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Landscape-level Analysis of Mountain Goat Population Connectivity in Washington and Southern British Columbia

David O. Wallin
Western Washington University, david.wallin@wwu.edu

Leslie C. Parks

Samuel A. Cushman

Brad H. McRae

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Leslie C. Parks, David O. Wallin, Samuel A. Cushman, Brad H. McRae

L. C. Parks, D. O. Wallin (corresponding author)

Department of Environmental Sciences, Huxley College of the Environment, Western Washington University,

516 High St., Bellingham, WA 98225, USA

e-mail: david.wallin@wwu.edu, phone: (360) 650-7526, fax: (360) 650-7284

S. A. Cushman

Rocky Mountain Research Station, U.S. Forest Service,

2500 S. Pine Knoll Dr., Flagstaff, AZ 86001, USA

e-mail: scushman@fs.fed.us

B. H. McRae

The Nature Conservancy, North America Region,

117 Mountain Ave., Suite 201, Fort Collins, CO 80524, USA

e-mail: bmcrae@tnc.org

Abstract

Habitat fragmentation and habitat loss diminish population connectivity, reducing genetic diversity and increasing extinction risk over time. Improving connectivity is widely recommended to preserve the long-term viability of populations, but this requires accurate knowledge of how landscapes influence connectivity. Detectability of landscape effects on gene flow is highly dependent on landscape context, and drawing conclusions from single landscape studies may lead to ineffective management strategies. We present a novel approach to elucidate regional variation in the relative importance of landscape variable effects on gene flow. We demonstrate this approach by evaluating gene flow between isolated, genetically impoverished mountain goat (*Oreamnos americanus*) populations in Washington and much larger, genetically robust populations in southern British Columbia. We used GENELAND to identify steep genetic gradients and then employed individual-based landscape genetics in a causal modeling framework to independently evaluate landscape variables that may be generating each of these genetic gradients. Our results support previous findings that freeways, highways, water, agriculture and urban landcover limit gene flow in this species. Additionally, we found that a previously unsupported landscape variable, distance to escape terrain, also limits gene flow in some contexts. By integrating GENELAND and individual-based methods we effectively identified regional limiting factors that have landscape-level implications for population viability.

Keywords: population connectivity; GENELAND; circuit theory; causal modeling; *Oreamnos americanus*

Introduction

Anthropogenic landscape change, including habitat loss, habitat fragmentation and climate change, is driving the global loss of biodiversity (Thomas et al. 2004; Wiegand et al. 2005; Fischer and Lindenmayer 2007; Butchart et al. 2010). Habitat loss reduces population size while habitat fragmentation disrupts historical patterns of gene flow, increasing isolation and lowering effective population size (N_e) (Keyghobadi 2007). Climate change may further reduce and isolate populations by diminishing habitat quality, altering species' distribution and causing range shifts (Root et al. 2003).

Small populations isolated by inhospitable landscapes are more vulnerable to demographic variability, environmental stochasticity and genetic processes including inbreeding depression (Crnokrak and Roff 1999; Keller and Waller 2002; Mainguy et al. 2009; Dunn et al. 2011), the random fixation of deleterious alleles (Lynch et al. 1995; Lande 1998) and the loss of adaptive potential (Lande 1995; Willi et al. 2006), that further increase population extinction risk. Conversely, a landscape that is permeable to individual movement increases N_e, genetic diversity and adaptive potential, while providing movement routes for populations to respond to climate change (Krosby et al. 2010). Maintaining population connectivity facilitates the movement of individuals and genes across the landscape and is therefore critical to preserve population viability (Taylor et al. 1993; Crooks and Sanjayan 2006; Heller and Zavaleta 2009).

Landscape genetics provide powerful methods to evaluate the effects of multiple landscape variables on population connectivity (Manel et al. 2003; Holderegger and Wagner 2008; Segelbacher et al. 2010). The genetic relatedness among individuals sampled across broad landscapes can be used to test hypotheses of landscape resistance and hence infer connectivity among local populations (Cushman et al. 2006; McRae and Beier 2007; Shirk et al. 2010). Many landscape genetic studies are based on associations between genetic samples and landscape

variables within a single landscape (Segelbacher et al. 2010). This may lead to erroneous conclusions about the general response of a species to a landscape feature because detectability of landscape effects on gene flow relies heavily on context (Jaquiéry et al. 2011; Cushman et al. 2012; Balkenhol et al. 2013; Cushman et al. 2013a). Even when a species has a globally consistent response to a landscape feature, the effect of that feature will only be detectable when the pattern across the study area is highly variable and limiting to gene flow (Cushman et al. 2011; Shortbull et al. 2011). Thus, replication of landscape genetic analyses over the range of habitat variability is crucial when inferring landscape effects on gene flow. Previous studies have not developed a systematic approach to spatially focus replication in a manner that is likely to reveal local limiting factors within continuous landscapes.

We expand previous research (Shirk et al. 2010) to evaluate population connectivity between genetically impoverished mountain goat (*Oreamnos americanus*) populations in Washington (WA) and larger, more genetically diverse populations in British Columbia (BC). The WA populations have been greatly reduced (>50%) by historical overharvest and many have not recovered despite drastically reduced hunting pressure (Rice and Gay 2010). Shirk et al. (2010) found that mountain goat gene flow within the Cascade Range, WA is limited by an interstate, smaller highways, development in low elevation valleys and water, suggesting that anthropogenic landscape alterations may also diminish population connectivity between WA and southern BC. The expansive Okanagan Valley may also contribute to genetic isolation as this feature was found to limit cougar gene flow within the same area (Warren et al. 2014). We used GENELAND (Guillot et al. 2005) to identify genetic discontinuities and divide our large study area into regions, facilitating an analysis based on ecologically relevant boundaries rather than political boundaries. We then analyzed landscape resistance across regional boundaries within a

causal modeling framework to identify local limiting factors and infer gene flow across the study area. This enabled us to evaluate gene flow in a wider range of landscapes and combinations of landscape features. Research that transcends political boundaries also encourages interagency collaboration that is vital to plan and implement efforts to maintain viable populations confronted with habitat loss, habitat fragmentation and climate change (Beier et al. 2011).

