molecular Ecology* **7:** 1599–1604.
Beheregaray et al., 2000	Beheregaray, L. B., Sunnucks, P., Alpers, D. L., Banks, S. C., and Taylor, A. C. (2000). *Conservation Genetics* **1:** 89–92.
Belant et al., 2005	Belant, J. L., Van Stappen, J. F., and Paetkau, D. (2005). *Ursus* **16:** 85–92.
Bell and Okamura, 2005	Bell, J. J. and Okamura, B. (2005). *Proceedings of the Royal Society of London B-Biological Sciences* **272:** 1067–1074.
Benton et al., 1994	Benton, M. J., Diamond, S. A., and Guttman, S. I. (1994). *Ecotoxicology and Environmental Safety*. **29:** 20–37.

Appendix Table A1 continued

Abbreviated reference	Complete reference
Benton et al., 2002	Benton, M. J., Malott, M. L., Trybula, J., Dean, D. M., and Guttman, S. I. (2002). *Environmental Toxicology and Chemistry* **21:** 584–589.
Berckmoes et al., 2005	Berckmoes, V., Scheirs, J., Jordaens, K., Blust, R., Backeliau, T., and Verhagen, R. (2005). *Environmental Toxicology and Chemistry* **24:** 2898–2907.
Bérubé et al., 1998	Bérubé, M., Aguilar, A., Dendanto, D., Larsen, F., Notarbartolo Di Sciara, G., Sears, R., Sigurjónsson, J., Urban-R., J., and Palsbøll, P. J. (1998). *Molecular Ecology* **7:** 585–599.
Bérubé et al., 2000	Bérubé, M., Jørgensen, H., McEwing, R., and Palsbøll, P. J. (2000). *Molecular Ecology* **9:** 2181–2183.
Billington 1991	Billington, H. L. (1991). *Conservation Biology* **5:** 115–119.
Billot et al., 1998	Billot, C., Rousvoal, S., Estoup, A., Epplen, J. T., Saumitou-Laprade, P., Valero, M., and Kloareg, B. (1998). *Molecular Ecology* **7:** 1771–1788.
Blundell et al., 2002	Blundell, G. M., Ben-David, M., Groves, P., Bowyer, R. T., and Geffen, E. (2002). *Molecular Ecology* **11:** 289–303.
Bottin et al., 2005	Bottin, L., Verhaegen, D., Tassin, J., Olivieri, I., Vaillant, A., and Bouvet, J. M. (2005). *Molecular Ecology* **14:** 1989.
Bowyer et al., 2002	Bowyer, J. C., Newell, G. R., and Eldridge, M. D. B. (2002). *Conservation Genetics* **3:** 61–69.
Bradley et al., 2000	Bradley, B. J., Boesch, C., and Vigilant, L. (2000). *Conservation Genetics* **1:** 289–292.
Browning et al., 2001	Browning, T. L., Taggart, D. A., Rummery, C., Close, R. L., and Eldridge, M. D. B. (2001). *Conservation Genetics* **2:** 145–156.
Buchanan et al., 1996	Buchanan, F. C., Friesen, M. K., Littlejohn, R. P., and Clayton, J. W. (1996). *Molecular Ecology* **5:** 571–575.
Buchert et al., 1997	Buchert, G. P., Rajora, O. M. P., Hood, J. V., and Dancik, B. P. (1997). *Conservation Biology* **11:** 747–758.
Bulgin et al., 2003	Bulgin, N. L., Gibbs, H. L., Vickery, P., and Baker, A. J. (2003). *Molecular Ecology* **12:** 831–844.
Burland et al., 1998	Burland, T. M., Barratt, E. M., and Racey, P. A. (2002). *Molecular Ecology* **7:** 133–140.
Burton et al., 2002	Burton, C., Krebs, C. J., and Taylor, E. B. (2002). *Molecular Ecology* **11:** 1689–1701.
Caizergues et al., 2003	Caizergues, A., Rätti, O., Helle, P., Rotelli, L., Ellison, L., and Rasplus, J. (2003). *Molecular Ecology* **12:** 2297–2305.
