ORIGINAL PAPER

Modeling the spread of invasive species using dynamic network models

**Joseph R. Ferrari · Evan L. Preisser ·
Matthew C. Fitzpatrick**

Abstract Spread dynamics of biological invasions are influenced by both the availability and spatial arrangement of suitable habitat. As such, invasive spread can be considered to occur across a network of nodes, representing patches of suitable habitat, with linkages representing the potential for movement between habitat patches. While static network models can provide valuable insight into the potential framework of nodes and linkages across which spread could occur, they offer little information on the actual spatiotemporal dynamics of range expansion processes. Here, we explore the development and application of dynamic network models (DNMs) to model the spread of invasive species. DNMs accommodate temporal dynamics in the utilization of nodes and the connections between them and can flexibly perform simulations at the spatial scales of observational data. As case studies, we develop DNMs to simulate the spread of a generalist forest pathogen and the hemlock woolly adelgid (*Adelges tsugae* Annand). We highlight the utility of DNMs for identifying habitat patches that contribute most to spread across the landscape and for visualizing emergent spread dynamics. While currently underutilized in ecology as compared to static network models, DNMs are potentially applicable to numerous research and management questions relevant to biological invasions and the more general phenomena of range expansion.

Keywords *Adelges tsugae* · Forest · Graph theory · Habitat · Hemlock woolly adelgid · Landscape · Model · Risk assessment

J. R. Ferrari · M. C. Fitzpatrick (✉)
Appalachian Lab, University of Maryland Center for Environmental Science, 301 Braddock Road, Frostburg, MD 21532, USA
e-mail: mfitzpatrick@umces.edu

Present Address:
J. R. Ferrari
Wartsila North America, Inc., 900 Bestgate Road, Suite 400, Annapolis, MD 21401, USA

E. L. Preisser
Department of Biological Sciences, University of Rhode Island, Kingston, RI, USA

Introduction

For decades ecologists have conceptualized ecological systems as networks, with food web structure being one of the earliest and best-known examples (Pimm et al. 1991). More recently, landscape ecologists have used spatially explicit networks to quantify landscape connectivity and to identify potential pathways for dispersing organisms (e.g., Minor and Urban 2008; Urban et al. 2009; Lookingbill et al. 2010; Fletcher et al. 2011). The broadening application of networks in ecology has paralleled their increasing use in other disciplines such as sociology, public health, telecommunications, transportation, and biogeography (Shavitt and Singer 2007; Butts 2009; Cumming et al. 2010; Araújo et al. 2011).

Regardless of discipline, many applications of network theory rely on static networks to represent dynamic systems (Banks et al. 2010). Static network models emphasize the effects of network structure on stability and connectivity (i.e., *where* connections occur and their importance) rather than the temporal dynamics related to *when* connections arise or disappear and their probability of doing so. In landscape ecology, for example, static networks are often used to represent interpatch connectivity as a function of distance (Bunn et al. 2000; Ferrari et al. 2007; Minor and Urban 2007; Lookingbill et al. 2010). While static networks are widely used and often useful, many questions in invasion biology concern processes that are dynamic in both space and time. When an introduced species spreads across a landscape of suitable habitat patches, for example, a static network can be developed using information on the species' dispersal ability to determine the underlying network of habitat patches and potential interpatch connections (Ferrari and Lookingbill 2009). Although such static networks can identify possible spread pathways and inform questions regarding network structure, they provide little insight regarding how interpatch connections are likely to evolve as the invasion proceeds. From a management perspective, knowing when an invader is most likely to arrive in a new location, and which patches may contribute most to spread, are likely to be more useful than a simple delineation of invasion-prone locations based on habitat suitability or interpatch distances alone.

An alternate framework for modeling range expansion involves dynamic network models (DNMs), which have seen broad application in other disciplines. DNMs share several features with their static counterparts; for instance, both consider connections between interacting entities and can rely on a predefined potential network that identifies links and nodes. Unlike static networks, however, DNMs allow connections to change through time by temporally mapping when each node and link become active in the simulated dynamic process. The emergent dynamic network may only constitute a subset of the total potential static network and can be highly dependent on initial conditions of the ecological process of interest (Ferrari and Lookingbill 2009). When applied to biological invasions, DNMs may provide a new suite of analytical techniques for answering questions regarding spread dynamics and for intuitively visualizing range expansion to provide information which

may prove useful in developing and applying control measures (e.g., Sharov et al. 2002) and which may not be readily obtainable from more established methods of modeling spread.

We describe the application of DNMs to the problem of modeling range expansion, an inherently dynamic ecological process. We focus on the spread of invasive species generally and forest pests in particular, although the approach is general and applicable to a wide array of dynamic ecological processes. To introduce the approach, we first develop a DNM for a hypothetical forest pest and then simulate its spread across a landscape of forest patches. We then develop a DNM for the spread of hemlock woolly adelgid ('HWA', *Adelges tsugae*; Hemiptera: Adelgidae), an introduced pest of eastern hemlock (*Tsuga canadensis*) and Carolina hemlock (*Tsuga caroliniana*) in the eastern United States (Orwig 2002). We use these case studies to examine the dynamics of invasive spread and to demonstrate the utility of DNMs for quantifying and visualizing these dynamics.

Methods

Network representations of landscapes typically discriminate ecological resources, often as pixels of specific habitat types in gridded, digital maps, from unsuitable background areas. Collections of suitable pixels are aggregated into patches that are defined as "nodes" in network parlance. For invasive species, nodes might represent patches of disturbed habitat, or, for forest pests, forested patches containing host tree species. Patterns of potential connectivity among nodes are identified as "links". While such connections are commonly referred to as "edges" in network science, we prefer the term link to avoid confusion with other common uses of "edge" in ecology and invasion biology (e.g., the edge of the advancing population of a range-expanding species). For a static network, links are typically a function of distance alone and the resulting link patterns form the basis of a pairwise patch adjacency matrix A. Under a static network paradigm, the matrix A is constant in time (e.g., Keitt et al. 1997), with entries that are either binary (0/1; not connected/connected) or weighted (0–1 corresponding to connection strength). The matrix A is the fundamental basis of static network models.