Methods

Study area

The study area encompasses 151,760 km^2, including the Cascade Range of WA and the Coast, Selkirk and Purcell mountain ranges of BC (Fig. 1). Elevation varies widely with heavily forested valleys dissecting rugged alpine terrain. Interstate 90 (I90) cuts across the Cascades east-west, and the Coquihalla Highway (Hwy. 5) cuts across BC north-southwest. Several secondary highways and numerous other roads also transect the study area. Developed areas and agriculture are present at lower elevations and along transportation corridors. At higher elevations, ski resorts and residential areas have developed near major passes.

Sample collection

We used protocol developed by Rutledge et al. (2009) to collect 250 scat samples in the summers of 2007, 2008, 2010 and 2011. We swabbed the pellet surface with a cotton-tipped applicator moistened with DET salt solution (20% dimethyl sulfoxide, 0.25 M sodium-ethylenediaminetetraacetic acid [EDTA], 100 mM TRIS [tris (hydroxymethyl) aminomethane], pH 7.5 and saturated NaCl; Seutin et al. [1991]). The applicator tip was broken off into a 2 ml vial containing 99% alcohol to preserve the sample. We opportunistically collected 2 hair samples, 1 tissue sample and 1 bone sample. We obtained DNA from 24 tissue samples from the Selkirk and Purcell mountains that were acquired by Shafer et al. (2011) from legally permitted

hunters from 2005 to 2007 and 16 genetic samples from the Coast Range of BC that were acquired by Poole and Reynolds (2010) in 2009 from scat and hair. We used 147 genotypes from genetic samples (96 tissue samples, 50 blood samples and 1 bone sample) collected from 2003 to 2008 by Shirk et al. (2010) in collaboration with the National Park Service (NPS) and the Washington Department of Fish and Wildlife (WDFW). All procedures were approved by the Animal Care and Use Committee at Western Washington University and permitted by the WDFW, NPS, United States Department of Agriculture, BC Ministry of the Environment and BC Ministry of Forests Lands and Natural Resource Operations.

Genotyping

We conducted all laboratory procedures at the WDFW molecular genetics lab in Olympia, WA. We used laboratory techniques previously described by Shirk et al. (2010) with these exceptions for scat samples: ethanol was evaporated from the collection vial prior to extraction, initial extraction steps were conducted in the vial to maximize DNA collection and lysis buffer volumes were doubled to cover the entire swab in liquid. We used MICRO-CHECKER 2.2.3 (Van Oosterhout et al. 2004) to screen for allelic dropout, null alleles and stuttering, GENEPOP 4.1.3 (Raymond and Rousset 1995; Rousset 2008) to detect deviations from Hardy-Weinberg equilibrium (HWE) and linkage equilibrium (LE) and GENALEX 6.4 (Peakall and Smouse 2006) to identify samples potentially from the same individual.

Genetic gradients and diversity

Olympic National Park (ONP) hosts an introduced population of mountain goats derived from animals captured in southeast Alaska and the Selkirks in the 1920s. In the 1980s, 130 individuals were translocated from ONP to the Cascades (Houston et al. 1994), where the population was estimated at 8,500 individuals in 1961 (Rice and Gay 2010). We obtained 12 genotypes collected

by Shirk et al. (2010) from ONP and used STRUCTURE 2.3.3 (Pritchard et al. 2000) as described by Shirk et al. (2010) to identify individuals highly admixed with the ONP population and remove those genotypes from this analysis because they do not represent natural population structure or gene flow within the region (Parks 2013).

We used GENELAND 4.0.2 (Guillot et al. 2005) to detect genetic gradients because it outperforms similar methods for detecting barriers in continuous populations with high dispersal ability (Blair et al. 2012). GENELAND uses Bayesian inference to estimate the number of panmictic groups by minimizing Hardy-Weinberg and linkage disequilibrium, while allowing spatial coordinates to inform prior distribution. We used the uncorrelated allele frequency model and evaluated the support for 1 to 10 populations with 10^6 iterations and a burn-in of 1,000. Every 100^{th} observation was sampled to reduce sample autocorrelation. After estimating the value of *K*, we simulated fixed *K* using the above parameters to determine population membership and generate posterior probability maps.

We used the software package sGD (Shirk and Cushman 2011) to estimate spatially explicit indices of genetic diversity and detect fine-scale spatial heterogeneity in diversity across the study area. This approach groups individuals into genetic neighborhoods and is more appropriate for continuous populations. We used a Mantel correlogram depicting autocorrelation in genetic distance between individuals across distance classes, based on Euclidean distance, to estimate the genetic neighborhood diameter, defined as the largest distance class that has a significant (α=0.05) positive correlation with genetic distance (described below). We set the minimum population size to 10 individuals to minimize sampling error.

Modeling framework

We hypothesized that genetic gradients are a function of isolation by resistance (IBR) where genetic distance between individuals is dictated by resistance of a heterogeneous landscape to gene flow (Cushman et al. 2006; McRae 2006). Furthermore, we hypothesized that the relative contribution of landscape variables to genetic distance varies across our large study area due to different local limiting factors (Short Bull et al. 2011). We modeled IBR across the study area and then independently modeled IBR for each pair of adjacent populations identified by GENELAND because this variability may be masked by a single, global analysis. We evaluated the support for multiple hypotheses of IBR accumulated by four landscape variables: distance to escape terrain (D_{et}), roads, landcover and elevation, selected *a priori* as potential factors influencing mountain goat movement (Festa-Bianchet and Côté 2008; Shirk 2009; Shirk et al. 2010; Shafer et al. 2012; Wells 2012). We transformed each variable into alternative hypotheses of landscape resistance using mathematical functions that allowed us to systematically vary model parameters and resistance values (e.g. Shirk et al. 2010). We based resistance and parameter values on previous research by Shirk et al. (2010) and adjusted values accordingly to reach a unimodal peak of support in correlation between genetic distance and resistance distance. We then identified the IBR model most related to genetic distance in each region and tested the support for IBR models against the null model of isolation by distance (IBD).

