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Short communication

Relative effects of road mortality and decreased connectivity on population genetic diversity

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ABSTRACT

Roads can have two important effects on populations that impact genetic variation: reduced gene flow and reduced abundance. Reduced gene flow ("barrier effects") due to road avoidance behavior or road mortality can lead to reduced genetic diversity because genetic drift is enhanced in fragmented populations. Road mortality can also reduce population abundance ("depletion effects") whenever road-caused mortality outpaces recruitment, also lowering diversity even when barrier effects are inconsequential. Although roads are expected to affect both genetic diversity and fragmentation, most research focuses only on fragmentation. Furthermore, in studies that do investigate road effects on genetic diversity, correlations are usually attributed to barrier effects and little attention is paid to the potentially confounding influence of mortality-caused depletion effects. Here we investigate the relative importance of barrier and depletion effects on genetic diversity of populations separated by a road by performing coalescent simulations wherein these two road effects are varied independently. By simulating wide ranging rates of migration and population decline, we also determine how the importance of these forces changes depending on their relative magnitude. We show that the vast majority of potential variation in genetic diversity is governed by depletion (mortality) rather than barrier effects. We also show that unless migration is sufficiently high and population decline due to mortality is sufficiently low, increasing migration across roads will generally not recoup genetic variation lost due to road mortality. We argue that the genetic effects of road-mediated mortality have been underappreciated and should be more often considered before prioritizing road-mitigation measures.

1. Introduction

One of the fundamental goals of conservation biology is to preserve genetic diversity (Avise, 1996; Schonewald-Cox et al., 1983). Consequently, when evaluating the influence of roads and traffic on patterns of population genetic variation, genetic diversity is one of the parameters we are most interested in tracking. The impact of roads on population connectivity and abundance are of concern to conservation geneticists largely because they are thought to ultimately reduce genetic diversity (Frankel and Soulé, 1981). By understanding the relative contribution of different road effects to the erosion of population genetic diversity, we can better devise measures of road mitigation.

When organisms interact with roads, road avoidance and mortality are two well-known outcomes that can contribute to population genetic diversity decline (Forman et al., 2003). Road avoidance behavior limits dispersal across roads. Reduced gene flow between populations separated by roads genetically fragments populations, leading to lower effective population sizes. This heightens the power of genetic drift to erode genetic diversity within populations (Wright, 1931). Road avoidance thus results in a "barrier effect" that is expected to decrease diversity within populations while increasing divergence between populations.

Mortality on roads due to collision of individuals with on-coming vehicles has been well-documented in many animal species (reviewed in Forman et al., 2003). Mortality can depress population abundance if sufficiently high relative to background mortality rates (reviewed in Fahrig and Rytwinski, 2009). This decreased abundance or population "depletion effect" also leads to loss of genetic variation due to genetic drift. However, mortality on roads also entails a barrier effect because it eliminates would-be road-crossers. Thus, if road-kill rates are high, an especially negative effect on genetic diversity is expected given that a reduction in abundance and connectivity may be occurring simultaneously.

Despite the fact that both depletion and barrier effects of roads can contribute to a decline in genetic diversity, barrier effects have attracted a majority of the attention from landscape and conservation geneticists. A 2009 review of road genetics research found that nearly twice as many studies tested the influence of roads on gene flow or genetic fragmentation (consequences of a barrier effect) as

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on genetic diversity (Balkenhol and Waits, 2009). Our own examination of the literature since that review (August 2009–April 2011) yielded 21 additional studies that investigate the genetic effects of roads, only two of which report road effects on genetic diversity (see Table A.1 in Supplementary material). Of studies that do examine road effects on diversity, most attribute a detected negative relationship to diminished connectivity across roads (e.g., Epps et al., 2005; Keller et al., 2005; Reh and Seitz, 1990; Tamura and Hayashi, 2007), even though such a relationship is also consistent with the effects of lower abundance alone (i.e., in the absence of reduced connectivity).

Thus, it is unclear whether this focus on the barrier effect of roads is proportional to its relative influence on genetic diversity. For example, although barrier effects may be more pervasive than depletion effects (given that both mortality and road avoidance can lead to reduced gene flow across roads) and can quickly generate genetic divergence (Keyghobadi, 2007), continual reduction in population abundance due to road mortality is potentially an additive process and may have a greater long-term impact on genetic diversity. Also, the relative importance of barrier and depletion effects may depend upon the particular levels of road avoidance and mortality in a population. However, given that the effects of avoidance and mortality are confounded, it is difficult to tease apart their relative impacts on population genetic diversity by analyzing samples from natural populations.