The spread of a forest obligate invasive species across a habitat network can be cast in an epidemiological context (Mollison 1977) that identifies nodes as forest patches (depending on the scale of analysis, nodes could also be individual trees in a stand or geopolitical units such as counties) that are assigned a status of either Susceptible (*S*) or Infected (*I*). This approach is conceptually similar to epidemiological models advocated for human diseases (e.g., Watts and Strogatz 1998; Mack et al. 2000; Vazquez 2006), invasive species (Gilligan and van den Bosch 2008) and, in particular, pathogens (Brooks et al. 2008) and forest pests (Meentemeyer et al. 2011). Each *I* node contains a population capable of increasing with time, and has the potential to emit propagules that can spread the infection to *S* nodes. Borrowing from social network analysis (Leenders 1995; Snijders 2005), we chose a Markovian framework for the DNM. The R programming language (R Development Core Team 2011) was used for all simulations.

Markov analysis addresses transitions between states (e.g., from *S* to *I*) and requires conditions at time $t + 1$ be dependent on conditions at time t, a plausible precondition for spread of an invasive species. Addressing spatial spread, the vector (Eq. 1) represents the number of propagules leaving *I* nodes at time t. The matrix T contains pairwise probabilities (τ_{ij}) of propagule movement from nodes *i* to nodes *j*. The vector represents the number of propagules reaching nodes at time $t + 1$ and can be used to evaluate the timing of spread between nodes (i.e., node status change *S* to *I*), calculated as

$$\vec{w}_{t+1} = \vec{m}_t T \tag{1}$$

In essence, Eq. 1 links a within-node population growth process ($\vec{m}_t$) with a between-node dispersal process (T) to model spread; more complex formulations can be accommodated. Any DNM used to simulate range expansion relies on such an underlying model of within node population growth and between node dispersal across the habitat network. Here, we model these processes using a simple cellular automaton model to simulate spread of a hypothetical forest pest and a more complex stochastic spread model to simulate the spread of HWA. To build the DNM, successful transfers of propagules between nodes ($\vec{w}_{t+1}$) and the timing of these transfers are recorded. When mapped in a time series, the time-dependent linkages between nodes reveal dynamic population

expansion as an evolving, emergent network of habitat connections. By running many stochastic simulations, the probability of links emerging at different times can be quantified.

Model development

As an initial demonstration, we used the DNM framework to model range expansion of a hypothetical forest-obligate invasive species across a landscape of forest patches. We used the 1,318 ha Antietam National Battlefield Park (ANTI), located in central Maryland, USA as an example landscape (Fig. 1), with forest classes from the National Land Cover Dataset (Homer et al. 2004) used to identify 50 forest patches at 30 m resolution. All other cells were considered 'non-forest'.

Dynamic network model for ANTI

To formulate the landscape and associated spread dynamics as a DNM, we took the following steps: (1) to define the spatial structure of the network, we considered each forest patch to be a node, resulting in 50 nodes; (2) using inter-node distances, we defined potential links among the 50 nodes in a 50×50 adjacency matrix A (i.e., the static network); and (3) we generated the transition probability matrix T, entries in which estimate the likelihood a link in the adjacency matrix A would be "realized" in a dynamic simulation.

To determine the potential links in A, we used a negative exponential probability density function, commonly used for dispersal estimation (e.g., Minor and Urban 2007), to calculate the probability P_{ij} a propagule will move a distance d_{ij} between nodes *i* and *j*, or

$$P_{ij} = e^{-\phi d_{ij}}, \tag{2}$$

where the coefficient ϕ determines the rate of decay of the probability with distance. Using LANDGRAPHS (Urban 2003), $d_{ij} = 720$ m was found to be the smallest inter-patch Euclidean distance for which each of the 50 nodes was connected to at least one other node. For our simulations, we set the probability P_{ij} (Eq. 2) to 0.05 for a tail distance of 720 m, and calculated the necessary decay coefficient ϕ to meet

Fig. 1 An evolving
network resulting from
1,000 stochastic simulations
of a dynamic network model
for the invasion of a
hypothetical forest pest
spreading across Antietam
National Battlefield Park.
Black circles indicate node
centroids of forest patches
(*dark grey shading*). Link
symbolization is scaled
relative to probability of
occurrence by time *t*, with
darker, *thicker lines*
indicating greater
probabilities. The *white
square* in the northwest
corner of the map indicates
the initial location of the
simulated infestation
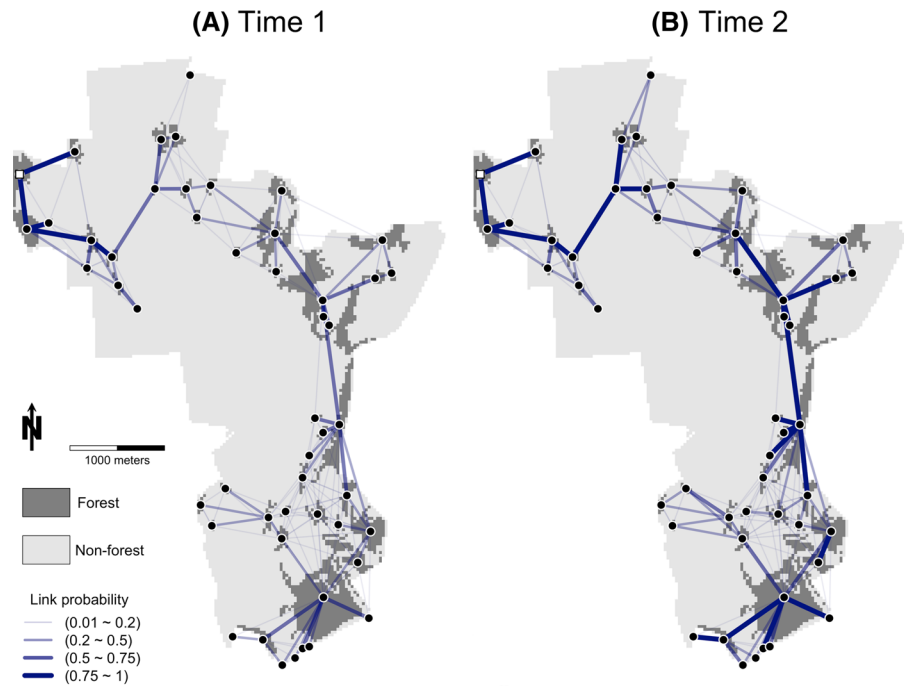
this condition. Every *i*–*j* node pair with $d_{ij} \leq 720$ m therefore had a corresponding entry in $\boldsymbol{A}$ and $\boldsymbol{T}$. As in any theoretical simulation, different values for these parameters would lead to different spread dynamics.