Mathematical functions for landscape resistance

We obtained a 30 m resolution digital elevation model (DEM) and 20 m resolution Canadian digital elevation data (CDED), using the nearest neighbor technique to resample the CDED to a 30 m resolution and combine the CDED with the DEM. We used focal statistics to assign elevation values to cells with no data based on neighboring cell values and fill a small data gap along the international border. Mountain goats are adapted to utilize an optimal elevation range

between suboptimal lowland valleys and high elevation summits (Festa-Bianchet and Côté 2008; Shirk et al. 2010; Wells 2012). Thus, we modeled landscape resistance due to elevation based on the Gaussian function:

$$R = R_{\max} - R_{\max} * e^{\frac{-(elevation - - E_{opt})^2}{2*E_{SD}^2}} + 1$$

where R is the pixel resistance, R_{max} dictates maximum resistance, E_{opt} is the optimal elevation and E_{SD} is the standard deviation. As elevation moves away from E_{opt}, resistance increases from 1 to R_{max} at a rate dictated by E_{SD}. We evaluated five E_{opt} values (1,200, 1,400, 1,600, 1,800 and 2,000), three values of R_{max} (5, 10 and 25) and three rates of E_{SD} (500, 1,000 and 1,500).

We modeled landscape resistance due to distance to escape terrain (D_{et}) by reclassifying a raster representing Euclidean D_{et}, with escape terrain defined as slope ≥ 50° (Smith 1994), according to the following function:

$$R = (D_{et} / V_{\max})^x * R_{\max} + 1$$

where x is the response shape exponent, R_{max} dictates maximum resistance and V_{max} is a constant representing the maximum value of D_{et}. As the variable increases to V_{max}, the resistance increases to R_{max} at a rate dictated by x. When x is equal to one, the increase to R_{max} is linear, and when x is not equal to one, the increase is nonlinear. We evaluated four different response shape exponents (0.1, 0.25, 0.5 and 1) and seven different values of R_{max} (4, 9, 24, 49, 99, 249 and 449).

We obtained road data at 100 m resolution from the Washington Wildlife Habitat Connectivity Working Group (WHCWG 2010). We classified roads as pixels within 500 m of the road centerline for the following categories: freeway, major highway, secondary highway, local road and no road. We ranked the five road categories from 0 to 4 in order of increasing resistance: no road, local road, secondary highway, major highway and freeway. We modeled landscape resistance due to roads according to the following function:

$$R = (Rank / V_{\max})^{x} * R_{\max} + 1$$

where x is the response shape exponent, R_{max} dictates maximum resistance and V_{max} is a constant representing the highest road resistance rank (4). As the variable increases to V_{max}, the resistance increases to R_{max} at a rate dictated by x. We evaluated five different response shape exponents (1, 3, 8, 10 and infinite) and eight different values of R_{max} (4, 9, 24, 49, 99, 249, 499 and 999).

We obtained landcover data at 100 m resolution from the WHCWG (2010) and classified landcover into seven categories: alpine/sparsely vegetated, grass-dominated, wet forest/dry forest, shrub-dominated, water/wetland/riparian, agriculture and urban/developed. We reclassified urban/developed to no data (complete barrier) because no successful movement would likely occur through this landcover type. The remaining six landcover categories were ranked from 0 to 5 in order of increasing resistance: alpine/sparsely vegetated, grass-dominated, wet forest/dry forest, shrub-dominated, water/wetland/riparian and agriculture. We modeled landscape resistance due to landcover according to the following function:

$$R = (Rank / V_{\max})^{x} * R_{\max} + 1$$

where x is the response shape exponent, R_{max} dictates maximum resistance and V_{max} is a constant representing the highest landcover resistance rank (5). As the variable increases to V_{max}, the resistance increases to R_{max} at a rate dictated by x. We evaluated five different response shape exponents (1, 5, 10, 15 and infinite) and eight different values of R_{max} (4, 9, 24, 49, 99, 249, 499 and 999).

We projected all GIS data to Albers Equal Area Conic GCS North America Datum of 1983. Data layers were resampled to a cell size of 150 m prior to reclassification into resistance surfaces to attain reasonable computation time when calculating pairwise resistance distance. Elevation and D_{et} resistance surfaces were converted to this cell size by aggregating 5 x 5 blocks

of 30 m pixels into a single pixel (based on average aggregation technique and minimum aggregation technique, respectively). The landcover and road rasters were converted from 100 m resolution to 150 m resolution using the nearest neighbor resample technique.

Model evaluation

We selected principle component analysis (PCA) to quantify genetic distance because Shirk et al. (2010) found PCA yielded the highest correlation values with landscape resistance compared to proportion of shared alleles (Bowcock et al. 1994) and Rousset's *a* (Rousset 2000). PCA is theoretically more sensitive to genetic dissimilarity because it reduces multidimensional data into one dimension containing most of the variance, allowing alleles with the most genetic variation to contribute more to genetic distance than common alleles (Shirk et al. 2010). We generated a genetic data matrix $\boldsymbol{Y}$ with n rows and m columns, where n is the number of individuals in the analysis and m is the number of alleles present within the dataset. Each element in the matrix $\boldsymbol{Y}$ (i,j) is populated for individual i by the number of occurrences for the jth allele. The eigenvectors of $\boldsymbol{Y}$ were then computed in R 2.14.2 (R Development Core Team 2012), and the R software package Ecodist (Goslee and Urban 2007) was implemented to generate a n x n pairwise genetic distance matrix ($\boldsymbol{G}$) based on distance between individuals along the first eigenvector (Patterson et al. 2006).

