

NEWS AND VIEWS

PERSPECTIVE

Pushing the envelope in genetic analysis of species invasion

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Pushing the envelope

How a species spreads once introduced into a new environment is a major question in landscape genetics. When the species in question is a potential disease vector, the findings are important not only for fundamental science, but can impact applied science and public health as well. In this issue of *Molecular Ecology* Medley *et al.* (2014) study, the invasion patterns of the Asian tiger mosquito (*Aedes albopictus*), which is thought to have been introduced in Houston, Texas in the 1980s and can be a vector for human disease including dengue fever, West Nile virus, yellow fever, encephalomyelitis and the Chikungunya virus. This topic is at the extreme cutting edge of landscape genetics. By combining multiple scales of analysis within a rangewide study of genetic differentiation of an invasive species, Medley *et al.* (2014) undertake a truly ambitious analysis that pushes the envelope in methodology and scope of inference in the study of the genetics of range expansion.

The Asian tiger mosquito is a container-breeding species that is associated with areas of human development. The rapid spread of the species across the US southeast is thought to have been facilitated by long-distance transport of used tires between cities on trucks. Medley *et al.* (2014) use an individual-based landscape genetic approach to test a range of hypotheses about the effects that different landscape features, such as forest cover, agriculture and urban development, and the transportation network. Their results suggest that broad-scale patterns of colonization and range expansion are driven by long-distance transport on trucks, while the spread and expansion of those colonization

events is governed by contagious spread as a function of the resistance of local landscape features to movement and gene flow.

There are several aspects of their study that are particularly interesting. First, there have been very few attempts to identify the factors that govern range expansion and gene flow of an invasive species at multiple scales. The multiple scale analysis Medley *et al.* (2014) present is extremely interesting and suggests different factors may limit gene flow at the core vs. the periphery of the range. An additional interesting idea is the potential role that antagonistic interspecific interactions play in limiting dispersal. Furthermore, the authors' comparison of least cost distance and circuit distance is intriguing, with very interesting result that cost distance was the more effective measure for broad-scale core-edge patterns reflecting recent expansion, while circuit distance was better for predicting genetic structure of the core populations. The authors make a good case for why this would be expected given that large-scale patterns of population spread are likely driven by long-distance transport along highways, such that least cost distance would be a better measure, while core population structure is governed by diffuse and continuous patterns of dispersal along gradients of landscape resistance.

Landscape genetics of range expansion, founder effects, vicariance and secondary contact

Separating the genetic effects of range expansion from anisotropic landscape resistance is a major challenge, and the study seems, like many studies on the cutting edge, to raise as many questions as it answers. Genetic study of an invasive species experiencing rapid range expansion is complicated by several factors, including the influences of the original founder event, the disequilibrium process of contagious population spread from the initial site of colonization, interactions with heterospecific competitors and long-distance founder events establishing new subpopulations.

To better illustrate the spatial dynamism and complexity, this entails I have simulated population expansion from the putative initial colonization site across a 25-year simulation period (Houston Texas; Fig. 1a; See Supporting Information). Following Medley *et al.* (2014), I coded urban and developed areas with the lowest landscape resistance, and forest land, agricultural land, and large water bodies with higher resistance (Table S1, Supporting information). I modelled contagious range expansion from the initial source as a function of landscape resistance with the resistant kernel functionality in the UNICOR software (Landguth *et al.* 2012b) which predicts the frequency of dispersal events as a function of the source population size, location

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and cumulative cost of movement through the landscape. In addition, given the important role of long-distance founder events, I modelled a stochastic process of colonization of additional urban areas as a probabilistic function of connectivity to extant populations through the interstate highway network (blue patches in Fig. 1a).

This original founder effect would likely be characterized by small population size with a relatively small number of alleles relative to the source population, because of stochastic sampling of a small founder population from the large source population. As the founder population expanded rapidly in the new landscape, this low initial genetic diversity would potentially limit researchers' abilities to reliably ascertain the effects of landscape features on gene flow (e.g. Landguth *et al.* 2012a).