We estimated transition probabilities (τ_{ij}) in $\boldsymbol{T}$ from any source node *i* to any potential receiver node *j* as a function of d_{ij} and the patch area of node *j* (F_j). For tractability, we assumed nodes were circular as our goal was to construct a straightforward DNM for evaluation purposes, and the circular approximation was sufficient for this purpose. The probability of any propagule from node *i* landing in node *j* is therefore

$$\tau_{ij} = \frac{1}{2\pi} \int_0^{2\pi} \int_0^{\infty} e^{-\phi d_{ij}} dr \, d\theta, \quad \text{for all } r, \quad \theta \in F_j. \quad (3)$$

The dividend 2π forces $0 \leq \tau_{ij} \leq 1.0$. Because no two patches have identical area, $\tau_{ij} \neq \tau_{ji}$.

Infestation status (*S* or *I*) and spread were considered at both the patch (node) scale and for each forest cell within nodes. We assumed node carrying capacity equaled the number of cells in the respective forest patch, and at any time each cell was assigned *S* or *I* status. Only *I* cells can lead to within-node population growth or to between-node dispersal. Any node containing a single *I* cell was considered infected;

otherwise, the node contained all *S* cells and the entire node was considered susceptible.

The number of infested cells in an *I* node was assumed to start with one cell initially at the node centroid and increase deterministically at each time step to adjacent *S* cells within the node (using an 8-neighbor rule), assuming homogeneous forest structure within the node. At the end of the population growth phase, we assumed each infested cell had a 20 % probability of contributing to a between-node long-distance dispersal event. A random number μ was drawn for each of these events; if $\mu \leq$ the transfer probability then a successful transfer from node *i* to *j* occurred. If node *j* had status *S* when the transfer was made, the event was considered a *founder event*; the status of the central cell in node *j* changed from *S* to *I*, and status for node *j* changed from *S* to *I* as well. For each founder event, we recorded the node pairs involved in the transfer and the timing of the event as a "realized link-list", which constituted the primary output of the DNM.

Summarizing DNM output

We summarized output from the DNM by evaluating realized link-lists and by mapping time series of

realized links. Node utilization during the invasion process was quantified using a variant of the centrality measure "source strength" (Yook et al. 2001), defined as:

$$S_i = \sum_{k=1}^{D_i} Z_k \qquad (4)$$

The subscript k ranges from 1 to the degree of node i (D_i, the number of neighboring nodes joined to node i). The value of Z_k is given as

$$Z_k = \frac{H}{w} \qquad (5)$$

where H is the number of occurrences of link k with source node i and w is the number of stochastic simulations. For our analyses, w equaled 1,000. If a link was realized in all w replicate simulations, $Z_k = 1.0$, thus $0 \leq S_i \leq D_i$. Degree and source strength are measures of centrality, the extent to which a node is connected within the network, a metric often used for network analyses (Opsahl et al. 2010). In the context of biological invasions, S_i measures the extent to which a node acts as an infection source for other nodes; nodes with relatively high S_i values promote spread throughout the network.

Model application to hemlock woolly adelgid

For a real world application, we developed a DNM to simulate and visualize the spread of the hemlock woolly adelgid (HWA), a rapidly spreading invasive pest of eastern and Carolina hemlock in the eastern United States (McClure and Cheah 1999). Since its introduction in Richmond, Virginia in 1951 (Stoetzel 2002), HWA has rapidly invaded eastern US forests, causing widespread hemlock mortality from Maine to Georgia (Orwig 2002). Spread is driven by passive dispersal, with birds, deer and humans acting as the primary dispersal agents (McClure 1990; Turner et al. 2011).

Any DNM used to simulate range expansion relies on an underlying model of within node population growth and between node dispersal across the habitat network. To simulate spread of HWA in a DNM framework, we used a modified version of an existing spread model conceptually analogous to that described for ANTI above, but which incorporated greater complexity in terms of intra-node population dynamics and between-node dispersal. The model, which is described in detail in Fitzpatrick et al. (2012), couples a stochastic population model that simulates reproduction and mortality of the different stages of HWA's lifecycle with stochastic dispersal functions that model the movement of propagules between locations. Population and dispersal dynamics were parameterized using multiyear field surveys of HWA reproduction and survival rates in New England and multiple data sets documenting the spread of HWA across different regions of the eastern United States. When placed in a DNM framework, the model produces a series of time-dependent adjacency matrices. Analysis of these matrices reveals critical dispersal pathways and identifies nodes that contribute most to spread across the landscape. These key insights are valuable for understanding spread dynamics and potentially for management, yet are not readily obtainable from similar metapopulation or cell-based models of range expansion, including the model of Fitzpatrick et al. (2012) or from analysis of static graph adjacency structure.

The model simulates HWA population growth and dispersal on an annual time step; spatiotemporal variability in habitat and climate is accommodated using high-resolution raster maps (1 km × 1 km) of hemlock abundance and winter temperature respectively, which constitute the primary controls on HWA spread (Evans and Gregoire 2007; Morin et al. 2009; Fitzpatrick et al. 2010). For our purposes, the hemlock and temperature maps used in Fitzpatrick et al. (2012) were aggregated to the county level (see below). Hemlock abundance in each county determines the probability that dispersing HWA establish and also sets the upper limit to HWA population growth once a county becomes infested. Winter temperatures (December–January) change annually following observed temperature fluctuations during the period 1951–2008 and influence population growth by causing HWA mortality.