Castella et al., 2000	Castella, V., Ruedi, M., Excoffier, L., Ibáñez, C., Arlettaz, R., and Hausser, J. (2000). *Molecular Ecology* **9:** 1761–1772.
Cespedes et al., 2003	Céspedes, M., Gutierrez, M. V., Holbrook, N. M., and Rocha, O. J. (2003). *Molecular Ecology* **12:** 3201–3212.
Chase et al., 1996	Chase, M., Kesseli, R., and Bawa, K. (1996). *American Journal of Botany* **83:** 51–57.
Chen et al., 2003	Chen, X., Li, N., Shen, L., and Li, Y. (2003). *Environmental Pollution* **124:** 449–455.
Cimmaruta et al., 2003	Cimmaruta, R., Scialanca, F., Luccioli, F., and Nascetti, G. (2003). *Oceanologica Acta* **26:** 101–110.
Ciofi et al., 2002	Ciofi, C., and Bruford, M. W. (2002). *Molecular Ecology* **7:** 133–140.
Colson and Hughes, 2004	Colson, I., and Hughes, R. N. (2004). *Molecular Ecology* **13:** 2223–2233.
Coltman et al., 1998	Coltman, D. W., Bowen, W. D., and Wright, J. M. (1998). *Proceedings of the Royal Society of London B-Biological Sciences* **265:** 803–809.
Coltman et al., 1999	Coltman, D. W., Bancroft, D. R., Robertson, A., Smith, J. A., Clutton-Brock, T. H., and Pemberton, J. M. (1999). *Molecular Ecology* **8:** 1199–1209.
Comer et al., 2005	Comer, C. E., Kilgo, J. C., D'Angelo, G. J., Glenn, T. C., Miller, K. V. (2005). *Journal of Wildlife Management* **69:** 332–344.
Cronin et al., 2005	Cronin, M. A., Shideler, R., Waits, L., and Nelson, R. J. (2005). *Ursus* **16:** 70–84.
Cross and Rebordinos, 2003	Cross, I., and Rebordinos, L. (2003). *Ciencias Marinas* **29:** 239–250.
Davis and Strobeck, 1998	Davis, C. S., and Strobeck, C. (1998). *Molecular Ecology* **7:** 1771–1788.
De Oliveira et al., 2005	de Oliveira, M., Russo, C., Lazoski, C., Vianna, P., and Solé-Cava, A. (2005). *Genetics and Molecular Research* **4:** 197–202.
Descimon and Napolitano, 1993	Descimon, H. and Napolitano, M. (1993). *Biological Conservation* **66:** 117–123.
Dhuyvetter et al., 2005	Dhuyvetter, H., Gaublomme, E., Verdyck, P., and Desender, K. (2005). *Journal of Heredity* **96:** 381–387.
Diniz et al., 2005	Diniz, F. M., Maclean, N., Ogawa, M., Paterson, I. G., and Bentzen, P. (2005). *Conservation Genetics* **6:** 637–641.
Edwards et al., 2003	Edwards, T., Goldberg, C. S., Kaplan, M. E., Schwalbe, C. R., and Swann, D. E. (2003). *Molecular Ecology Notes* **3:** 589–591.

Appendix Table A1 continued

Abbreviated reference	Complete reference
Eggert et el, 2003	Eggert, L. S., Eggert, J. A., and Woodruff, D. S. (2003). *Molecular Ecology* **12:** 1389–1402.
Eizirik et al., 2001	Eizirik, E., Kim, J., Menotti-Raymond, M., Crawshaw Jr., P. G., O'Brien, S. J., and Johnson, W. E. (2001). *Molecular Ecology* **10:** 65–79.
Eldridge et al., 2004	Eldridge, M. D., Kinnear, J. E., Zenger, K. R., McKenzie, L. M., and Spencer, P. B. (2004). *Conservation Genetics* **5:** 325–328.
Escorza-Treviño and Dizon, 2000	Escorza-Treviño, S., and Dizon, A. E. (2000). *Molecular Ecology* **9:** 1049–1060.
Favre and Balloux, 1997	Favre, L., and Balloux, F. (1997). *Molecular Ecology* **6:** 595–596.
Fickel et al., 2005	Fickel, J., Schmidt, A., Putze, M., Spittler, H., Ludwig, A., Juergen Streich, W., and Pitra, C. (2005). *Journal of Wildlife Management* **69:** 760–770.