The rapid range expansion from the initial site of colonization would potentially drive a disequilibrium between the genetic structure of the population and landscape features, because of the time lag in population genetic response (e.g. Landguth *et al.* 2010; Reding *et al.* 2013). Allelic richness and heterozygosity are typically reduced at the edge of a population (particularly an expanding population) relative to the centre, which would further limit statistical power to understand landscape controls on gene flow and dispersal (Fig. 1a).

Medley *et al.* (2014) suggest gene flow is influenced by landscape features and antagonistic interactions with a congeneric competitor. The heterospecific competitor is hypothesized to occupy suitable niches in forested habitats, such that forest land is seen to resist gene flow and range expansion. However, the forest as surrogate for competitor model is scale dependent and will break down at unknown extents, which may limit utility as a predictor.

Perhaps most challenging, the population spread of the Asian tiger mosquito appears to be driven both by local contagious spread from core populations (e.g. Fig. 1a) and also by long-distance founder events (e.g. blue patches Fig. 1a). These long-distance founder events might further reduce genetic diversity, and random genetic differences between source and colonized sites might add difficulty to reliably identifying drivers of gene flow and range expansion.

A further challenge arises when the dispersed founder populations begin to merge in zones of secondary contact (Fig. 1b). The combination of vicariance and secondary contact, if not accounted for, will introduce bias into biological inferences (e.g. Dyer *et al.* 2010). This is particularly relevant in the context of the Asian tiger mosquito, where local dispersal from core populations coupled with frequent, stochastic, long-distance founder events jointly drive range expansion.

One way to account for these interactions would be to condition the genetic covariance on founder event history (e.g. Dyer & Nason 2004) and then analyse the residual influence of landscape features. However, the challenge in the Asian tiger mosquito system is that the locations and timing of the founder events are unknown and probably unknowable, making it impossible to formally separate the