We used Circuitscape 3.5.8 (McRae and Shah 2009) to quantify resistance distance between sample locations because Circuitscape does not assume gene flow is mediated by single, optimal pathways, but instead takes into account how alleles move over multiple pathways through intervening populations over many generations (McRae 2006). We generated an n x n pairwise matrix ($\boldsymbol{X}$) of resistance distance between genetic sample locations for each landscape resistance surface tested. We allowed gene flow to the eight nearest cells (i.e. diagonal

connections enabled) and calculated resistance between two cells as the average of the resistance value assigned to both cells. To model IBD, we used the Landscape Genetics Arc Toolbox (Etherington 2011) distance matrix tool to generate an *n* x *n* matrix of Euclidean distance between all sample locations. We also considered a Log_{10} transformed *n* x *n* matrix of Euclidean distance because the logarithm of geographic distance would theoretically have a higher correlation with genetic distance in two-dimensional landscapes (Rousset 1997).

We used Mantel tests (Mantel 1967) with 10,000 permutations in the R package Ecodist (Goslee and Urban 2007) to calculate the correlation between genetic distance and resistance distance ($\boldsymbol{X}_{Elev}$, $\boldsymbol{X}_{Det}$, $\boldsymbol{X}_{Road}$ or $\boldsymbol{X}_{Land}$). We chose the optimized model of genetic isolation as the model with the highest, significant (*P*-value <0.05) correlation that also reached a unimodal peak of support (Cushman et al. 2006; Shirk et al. 2010).

Causal Modeling

After we identified the optimized IBR model for each region, we evaluated the relative support of IBR against the null model of IBD by employing partial Mantel tests (Smouse et al. 1986) in the R software package Ecodist. This allowed us to evaluate the relative support for IBR and IBD (Cushman et al. 2006; Cushman and Landguth 2010; Shirk et al. 2010). We expected that causal IBR models would retain a significant, positive relationship with genetic distance after partialling out the effect of IBD and have a higher partial Mantel *r* than IBD (Cushman et al. 2013b).

Results

Genotyping

Of the 250 scat samples collected, we deleted 127 genotypes that were less than 63% complete, 40 genotypes potentially from the same individual and 2 genotypes that were highly related to

the ONP population. We retained genotypes from 81 scat samples, 2 hair samples, 1 tissue sample and 1 bone sample. On average, these 85 genotypes were 92% complete. We deleted 11 genotypes from Shirk et al. (2010) that were highly related to the ONP population, leaving 136 genotypes that were 98% complete. The 24 genotypes from Shafer et al. (2011) were 92% complete and the 16 genotypes from Poole and Reynolds (2010) were 98% complete. From all sources, 261 genotypes were used in our analysis. We excluded URB038 because it was monomorphic and McM527 because all samples from the Selkirk and Purcell mountains failed to amplify at this locus. We retained the remaining 17 polymorphic loci.

When the dataset was divided according to the highest level of substructure detected by STRUCTURE 2.3.3 (Pritchard et al. 2000), there was no evidence of allelic dropout or stuttering (Parks 2013). Nine loci (BM203, BM1225, BM1818, BM4107, BM4513, BMC1009, HEL10, OarCP26 and RT9) did show significant homozygote excess in one or two of the seven subpopulations, but because this problem was not systematic we retained all nine loci. We found no significant departure from LE or HWE after dividing the data according to GENELAND population assignments, after Bonferroni correction for multiple comparisons.

Genetic structure and diversity

GENELAND supported the presence of four populations based on K=4 being the most frequent value along the simulation chain and detected three steep genetic gradients that differentiate the Coast Range (CR), Selkirk and Purcell mountains (SP), Okanagan Valley and north Cascades (ONC) and south Cascades (SC) (Fig. 2). Genetic diversity was generally highest in the CR and lowest in the SC, but we also observed fine-scale spatial heterogeneity in genetic diversity across the study area (Fig. 3). The genetic neighborhood diameter was 165 km.

Model optimization and causal modeling

The optimized model of IBR_{Elev} was nearly identical for all three regions, indicating a consistent relation to elevation with R_{max} of 5. E_{opt} is higher in the ONC/SC and the study area, but this is consistent with latitudinal variation in tree line. In contrast, the optimized models of IBR_{Det}, IBR_{Road} and IBR_{Land} varied considerably among regions (Table 1). All optimized models of landscape resistance were highly correlated with genetic distance (r=0.628-0.842, P-value <0.001, Table 2) and all showed unimodal peaks of support. The null model of IBD was also highly correlated with genetic distance and was more highly correlated with genetic distance than the log transform of IBD in all cases (Table 2).

In the CR/ONC (n=163), only the IBR_{Road} model met expectations as a causal model (Table 2). In this model, freeways, major highways and secondary highways contribute resistance of 1,000, 57 and 2, respectively (Fig. 4a). Local roads contribute resistance of 1, equivalent to the resistance contributed by IBD. In the SP/ONC (n=144), both IBR_{Det} and IBR_{Land} met expectations as causal models (Table 2). Resistance due to D_{et} increases linearly to a maximum of 450 (Fig. 4b). In the optimized IBR_{Land} model, alpine/sparsely vegetated, grass-dominated and wet forest/dry forest contribute resistance of 1, while shrub-dominated, water/wetland/riparian and agriculture contribute resistance of 7, 108 and 1,000, respectively. Urban/developed landcover was modeled as a complete barrier (Fig. 4c). In the ONC/SC (n=175), IBR_{Road} met expectations as a causal model (Table 2). In this model, only I90 with resistance of 100 contributes significantly to genetic isolation (Fig. 4d). We removed the 17 northernmost samples from the ONC/SC because gene flow through the Okanagan was modeled in the CR/ONC and SP/ONC.

When we considered the entire study area (n=261, Table 2.), IBR_{Elev}, IBR_{Road} and IBR_{Land} all met expectations as causal models. In the optimized IBR_{Elev} model, elevation contributed

resistance of 1 at E_{opt} of 1,600 and resistance increased to 5 as elevation moves away from E_{opt} at a rate governed by E_{SD} of 1,500, results consistent with Shirk et al. (2010). Both the optimized models IBR_{Road} and IBR_{Land} had infinite shape exponents, where only freeways and agriculture contribute the maximum resistance of 25.