Fitzsimmons et al., 1995	Fitzsimmons, N. N., Buskirk, S. W., and Smith, M. H. (1995). *Conservation Biology* **9:** 314–323.
Flagstad et al., 2000	Flagstad, O., Syvertsen, P., Stenseth, N., Stacy, J. E., Olsaker, I., Roed, K. H., and Jakobsen, K. S. (2000). *Conservation Biology* **14:** 254–264.
Forbes and Boyd, 1996	Forbes, S. H., and Boyd, D. K. (1996). *Conservation Biology* **10:** 1082–1090.
Foré et al., 1992	Foré, S. A., Hickey, R. J., Vankat, J. L., Guttman, S. I., and Schaeffer, R. L. (1992). *Canadian Journal of Botany* **70:** 1659–1668.
Foré et al., 1995	Foré, S. A., Guttman, S. I., Bailer, A. J., Altfater, D. J., and Counts, B. V. (1995). *Ecotoxicology and Environmental Safety* **30:** 24–35.
Fraser et al., 2005	Fraser, D. J., Duchesne, P., and Bernatchez, L. (2005). *Molecular Ecology* **14:** 3133–3146.
Frati et al., 1992	Frati, F., Fanciulli, P. P., and Posthuma, L. (1992). *Biochemical Systematics and Evolution* **20:** 297–310.
Freville et al., 2001	Freville, H., Justy, F., and Olivieri, I. (2001). *Molecular Ecology* **10:** 879–889.
Fullard et al., 2000	Fullard, K. J., Early, G., Heide-Jørgensen, M. P., Bloch, D., Rosing-Asvid, A., and Amos, W. (2000). *Molecular Ecology* **9:** 949–958.
Funk et al., 1999	Funk, W. C., Tallmon, D. A., and Allendorf, F. W. (1999). *Molecular Ecology* **8:** 1633–1640.
Galeuchet et al., 2005	Galeuchet, D. J., Perrett, and C., Fischer, M. (2005). *Molecular Ecology* **14:** 991–1000.
Gao 2005	Gao, L. (2005). *Molecular Ecology* **14:** 4287–4297.
Garcia-Rodriguez et al., 2000	Garcia-Rodriguez, A. I., Moraga-Amador, D., Farmerie, W., McGuire, P., and King, T. L. (2000). *Molecular Ecology* **9:** 2155–2234.
Garza et al., 1997	Garza, J. C., Dallas, J., Duryadi, D., Gerasimov, S., Croset, H., and Boursot, P. (1997). *Molecular Ecology* **6:** 1009–1117.
Ge et al., 1998	Ge, S., Wang, K., Hong, D., Zang, W., and Zu, Y. (1999). *Conservation Biology* **13:** 509–513.
Gerlach and Hoeck, 2001	Gerlach, G., and Hoeck, H. N. (2001). *Molecular Ecology* **10:** 2307–2317.
Gerlach and Musolf, 2000	Gerlach, G., and Musolf, K. (2000). *Conservation Biology* **14:** 1066–1074.
Girman et al., 2001	Girman, D. J., Vilà, C., Geffen, E., Creel, S., Mills, M. G. L., McNutt, J. W., Ginsberg, J., Kat, P. W., Mamiya, K. H., and Wayne, R. K. (2001). *Molecular Ecology* **10:** 1703–1723.
Godt et al., 1996	Godt, M. J. W., Johnson, B. R., and Hamrick, J. L. (1996). *Conservation Biology* **10:** 796–805.
Godt and Hamrick, 1998	Godt, M. J. W., and Hamrick, J. L. (1998). *American Journal of Botany* **85:** 802–810.
Goldberg et al., 2003	Goldberg, C. S., Edwards, T., Kaplan, M. E., and Goode, M. (2003). *Molecular Ecology Notes* **3:** 539–541.
González-Astorga and Castillo-Campos, 2004	González-Astorga, J., and Castillo-Capos, G. (2004). *Annals of Botany* **93:** 521–528.
González-Astorga and Núñez-Farfán, 2001	González-Astorga, J., and Núñez-Farfán, J. (2001). *Evolutionary Ecology Research* **3:** 861–872.
