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# Using a genetic network to parameterize a landscape resistance surface for fishers, *Martes pennanti*

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## Abstract

**Knowledge of dispersal-related gene flow is important for addressing many basic and applied questions in ecology and evolution. We used landscape genetics to understand the recovery of a recently expanded population of fishers (*Martes pennanti*) in Ontario, Canada. An important focus of landscape genetics is modelling the effects of landscape features on gene flow. Most often resistance surfaces in landscape genetic studies are built a priori based upon nongenetic field data or expert opinion. The resistance surface that best fits genetic data is then selected and interpreted. Given inherent biases in using expert opinion or movement data to model gene flow, we sought an alternative approach. We used estimates of conditional genetic distance derived from a network of genetic connectivity to parameterize landscape resistance and build a final resistance surface based upon information-theoretic model selection and multi-model averaging. We sampled 657 fishers from 31 landscapes, genotyped them at 16 microsatellite loci, and modelled the effects of snow depth, road density, river density, and coniferous forest on gene flow. Our final model suggested that road density, river density, and snow depth impeded gene flow during the fisher population expansion demonstrating that both human impacts and seasonal habitat variation affect gene flow for fishers. Our approach to building landscape genetic resistance surfaces mitigates many of the problems and caveats associated with using either nongenetic field data or expert opinion to derive resistance surfaces.**

*Keywords*: circuit theory, graph theory, landscape genetics, landscape resistance, *Martes pennanti*, network analysis

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## Introduction

Identifying factors that affect dispersal is important for both fundamental and applied questions in ecology and evolution as dispersal has important implications for the genetic structure and dynamics of populations (Slatkin 1985; Clobert *et al*. 2001). Because dispersal patterns can induce genetic structure and are often affected by habitat, we can use genetic structure to infer important habitat characteristics that influence dispersal (Manel *et al*. 2003; Storfer *et al*. 2007). Increasingly, landscape geneticists are seeking to use resistance surfaces to test hypotheses about relationships among gene flow, spatial population structure, and habitat suitability (e.g. Cushman *et al*. 2006; McRae 2006; Spear *et al*. 2010). Resistance surfaces are simply maps where the ease of movement (i.e. resistance) through map elements is identified. Such surfaces are frequently used in corridor design (Beier *et al*. 2009) and more generally, for landscape planning (Urban & Keitt 2001; Fall *et al*. 2007; Minor & Lookingbill 2010). Thus, resistance surfaces can provide a framework for testing hypotheses about landscape structures facilitating or impeding gene flow.

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We were interested in using measures of genetic structure to understand how landscape features affected fisher (*Martes pennanti*) gene flow during a population expansion in Ontario, Canada. By the mid-1900s, fishers were extirpated from much of Ontario as a result of fur harvest, predator control, and habitat loss (Powell 1993). Since then populations have recovered, likely owing to a reduction in the extent of agriculture and improved fur harvest management (de Vos 1964; Lancaster *et al.* 2008). Previous landscape genetic studies of these data have shown that contemporary fisher populations in Ontario expanded from remnant populations (Carr *et al.* 2007a; b; Garroway *et al.* 2008). During the expansion, high-quality habitat patches were net producers of emigrants and dispersal was likely density dependent, from high to low quality habitat (Carr *et al.* 2007a; b; Garroway *et al.* 2008). Our previous analyses focused on habitat quality within the patches sampled for fishers; however, we also hypothesized that during the population expansion, fisher gene flow was influenced by landscape characteristics beyond the sampled patches, and testing this landscape hypothesis is the focus of our current study. We predicted that the fisher gene flow was facilitated by high-quality habitat between sampled patches, and impeded by linear barriers.

Habitat use by fishers is well studied; thus, we were able to select a set of candidate variables that we suspected to be generally important for fishers. High-quality fisher habitat is characterized by low snow depth, which likely reduces the cost of locomotion, and the extent of coniferous forest cover, which is likely related to food availability (Krohn *et al.* 1995; Carr *et al.* 2007b; Garroway *et al.* 2008). We suspected that rivers that remain ice-free in the winter would impede gene flow as juvenile fishers disperse during winter. Similarly, we suspected that roads represent barriers to fisher gene flow, both because of a direct barrier effect of roads, but also because of indirect effects of increased human activity near roads (Kelly 1977; Hodgman *et al.* 1994; Wisely *et al.* 2004; Jaeger *et al.* 2005). Thus, we used snow depth, coniferous forest cover, river density, and road density as independent variables to test hypotheses about the effects of landscape resistance on fisher gene flow during the range expansion. We expected that coniferous forest would facilitate gene flow, and that deep snow and high densities of paved roads and ice-free rivers would impede gene flow. By limiting our variables to those that we hypothesized a priori to be generally important, we aimed to avoid over-fitting our models (Burnham & Anderson 2002); that is, we aimed to avoid developing a surface that predicted gene flow only in the study area. With this model we built a multivariate, continuous surface depicting landscape resistance to fisher gene flow.

A major challenge in using resistance surfaces is parameterizing resistance values of different landscape elements. There are two prevailing approaches for assigning resistance values to surfaces: nongenetic field data and expert opinion (Spear *et al.* 2010). Nongenetic field data have long been used to assess landscape resistance to animal movement (Adriaensen *et al.* 2003; O'Brien *et al.* 2006); however, their utility for developing maps of genetic resistance has been questioned on at least two counts (Spear *et al.* 2010; Wang & Summers 2010). Typically, such data are available on a much smaller spatial scale than are required for landscape level inference in genetic studies. In addition, movement or other demographic data may not be relevant in predicting genetic structures. The second approach, expert opinion, involves assigning arbitrary, but relative, costs of travelling through different landscape elements followed by the estimation of resistance distances. This is the approach often taken in least cost modelling (e.g. Broquet *et al.* 2006). Although expert opinion is widely used to generate landscape resistances, it is potentially inaccurate, and rarely transparent (Rayfield *et al.* 2010; Spear *et al.* 2010).