To simulate relevant population dynamics of HWA, multiyear field surveys of HWA reproduction and survival rates in Massachusetts and Connecticut were used to estimate probability distributions, which were sampled to simulate stochastic reproduction and mortality of different life stages of HWA. Parameters included average number of new individuals produced by each overwintering HWA and the associated mortality rates of progrediens (the generation present in spring and produced by overwintering sistens). For the

sistens generation (present from early summer to late spring and produced by progrediens), parameters included the average number of sistens produced by progrediens and the mortality rates of dispersing and aestivating sistens. For details regarding the life cycle of HWA see McClure (1989). To simulate stochastic population growth, we sampled Poisson distributions and multiplied these values by the existing population size of either sistens or progrediens. Binomial distributions were used to simulate mortality of progrediens and sistens, except for overwintering mortality of sistens, for which we used a linear model developed by Paradis et al. (2008) to relate winter temperature to overwintering mortality of sistens. Lastly, we incorporated the influence of density dependence on population dynamics by allowing HWA density to increase to the carry capacity set by hemlock abundance. See (Fitzpatrick et al. 2012) for further details regarding the model.

Creating nodes for the DNM

Nodes for the HWA DNM were considered to be any of the 1,168 counties in the eastern United States that contain hemlock. Simulating spread at the county level reflects the scale of the long-term observational spread data for HWA, which are recorded as the date of first observation in a county (Fitzpatrick et al. 2010). To quantify hemlock abundance in each county, we calculated the mean basal area ($\overline{BA}$) of all 1 km × 1 km cells from the hemlock abundance map containing hemlock in each county. We also used the hemlock abundance values to determine a basal-area-weighted centroid of all hemlock cells, thereby locating the node centroid closest to regions with greatest hemlock abundance rather than at the county centroid. Between-node distances were calculated as the Euclidean distance between the basal-area-weighted centroid of each county. In sum, the manner in which we model population and dispersal dynamics of HWA are the same as in Fitzpatrick et al. (2012), but the implementation and landscape are different.

The dynamic network model for HWA

To determine the potential links in the node adjacency matrix A (1,168 × 1,168), we applied a between-node cutoff distance of 200 km, representing a 0.01 % tail probability of transmission as calculated using the log-

normal probability density function that best described the distribution of observed dispersal distances (Fitzpatrick et al. 2012). For each county pair with centroid–centroid distances <200 km, transfer probabilities in T were calculated using numerical integration of the log-normal kernel similar to the approach described above for forest patches at ANTI and assuming counties were circles of the appropriate area.

One thousand stochastic simulations of the DNM were initiated by seeding a population in Richmond, VA, where HWA was first detected in 1951. For each time step, the total number of HWA produced (N_i) in each infested county (I nodes) was multiplied by a random number β ($0 < \beta \leq \beta_{max}$) to estimate the number of propagules subject to between-node long-distance dispersal. To tune β_{max}, which determines the upper limit to the proportion of HWA propagules subject to between-node dispersal, we performed sensitivity analyses. We determined the optimal value for β_{max} was 7.5×10^{-4}; values smaller than this led to more than 50 % of model runs having no expansion beyond Richmond, VA, and larger values yielded accelerating rates of spread that exceeded those observed for HWA (e.g., 10–20 km per year; Evans and Gregoire 2007). The product $N_i * \beta$ was multiplied by all transition probabilities with node i as the source, and each product was multiplied by the mean basal area of each potential recipient node j to accommodate the assumption that the probability of HWA landing and establishing in a node is linearly proportional to the amount of hemlock in that node. If the product was greater than one for any node j previously having S status, HWA was assumed to have established in county j and the status of that node was changed to I. All such realized links were recorded, allowing multiple founder events to occur simultaneously. Simulations were ceased when the northernmost county with observed presence of HWA as of 2008 was infested. Most simulations ceased by the mid-1990's.

To summarize output from the HWA DNM, a metric similar to source strength (Eq. 5) was evaluated:

$$\gamma_t = \frac{\sum_{i=1}^{k_t} \left(\frac{n_i}{w}\right)}{k_t} \qquad (6)$$

where k_t is the number of realized links at time t, w is the number of stochastic simulations, and n_i is the number of

times each of the k_t links were realized out of w simulations. The numerator of Eq. 6 is the sum of the probabilities of links occurring at time t and the denominator is the number of realized links as of time t. If all realized links are predicted to occur in all simulations at time t, $\gamma_t = 1.0$. While source strength evaluates the characteristics of each node, γ_t is a measure of the stochasticity of the emergent network in time.

Results

DNM for ANTI

The DNM for ANTI revealed an emerging, evolving network that differed from the static network representation. Links between nodes closest to the initial infection had the highest probabilities of realization early in the simulation, while probability of link realization was generally low elsewhere (Fig. 1a). As the invasion proceeded, link probabilities increased for nodes more distant from the node where the invasion was initiated (white square, Fig. 1), though probabilities generally remained low for smaller, isolated nodes (Fig. 1b). Source strength values from the DNM were always less than node degree (Fig. 2), indicating that none of the 50 nodes had all potential links realized in all 1,000 simulations. Source strength represents the extent to which the node tended to act as a source or a sink. Most nodes tended to act as sinks (zero or low source strength), with fewer than ten nodes strongly acting as a source of infection to other nodes. The strongest source nodes tended to be large patches in close proximity to many adjacent patches, while some smaller nodes acted as sources mainly if they were near the initial source of the invasion.

The frequency at which links were realized across the 1,000 simulations measures the extent to which links are likely to be utilized in the spread process. Of the 340 potential links possible in the static network, 154 were never realized in any of the 1,000 simulations (Fig. 3), indicating clear differences between the static network and the DNM. Fewer than 50 links were realized in more than 50 % of the simulations.