Here we simulate the evolution of populations separated by a road where we allow depletion (reduced abundance) and barrier effects (reduced gene flow) to vary independently. This allows us to investigate the potential relative influence of these two effects on patterns of genetic variation across a large region of the parameter space. We expect that free crossing of roads will result in increased genetic diversity relative to complete road-avoidance if there is no mortality risk, but will lead to decreased diversity relative to road-avoidance if reduction in population size due to mortality of crossers is extremely high. However, when both mortality and gene flow rates are moderate, the dynamics that drive genetic diversity patterns are less clear. In this study, by monitoring genetic diversity outcomes across factorial combinations of migration and depletion rates, we address the questions, (1) what level of successful migration across roads must be attained to recoup the genetic diversity lost due to road-induced mortality and (2) are genetic diversity outcomes more sensitive to changing rates of connectivity or mortality-caused population decline?

2. Materials and methods

2.1. The simulation model

We used the program Simcoal v2.1.2 (Laval and Excoffier, 2004) to simulate the evolution of microsatellite loci under a variety of scenarios involving a road that imposes varying depletion and barrier effects on a population. In all cases, an ancestral, randomly mating population was divided into two by a road at a particular time t in the past. We performed the simulation across two different sample sizes ($2N$ = 250 and 2500 diploid individuals within each daughter population). To simulate depletion effects, we applied varying rates of negative exponential population growth (r = 0, −0.0001, −0.0005, −0.001, −0.005, −0.01, −0.05, and −0.1, where r equals the percent change in population size per generation). We therefore assumed that road mortality is sufficient to result in continuous population decline and that reproductive rate is not compensatory, and we varied the rate of that decline to represent different mortality rates. To simulate barrier effects, we varied rates of migration between the two populations (m = 0, 0.0001, 0.001, 0.01, 0.1, and 0.25, where m equals the proportion of migrants that successfully cross the road per generation); 0 represents a complete barrier effect whereas 0.25 represents no barrier. Time since construction of the road was also varied (t = 10, 50, 100, and 500 years before present, assuming that generation time was 1 year). We performed the simulation using a full factorial design ($8r \times 6m \times 4t \times 2(2N)$ = 384 total factor combinations) where each scenario was simulated 1000 times using a different random number seed each time.

For each iteration, 50 unlinked microsatellite loci were simulated within each of the two populations. Microsatellites evolved at a rate of $\mu = 1 \times 10^{-4}$ mutations per locus per generation, a value typically observed within vertebrates (Bhargava and Fuentes, 2010). Microsatellites were simulated under a strict stepwise mutation model without range constraints and without recombination. The program Simcoal simulates under a coalescent model (Kingman, 1982a,b), and thus, although we have described the model forward in time, scenarios were simulated in the reverse direction.

2.2. Data analysis

Upon completion of each simulation, 100 diploid samples were drawn from each daughter population from which diversity and divergence metrics were calculated. Using Arlcore v3.5.1.2 (Excoffier and Lischer, 2010), we calculated two common metrics of genetic diversity (H = mean expected heterozygosity and A = mean allelic richness) and one metric of divergence (F_{ST}) to assess the effects of factor combinations on patterns of genetic variation.

To determine the relative influence of depletion and barrier effects on genetic outcomes, we performed multiple regressions where response variables were the three diversity/divergence metrics and predictor variables were levels of migration (m) and population decline (r; both treated as categorical data). Response variables were standardized to facilitate their comparison. For each response, we repeated three regressions where m and r were included separately and jointly in the model. This was repeated for t = 10, 50, 100, and 500 generations. Akaike information criterion (AIC) values were calculated to determine the information value of each variable when predicting genetic diversity outcomes, and R^2 values were calculated to compare model fit. Variable coefficients from the full model were used to determine the impact of depletion and barrier effects at each simulated level. We carried out analyses using the R package (R Development Core Team, 2010).