Model optimization is a valuable tool that has been used recently for developing resistance surfaces. Optimization typically involves comparing a set of alternative resistance surfaces, each considered an a priori hypothesis, using model selection procedures. Recently, a two-step causal modelling approach has also been advocated which involves first fitting models derived from expert opinion and second, iteratively altering landscape costs to increase model fit (Shirk *et al.* 2010; Wasserman *et al.* 2010). Optimization methods have been used to develop models of landscape resistance to movement and gene flow based upon both nongenetic field data (e.g. Driezen *et al.* 2007; Chietkiewicz & Boyce 2009) and expert opinion derived (e.g. Cushman *et al.* 2006) resistance surfaces. To date however, there are few examples where model optimization methods have used genetic data to empirically select habitat variables and develop parameter estimates which can then be used to build a resistance surface (but see Dyer *et al.* 2010; Murphy & Evans 2011). Thus, the limitations inherent in using nongenetic field data or expert opinion to derive genetic resistance surfaces remain apparent in many studies (Spear *et al.* 2010). Here, we incorporate several recently established landscape genetic techniques to a build a continuous resistance surface based upon models of genetic structure without having to rely on arbitrary nongenetic costs, iterative model fitting, or inference from a single model. Model selection uncertainty, a source of variance for parameter estimation, arises during model optimization when only a single top model is selected (Burnham & Anderson

2002; Diniz-Filho *et al.* 2008). Thus, we used multimodel averaging, which consists of averaging coefficients across a set of candidate models and build our resistance surface based upon and set of well supported models (Burnham & Anderson 2002).

To model landscape resistance for fishers, we first developed a resistance surface for each of the four habitat variables that we had selected a priori. Raster values for these surfaces were the raw values for each variable standardized to make parameter estimates comparable. Following this we used circuit theory (McRae 2006) to make pairwise estimates of landscape resistance between sampled patches for each variable. To develop parameter estimates for resistance, we used multiple regression on distance matrices (MRDM; Legendre *et al.* 1994; Legendre & Legendre 1998, p. 559), where pairwise estimates of conditional genetic distance (Dyer *et al.* 2010) was the dependent variable and the four resistance surfaces were independent variables. MRDM performs well for landscape genetic data (Balkenhol *et al.* 2009). Finally, we used model-averaged parameter estimates to build a multivariate resistance surface.

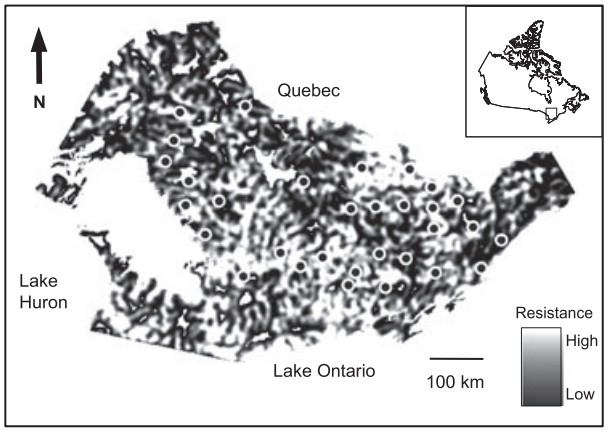


**Fig. 1** Map of landscape resistance to fisher (*Martes pennanti*) gene flow in Ontario, Canada. Information-theoretic model selection and multi-model inference suggested that deep snow, roads, and rivers impeded gene flow. Resistance to gene flow was parameterized with model averaged parameter estimates for these variables. White cells have high resistance and black cells have low resistance. Model uncertainty could be explored visually by mapping upper and lower confidence intervals for model coefficients.

## Methods

Our data set consisted of 657 fishers genotyped at 16 microsatellite loci (see Carr *et al.* 2007a; b for a complete description of molecular techniques) and sampled from 31 patches (hereafter called sample sites) in Ontario, Canada. Sample sites were of comparable size (approximately 300 $km^2$ each) with differing ecological and environmental characteristics. The spatial resolution of samples was at the level of Ontario's geographic townships and so we used these townships to stratify our sampling. We systematically selected townships to sample in an approximate grid such that daily fisher movements and home ranges would not connect the sites (Fig. 1; Arthur *et al.* 1993; Bowman *et al.* 2002); thus, nodes were functionally independent. The mean [SE] distance between sites was 163 [91] km. The mean [SE] number of fishers sampled per site was 21 [7] (Fig. S1). Each sample site was a node on the network of genetic connectivity (Fig. S2; Garroway *et al.* 2008). These 31 sites were a subset of those analysed by Garroway *et al.* (2008). We omitted samples from the Adirondack region of New York and Gatineau, Quebec because we did not have appropriate land cover data as collection techniques were different across political boundaries (outside of Ontario). We also omitted samples from Ontario's Bruce Peninsula to avoid artificially inflated resistance values due to map edge effects associated with the circuit theory algorithms (Koen *et al.* 2010).

We have used a focal patch sampling design, where we assumed samples were point estimates (Brennan *et al.* 2002). Thus, we assumed zero between-individual distance for samples within nodes. We note that this is analogous to individual-level analyses that assign point locations to samples and ignore the extent of home range size in spatial models. In both approaches, variation due to the point sample assumption will contribute to the unexplained nugget variation in spatial models. Garroway *et al.* (2008) address how the features of nodes effect landscape genetic structure in this fisher network.

### *Genetic distance*

We used a network of genetic connectivity constructed by Garroway *et al.* (2008), following Dyer & Nason (2004) and Dyer (2009) to calculate genetic distance between landscapes. Briefly, the network is a minimal edge set that describes among-node genetic covariance. Individual genotypes within nodes were used to define multi-dimensional node centroids with unique coordinates in multi-dimensional space. Pairwise distances among centroids in multi-dimensional space were written as a distance matrix with the off-diagonal values representing network edges; the statistical distance between nodes. This gave a saturated network with all nodes connected. The network was then pruned such that only edges contributing to the overall genetic covariance structure were retained. In this manner, networks can be constructed across spatial scales relevant for landscape planning on the basis of empirical genetic relationships among nodes for the species in question.