DNM for HWA

The mean simulated maximum number of counties infested in any 1 year was approximately 28 (between
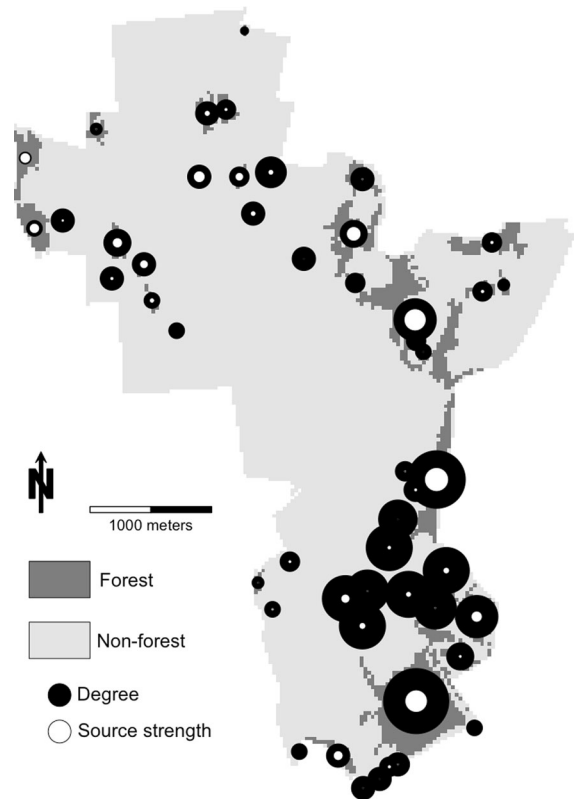

Fig. 2 Nodes scaled to (*black*) degree (i.e., the number of potential connections to neighboring nodes based on distance alone) and (*white*) source strength, a measure of the extent to which nodes act as a source of infection to other nodes across 1,000 stochastic simulations. Nodes represented by *large white circles* tended to act as strong sources of secondary infection

1970 and 1980), compared to observed maxima of ~ 35 counties/year occurring in the early 1990s and the mid 2000's (Fig. 4a). The cumulative simulated number of counties invaded per year (Fig. 4b) followed a classic logistic growth pattern, characteristic of a 'Type 3' invasion process (logistic growth) identified by Shigesada et al. (1995) that begins with a prolonged initial establishment, or "lag" phase followed by rapid range expansion driven by long-distance dispersal until most suitable habitats are occupied and spread slows. Mean simulated and observed rates of spread for post-lag years were strongly related ($r^2 > 0.94$), though the simulated values had a slope of 18.1 counties per year versus 12.0 counties per year for the observed data. The lag phase was shorter in the simulations (approximately 10 years; 1951–1961) than in the observed invasion (29 years; 1951–1980). After the lag-phase and before
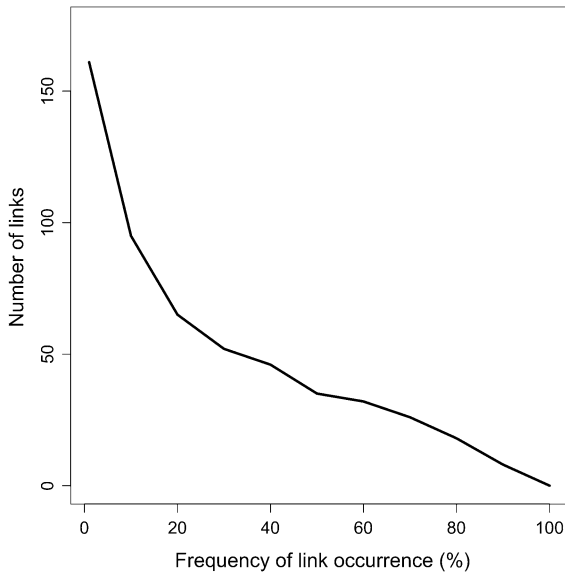
Fig. 3 Number of links versus frequency of occurrence for 1,000 stochastic simulations of a dynamic network model for the invasion of a hypothetical forest pest spreading across Antietam National Battlefield Park

the maximum rate of spread was realized, both the simulated and observed spread patterns indicate a period of intermediate acceleration of HWA spread (~1960–1966 simulated, ~1985–1991 observed, Fig. 4b). The post-lag acceleration of the invasion front was associated with a moderate increase in the number of new realized links followed by a rapid increase in new realized links in the mid-1960's (Fig. 4c).

On average, 43 % of the 1,168 counties containing hemlock had been infected by the end of the simulation. This resulted in a transition probability matrix T having 97,261 non-zero entries, of which only 15,561 (16 %) were realized in all 1,000 simulations. When links were mapped as an evolving network (Fig. 5), the lag phase is associated with low-probability spread (i.e., all links have low probability of occurrence) to counties surrounding and predominantly west and north of Richmond, VA (Fig. 5a, b). Early acceleration of spread rate corresponds to the simulated arrival of HWA by 1966 to counties west and north of Richmond, VA with relatively high