Discussion

Partitioning our large study area into regions of rapid genetic change with GENELAND revealed patterns that were concealed in the global analysis. This approach enabled us to account for landscape-level population connectivity, while controlling for regional variation in the relative importance of landscape variables. We identified local limiting factors within each region and found that the landscape variables influencing gene flow varied regionally. This suggests that the power to detect landscape effects on gene flow is highly dependent on landscape context, i.e. landscape variables present a detectable relationship with genetic differentiation only when the pattern across the landscape varies substantially enough to limit to gene flow.

Genetic diversity

We observed patterns of genetic diversity consistent with the distribution of high-elevation alpine habitat as "sky islands" (Galbreath et al. 2009) across the study area. Genetic diversity was higher in the CR and SP, where patches of alpine habitat are larger and locally well connected, but declined across the Cascades moving toward the southern periphery of the species' distribution, where alpine habitat is less abundant and more fragmented, results that were consistent with a recent study by Shafer et al. (2011) of genetic diversity across the species' range. We also detected fine-scale spatial heterogeneity in genetic diversity. In particular, the Okanagan and northwest region in the north Cascades both exhibited relatively low indices of diversity, likely reflecting limited connectivity to other populations. Genetic diversity was

relatively higher in the central Cascades of the ONC, but declined towards the south Cascades, where we observed the lowest genetic diversity (Fig. 3). The patchy distribution of alpine habitat across WA, isolation at the southern extreme of the species' distribution and historical overharvest (Rice and Gay 2010) likely all contribute to the observed patterns of genetic diversity across the Cascades.

Reduced hunting pressure beginning in the 1990s allowed for the recovery of some WA populations, but large areas of historical habitat remain sparsely populated or unoccupied (Rice and Gay 2010). Although alpine habitat throughout WA is largely intact (approximately 80% of the study area in WA is protected, National Gap Analysis Program), the intervening low elevation habitat has undergone varying degrees of anthropogenic alterations that potentially diminish or sever historical linkages. Consequently, resistance to landscape-level gene flow may further erode genetic diversity and limit the ability of WA populations to recover. Indeed, low heterozygosity has been associated with reduced juvenile survivorship in another small and isolated mountain goat population in Caw Ridge, Alberta (Mainguy et al. 2009). Ortega et al. (2011) observed a temporal decline in genetic diversity in the Caw Ridge population concurrent with increasing population size, but higher heterozygosity in the offspring of individuals that migrated to Caw Ridge. This suggests that increasing population size inadequately compensates for small N_e and that immigration is critical to increase genetic diversity. Furthermore, Hampe and Petit (2005) found that populations residing at the low-latitude margins of a species' distribution, such as those in WA, may be disproportionately important for the long-term conservation of a species' genetic diversity, phylogenetic history and evolutionary potential.

Causal modeling outcomes

Causal modeling supported freeways as the most resistant, significant landscape feature in the CR/ONC, with major highways contributing additional resistance. The inclusion of major highways in this model is not surprising given that Hwy. 99, which links Vancouver, BC to Whistler, a major ski area, cuts across core habitat in the CR. Two additional major highways, Hwy. 1 and Hwy. 3, further inhibit gene flow through smaller habitat patches in the CR and into the ONC (Fig. 4a).

In the SP/ONC, urban/developed was modeled as a complete barrier and agriculture was the most resistant landscape variable, with shrub-dominated, water/wetland/riparian landcover types contributing further landscape resistance. As expected, development and agriculture in the Okanagan Valley severely restrict gene flow into the ONC from the SP, with three large lakes limiting gene flow within the SP (Fig. 4c). D_{et} was also identified as a significant contributor to landscape resistance in the SP/ONC (Fig. 4b). D_{et} is widely expected by expert opinion to potentially contribute to IBR (Festa-Bianchet and Côté 2008; Shirk et al. 2010; Shafer et al. 2012), but Shirk et al. (2010) noted the surprising lack of support for this variable in their WA analysis. Our results suggest that D_{et} does influence individual movement, but only in landscapes where it is a limited resource, as it is in the Okanagan Valley.

Causal modeling supported I90 as the significant contributor to genetic isolation in the ONC/SC. This result is consistent with Shirk et al. 2010, but causal modeling did not support the inclusion of IBR_{Elev} or IBR_{Land}, variables found by Shirk et al. 2010 and our global analysis to significantly influence gene flow. While this is surprising, we had higher correlation between the IBD null model and genetic distance in the ONC/SC (r=0.716) than Shirk et al. (2010) (r=0.686) or our global analysis (r=0.684). Consequently, IBR hypotheses needed a higher correlation with genetic distance in the ONC/SC to be supported as a causal model.

Our approach revealed regional variation in both the shape and magnitude of relationships between landscape variables and genetic distance. Our global analysis only identified the landscape variables that contributed the strongest resistance to gene flow within each region (e.g. freeways, urban/developed and agriculture) and produced estimates of maximum resistance that were below those identified in regional subsets (Table 1). In the case of the IBR_{Road} model, the global analysis underestimated the maximum resistance of freeways relative to the CR/ONC and ONC/SC (25, 1,000 and 100, respectively), possibly because freeways were not supported as a local limiting factor in the SP/ONC, diluting the global signal. Global analysis also drastically underestimated the resistance of agriculture in the SP (25 and 1,000, respectively). We did not find significant support for IBR_{Det} in the global model, likely because escape terrain is not a limiting factor in the CR or ONC. Major highways and secondary highways in the CR and water/wetland/riparian and shrub-dominated landcover types in the SP were other regionally significant landscape variables that were not supported in the global model because regional optimized models identified differing response shape exponents than the global model.