From our fisher network, we calculated the genetic distance between nodes as the sum of edge weights along the shortest path through the network. This genetic distance measure has recently been called both 'graph distance' (Dyer & Nason 2004; Garroway *et al.* 2008) and 'conditional genetic distance' (cGD; Dyer *et al.* 2010) in applications of graph theory to landscape genetics. However in principle, any appropriate genetic distance could be used here. We chose this measure as it has recently been shown to have desirable characteristics for landscape genetics studies (Dyer *et al.* 2010) and to maintain a consistent analytical framework with our previous work on this population (Garroway *et al.* 2008).

### *Model selection for landscape resistance*

We modelled landscape resistance to gene flow within an information-theoretic model selection framework (Burnham & Anderson 2002). We built 15 a priori models comprised of various combinations of four variables hypothesized to affect fisher gene flow among landscapes (extent of coniferous forest cover; snow depth; density of ice-free rivers; density of paved roads; Table 1). We created raster layers for each variable with a cell size of 1000 m × 1000 m. Coniferous forest cover was assessed from a Landsat image with an unsupervised classification (dense coniferous category in the 28-class Ontario Provincial Landcover). Snow depth records for the years 1993 through 2002 were obtained from Ontario Ministry of Natural Resources monitoring stations and compiled as mean weekly snow depth from January to April, and interpolated (Carr *et al.* 2007b). Road density was calculated as the density of paved roads per cell based upon Ontario Road Network data (Ontario Ministry of Natural Resources, unpublished data). Finally, the river density layer was calculated as the density of ice-free rivers (based upon the Strahler scale; Strahler 1957) per cell using data from the Water Resource Inventory (obtained from the Ontario Ministry of Natural Resources).

We used circuit theory (McRae 2006; McRae *et al.* 2008) to estimate the potential resistance to gene flow between sample sites for each of the variables thought to impede (snow depth, road density, and river density) or facilitate (coniferous cover) gene flow. In circuit theory, raster cells are treated as nodes on a network and are connected to adjacent nodes (cells) by resistors (analogous to edge weights). Resistor values are based on the mean values for each pair of cells. In our case, cell values were the landcover data related to our hypotheses. We coded the three variables thought to impede gene flow as resistances with edges coded as resistors, and we coded the coniferous variable as a conductance surface with edges coded as conductors. Conductive surfaces are simply the inverse of resistance surfaces. We then calculated effective resistance between sample sites, which is estimated as voltage standardized by current. Because electricity on a network has properties of a random walk, effective resistance can be interpreted as the probability of a random walker travelling through the network (Doyle & Snell 1984). Cells were connected to their eight neighbouring cells. For each of the four raster surfaces, we used circuit theory to estimate the mean effective resistance between all pairs of sample sites (pairwise mode in the

**Table 1** Ranked models explaining landscape resistance to gene flow for fishers (*Martes pennanti*) in Ontario, Canada. Models are ranked based upon the difference between Akaike's Information Criterion (AIC) ranked *i*th and the top-ranked model (ΔAIC). Akaike weights ($w_i$) are the probability that the model is the best in the model set

| Model statement | AIC | ΔAIC | $w_i$ |
|---|---|---|---|
| River density + road density + snow depth | 473.80 | 0.00 | 0.53 |
| Conifer cover + river density + road density + snow depth | 474.00 | 0.20 | 0.47 |
| River density + snow depth | 485.90 | 12.10 | 0.00 |
| Conifer cover + river density + road density | 486.00 | 12.20 | 0.00 |
| Conifer cover + river density + snow depth | 487.80 | 14.00 | 0.00 |
| Road density + snow depth | 495.80 | 22.00 | 0.00 |
| Conifer cover + road density + snow depth | 497.50 | 23.70 | 0.00 |
| River density + road density | 499.60 | 25.80 | 0.00 |
| Conifer cover + river density | 500.50 | 26.77 | 0.00 |
| River density | 506.80 | 33.00 | 0.00 |
| Conifer cover + snow depth | 528.30 | 54.50 | 0.00 |
| Snow depth | 528.50 | 54.70 | 0.00 |
| Conifer cover + road density | 534.10 | 60.30 | 0.00 |
| Road density | 551.00 | 77.20 | 0.00 |
| Conifer cover | 578.30 | 104.50 | 0.00 |

Circuitscape software; McRae 2006). Using a *z*-transformation, we then standardized the pairwise values of mean effective resistance between sites for the four variables and used these standardized values as independent variables in linear regressions. We standardized resistances so that parameter estimates for each variable would be comparable. We did not include a separate measure of Euclidean distance in our models, because each resistance surface already accounted for distance (i.e. Euclidean distance is one of the factors contributing to effective resistance). Thus, variation in distance was controlled for across our independent variables.

We used MRDM to fit linear models of landscape resistance to cGD between sites (e.g. Legendre *et al.* 1994; Legendre & Legendre 1998, p. 559; Holzhauer *et al.* 2006; Balkenhol *et al.* 2009; Dyer *et al.* 2010). All Pearson correlations between independent variables were $r \leq 0.25$, and the variance inflation factor was 1.6. Both of these lines of evidence suggested a lack of multicollinearity (e.g. Graham 2003; Kutner *et al.* 2004; ), so we did not exclude any variables from regression analyses. The MRDM approach uses permutation tests of linear regressions upon unfolded distance matrices (rectangular data tables; e.g. Legendre *et al.* 1994; Legendre & Legendre 1998, p. 559). We permuted the data 1000 times. Variables were considered to have nonrandom effects if parameter estimates were greater than or less than 95% of permuted values. Parameter estimates arising from linear regression of such unfolded distance matrices are unbiased (Legendre *et al.* 1994; Hawkins *et al.* 2007; Lichstein 2007). We estimated standard errors of parameters via basic bootstrap resampling with replacement 1000 times (Davison & Hinkley 1997).