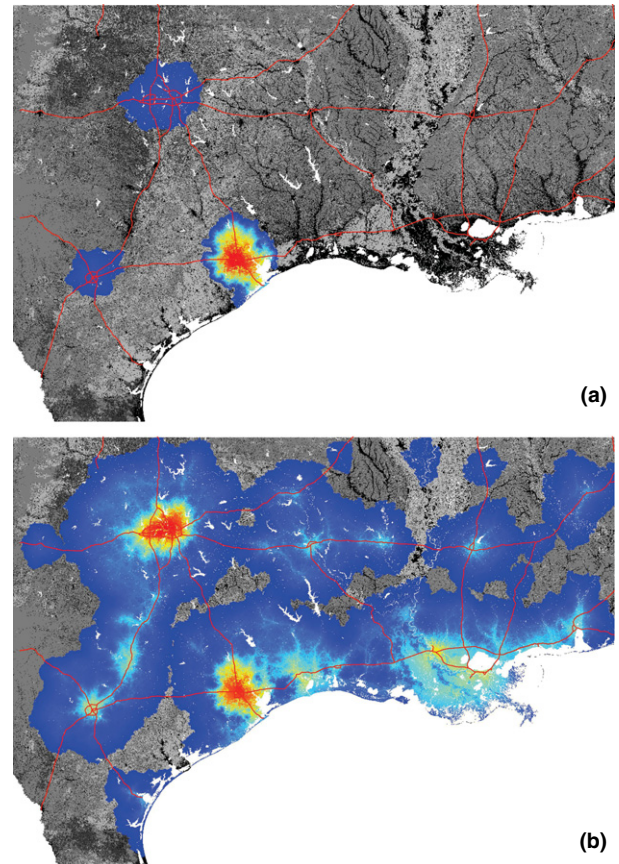


Fig. 1 Results of resistant kernel modelling of population colonization and contagious spread to illustrate aspects of population dynamism relevant in the *Aedes albopictus* system. Resistant kernel modelling predicted contagious spread from an initial colonization event in Houston Texas as a function of cumulative movement cost on a resistance surface. Landscape resistance is shown in a grey scale with high resistance in white and low resistance in black. The interstate highway network is shown as red lines. The red area in panel (a) shows the contagious spread and population growth surrounding the initial colonization site in Houston Texas. Long-distance colonization of new sites was modelled as a stochastic process where probability of colonization was a function of connectivity through the interstate highway network from the extant populations (blue patches in panel a). The simulation produced complex pattern of contagious spread from core populations, continual colonization of new locations through long-distance transport, and resulting range expansion, vicariance, and secondary contact (panel b). The colour gradient in each panel represents the relative abundance in each pixel as predicted by the resistant kernel spread model at that time step, with red indicating the highest population density areas and blue the lowest, and grey areas predicted to be unoccupied.

effects of founder event vicariance, secondary contact and contagious range expansion through resistant landscapes. Failure to separate these effects likely results in observed correlations that are simultaneously the result of several

processes, each governed by different ecological and environmental factors acting at different scales.

Combining simulation modelling and experimentation to improve understanding

The most important future advances in evolutionary and population genetics may depend on integrating genomics, bioinformatics, modelling and experimentation (Cushman 2014). This seems to particularly apply to the Asian tiger mosquito system, in which stochastic founder events, contagious spread, antagonistic interspecific interactions and secondary contact interact in complex and unknown ways. Combining focused experimentation with thoughtful simulation modelling would help resolving these complex interactions (Cushman 2014).

In the Asian tiger mosquito system, several modes of experimentation would be particularly helpful. First, the putative effect of the congeneric competitor mediating apparent resistance of forest to gene flow could be investigated in controlled and replicated experiments. Additionally, the competition hypothesis could be tested with comparative mensurative replicated landscape experiments (e.g. McGarigal & Cushman 2002) where landscape genetic studies are metareplicated (e.g. Short Bull *et al.* 2011) in areas both with and without the competitor. Additionally, both manipulative and mensurative experiments could investigate the effects of founder events, contagious spread from colonization sites and secondary contact on landscape genetic inferences.

Simulation modelling provides tremendous abilities to explore the implications of alternative hypotheses and generalize the results of experiments (Epperson *et al.* 2010). In the context of the Asian tiger mosquito system, spatially explicit, individual-based, cost-distance-driven simulation models such as CDPOP (Landguth & Cushman 2010) could be used to model founder events, vicariance, contagious spread, stochastic long-distance dispersal and secondary contact across complex landscapes. Simulation studies could be designed to evaluate the effects of each of these factors in isolation and then in combination to untangle the processes that drive genetic differentiation and genetic diversity.

Ecosystems are the stage on which the play of evolution is acted (R. May), and ecosystems are complex, spatially structured and temporally varying. Experiments are needed to test hypotheses mechanistically at small scales over short time periods. Simulation modelling is critical to explore how these pattern–process relationships propagate across scale and how variation across space and through time influences their outcomes (Fig. 2). Little is known about how founder effects, stochastic long-distance dispersal, resulting patterns of vicariance, contagious population expansion across resistant landscapes and secondary contact interact to affect the genetic diversity and genetic differentiation across populations. The Medley *et al.* (2014) study pushes the envelope into exciting and important frontiers that are critical to deepening our understanding of spatial genetics. The study presents a number of

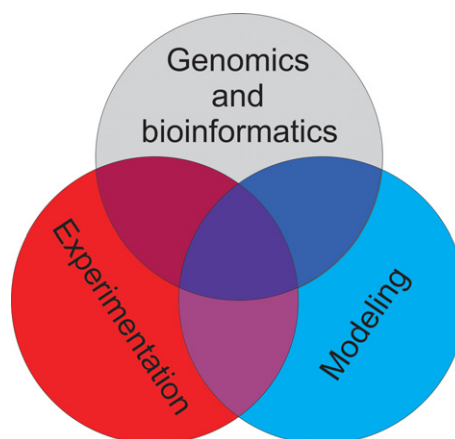


Fig. 2 The resolving the major challenges in understanding the spatial dynamics of founder events, subsequent range expansion through the combination of contagious spread and long-distance colonization, and resulting patterns of vicariance and secondary contact would be facilitated by combining research in genomics and bioinformatics with genetic experiments and spatially explicit simulation modelling. Experiments could be used to test hypotheses and verify causal relationships guide the parameterization and calibration of models. Modelling could generalize and extend experimental results to explore implications of pattern–process relationships across scale.