3. Results

3.1. Relative influence of changing depletion and barrier effects on diversity

Nearly all variation in genetic diversity is governed by depletion rather than barrier effects (Fig. 1 and Table 1). When initial population size is large (2N = 2500), rate of negative population growth (r) alone explains most of the variation in H (R^2 = 0.81, 0.98, 0.98, and 0.99 for t = 10, 50, 100, and 500 datasets, respectively) and A (R^2 = 0.82, 0.95, 0.96, and 0.97 across the four time periods). Because r and m are completely uncorrelated, R^2 values can be attributed completely to these marginal effects.

In accordance with expectations supported by theory (Wright, 1931) and previous simulation studies (Chakraborty and Nei, 1977; Maruyama and Fuerst, 1985; Nei et al., 1975), the effect of r on H and A is small at high r values, but promotes large declines in H and A at low r values (particularly when $r \leqslant -0.01$; Fig. 1 and Table A.2). After 10 generations of road effects, decreasing r slightly (from 0 to −0.0001) results in essentially no change in H or A, but decreasing r dramatically (from 0 to −0.1) results in a 1.05 SD

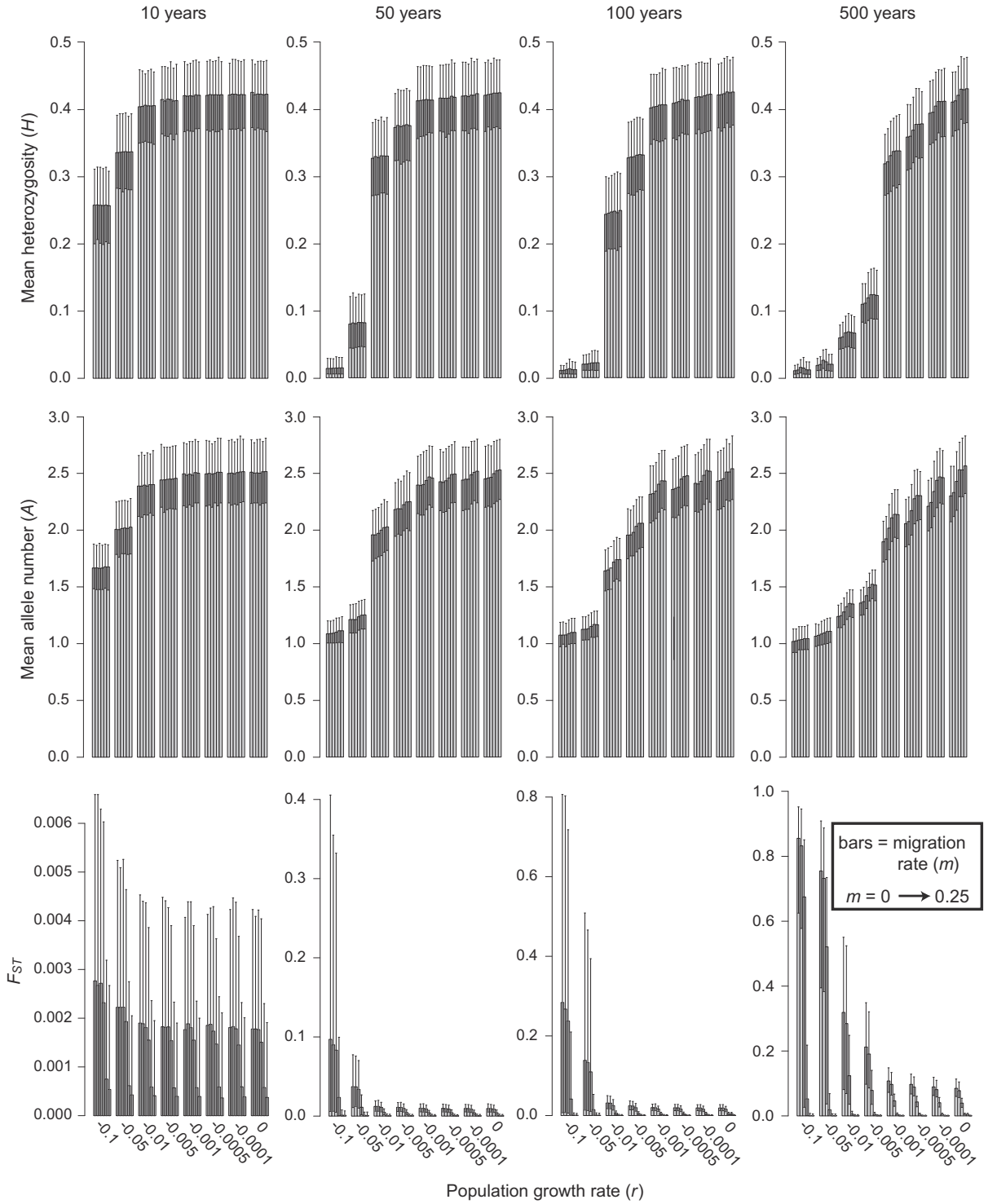

Fig. 1. Relative influence of road effects on genetic diversity. Estimates of mean heterozygosity, allelic richness, and F_{ST} across varying simulated road effects: migration rate, negative growth rate, and time period since road effects were implemented. Population size = 2500 diploid individuals in all simulations. Individual bars indicate values across increasing migration rate (from left to right: $m = 0$, 0.0001, 0.001, 0.01, 0.1, 0.25). Groups of bars indicate values across increasing rates of population growth (as indicated on the *x*-axis). Ninety-five percent confidence intervals are shown using bars for each simulation. Note the differences in scale for plots of F_{ST} across time periods.

increase in H or a 1.52 SD increase in A. Also, the influence of r on genetic diversity becomes much larger as road effects are in place for longer periods of time. After 500 generations of road effects, decreasing r slightly (from 0 to −0.0001) results in a 0.11 SD increase in H or a 0.17 SD increase in A, but decreasing r dramatically (from 0 to −0.25) results in a 2.66 SD increase in H or a 2.79 SD increase in A.