We considered models with ΔAIC (difference between the AIC of each model and that of the top ranked model ($\Delta i$)) of less than 7 to be our candidate model set. Sampling error will lead to situations where the model with the lowest AIC value is not the Kullback–Leibler best model (what AIC attempts to estimate). We chose the more conservative $\Delta i$ of 7 as a threshold to consider models important rather than the more typical 2 as Richards (2005) show that the threshold of 7 will retain the top Kullback–Leibler model with an approximate a 95% probability. For each model we calculated Akaike weights ($w_i$), the probability that the *i*th model is the best approximating model among the candidate models (Burnham & Anderson 2002). Model-averaged parameter estimates often have less bias and more precision than parameter estimates from a single model (Burnham & Anderson 2002). Therefore, for all variables within the candidate model set we estimated the model averaged parameter estimates (β) with estimated unconditional 95% confidence intervals (CI; model averaged bootstrap CIs). We assessed the biological importance of model averaged parameter estimates by examining the breadth and position of 95% confidence intervals. If CIs overlapped zero or had minimal impact given the range of resistance values we considered their biological importance to be minimal. We then used the model averaged parameter estimates to parameterize their corresponding resistance surfaces within a linear regression equation to build the final multivariate resistance surface. Model averaging is an important aspect of our approach. There is uncertainty in parameter estimation that is lost by drawing inference from a single top model as parameter estimates from single models are conditional upon the other variables included in the model (Burnham & Anderson 2002). Model averaging attempts to derive unconditional parameter estimates and error (although they are conditional upon the a priori model set) by averaging parameter estimates across the model set. By averaging the top models we acknowledge model selection uncertainty by model averaging and build parameter estimates that are not conditional on the presence of other variables in the model (Burnham & Anderson 2002). As a final step, we tested for the contribution of isolation-by-distance to our modelled estimates of effective resistance. First, we compared conditional genetic distance to effective resistance between all pairs of points using a simple Mantel test; second, we used a partial Mantel test to test for an effect of our resistance surface controlling for geographic distance. All statistics related to model fitting and model selection were calculated using the R statistical language (R Development Core Team 2010).

## Results

Two, nearly equally well supported models clearly outperformed the other models (Tables 1 and 2). Ranked first was the model containing the variables for river density, road density, and snow depth with 52% probability of being the best approximating model in the model set. Ranked second was the global model that contained the same three variables as the top ranked model with the addition of coniferous forest. This second model had a 47% probability of being the best approximating model in the model set and had a ΔAIC of just 0.20. The next highest ranked model had a ΔAIC greater than 12 suggesting that there was very little support for any of the other models (Burnham & Anderson 2002; Richards 2005). The direction of effects and effect sizes for the variables shared by both models (ice-free river density, road density, and snow depth) were consistent and did not overlap zero suggesting that each of the variables was meaningful (Table 2).

**Table 2** Parameter estimates ($\beta$), 95% confidence intervals (95% CI), permuted *P*-values (*P*; the proportion of randomized parameter estimates greater than those based upon the original data), $R^2$ for the top two ranked models explaining fisher gene flow

| | $\beta$ | 95% CI | *P* |
|---|---|---|---|
| Intercept | 11.12 | (10.70, 11.52) | 0.0000 |
| River density | 0.99 | (0.61, 1.36) | 0.0000 |
| Road density | 0.71 | (0.34, 1.09) | 0.0010 |
| Snow depth | 1 | (0.65, 1.36) | 0.0064 |
| $\Delta$AIC = 0, $w_i$ = 0.52, $R^2$ = 0.19 | | | |
| Intercept | 11.12 | (10.71, 11.50) | 0.0000 |
| River density | 1.03 | (0.64, 1.44) | 0.0000 |
| Road density | 0.78 | (0.38, 1.19) | 0.0001 |
| Snow depth | 0.84 | (0.41, 1.30) | 0.0020 |
| Conifer cover | 0.29 | (−0.13, 0.71) | 0.1825 |
| $\Delta$AIC = 0.2, $w_i$ = 0.47, $R^2$ = 0.19 | | | |

Permutation tests demonstrated that parameter estimates for these three variables were different than that expected by chance (Table 2). Coniferous forest cover was contained only in the second model and its confidence intervals overlapped zero suggesting that it likely did not have a biologically meaningful effect (Table 2). Similarly, permutation tests suggested that its parameter estimate was not different than that expected by chance (Table 2). We therefore did not consider the coniferous forest cover variable further.

Model-averaged parameter estimates of the standardized resistance values suggested that a high density of ice-free rivers ($\beta_{\text{intercept}}$ = 11.01; CI: 10.61–11.42, $\beta_{\text{rivers}}$ = 1.00; CI: 0.60–1.40), deep snow ($\beta_{\text{snow}}$ = 0.92; CI: 0.49–1.35) and a high density of paved roads ($\beta_{\text{roads}}$ = 0.74; CI: 0.36–1.12) impeded gene flow between sample sites for fishers. Confidence intervals of parameter estimates for these three variables overlapped extensively indicating that no one variable was driving the relationship; however, the trend suggested that river density may have had the largest effect, followed by snow depth, and then road density. We applied the model-averaged parameter estimates in a linear equation to create a new raster of cells, where cell values depict overall model resistance to fisher gene flow. Although some regions of high resistance were apparent, sample sites appeared well connected (Fig. 1). Simple (cGD $\sim$ modelled surface; Mantel *r* [CI]: 0.32 [0.25–0.39]; $P$ = 0.001) and partial (cGD $\sim$ modelled surface | geographic distance; Mantel *r* [CI]: 0.24 [ 0.18–0.30]) Mantel tests suggested that our final model fit the genetic data well and that the relationship to landscape structure was retained after partialling out the effects of geographic distance among sample sites.