Goodman et al., 2001	Goodman, S. J., Tamate, H. B., Wilson, R., Nagata, J., Tatsuzawa, S., Swanson, G. M., Pemberton, J. M., and McCullough, D. R. (2001). *Molecular Ecology* **10:** 1357–1370.
Goosens et al., 2001	Goosens, B., Chikhi, L., Taberlet, P., Waits, L. P., and Allainé, D. (2001). *Molecular Ecology* **10:** 41–52.
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Gottelli et al., 1994	Gottelli, D., Sillerozubiri, C., Applebaum, G. D., Roy, M. S., Girman, D. J., Garciamoreno, J., Ostrander, E. A., and Wayne, R. K. (1994). *Molecular Ecology Notes* **3:** 301–312.
Guillemin et al., 2000	Guillemin, M., Lavergne, A., and Catzeflis, F. (2000). *Molecular Ecology* **9:** 1433–1449.
Guinand et al., 2003	Guinand, B., Scribner, K. T., Page, K. S., and Burnham-Curtis, M. K. (2003). *Proceedings of the Royal Society of London B-Biological Sciences***270:** 425–433.

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Abbreviated reference	Complete reference
Gutiérrez-Espeleta et al., 2000	Gutiérrez-Espeleta, G. A., Kalinowski, S. T., Boyce, W. M., and Hedrick, P. W. (2000). *Conservation Genetics* **1:** 3–15.
Gutiérrez-Rodríguez and Lasker, 2004	Gutiérrez-Rodríguez, C., and Lasker, H. R. (2004). *Molecular Ecology* **13:** 2211–2221.
Hanfling et al., 2004	Hänfling, B., Durka, W., and Brandl, R. (2004). *Conservation Genetics* **5:** 247–257.
Hansen et al., 2002	Hansen, M. M., Ruzzante, D. E., Nielsen, E. E., Bekkevold, D., and Mensberg, K. D. (2002). *Molecular Ecology* **11:** 2523–2535.
Harley et al., 2005	Harley, E. H., Baumgarten, I., Cunningham, J., and O'Ryan, C. (2005). *Molecular Ecology* **14:** 2981–2990.
Hauser et al., 2002	Hauser, L., Adcock, G. J., Smith, P. J., Ramirez, J. H., and Carvalho, G. R. (2002). *Proceedings of the National Academy of Sciences of the United States of America* **99:** 11742–11747.
Heath et al., 2002	Heath, D. D., Busch, C., Kelly, J., and Atagi, D. Y. (2002). *Molecular Ecology* **11:** 197–214.
Heckel et al., 2000	Heckel, G., Achmann, R., and Mayer, F. (2000). *Molecular Ecology* **9:** 242.
Hedgecock 1978	Hedgecock, D. (1978). *Evolution* **32:** 271–286.
Hellborg et al., 2002	Hellborg, L., Walker, C. W., Rueness, E. K., Stacy, J. E., Kojola, I., Valdmann, H., Vilà, C., Zimmermann, B., Jakobsen, K. S., and Ellegren, H. (2002). *Conservation Genetics* **3:** 97–111.
Heuertz et al., 2001	Heuertz, M., Hausman, J. F., Tsvetkov, I., Frascaria-Lacoste, N., and Vekemans, X. (2001). *Molecular Ecology* **10:** 1615–1623.
Houlden et al., 1996	Houlden, B. A., England, P. R., Taylor, A. C., Greville, W. D., and Sherwin, W. B. (1996). *Molecular Ecology* **5:** 269–281.
Hughes et al., 1998	Hughes, C. R., Melland, R. R., and Beissinger, S. R. (1998). *Molecular Ecology* **7:** 1247–1248.
Hughes et al., 2003	Hughes, J. M., Mather, P. B., Toon, A., Ma, J., Rowley, I., and Russell, E. (2003). *Molecular Ecology* **12:** 3441–3450.
Ishibashi et al., 1996	Ishibashi, Y., Saitoh, Y., Abe, S., and Yoshida, M. C. (1995). *Molecular Ecology* **5:** 589–590.