Study limitations

The use of Mantel testing in landscape genetics is controversial (Raufaste and Rousset 2001; Guillot and Rousset 2013; Graves et al. 2013), but multiple analyses defend the use of this method within a causal modeling framework (Cushman and Landguth 2010; Shirk et al. 2010; Cushman et al. 2013b; Castillo et al. 2014). Legendre and Fortin (2010) warn that Mantel tests lead to a large loss of statistical power, and Balkenhol et al. (2009) found simple Mantel tests have high Type I error rates when assessing the relative importance of landscape variables due to high correlation among distance matrices. Cushman and Landguth (2010) found simple Mantel

tests do produce spurious correlations, but partial Mantel tests effectively rejected incorrect explanations and identified the true causal process. Additionally, Mantel tests may be biased when there is spatial correlation in resistance models (Guillot and Rousset 2011; Amos et al. 2012; Meirmans 2012). Cushman et al. (2013b) further evaluated the ability of causal modeling to identify the true driver of genetic isolation and found partial Mantel tests have very low Type II error rates, but elevated Type I error rates when there is high correlation among alternative landscape resistance models. They proposed basing model comparison on partial Mantel *r* values rather than *p*-values, effectively lowering Type I error. With this approach, we detected support for variables significantly limiting gene flow in each region that correspond with GENELAND genetic gradients and global patterns of genetic diversity.

The effect of genetic distance metric choice on causal modeling outcomes has not been evaluated within the field of landscape genetics. It is therefore difficult to anticipate biases in PCA-based conclusions. Although PCA has not been widely applied in landscape genetic studies, Shirk et al. (2010) found consistent causal modeling outcomes when using PCA, proportion of shared alleles and Rousset's *a* (Rousset 2000), and Castillo et al (2014) found genetic distance based on Bray-Curtis percent dissimilarity (Legendre and Legendre 1998) was similar to PCA genetic distance. We detected significant landscape variables within all three regions despite relatively low sample size, supporting the utility of PCA in landscape genetic studies of continuously distributed species.

We did not explore multivariate space in order to maintain reasonable computation time. Only the SP/ONC supported the inclusion of more than one variable, but interactions between D_{et} and landcover may add complexity to the system that is not captured with univariate optimization. Genetic algorithms that more efficiently search parameter space to fit landscape

resistance surfaces to spatial genetic patterns may soon be readily available as computer capabilities increase (Spear et al. 2010). This could enable the development of a multivariate, moving-window analysis that better accounts for complex landscape configuration. Recently, Castillo et al. (2014) found that causal model outcomes based solely on relative support underestimated the magnitude of resistance compared to reciprocal causal modeling. The application of reciprocal causal modeling could increase confidence in our assigned resistance values. Finally, low sample density, particularly in BC, may have limited our ability to detect landscape variable effects. We caution against concluding variables are not important to population viability based on a nonsignificant relationship in this analysis due to biases inherent in modeling complex landscapes and patterns of genetic diversity. For these reasons, management decisions based on our results should be carefully evaluated.

Conclusions

Gene flow is not necessarily bound by regional, state or international boundaries. Additionally, the landscape features that control gene flow may differ across a species' range due to changing limiting factors. Our study attempted to address these issues by comparing a global scale analysis to regional analyses of how landscape features influence gene flow. Dominant landscape variables limiting gene flow varied across the study area, insight that only became apparent through the analysis of subsets of the larger study area. We suggest that landscape-level genetic studies should be carefully designed to account for regional landscape variation. Our results have important conservation implications since local gene flow may be insufficient to counterbalance the genetic consequences of low N_e, making it imperative to understand how the landscape is limiting landscape-level gene flow. Given anthropogenic landscape change, immigration into the Cascades and Okanagan may be insufficient to counterbalance low N_e. Insight gained from our

research better informs habitat connectivity planning for mountain goats in WA and southern BC, where gene flow among these populations at the southern periphery of the species' range can bolster population viability and adaptive potential in response to climate change (Sexton et al 2011).

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Compliance with ethical standards

The authors declare that they have no conflict of interest.

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Table 1 The most highly supported models of IBR due to elevation, D_{et}, roads and landcover within each landscape

	Elevation			D_{et}		Roads		Landcover	
	E_{SD}	E_{opt}	R_{max}	x	R_{max}	x	R_{max}	x	R_{max}

CR/ONC	1500	1400	5	1	5	10	1000	Inf	1000
SP/ONC	1500	1400	5	1	450	3	5	10	1000
ONC/SC	1500	1600	5	1	10	Inf	100	Inf	1000
Study area	1500	1600	5	1	5	Inf	25	Inf	25

Table 2 Causal modeling results for the candidate models of IBR and the null model of IBD

	Model	G~L *r*	G~L *P* value	G~L|D Partial *r*	G~L|D *P* value	G~D|L Partial *r*	G~D|L *P* value	Causal model?
CR/ONC	Elevation	0.628	0.0001	-0.036	0.8198	0.403	0.0001	N

	D_{et}	0.638	0.0001	-0.141	1.0000	0.399	0.0001	N
	Roads	0.783	0.0001	0.491	0.0001	-0.055	0.9538	**Y**
	Landcover	0.662	0.0001	0.105	0.0202	0.326	0.0001	N
	IBD	0.701	0.0001	-	-	-	-	-
	IBD_{log}	0.446	0.0001	-	-	-	-	-
SP/ONC	Elevation	0.668	0.0001	-0.112	0.9807	0.545	0.0001	N
	D_{et}	0.826	0.0001	0.452	0.0001	0.125	0.0062	**Y**
	Roads	0.701	0.0001	-0.278	1.0000	0.534	0.0001	N
	Landcover	0.842	0.0001	0.512	0.0001	0.039	0.2208	**Y**
	IBD	0.779	0.0001	-	-	-	-	-
	IBD_{log}	0.499	0.0001	-	-	-	-	-
ONC/SC	Elevation	0.681	0.0001	-0.027	0.8199	0.303	0.0001	N
	D_{et}	0.688	0.0001	0.053	0.0335	0.278	0.0001	N
	Roads	0.725	0.0001	0.252	0.0001	0.199	0.0001	**Y**
	Landcover	0.692	0.0001	-0.009	0.6756	0.254	0.0001	N
	IBD	0.716	0.0001	-	-	-	-	-
	IBD_{log}	0.537	0.0001	-	-	-	-	-
Study area	Elevation	0.697	0.0001	0.217	0.0001	0.117	0.0003	**Y**
	D_{et}	0.671	0.0001	0.064	0.0067	0.188	0.0001	N
	Roads	0.711	0.0001	0.281	0.0001	0.084	0.0061	**Y**
	Landcover	0.713	0.0001	0.284	0.0001	0.066	0.0195	**Y**
	IBD	0.684	0.0001	-	-	-	-	-
	IBD_{log}	0.523	0.0001	-	-	-	-	-