## Discussion

Our findings suggest that rivers, roads, and deep snow have all impeded fisher gene flow to varying extents during the species' population recovery in Ontario, Canada. It appears, however, that coniferous forest has had relatively little effect on fisher gene flow. Rivers that remained open in winter seemed to create substantial landscape resistance for fishers. This result is consistent with findings from two previous studies, which have suggested that rivers can act as barriers to fisher movement (Kelly 1977; Wisely *et al.* 2004). In contrast, Carr *et al.* (2007a) found that fishers immigrated to Ontario across the large St. Lawrence River from Adirondack, New York. The St. Lawrence River often freezes during winter however, which likely facilitated crossing. Winter is when most juvenile fishers disperse, and open, fast flowing rivers likely limit movements of dispersers. Our findings concerning snow depth are consistent with several previous studies suggesting that deep snow limits fisher populations (Krohn *et al.* 1995, 1997; Carr *et al.* 2007b). Fishers have relatively small feet for their body size, and a high foot load, which makes travelling in deep snow inefficient (Krohn *et al.* 2003). We are aware of no other studies demonstrating that roads contribute to landscape resistance for fishers, although demographic data suggest road effects in the closely related marten (*Martes americana*) (Hodgman *et al.* 1994; Robitaille & Aubry 2000). Road density is related to human activity and so this variable is likely correlated with a number of human related activities (such as trapping) that may impede gene flow. Increasingly, genetic impacts of various road features are being documented (Balkenhol & Waits 2009; Holderegger & DiGiulio 2010). It is important that the negative relationship between road density and gene flow be considered in future management of fishers.

Our approach has allowed us to examine hypotheses about the influence of landscape features, both anthropogenic and natural, on fisher gene flow. The final resistance surface can also be considered a hypothesis, which could be tested within an adaptive management framework. Based upon this hypothesized resistance surface, one could proceed with either simulations or real world manipulations of predictor variables in order to modify the hypothesis and further reduce uncertainty about fisher gene flow. Thus, we envision using resistance surfaces in an iterative management process, with the aim of actively accruing information over time through ongoing monitoring, model testing and experiments.

Although the explanatory power of our models may seem low compared to some other landscape genetic studies, we emphasize that the models should have

broad applicability for fishers beyond just our study area, because of the hypothetico-deductive model selection framework we used. We selected candidate models a priori, which favours parsimony over complexity. The tradeoff, however, is that we did not explain as much variation in our data as we could have. Post hoc efforts to maximally explain variation risk model over-fitting, where spurious effects may be added to models and model applicability is limited (Burnham & Anderson 2002). Although rare in population genetics studies, a valuable technique for testing the generality of landscape genetics models would the dividing of data into testing and evaluation sets. As the costs of genotyping decrease these approaches should become more feasible.

We used a genetic network to make inferences concerning the genetic structure of fisher populations in Ontario. Networks offer several benefits over other, similar techniques for landscape genetic comparisons. For example, conditional genetic distance, which simultaneously accounts for the genetic covariance among all samples, achieves more statistical power than the most commonly used measure of genetic distance, $F_{ST}$ (Dyer *et al.* 2010). Further, parameters estimated from pairwise relationships, such as $F_{ST}$, have higher variance than parameters estimated from the totality of the data, such as is done in the estimation of conditional genetic distance (Dyer *et al.* 2010). This is likely partially a result of not assuming a priori that all landscapes are connected.

We used genetic distance as a response variable, which allowed us to objectively parameterize the resistance surface, without relying on expert-based costs or *post hoc* iterative increasing of model fit. The method appears to avoid many potential pitfalls associated with deriving resistance surfaces for landscape genetic studies, including what Spear *et al.* (2010) considered the biggest challenge for calculating resistance surfaces—the assignment of resistance values to different landscape features. Rather than defining a resistance surface a priori using nongenetic field data or expert opinion, we simply used regressions of the relationship between habitat features and genetic topology to estimate landscape resistance. The resulting model-averaged resistance surface (Fig. 1) was the best estimate of landscape resistance for the model set, and the distribution of these raster resistances reflected the true value of the underlying data, rather than values derived from expert opinion, including potential biases inherent in such data. Although we used conditional genetic distance as our measure of genetic distance, we note that any appropriate pairwise measure of genetic distance could be used as a response variable.

Our approach to building a resistance surface has more in common with ecological modelling than with current landscape genetic practices. There is a large literature about the merits of information theoretic approaches to model selection and multi-model inference (Yoccoz 1991; Burnham & Anderson 2002; Johnson & Omland 2004; Stephens *et al.* 2007). These techniques have become widespread in ecology and are now beginning to be applied to landscape genetics (e.g. Bruggeman *et al.* 2010; Goldberg & Waits 2010; Murphy *et al.* 2010; Selkoe *et al.* 2010). Briefly, an information theoretic approach to model building restricts the number of models analysed to minimize the potential of identifying spurious relationships, provides a method to rank models and quantify model selection uncertainty, and a means to draw inference from multiple models rather than a single top model (Burnham & Anderson 2002). The application of model selection and multimodel inference to landscape genetics requires an understanding of the potential effects of nonindependence inherent in genetic data. There have been many approaches suggested to deal with such nonindependence (e.g. Goldberg & Waits 2010; Selkoe *et al.* 2010). To add to this discussion, we point out that model-averaged parameter estimates derived from systematic samples of spatially autocorrelated data are unbiased (Diniz-Filho *et al.* 2008), and that risks of inflated Type-1 error can be mitigated with bootstrapping to calculate standard errors (Davison & Hinkley 1997) and permutation tests (Legendre *et al.* 1994; Balkenhol *et al.* 2009).

It is also worth noting that by using circuit theory, we assessed resistance along multiple paths between each graph node. We considered this the most the appropriate approach to estimate landscape resistance to gene flow for our data because we compared populations rather than individuals at each node; more than one single route (e.g. the least-cost route) was likely taken by multiple dispersers and it is unlikely that dispersers would have knowledge of the single, least-cost path. Moreover, single least-cost paths may bias resistance estimates in heterogeneous landscapes such as those in this study (Rayfield *et al.* 2010). On the other hand, caution is warranted when applying multiple path approaches to avoid biased resistance estimates near map edges (Koen *et al.* 2010).