Israel et al., 2004	Israel, J. A., Cordes, J. F., Blumberg, M. A., and May, B. (2004). *North American Journal of Fisheries Management* **24:** 922–931.
Jekielek and Strobeck, 1999	Jekielek, J., and Strobeck, C. (1999). *Molecular Ecology* **8:** 895–906.
Johnson et al., 1999	Johnson, W. E., Slattery, J. P., Eizirik, E., Kim, J., Raymond, M. M., Bonacic, C., Cambre, R., Crawshaw, P., Nunes, A., Seuánez, H. N., Moreira, M., Seymour, K. L., Simon, F., Swanson, W., and O'Brien, S. J. (1999). *Molecular Ecology* **8:** S79-S94.
Johnson et al., 2003	Johnson, J. A., Tpe[fer, J. E., and Dunn, P. O. (2003). *Molecular Ecology* **12:** 3335–3347.
Jones and Gliddon, 1999	Jones, B., and Gliddon, C. (1999). *Plant Ecology* **141:** 151–161.
Kang et al., 2005	Kang, M., Jiang, M., and Huang, H. (2005). *Annals of Botany* **95:** 1145–1151.
Kays et al., 2000	Kays, R. W., Gittleman, J. L., and Wayne, R. K. (2000). *Molecular Ecology* **9:** 743–751.
Keklak et al., 1994	Keklak, M. M., Newman, M. C., and Mulvey, M. (1994). *Archives of Environmental Contamination and Toxicology* **27:** 20–24.
Keller and Largiadèr, 2003	Keller, I. and Largiadèr, C. R. (2003). *Proceedings of the Royal Society of London B-Biological Sciences* **270:** 417–423.
Ketmaier et al., 2003	Ketmaier, V., Scapini, F., and De Matthaeis, E. (2003). *Estuarine, Coastal and Shelf Studies* **58S:** 159–167.
Kim and Sappington, 2005	Kim, K. S., and Sappington, T. W. (2005). *Environmental Entomology* **34:** 494–503.
Kirchhoff et al., 1999	Kirchhoff, S., Sévigny, J. M., and Couillard, C. M. (1999). *Marine Environmental Research* **47:** 261–283.
Knaepkens et al., 2004	Knaepkens, G., Bervoets, L., Verheyen, E., and Eens, M. (2004). *Biological Conservation* **115:** 403–410.
Korfanta et al., 2005	Korfanta, N. M., McDonald, D. B., and Glenn, T. C. (2005). *The auk* **122:** 464–478.
Kraaijeveld-Smit et al., 2005	Kraaijeveld-Smit, F. J. L., Beebee, T. J. C., Griffiths, R. A., Moore, R. D., and Schley, L. (2005). *Molecular Ecology* **14:** 3307–3315.
Kronforst and Fleming, 2001	Kronforst, M. R. and Fleming, T. H. (2001). *Heredity* **86:** 243–250.
Krutzen et al., 2004	Krützen, M., Barré, L. M., Connor, R. C., Mann, J., and Sherwin, W. B. (2004). *Molecular Ecology* **13:** 1975–1990.
Kuehn et al., 2004	Kuehn, R., Haller, H., Schroeder, W., and Rottmann, O. (2004). *Journal of Heredity* **95:** 136–143.
Kyle et al., 2004	Kyle, C. J., Weir, R. D., Newhouse, N. J., Davis, H., and Strobeck, C. (2004). *Journal of Mammology* **85:** 633–639.
Lacey 2001	Lacey, E. A. (2001). *Heredity* **86:** 629–637.
Lade et al., 1996	Lade, J. A., Murray, N. D., Marks, C. A., and Robinson, N. A. (1996). *Molecular Ecology* **5:** 81–87.

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Abbreviated reference	Complete reference
Lampert et al., 2003	Lampert, K. P., Rand, A. S., Mueller, U. G., and Ryan, M. J. (2003). *Molecular Ecology* **12:** 3325–3334.
Larno et al., 2001	Larno, V., Laroche, J., Launey, S., Flammarion, P., and Devaux, A. (2001). *Ecotoxicology* **10:** 145–158.
Larson et al., 2002	Larson, S., Jameson, R., Bodkin, J., Staedler, M., and Bentzen, P. (2002). *Journal of Mammology* **83:** 893–906.