Fig. 4 Comparisons of observed and simulated invasion patterns from 1,000 stochastic simulations of a dynamic network model for the spread of the hemlock woolly adelgid in US counties containing hemlock. a Observed and mean simulated number of new counties invaded by HWA per year; b cumulative number of observed and mean simulated counties invaded by HWA for the period 1951–2008; c mean simulated number of realized links per year; and d gamma ratio (see text for description). Dashed lines in a and c represent ±1 standard deviation
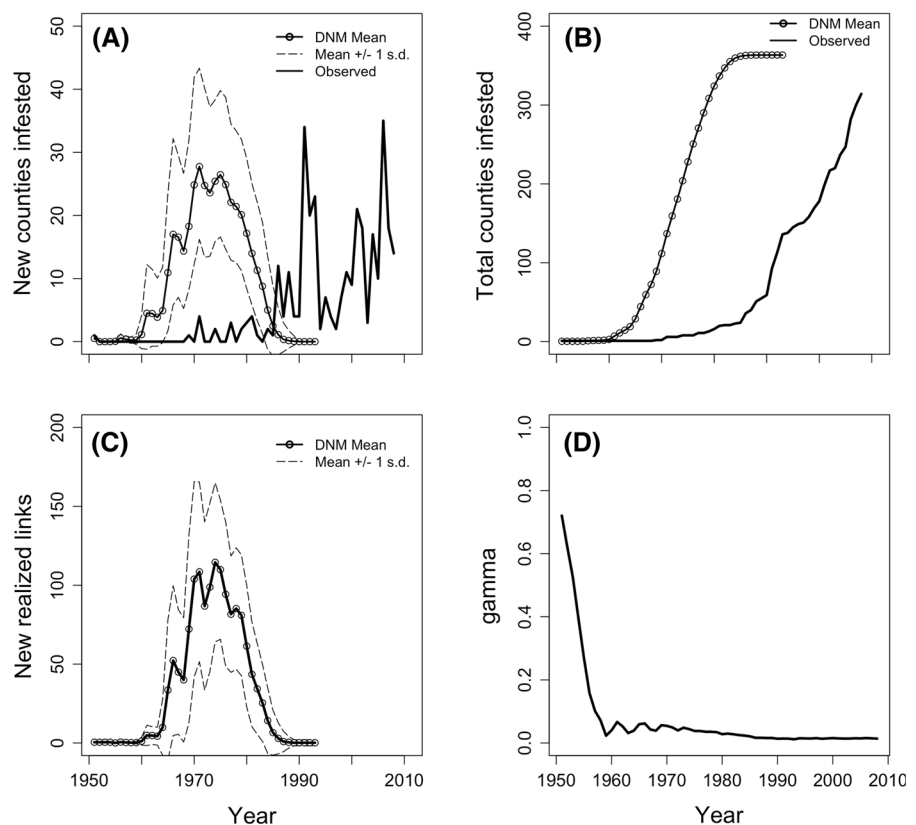
Fig. 5 Time series of an evolving network from the initial stages of 1,000 stochastic simulations of a dynamic network model for the invasion of the hemlock woolly adelgid. *Hollow circles* indicate uninfested US counties containing hemlock. *Black circles* are scaled to source strength and indicate nodes simulated to become infested by time t in at least 1 simulation. Link symbolization is scaled relative to probability of occurrence by time t, with *darker*, *thicker lines* indicating greater probabilities as in Fig. 1. The *white square* indicates the initial location of the infestation in Richmond, VA in 1951
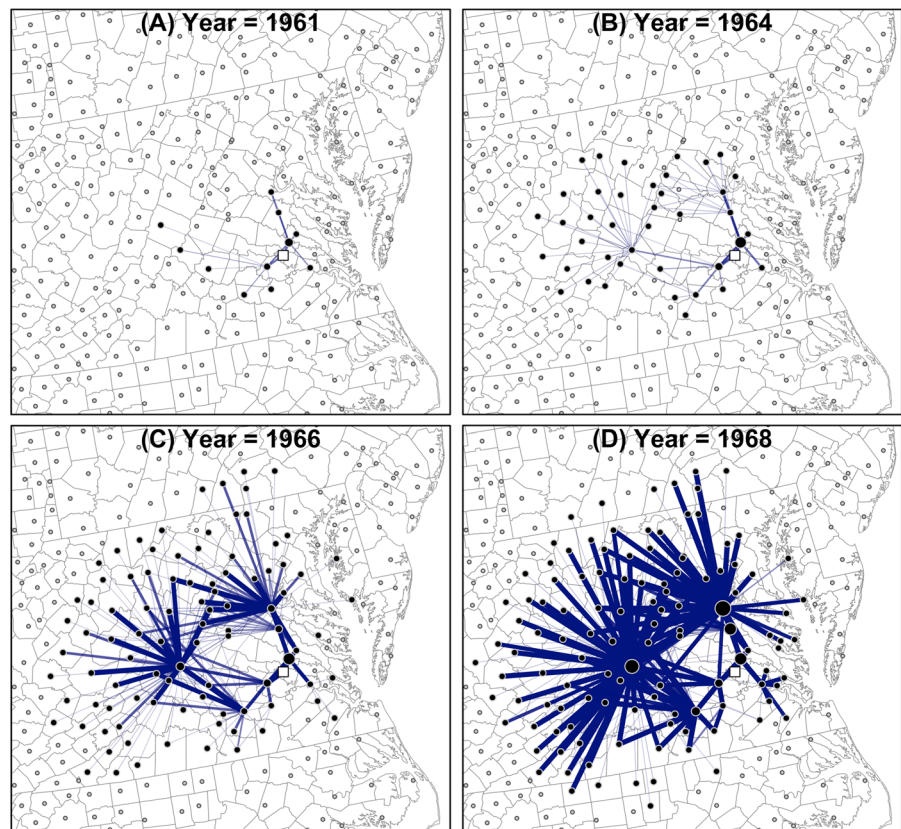
hemlock abundance (Fig. 5c). Once infested, these counties acts as sources of infestation for surrounding counties and initiate rapid and high-probability spread throughout the network (Fig. 5d).

The gamma ratio (γ_t, Eq. 6) measures realized link frequency relative to the total number of realized links. The closer γ_t is to 1.0, the more consistently the DNM predicts the timing of link utilization. For the HWA DNM, γ_t declined rapidly with time, indicating a rapid decrease in the temporal consistency of the model in predicting realized links associated with spread of HWA and increasing temporal stochasticity in the simulated spread dynamics.

Discussion

While relatively new to ecology, DNMs hold great promise for understanding biological invasions and other spatially explicit processes. The DNM approach, borrowing from a rich history of graph theory applications, can leverage powerful visualization and analytical approaches largely lacking in existing metapopulation or cell-based modeling paradigms to provide scientists, managers, and policy makers visually intuitive output to inform decisions. For example, by evaluating the probabilistic emergent network as a series of time-dependent adjacency matrices and mapping time sequences of the evolving network, DNMs can identify locations likely to act as an infection source and promote spread across the landscape, as well as visualize the prominent pathways of spread and their timing of utilization. Such insights are not readily obtainable from analyses of the static graph adjacency structure or from other dynamic models of range expansion—such as that described and implemented by Fitzpatrick et al. (2012). For example, while Fitzpatrick et al. (2012) generated estimates of the probability and timing of infestation of HWA, their model is mute on which infestations contributed most to spread and the timing of these events. By allowing nodes to vary in habitat characteristics, DNMs can also readily accommodate spatial and temporal heterogeneities in habitat. Lastly, DNMs provide a framework for risk assessment congruent with the spatial scale of observation (e.g., individual,

patch, county, etc.), while retaining essential biological features of the organism and spatial data associated with habitat resources.