We have proposed a general strategy, incorporating existing landscape genetic approaches, for using genetic data to parameterize landscape resistance surfaces. Some additional considerations are warranted. First, it should be noted that in many systems, it will be impractical to sample all geographic locations. Thus, unsampled 'ghost populations' may affect estimates of migration rates and population connectivity

(Beerli 2004). The effects of ghost populations on estimates of conditional genetic distance are currently unknown, and this points toward an interesting avenue of research. Perhaps instructive in this context, to identify sites important for maintaining gene flow, Garroway *et al.* (2008) performed two *in silico* node removal experiments. They first sequentially removed the eight best connected nodes and second sequentially removed eight nodes most likely to act as bottlenecks to gene flow (measured with 'betweenness') and examined effects of node removal at each step on the mean graph distance among all nodes. They found that the removal of well connected nodes had little effect on mean graph distance but the removal of high betweenness nodes caused graph distance to increase. This suggests that some nodes may be disproportionately important when measuring gene flow and that these nodes are not necessarily the best connected nodes. We suspect that systematic sampling should alleviate some of the potential problems associated with ghost populations. Second, it is important to recognize that although important for urban planning, corridor design, and reserve design, landscape genetic resistance surfaces are not a panacea. Gene flow will not always reflect important animal movement behaviour nor will movement behaviour necessarily reflect realized gene flow. For these purposes further work integrating animal movement data, with careful consideration of associated caveats (e.g. O'Brien *et al.* 2006) and landscape genetics will be important. An integration between landscape genetic graphs (e.g. Garroway *et al.* 2008; Murphy *et al.* 2010) and spatial movement and habitat-based graphs (Urban & Keitt 2001; O'Brien *et al.* 2006; Fall *et al.* 2007) will likely be promising with respect to identifying and conserving landscape features important for gene flow and animal movements.

By combining model selection techniques with a genetic response variable (the population network), we were able to draw inference about resistance surfaces affecting fisher gene flow. This approach could be easily adapted to different data sets and questions. For example, analyses within single landscapes could use the same technique, where the effects of habitat features on genetic distances among individuals are of interest. Although any pairwise measure of genetic distance could be used as a response variable, genetic networks offer some desirable analytical features (Garroway *et al.* 2008; Fortuna *et al.* 2009; Dyer *et al.* 2010; Murphy *et al.* 2010). These include providing a framework where the response variable can be used to model both gene flow along edges, and the impact of node features on genetic structure (Garroway *et al.* 2008; Murphy *et al.* 2010). In addition, networks allow for the estimation of a suite of node, edge, and system-level measures not available in pairwise analyses.

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## References

Adriaensen F, Chardron JP, DeBlust G *et al.* (2003) The application of 'least-cost' modelling as a functional landscape model. *Landscape and Urban Planning*, **64**, 233–247.

Arthur SM, Paragi TF, Krohn WB (1993) Dispersal of juvenile fishers in Maine. *Journal of Wildlife Management*, **57**, 868–874.

Balkenhol N, Waits LP (2009) Molecular road ecology: exploring the potential of genetics for investigating transportation impacts on wildlife. *Molecular Ecology*, **18**, 4151–4164.

Balkenhol N, Waits LP, Dezzani R (2009) Statistical approaches in landscape genetics: an evaluation of methods for linking landscape and genetic data. *Ecography*, **32**, 818–830.

Beerli P (2004) Effect of unsampled populations on the estimation of population sizes and migration rates between sampled populations. *Molecular Ecology*, **13**, 827–836.

Beier P, Majka DR, Newell SL (2009) Uncertainty analysis of a least-cost modeling approach for designing wildlife linkages. *Ecological Applications*, **19**, 2067–2077.

Bowman J, Jaeger JAG, Fahrig L (2002) Dispersal distance of mammals is proportional to home range size. *Ecology*, **83**, 2049–2055.

Brennan JM, Bender DJ, Contreras TA, Fahrig L (2002) Focal patch landscape studies for wildlife management: optimizing sampling effort across scales. In: *Integrating Landscape Ecology into Natural Resource Management* (eds Liu J, Taylor WW), pp. 68–91, 480pp. Cambridge University Press, Cambridge, MA.

Broquet T, Ray N, Petit E, Fryzell JM, Burel F (2006) Genetic isolation by distance and landscape connectivity in the American marten (*Martes americana*). *Landscape Ecology*, **21**, 877–889.

Bruggeman DJ, Wiegand T, Fernandez N (2010) The relative effects of habitat loss and fragmentation on population genetic variation in the red-cockaded woodpecker (*Picoides borealis*). *Molecular Ecology*, **19**, 3679–3691.

Burnham KP, Anderson DR (2002) *Model Selection and Multimodel Inference: A Practical Information–Theoretic Approach*. Springer-Verlag, New York.

Carr D, Bowman J, Kyle CJ *et al.* (2007a) Rapid homogenization of multiple sources: genetic structure of a recolonizing population of fishers. *Journal of Wildlife Management*, **71**, 1853–1861.

Carr D, Bowman J, Wilson PJ (2007b) Density-dependent dispersal suggests a genetic measure of habitat suitability. *Oikos*, **116**, 629–635.

Chietkiewicz CB, Boyce MS (2009) Use of resource selection functions to identify conservation corridors. *Journal of Applied Ecology*, **46**, 1036–1047.

Clobert J, Danchin E, Dhondt AA, Nichols JD (2001) *Dispersal*. Oxford University Press, Oxford.

Cushman SA, McKelvey KS, Hayden J, Schwartz MK (2006) Gene flow in complex landscapes: testing multiple hypotheses with causal modeling. *American Naturalist*, **168**, 486–499.

Davison AC, Hinkley DV (1997) *Bootstrap Methods and Their Application*, Cambridge University Press, New York, NY.

Diniz-Filho JAF, Rangel TFLVB, Bini LM (2008) Model selection and information theory in geographical ecology. *Global Ecology and Biogeography*, **17**, 479–488.

Doyle PG, Snell JL (1984) *Random Walks and Electric Networks*, 159 pp. Mathematical Association of America, Washington, DC.

Driezen K, Adriaensen F, Rondinini C, Doncaster CP, Matthysen E (2007) Evaluating least-cost model predictions with empirical dispersal data: a case-study using radiotracking data of hedgehogs (*Erinaceus europaeus*). *Ecological Modelling*, **209**, 314–322.

Dyer RJ (2009) GeneticStudio: a suite of programs for spatial analysis of genetic-marker data. *Molecular Ecology Resources*, **9**, 110–113.