In contrast, migration (m) alone explains a trivial amount of the variation in H ($R^2 = 0.00$ for all four time periods) and A ($R^2 = 0.00$, 0.00, 0.01, and 0.02 across the four time periods), regardless of the number of generations the road is in place (Fig. 1 and Table 1). In the short run (10 generations), the effects of m on H and A are negligible and generally not significant (Wald's test $p \geqslant 0.05$; Table A.2). However, in the long run (500 generations), effects are small

Table 1
Absolute AIC scores, changes in AIC score, and R^2 values for all models analyzed using regression when initial population size is large (2N = 2500). AIC scores are compared for full (m = migration + r = growth) and reduced models. Regressions were repeated across three different response variables (H = heterozygosity, A = allelic richness, and F_{ST}) and across four time periods. Model weights (i.e., probability a model is the "true" model) are 1.0 for the full model in all cases except for H at time 10, where the weight for model $H \sim r$ = 0.96.

Time	Heterozygosity (H)				Allelic richness (A)				F_{ST}			
	Model	AIC	ΔAIC	R^2	Model	AIC	ΔAIC	R^2	Model	AIC	ΔAIC	R^2
10	$H \sim r$	−33,086	0	0.81	$A \sim m + r$	1947	0	0.82	$F_{ST} \sim m + r$	−332,981	0	0.30
10	$H \sim m + r$	−33,080	6	0.81	$A \sim r$	2013	66	0.82	$F_{ST} \sim m$	−331,087	1894	0.27
10	$H \sim m$	46,841	79,927	0.00	$A \sim m$	84,089	82,141	0.00	$F_{ST} \sim r$	−317,624	15,357	0.03
50	$H \sim m + r$	−43,270	0	0.98	$A \sim m + r$	−8816	0	0.95	$F_{ST} \sim m + r$	−21,883	0	0.29
50	$H \sim r$	−43,214	56	0.98	$A \sim r$	−6615	2201	0.95	$F_{ST} \sim r$	−15,554	6329	0.19
50	$H \sim m$	135,357	178,627	0.00	$A \sim m$	137,365	146,181	0.00	$F_{ST} \sim m$	−10,376	11,507	0.10
100	$H \sim m + r$	−47,280	0	0.98	$A \sim m + r$	−13,396	0	0.96	$F_{ST} \sim m + r$	87,559	0	0.27
100	$H \sim r$	−47,095	185	0.98	$A \sim r$	−8192	5204	0.96	$F_{ST} \sim r$	93,127	5569	0.18
100	$H \sim m$	140,873	188,153	0.00	$A \sim m$	140,556	153,952	0.01	$F_{ST} \sim m$	98,221	10,662	0.09
500	$H \sim m + r$	−61,798	0	0.99	$A \sim m + r$	−20,467	0	0.97	$F_{ST} \sim m + r$	139,407	0	0.63
500	$H \sim r$	−58,318	3479	0.99	$A \sim r$	−1856	18,611	0.95	$F_{ST} \sim r$	168,939	29,532	0.32
500	$H \sim m$	145,397	207,195	0.00	$A \sim m$	144,871	165,338	0.02	$F_{ST} \sim m$	169,213	29,806	0.31