Lee et al., 2001	Lee, P. L. M., Bradbury, R. B., Wilson, J. D., Flanagan, N. S., Richardson, L., Perkins, A. J., and Krebs, J. R. (2001). *Molecular Ecology* **10:** 1633–1644.
Lefant 2003	Lefant, P. (2003). *Comptes Rendus Biologies* **326:** 751–760.
Lesbarèrres et al., 2003	Lesbarèrres, D., Pagano, A., and Lodé, T. (2003). *Comptes Rendus Biologies* **326:** S68-S72.
Libants et al., 2000	Libants, S., Olle, E., Oswald, K., and Scribner, K. T. (2000). *Molecular Ecology* **9:** 1433–1449.
Longauer et al., 2004	Longauer, R., Gömöry, D., Paule, L., Blada, I., Popescu, F., Mankovska, B., Müller-Starck, G., Schubert, R., Percy, K., Szaro, R. C., and Karnosky, D. F. (2004). *Environmental Pollution* **130:** 85–92.
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Appendix Table A1 continued

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Rajora et al., 2000	Rajora, O. P., Rahman, M. H., Buchert, G. P., and Dancik, B. P. (2000). *Molecular Ecology* **9:** 339–348.
Ranker et al., 1994	Ranker, T. A. (1994). *Biological Conservation* **70:** 19–24.
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Roach et al., 2001	Roach, J. L., Stapp, P., Van Horne, B., and Antolin, M. F. (2001). *Journal of Mammology* **82:** 946–959.
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Røed and Midthjell, 1998	Røed, K. H., and Midthjell, L. (1998). *Molecular Ecology* **7:** 1771–1788.
Rooney et al., 1999	Rooney, A. P., Honeycutt, R. L., Davis, S. K., and Derr, J. N. (1999). *Journal of Molecular Evolution* **49:** 682–690.
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Roy et al., 1994	Roy, M. S., Geffen, E., Smith, D., Ostrander, E. A., and Wayne, R. K. (1994). *Molecular Biology and Evolution* **11:** 553–570.
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Sarno et al., 2001	Sarno, R. J., Franklin, W. L., O'Brien, S. J., and Johnson, W. E. (2001). *Animal Conservation* **4:** 93–101.
Schmidt 1999	Schmidt, C. A. (1999). *Journal of Mammology* **80:** 522–529.
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Stevens et al., 1997	Stevens, S., Coffin, J., and Strobeck, C. (1997). *Molecular Ecology* **6:** 493–495.
Stewart et al., 1999	Stewart, W. A., Dallas, J. F., Piertney, S. B., Marshall, F., Lambin, X., and Telfer, S. (1999). *Biological Journal of the Linnean Society* **68:** 159–171.
Stow et al., 2001	Stow, A. J., Sunnucks, P., Briscoe, D. A., and Gardner, M. G. (2001). *Molecular Ecology* **10:** 867–878.
Sumner et al., 2004	Sumner, J., Jessop, T., Paetkau, D., and Moritz, C. (2004). *Molecular Ecology* **13:** 259–269.
Tallmon et al., 2002	Tallmon, D. A., Draheim, H. M., Mills, L. S., and Allendorf, F. W. (2002). *Molecular Ecology* **11:** 699–709.
Taylor et al., 2000	Taylor, A. C., Cowan, P. E., Fricke, B. L., Cooper, and D. W. (2000). *Molecular Ecology* **9:** 869–879.
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Appendix Table A1 continued

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Thomas et al., 1999	Thomas, B. R., Macdonald, S. E., Hicks, M., Adams, D. L., and Hodgetts, R. B. (1999). *Theoretical and Applied Genetics* **98:** 793–801.
Tsuda and Ide, 2005	Tsuda, Y., and Ide, Y. (2005). *Molecular Ecology* **14:** 3929–3941.
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Van Dongen et al., 1998	Van Dongen, S., Backeljau, T., Matthysen, E., and Dhondt, A. A. (1998).*Heredity* **80:** 92–100.
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Waldick et al., 2002	Waldick, R. C., Kraus, S., Brown, M., and White, B. N. (2002). *Molecular Ecology* **11:** 2241–2249.
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