Dynamic network models share features with spatially explicit cell based models, but are distinct in that DNMs, like static networks, aggregate information from multiple cells into a much smaller number of nodes and generate output in a network format. In essence, a cell-based model can be considered a DNM operating on a regular, lattice network (a homogeneous adjacency structure). The DNMs developed in this work are network models operating across a heterogeneous landscape with potential complex adjacency structure. When the operational unit of the DNM is a collection of cells, the DNM operates on a far smaller number of nodes relative to the number of cells, with potential benefits in terms of computation and interpretation. For example, Fitzpatrick et al. (2012) simulated spread of HWA on a grid comprised of millions of cells whereas the DNM implementation considered 1,164 nodes.

Our findings also suggest that the results of static graph analyses, which are based on an equilibrium assumption, should be interpreted with caution in the context of non-equilibrium processes such as range expansion. Minor and Urban (2007) compared the equilibrium output of a spatially explicit metapopulation model to a static network model and found close agreement among the methods and concluded that static network models have great value in general conservation contexts. Our results suggest that the application of static network models in non-equilibrium contexts may identify landscape features (e.g., patches and potential connections between them) that have little relevance for dynamic population expansion. For example, our application of a DNM to a hypothetical invader in ANTI showed that node degree (i.e., the number of links based on distance only; equivalent to a static network model) is rarely equivalent to node utilization (Fig. 2). The primary reason for this difference is that DNMs consider initial conditions (e.g., location of introduction) and resultant temporal dynamics while static network models do not. A DNM, like an actual invasion, begins with a founder population in a specific geographic location from which spread proceeds. Initial conditions, along with spatiotemporal heterogeneity in habitat, influences the resulting spread dynamics and produces links with differences in the timing and strength of utilization; findings not readily apparent

from a static network model. In a broader sense, our work highlights meaningful regimes of applicability for both static and dynamic approaches; static network models may be most appropriate when the goal is to estimate the potential of patches to contribute to spread as a function of only their position within the network, while DNMs can complement static network models by evaluating the most likely utilization of patches during range expansion.

A key challenge for forecasts of invasive spread, and of range expansion more generally, is to move beyond static habitat suitability maps by incorporating spatio-temporal dynamics in order to estimate the probability and timing of establishment of populations in specific locations (Yemshanov et al. 2009; Gallien et al. 2010; Meentemeyer et al. 2011; Fitzpatrick et al. 2012). A major hurdle in this regard is that where and when an invader is introduced can dramatically impact the extent (Minor and Gardner 2011) and speed (Ferrari and Lookingbill 2009) of the subsequent invasion. DNMs offer a means to address this uncertainty. By independently simulating each node in a landscape as the starting point of a potential invasion, DNMs can be used to determine, for each initial condition, how long it takes an invasion to reach equilibrium and the resulting areal extent of the invasion. Introductions to nodes that result in rapid spread and a large areal extent of the invasion pose the greatest risk of facilitating population expansion across the newly invaded range. Such information would be a valuable supplement to existing risk assessment frameworks that are based solely on static representations of habitat suitability and/or connectivity.

In a similar fashion, DNMs can inform the management of ongoing invasions by identifying locations likely to act as strong sources of secondary infection. In the HWA case study for example, the placement of the stochastic spread model in a DNM framework identified two counties that, once infested, were consistently predicted to initiate rapid spread throughout the network (Fig. 5c). When used in isolation, the HWA spread model estimated only the probability and timing of infestation (Fitzpatrick et al. 2012). In a management context, aggressively monitoring or treating locations predicted to initiate rapid spread across the landscape could represent a cost effective means of slowing invasions.

One of the metrics we evaluated for the HWA DNM was γ_t (Eq. 6), a measure of the consistency of a

stochastic model to simulate realized links of an emergent network in time. Values approaching zero indicate high stochasticity of an invasion. While it is generally understood that the early stages of the invasion are the most stochastic and uncertain (Facon and David 2006), we observed relatively few high-probability pathways for HWA spread out of Richmond, VA. As the number of realized links and infected nodes increased with time, the value of γ_t decreased, indicating that, while the invasion may have proceeded along certain high-probability pathways (as indicated by the high probability links in Fig. 5), the timing of the formation of most links tended to be inconsistent between simulations.

Taken together, our results suggest that DNMs can provide valuable insights and visualization capabilities not readily offered by approaches typically used to model the spread of invasive species and which may differ in important ways form static network representations of spread potential. Although our DNMs focused on quantifying the emergent dynamics of biological invasions across a heterogeneous landscape, this general formulation is relevant to any process of range expansion. For example, similar DNMs can be envisioned to estimate the response of species to shifting habitat resources as a result of climate change or to determine optimal reintroduction points for the reestablishment of extirpated species. More broadly, DNMs represent a reasonable and informative next step of the standard graph theory paradigm in the ecological sciences, and provide a novel framework for the pursuit of an array of ecological questions.

References

Araújo MB, Rozenfeld A, Rahbek C, Marquet PA (2011) Using species co-occurrence networks to assess the impacts of climate change. Ecography 34:897–908

Banks HT, Rehm KL, Sutton KL (2010) Dynamic social network models incorporating stochasticity and delays. Q Appl Math 68:783–802

Brooks CP, Antonovics J, Keitt TH (2008) Spatial and temporal heterogeneity explain disease dynamics in a spatially explicit network model. Am Nat 172:149–159

Bunn AG, Urban DL, Keitt TH (2000) Landscape connectivity: a conservation application of graph theory. J Environ Manage 59:265–278

Butts CT (2009) Revisiting the foundations of network analysis. Science 325:414

Cumming GS, Bodin Ö, Ernstson H, Elmqvist T (2010) Network analysis in conservation biogeography: challenges and opportunities. Divers Distrib 16:414–425

Evans A, Gregoire T (2007) A geographically variable model of hemlock woolly adelgid spread. Biol Invasions 9:369–382

Facon B, David P (2006) Metapopulation dynamics and biological invasions: a spatially explicit model applied to a freshwater snail. Am Nat 168:769–783

Ferrari J, Lookingbill T (2009) Initial conditions and their effect on invasion velocity across heterogeneous landscapes. Biol Invasions 11:1247–1258

Ferrari JR, Lookingbill TR, Neel MC (2007) Two measures of landscape-graph connectivity: assessment across gradients in area and configuration. Landsc Ecol 22:1315–1323