but significant: increasing m slightly (from 0 to 0.0001) results in a 0.01 SD increase in H or a 0.04 SD increase in A, but increasing m dramatically (from 0 to 0.25) results in a 0.08 SD increase in H or a 0.33 SD increase in A.

Although the effects of population decline and migration on H and A are similar, A responds to these effects more quickly (see effect sizes in Table A.2). This is in line with previous studies (Cornuet and Luikart, 1996; Nei et al., 1975) and stems from the fact that many rare alleles are lost in the first few generations after effective population size reduction, whereas more time is required for remaining alleles to stochastically shift away from intermediate frequencies.

Similar patterns were observed when initial population size is small (2N = 250): r alone explained most of the variation in H and A (R^2 for H = 0.40, 0.75, 0.77, and 0.80 and R^2 for A = 0.14, 0.34, 0.34, and 0.34 across the four consecutive time periods; see Fig. A.1 and Tables A.3 and A.4). Migration (m) alone explained little variation in H and A (R^2 for H = 0.00, 0.00, 0.01, 0.03 and R^2 for A = 0.01, 0.03, 0.04, and 0.06). The reduced explanatory value of r in smaller (2N = 250) relative to larger (2N = 2500) populations likely reflects the lower amount of starting variation in smaller populations.

Although migration generally has a much smaller effect than population depletion on patterns of diversity, an impact of migration is still nearly always detectable. Except in one case, AIC favors models that include both m and r over models that only include r (Table 1), indicating that, although its effect is usually very small (as indicated by low R^2 values), migration does provide information regarding genetic diversity outcomes. Furthermore, although depletion effects always dominate over migration effects in the short term (t = 10 or 50 years), in the long term there are special cases when moderate to high migration across a road can make up for moderate losses in diversity due to occasional mortality. For example, after 500 generations, if there is a very small risk of population decline (no steeper than $r = -0.0005$), as long as road-crossing will lead to moderate or high levels of inter-population migration (m = 0.001 or greater), then a population tends to be at least as well off (if not better off) crossing roads than avoiding them (Fig. 2). However, this is only true when initial population size is large (2N = 2500). When initial population size is small (2N = 250), no strategy results in greater genetic diversity than complete road avoidance, even in the long run (i.e., after 500 generations).

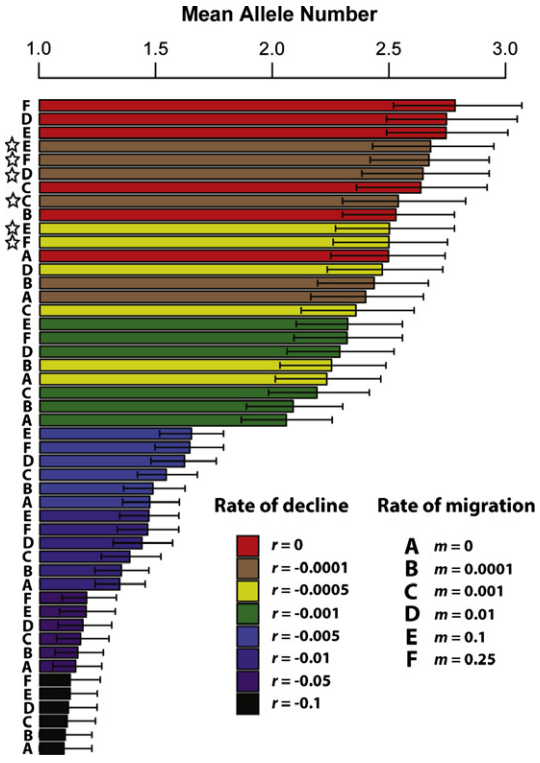

Fig. 2. The combined effects of population migration and depletion as ranked by their long term impact on genetic diversity. Mean allelic richness outcomes from coalescent simulations are shown, where two populations separated by a road experienced varying rates of population decline (r) and migration (m), and where simulations were run for 500 generations with large initial population size (2N = 2500). Scenarios are ranked by their effect on allelic richness. There are only six cases (indicated with a star) where non-zero population decline yields higher allelic richness than cases where population decline equals zero. Only when very low decline risk ($r \leqslant -0.0005$) is coupled with fairly high rates of migration ($m \geqslant 0.001$) can migration potentially compensate for diversity lost due to population decline.