Dyer RJ, Nason JD (2004) Population graphs: the graph theoretic shape of genetic structure. *Molecular Ecology*, **13**, 1713–1727.

Dyer RJ, Nason JD, Garrick RC (2010) Landscape modelling of gene flow: improved power using conditional genetic distance derived from the topology of population networks. *Molecular Ecology*, **19**, 3746–3759.

Fall A, Fortin MJ, Manseau M, O'Brian D (2007) Spatial graphs: Principles and applications for habitat connectivity. *Ecosystems*, **10**, 448–461.

Fortuna MA, Albaladejo RG, Fernández L, Aparicio A, Bascompte J (2009) Networks of spatial genetic variation across species. *Proceedings of the National Academy of Sciences, USA*, **106**, 19044–19049.

Garroway CJ, Bowman J, Carr D, Wilson PJ (2008) Applications of graph theory to landscape genetics. *Evolutionary Applications*, **1**, 620–630.

Goldberg CS, Waits LP (2010) Comparative landscape genetics of two pond-breeding amphibian species in a highly modified agricultural landscape. *Molecular Ecology*, **19**, 3650–3663.

Graham MH (2003) Confronting multicollinearity in ecological multiple regression. *Ecology*, **84**, 2809–2815.

Hawkins BA, Diniz-Filho JAF, Bini LM, De Marco P, Blackburn TM (2007) Red herrings revisited: spatial autocorrelation and parameter estimation in geographical ecology. *Ecography*, **30**, 375–384.

Hodgman TP, Harrison DJ, Katnik DD, Elowe KD (1994) Survival in an intensively trapped marten population in Maine. *Journal of Wildlife Management*, **58**, 593–600.

Holderegger R, DiGiulio M (2010) The genetic effects of roads: a review of empirical evidence. *Basic and Applied Ecology*, **11**, 522–531.

Holzhauer SIJ, Ekschmitt K, Sander A-C, Dauber J, Wolters V (2006) Effect of historic landscape change on the genetic structure of the bush-cricket *Metrioptera roeseli*. *Landscape Ecology*, **21**, 891–899.

Jaeger JAG, Bowman J, Brennan J *et al.* (2005) Predicting when animal populations are at risk from roads: an interactive model of road avoidance behavior. *Ecological Modeling*, **185**, 329–348.

Johnson JB, Omland KS (2004) Model selection in ecology and evolution. *Trends in Ecology and Evolution*, **19**, 101–108.

Kelly BM (1977) *Fisher (Martes pennanti) biology in the White Mountain National forest and adjacent areas*. PhD Dissertation, University of Massachusetts, Amherst. 178pp.

Koen EL, Garroway CJ, Wilson PJ, Bowman J (2010) The effect of map boundary on estimates of landscape resistance to animal movement. *PLoS ONE*, **5**, e11785.

Krohn WB, Elowe KD, Boone RB (1995) Relations among fishers, snow, and martens –development and evaluation of 2 hypotheses. *Forestry Chronicle*, **71**, 97–105.

Krohn WB, Zielinski WJ, Boone RB (1997) Relations among fishers, snow, and martens in California: results from small scale comparisons. In: *Martes: Taxonomy, Ecology, Techniques, and Management* (eds Proulx G, Bryant HN, Woodard PM), pp. 211–232. The Provincial Museum of Alberta, Edmonton, AB.

Krohn W, Hoving C, Harrison D, Phillips D, Frost H (2003) *Martes* foot-loading and snowfall patterns in eastern North America: implications to broad-scale distributions and interactions of mesocarnivores. In: *Martens and Fishers (Martes) in Human-Altered Environments* (eds Harrison DJ, Fuller AK, Proulx G), pp. 115–131. Springer-Verlag, New York, NY.

Kutner MH, Nachtsheim CJ, Neter J (2004) *Applied Linear Regression Models*, 4th edn. McGraw Hill, New York.

Lancaster PA, Bowman J, Pond BA (2008) Fishers, farms, and forests in eastern North America. *Environmental Management*, **42**, 93–101.

Legendre P, Legendre L (1998) *Numerical Ecology*, 2nd edn. Elsevier Science, Amsterdam.

Legendre P, Lapointe FJ, Casgrain P (1994) Modeling brain evolution from behavior—a permutational regression approach. *Evolution*, **48**, 1487–1499.

Lichstein JW (2007) Multiple regression on distance matrices: a multivariate spatial analysis tool. *Plant Ecology*, **188**, 117–131.

Manel S, Schwartz MK, Luikart G, Taberlet P (2003) Landscape genetics: combining landscape ecology and population genetics. *Trends in Ecology and Evolution*, **18**, 189–197.

McRae BH (2006) Isolation by resistance. *Evolution*, **60**, 1551–1561.

McRae GH, Dickson BG, Keitt TH, Shah VB (2008) Using circuit theory to model connectivity in ecology, evolution, and conservation. *Ecology*, **89**, 2712–2724.

Minor ES, Lookingbill TR (2010) Connectivity of protected area networks in the contiguous United States. *Conservation Biology*, doi: 10.1111/j.1523-1739.2010.01558.x.

Murphy M, Evans J (2011) Genetic connectivity as a function of landscape connectivity: application of landscape genetics for predictive modeling. In:*Predictive Wildlife and Habitat Modeling in Landscape Ecolog* (eds Drew CA, Huettmann F, Wiersma Y), Springer, New York, NY.

Murphy MA, Dezzani R, Pilliod DS, Storfer A (2010) Landscape genetics of high mountain frog metapopulations. *Molecular Ecology*, **19**, 3634–3649.