Bold letters indicate candidate models that are supported as a causal model

(1) G~L—simple Mantel test between the candidate model and genetic distance; (2) G~L|D—partial Mantel test between the candidate model and genetic distance, partialling out Euclidean distance; (3) G~D|L—partial Mantel test between Euclidean distance and genetic distance, partialling out the candidate model. For a candidate model to be supported, (1) and (2) must be significant (α=0.05) and the partial Mantel value for (2) must be greater than the partial Mantel value for (3)

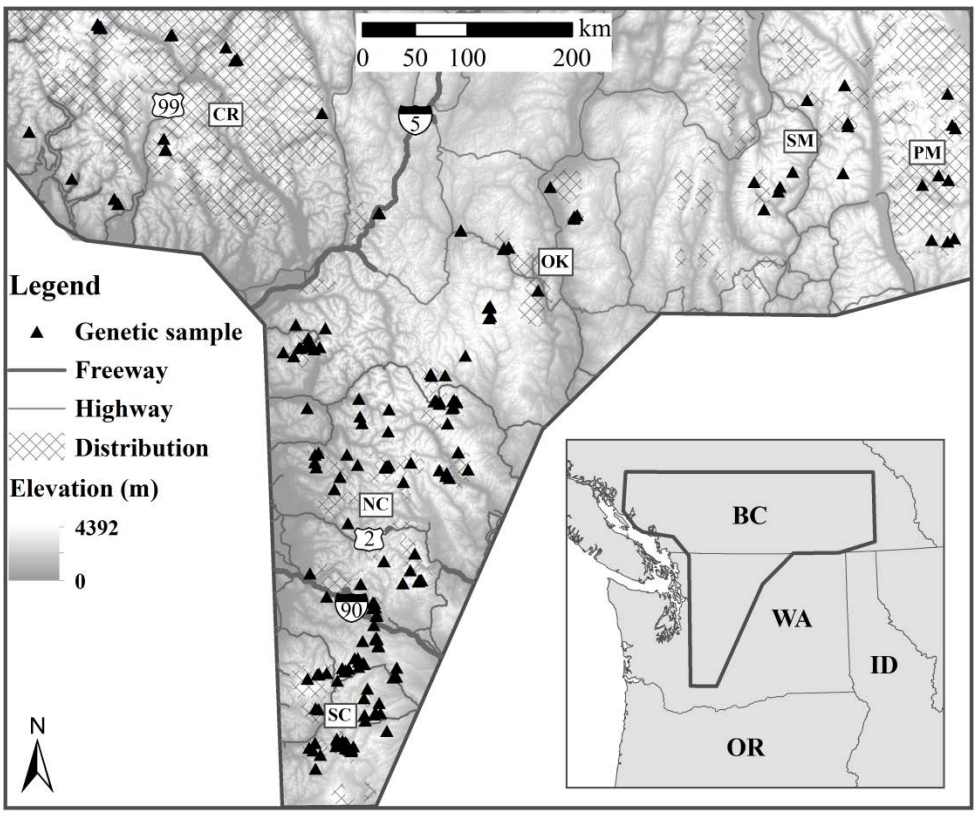

Fig. 1 The study area showing genetic sample locations (black triangles), freeways (thick grey lines), highways (thin grey lines), current mountain goat distribution, elevation and the study area extent orientation. CR: Coast Range; OK: Okanagan; SM: Selkirk Mountains; PM: Purcell Mountains; NC: North Cascades; SC: South Cascades

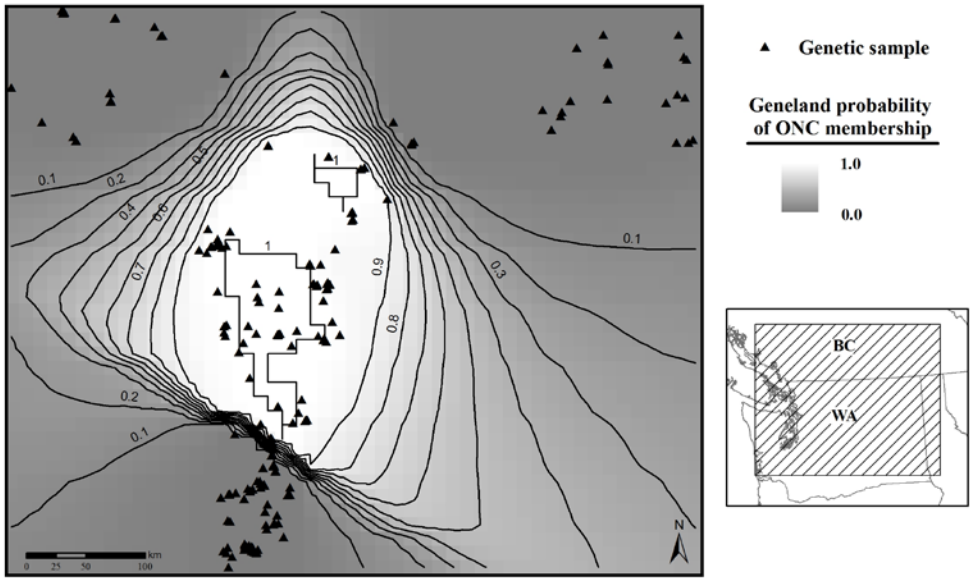

Fig. 2 GENELAND posterior probability map of membership for the ONC subpopulation