interesting findings and raises a large range of important and challenging questions. I believe that systematic work combining metareplicated field studies controlled laboratory and field experiments and carefully designed simulation studies offers the best means of making progress in untangling complex population genetic dynamics, such as those in the Asian tiger mosquito.

References

- Cushman SA (2014) Grand challenges in evolutionary and population genetics: the importance of integrating epigenetics, genomics, modeling and experimentation. *Frontiers in Genetics*, **5**, 197.
- Dyer RJ, Nason JD (2004) Population Graphs: the graph-theoretic shape of genetic structure. *Molecular Ecology*, **13**, 1713–1728.
- 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.
- Epperson BK, McRae B, Scribner K *et al.* (2010) Utility of computer simulations in landscape genetics. *Molecular Ecology*, **19**, 3540–3564.
- Landguth EL, Cushman SA (2010) CDPOP: an individual-based, cost-distance spatial population genetics model. *Molecular Ecology Resources*, **10**, 156–161.
- Landguth EL, Cushman SA, Luikart G, Murphy MA, Schwartz MK, McKelvey KS (2010) Quantifying the lag time to detect barriers in landscape genetics. *Molecular Ecology*, **19**, 4179–4191.
- Landguth EL, Fedy BC, Garey AL *et al.* (2012a) Effects of sample size, number of markers, and allelic richness on landscape genetic inference. *Molecular Ecology Resources*, **12**, 276–284.

- Landguth EL, Hand BK, Glassy J, Cushman SA (2012b) UNICOR: a species connectivity and corridor network simulator. *Ecography*, **35**, 9–14.
- McGarigal K, Cushman SA (2002) Comparative evaluation of experimental approaches to the study of habitat fragmentation effects. *Ecological Applications*, **12**, 335–345.
- Medley KA, Jenkins DG, Hoffman EA (2014) Human-aided and natural dispersal drive gene flow across the range of an invasive mosquito. *Molecular Ecology*, **24**, 284–295.
- Reding DM, Cushman SA, Gosselink TE, Clark WR (2013) The influence of landscape connectivity: discordance between movement behavior and patterns of gene flow in Linking movement behavior and fine-scale genetic structure to model landscape connectivity for bobcats (*Lynx rufus*). *Landscape Ecology*, **28**, 471–486.
- Short Bull RA, Cushman SA, Mace R *et al.* (2011) Why replication is important in landscape genetics: American black bear in the Rocky Mountains. *Molecular Ecology*, **20**, 1092–1107.

Samuel Cushman is solely responsible for all aspects of this manuscript.

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Supporting information

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

Appendix S1 Description of the resistant kernel approach used to model population expansion of *Aedes albopictus*.

Table S1 Resistance values assigned to NLCD cover types for resistant kernel dispersal modeling.

Fig. S1 Resistance map showing areas of low resistance in dark (resistance 1 in full black) and areas of high resistance in white (resistance 20 in full white).

Fig. S2. Illustration of first time step of contagious spread from a single location at the center of Houston Texas (a), plus the transformation through multiplying by the selection surface and resampling to produce a set of source points for the subsequent time step distributed at 10 km spacing within the occupied zone (b).

Fig. S3 Resistance map overlain with the least cost path density network among all major urban areas as function of resistance where resistance along the interstate highway network is 1 and elsewhere resistance is 100.

Video S1 AVI video file showing 25 years of simulated population expansion of *Aedes albopictus* across the U.S. Southeast as a function of contagious spread from focal colonization sites and stochastic long-distance colonization along the interstate highway network.

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