3.2. Road effects on divergence

The effects of population decline and migration on divergence conform to expectations (Keyghobadi, 2007) and are opposite their effects on diversity: increased migration rate and decreased population decline result in lower values of F_{ST} (Fig. 1 and Table 1). As observed in past simulation studies (Crow and Aoki, 1984; Varvio et al., 1986) F_{ST} responds more quickly to these effects relative to genetic diversity. Although 10–50 generations is generally not long enough for any parameter combination besides those involving extreme population decline to register a significant effect on diversity, this is sufficient time for dramatic effects on F_{ST} to take place. Also, in contrast to the diversity metrics,

migration and population decline have similar influences on F_{ST} (for 2N = 2500, R^2 for $F_{ST} \sim m$ = 0.27, 0.10, 0.09, and 0.31 across the four time periods, whereas R^2 for $F_{ST} \sim r$ = 0.03, 0.19, 0.18, and 0.32). The effect of migration is evident immediately (at t = 10), whereas the effect of decline requires more time (Fig. 1 and Tables 1 and A.2). The impact of population decline on F_{ST} is much lower relative to migration when populations are small (2N = 250; Fig. A.1 and Tables A.3 and A.4). This is likely due to the minimal genetic variation within small (and shrinking) populations from which divergence can arise. Note that population decline alone does not cause genetic fragmentation but rather heightens the rate of fragmentation already occurring due to restricted gene flow.

4. Discussion

Although the barrier effect of roads does contribute to reduced genetic diversity after a sufficient time lag, reduction in population size due to traffic mortality generally has a much larger impact on genetic diversity. Regardless whether gene flow is free or completely eliminated, genetic variation is usually preserved best when there is no population decline (no road mortality). Only when populations are sufficiently large (2N $\geqslant$ 2500), and enough time has passed ($t \geqslant 100$), and migration rates are moderate to high ($m \geqslant 0.001$), and rates of decline are low ($r \geqslant -0.0005$) can the genetic benefits of migration offset the genetic costs of population depletion. One possible reason for the dominance of mortality is the longer duration of its effects. In the worst case scenario for barrier effects (m = 0), population size is reduced (halved, in this case) in the first generation after road construction, after which diversity gradually declines to equilibrium levels expected in populations half the size of the original population. However, as simulated here, mortality on the road produces a continual decline in population size, resulting in an additive effect on diversity that can have huge consequences for populations in the long run.

If mortality imposes a disproportionately negative influence on genetic diversity, then why have genetic fragmentation and reduced gene flow—rather than genetic diversity—been the most commonly assessed effects of roads? First, sampling design can be more straightforward when testing for barrier effects. When investigating whether roads are reducing population genetic diversity, sampling of replicate roads and control sites (sites away from roads) is required because diversity near a road can only be reduced *relative* to other road and non-road sites. In contrast, one can test the hypothesis that a particular road is limiting gene flow by simply sampling sites on opposite sides of the road (although stronger inferences can be made when replicate road and control sites are also included; Balkenhol and Waits, 2009). In addition, because genetic divergence responds more quickly than genetic diversity to road effects, metrics that assess fragmentation are more likely to yield a pattern than metrics that assess diversity, and may thus be more attractive to (and more often reported by) researchers (although very rapid responses of genetic diversity to roads have been reported; Epps et al., 2005; Lesbarrères et al., 2006; Reh and Seitz, 1990).

Depletion effects may also have been ignored in previous studies because of the conflation of barrier and depletion effects resulting from disperser mortality. Because both road-mediated mortality and avoidance restrict dispersal across roads, it can superficially seem reasonable to assume that measuring fragmentation will adequately capture the impact of both of these forces on natural populations. This however ignores the strong possibility (based on our results) that depletion effects due to mortality have a much greater effect on genetic diversity than barrier effects resulting from both mortality and avoidance. Thus, it is important to assess genetic diversity of populations directly to obtain an accurate assessment of the overall effect of roads on patterns of genetic variation.