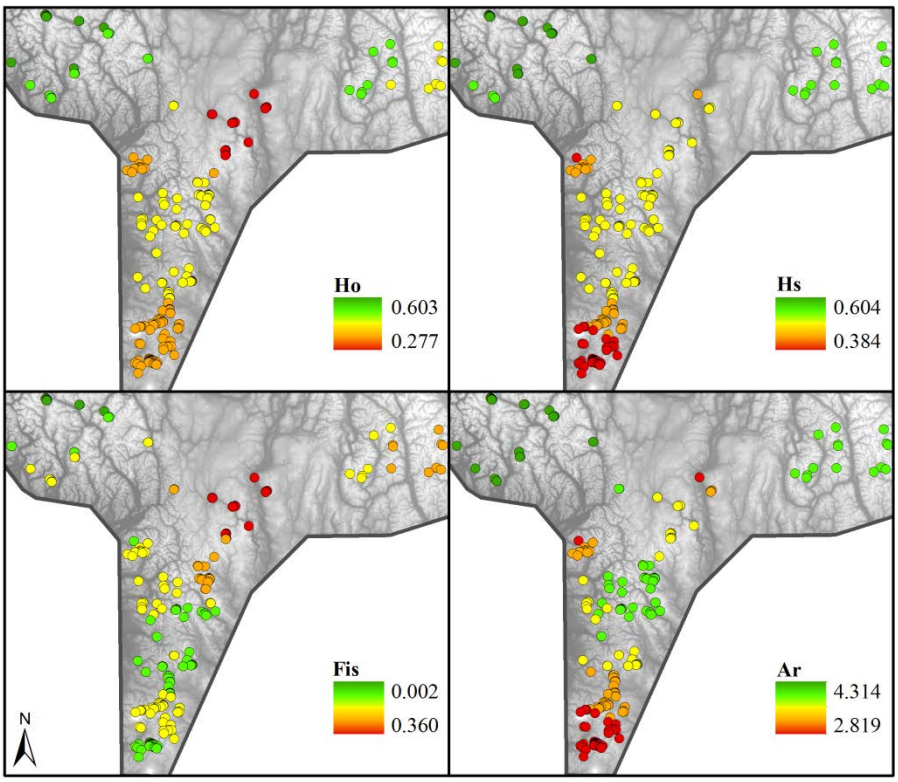

Fig. 3 Spatial patterns of genetic diversity calculated by sGD: observed heterozygosity (H_o), Nei's gene diversity (H_s), inbreeding coefficient (F_{IS}) and allelic richness (A_r)

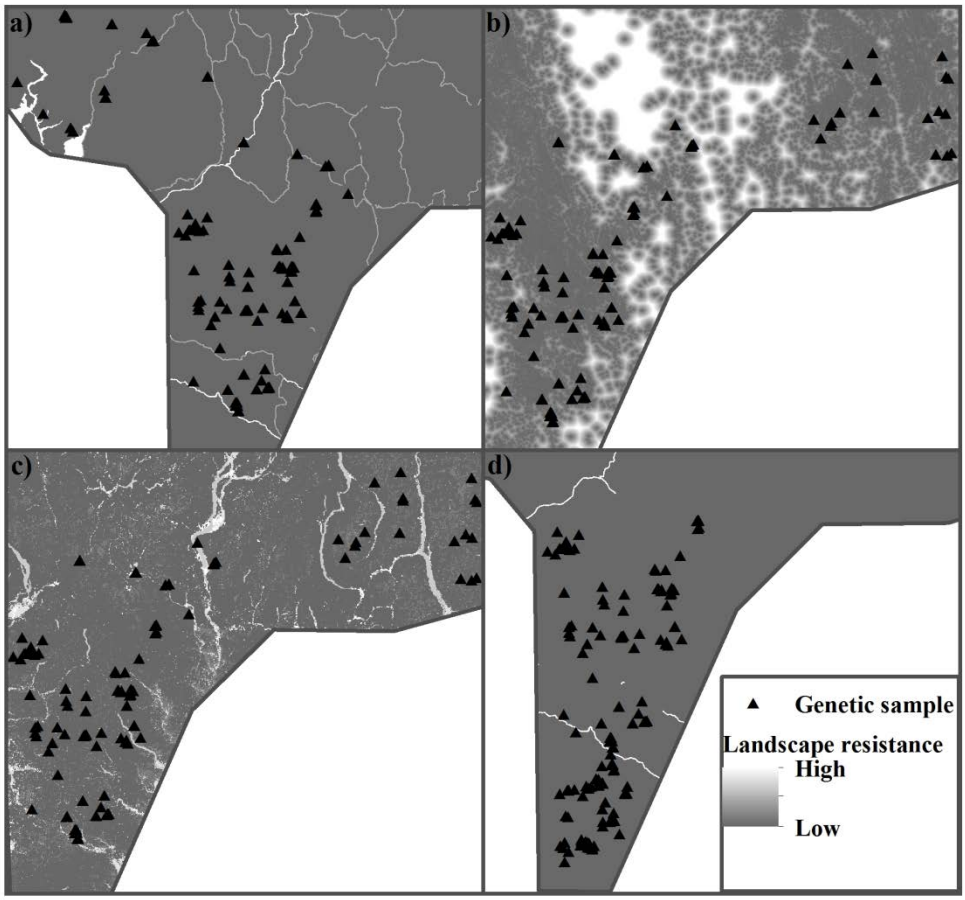

Fig. 4 Landscape resistance models that contribute significantly to genetic isolation. White represents the highest resistance and dark grey represents the lowest resistance. (a) Landscape resistance in the CR/ONC as dictated by roads. (b) Landscape resistance in the SP/ONC as dictated by D_{et}. (c) Landscape resistance in the SP/ONC as dictated by landcover. (d) Landscape resistance in the ONC/SC as dictated by roads