O'Brien D, Manseau M, Fall A, Fortin M-J (2006) Testing the importance of spatial configuration of winter habitat for

woodland caribou: an application of graph theory. *Biological Conservation*, **130**, 70–83.
Powell RA (1993) *The Fisher: Life History, Ecology, and Behavior*, 2nd edn. University of Minnesota Press, Minneapolis, USA.
R Development Core Team (2011) *R: A Language and Environment for Statistical Computing*. R Foundation for Statistical Computing, Vienna, Austria. ISBN 3-900051-07-0, URL http://www.R-project.org/.
Rayfield B, Fortin MJ, Fall A (2010) The sensitivity of least-cost habitat graphs to relative cost surface values. *Landscape Ecology*, **25**, 519–532.
Richards SA (2005) Testing ecological theory using the information-theoretic approach: examples and cautionary results. *Ecology*, **86**, 2805–2814.
Robitaille JF, Aubry K (2000) Occurrence and activity of American martens *Martes americana* in relation to roads and other routes. *Acta Theriologica*, **45**, 137–143.
Selkoe KA, Watson JR, White C *et al.* (2010) Taking the chaos out of genetic patchiness: seascape genetics reveals ecological and oceanographic drivers of genetic patterns in three temperate reef species. *Molecular Ecology*, **19**, 3708–3726.
Shannon P, Markiel A, Ozier O *et al.* (2003) Cytoscape: a software environment for integrated models of biomolecular interaction networks. *Genome Research*, **13**, 2498–2504.
Shirk AJ, Wallin DO, Cushman SA, Rice CG, Warheit KI (2010) Inferring landscape effects on gene flow: a new model selection framework. *Molecular Ecology*, **19**, 3603–3619.
Slatkin M (1985) Gene flow in natural populations. *Annual Review of Ecology and Systematics*, **16**, 393–430.
Spear SF, Balkenhol N, Fortin MJ, McRae BH, Scribner K (2010) Use of resistance surfaces for landscape genetic studies: considerations for parameterization and analysis. *Molecular Ecology*, **19**, 3576–3591.
Stephens PA, Buskirk SW, del Rio CM (2007) Inference in ecology and evolution. *Trends in Ecology and Evolution*, **22**, 192–197.
Storfer A, Murphy MA, Evans JS *et al.* (2007) Putting the 'landscape' in landscape genetics. *Heredity*, **98**, 128–142.
Strahler AN (1957) Quantitative analysis of watershed geomorphology. *American Geophysical Union Transactions*, **38**, 913–920.
Urban D, Keitt T (2001) Landscape connectivity: a graph-theoretic perspective. *Ecology*, **82**, 1205–1218.
de Vos A (1964) Range changes of mammals in the Great Lakes region. *American Midland Naturalist*, **71**, 210–231.
Wang IJ, Summers K (2010) Genetic structure is correlated with phenotypic divergence rather than geographic isolation in the highly polymorphic strawberry poison-dart frog. *Molecular Ecology*, **19**, 447–458.
Wasserman TN, Cushman SA, Schwartz MK, Wallin DO (2010) Spatial scaling and multi-model inference in landscape genetics: *Martes americana* in northern Idaho. *Landscape Ecology*, **25**, 1601–1612.
Weir BS, Cockerham CC (1984) Estimating F-statistics for the analysis of population structure. *Evolution*, **38**, 1358–1370.
Wisely SM, Buskirk SW, Russell GA, Aubry KB, Zielinski WJ (2004) Genetic diversity and structure of the fisher (*Martes pennanti*) in a peninsular and peripheral metapopulation. *Journal of Mammalogy*, **85**, 640–648.
Yoccoz NG (1991) Use, overuse, and misuse of significance tests in evolutionary biology and ecology. *Bulletin of the Ecological Society of America*, **72**, 106–111.

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## Data accessibility

Data supporting the results of this paper can be accessed from the Natural Resources and DNA Profiling Centre online database, hosted in Peterborough, Ontario, Canada, at http://nrdpfc.ca/. An account for this database can be obtained by emailing info@nrdpfc.ca.

## Supporting information

Additional supporting information may be found in the online version of this article.

**Fig. S1** A two-dimensional genetic network representing the genetic relationship among fishers (*Martes pennanti*) sampled from 3q locations in Ontario, Canada during 2000–2003 and profiled at 16 microsatellite loci. Edge length is proportional to the genetic distance between populations. The network was built following Dyer & Nason (2004) using the Genetstudio software (Dyer 2009). It was first calculated by Garroway *et al.* (2008). This network is modified in that here we did not include nodes for the Adirondack region of New York and Gatineau, Quebec, due to differences in data collection nor did we include the Bruce Peninsula, Ontario in order to reduce the effects of artificially inflated resistance values associated with map edges (Koen *et al.* 2010). We used the shortest path between nodes, with distance calculated as the sum of edge weights, as our measure of genetic distance. This measure has recently been called both 'graph distance' (Dyer & Nason 2004; Garroway *et al.* 2008) and 'conditional genetic distance (cGD; Dyer *et al.* 2010) in landscape genetics studies. We used this distance measure as a response variable to model landscape genetic resistance to gene flow in fishers. The network is projected using Cytoscape 2.7.0 (Shannon *et al.* 2003).

**Fig. S2** Labelled map of sample sites. Below are sample sizes from each site. Angelsea-Grimsthorpe (AG; $n = 16$), Anson-Lutterworth (AL; $n = 25$), Algonquin (AQ; $n = 20$), Anstruther (AS; $n = 24$), Badgerow (BA; $n = 22$), Burton-McKenzie (BK; $n = 16$), Belmont (BL; $n = 7$), Blair-Mowat (BM, $n = 26$), Broughman (BR; $n = 23$), Carlow-Bangor (CB; $n = 20$), Carling-

Ferguson (CF; $n = 8$), Conger-Freeman (CM; $n = 15$), Dalhousie (DL; $n = 20$), Darling (DR; $n = 22$), Escott-Yonge (EY; $n = 20$), Falconer (FL; $n = 22$), Fraser-Richards (FR; $n = 21$), Galway (GW; $n = 20$), Hungerford-Huntington (HH; $n = 14$), Kennebec (KB; $n = 23$); Loughborough-Bedford (LB; $n = 31$), Lyndoch (LN; $n = 19$), Monteith-Christie (MC; $n = 26$), Marmora-Lake (ML; $n = 32$), McNab (MN; $n = 24$), Montague (MT; $n = 21$), Olrig Cluster (OL; $n = 14$), Orillia-Ramara (OR; $n = 17$), Prescott (PR; $n = 48$), Ramsey-Huntley (RH; $n = 20$), Ross (RO; $n = 19$).

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