The relative influence of road-mediated mortality depends on the range of population depletion simulated. Although it is difficult to measure the rate of depletion due to roads in nature, there is some evidence that these rates can be very high. Significant proportions of crossing attempts result in death in a variety of organisms (e.g., Bouchard et al., 2009; Freeman, 2010; McCall et al., 2010; Row et al., 2007; Soluk, 2011) and roads are thought to be the largest cause of mortality in several medium to large-sized vertebrates (reviewed in Forman et al., 2003). Such mortality often leads to reduction in population size (reviewed in Benítez-López et al., 2010; Fahrig and Rytwinski, 2009). Hels and Buchwald (2001) estimated that roads kill 5–20% of the population per year for three species of frog, while Keller et al. (2005) found a 30–100-fold reduction in effective population size of flightless beetles over a time frame commensurate with the construction of major roads that divide forest habitat.

Even if depletion effects do not commonly occur at the high end of the range employed here, they still generally dominate barrier effects. When our regression analysis is restricted to $r > -0.01$, population decline (r) predicts 0.02–0.95 of the variation in H across the four time periods, whereas migration (m) predicts only 0.00–0.01 of the variation in H. When $r \geqslant -0.001$, r predicts 0.00–0.64 of the variation in H, whereas m predicts 0.00–0.03 of the variation in H. Also, if depletion effects are reduced by lower mortality risk, barrier effects would also decline due to increased dispersal success.

In our simulation, we have ignored some consequences of limited population connectivity due to roads that can also ultimately contribute to population depletion, and thus genetic diversity loss. For example, if roads divide habitat into isolated fragments such that resources vital to life history (e.g., eating, nesting, or mating) are inaccessible, then roads will have a greater impact on diversity than indicated here. Also, if road avoidance entails avoidance of areas near roads, barrier effects can also reduce habitat amount (thus leading to reductions in population abundance and genetic variation; e.g., Stewart et al., 2010).

There are also factors that may moderate the effects of mortality on population abundance decline. For example, mortality rates heightened due to roads may be somewhat compensated for by a population response to depressed population density (e.g., via reduced intraspecific competition). Also, populations depleted by road kills may be replenished by immigration from surrounding populations that are less affected by roads. This may be particularly common for abundant, wide-ranging species (Forman and Alexander, 1998).

Even so, this simulation demonstrates that the potential impact of mortality per se on genetic diversity of natural populations (i.e., when barrier effects of mortality are ignored) may be very high, a fact that has been underappreciated in road genetics research. We argue that more focus should be placed on assessing the influence of road-mediated mortality on genetic diversity. It is not sufficient to assess the influence of roads on genetic fragmentation, given that fragmentation can be caused by both road avoidance and mortality which can have different consequences on genetic variation. Thus, whenever possible, genetic diversity and divergence due to roads should be estimated simultaneously such that the relative influence of depletion and barrier effects can be assessed. Additionally, more studies are needed that investigate how often road mortality contributes to population decline and the shape of that decline over time (e.g., linear, exponential, density-dependent). This can be done by measuring population size at temporal intervals or by testing for a genetic signature of negative growth that coincides with road construction. Also, a contribution by road mortality should be considered whenever a pattern of declining genetic variation is observed.

The dominance of depletion effects indicated here suggests that when weighing road mitigation options, we should keep in mind that only in rare cases can successful migration across roads outweigh deleterious impacts of disperser mortality. Thus, mitigation measures that minimize mortality on roads (such as the construction of fences) should more effectively promote genetic diversity in the long run than measures that attempt to promote connectivity (such as construction of wildlife overpasses). When mortality risk is substantial, building fences is also expected to increase the probability of population persistence (Jaeger and Fahrig, 2004). By basing conservation policy on the barrier effects of roads, we are failing to address the aspect of roads that is contributing most to genetic diversity decline. By investigating and distinguishing the consequences of road-based mortality and avoidance on genetic diversity, we will be able to better develop ways to minimize the deleterious effects of roadways and traffic on natural populations.

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Appendix A. Supplementary material

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.biocon.2011.09.010.

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