Fitzpatrick MC, Preisser EL, Porter A, Elkinton J, Waller LA, Carlin BP, Ellison AM (2010) Ecological boundary detection using Bayesian areal wombling. Ecology 91:3448–3455

Fitzpatrick MC, Preisser EL, Porter A, Elkinton J, Ellison AM (2012) Modeling range dynamics in heterogeneous landscapes: invasion of the hemlock woolly adelgid in eastern North America. Ecol Appl 22:472–486

Fletcher RJ, Acevedo MA, Reichert BE, Pias KE, Kitchens WM (2011) Social network models predict movement and connectivity in ecological landscapes. Proc Natl Acad Sci 108:19282–19287

Gallien L, Münkemüller T, Albert CH, Boulangeat I, Thuiller W (2010) Predicting potential distributions of invasive species: where to go from here? Divers Distrib 16:331–342

Gilligan CA, van den Bosch F (2008) Epidemiological models for invasion and persistence of pathogens. Annu Rev Phytopathol 46:385–418

Homer C, Huang C, Yang L, Wylie B, Coan M (2004) Development of a 2001 national landcover database for the United States. Photogramm Eng Remote Sensing 70:829–840

Keitt TH, Urban DL, Milne BT (1997) Detecting critical scales in fragmented landscapes. Conserv Ecol 1:4

Leenders R (1995) Models for network dynamics: a Markovian framework. J Math Sociol 20:1–21

Lookingbill TR, Gardner RH, Ferrari JR, Keller CE (2010) Combining a dispersal model with network theory to assess habitat connectivity. Ecol Appl 20:427–441

Mack RN, Simberloff D, Lonsdale WM, Evans H, Clout M, Bazzaz FA (2000) Biotic invasions: causes, epidemiology, global consequences, and control. Ecol Appl 10:689–710

McClure M (1989) Evidence of a polymorphic life cycle in the hemlock woolly adelgid, *Adelges tsugae* (Homoptera: Adelgidae). Ann Entomol Soc Am 82:50–54

McClure M (1990) Role of wind, birds, deer, and humans in the dispersal of hemlock woolly adelgid (Homoptera: Adelgidae). Environ Entomol 19:36–43

McClure MS, Cheah CAS-J (1999) Reshaping the ecology of invading populations of hemlock woolly adelgid, *Adelges tsugae* (Homoptera: Adelgidae), in Eastern North America. Biol Invasions 1:247–254

Meentemeyer RK, Cunniffe NJ, Cook AR, Filipe JAN, Hunter RD, Rizzo DM, Gilligan CA (2011) Epidemiological modeling of invasion in heterogeneous landscapes: spread of sudden oak death in California (1990–2030). Ecosphere 2:art 17. doi:10.1890/ES10-00192.1

Minor ES, Gardner RH (2011) Landscape connectivity and seed dispersal characteristics inform the best management strategy for exotic plants. Ecol Appl 21:739–749

Minor ES, Urban DL (2007) Graph theory as a proxy for spatially explicit population models in conservation planning. Ecol Appl 17:1771–1782

Minor ES, Urban DL (2008) A graph-theory framework for evaluating landscape connectivity and conservation planning. Conserv Biol 22:297–307

Mollison D (1977) Spatial contact models for ecological and epidemic spread. J R Stat Soc Ser B Methodol 39:283–326

Morin R, Liebhold A, Gottschalk K (2009) Anisotropic spread of hemlock woolly adelgid in the eastern United States. Biol Invasions 11:2341–2350

Opsahl T, Agneessens F, Skvoretz J (2010) Node centrality in weighted networks: generalizing degree and shortest paths. Soc Netw 32:245–251

Orwig DA (2002) Ecosystem to regional impacts of introduced pests and pathogens: historical context, questions and issues. J Biogeogr 29:1471–1474

Paradis A, Elkinton J, Hayhoe K, Buonaccorsi J (2008) Role of winter temperature and climate change on the survival and future range expansion of the hemlock woolly adelgid (*Adelges tsugae*) in eastern North America. Mitig Adapt Strateg Glob Change 13:541–554

Pimm SL, Lawton JH, Cohen JE (1991) Food web patterns and their consequences. Nature 350:669–674

Sharov AA, Leonard D, Liebhold AM, Roberts EA, Dickerson W (2002) "Slow the spread": a national program to contain the gypsy moth. J For 100:30–35

Shavitt Y, Singer Y (2007) Beyond centrality—classifying topological significance using backup efficiency and alternative paths. New J Phys 9:266

Shigesada N, Kawasaki K, Takeda Y (1995) Modeling stratified diffusion in biological invasions. Am Nat 146:229–251

Snijders TA (2005) Models for longitudinal network data. In: Carrington PJ et al (eds) Models and methods in social network analysis. Cambridge University Press, Cambridge, pp 215–247

Stoetzel M (2002) History of the introduction of *Adelges tsugae* based on voucher specimens in the Smithsonian Institute National Collection of Insects. Symposium on the hemlock woolly adelgid in Eastern North America, US Forest Service, New Brunswick, NJ

R Development Core Team (2011) R: a language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. Retrieved from http://www.R-project.org

Turner JL, Fitzpatrick MC, Preisser EL (2011) Simulating the dispersal of hemlock woolly adelgid in the temperate forest understory. Entomol Exp Appl 141:216–223

Urban DL (2003) LANDGRAPHS: a package for graph theoretic analyses of landscapes. Landscape Ecology Laboratory, Duke University, Durham

Urban DL, Minor ES, Treml EA, Schick RS (2009) Graph models of habitat mosaics. Ecol Lett 12:260–273

Vazquez A (2006) Spreading dynamics on heterogeneous populations: multitype network approach. Phys Rev E 74:066114

Watts DJ, Strogatz SH (1998) Collective dynamics of "small-world" networks. Nature 393:440–442

Yemshanov D, Koch FH, McKenney DW, Downing MC, Sapio F (2009) Mapping invasive species risks with stochastic models: a cross border United States Canada application for *Sirex noctilio* Fabricius. Risk Anal 29:868–884

Yook SH, Jeong H, Barabási AL, Tu Y (2001) Weighted evolving networks. Phys Rev Lett 86:5835–5838