

The importance of transport hubs in stepping-stone invasions

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Summary

1. Biological invasions are characterized by occasional long-distance, human-assisted dispersal. Centres of human transportation that are connected by trade to a wide range of other locations ('transport hubs') may be important catalysts of the rate at which new populations of an invader are established.
2. We developed a spatially explicit stochastic model to simulate the spread of a hypothetical marine invader by hull fouling. The model was based on classic 'Susceptible–Infected–Resistant' models used in medical epidemiology. It was parameterized using empirical data on the colonization of vessel hulls by fouling organisms, and on maintenance and travel patterns of ~1300 domestic and international yachts around New Zealand. Thirty-six marinas were grouped into three categories that represented a gradient in the number of other transport nodes each marina was 'connected' to and the frequencies of yacht movements between them. Invasions were seeded in three locations from each category. Simulations were run over 10 years to determine differences in the trajectory of invasions originating from busy and less frequented transport nodes.
3. Busy 'hub' locations were 75% more likely to become infected by an invader than quieter locations. Infection of hub nodes occurred at an earlier average stage in the invasion sequence. This occurred irrespective of whether the initial source of the invasion was associated with low or high traffic volume and connectivity.
4. Biotic invasions originating from hub locations did not consistently result in faster spread, or a larger number of secondary infestations. However, the rate of spread from hubs was less variable than from quieter nodes and was less often preceded by a prolonged lag period.
5. *Synthesis and applications.* Rapid spread of invasive organisms can occur from busy and from seemingly unimportant transport nodes. Busy locations were consistently more likely to become infested by an invader and to accelerate spread to secondary locations faster. Busy transport hubs should be considered a priority for the allocation of preventative and management efforts, such as regular baseline or target surveys and the development of incursion response plans that minimize the risk of spread within the transport network.

Key-words: biofouling, epidemiology, hull fouling, invasive species, transport networks, SIR models, spreading rate, yachts

Introduction

Growth in international trade and travel has provided greater opportunities for species to move outside their natural geographic ranges (Jenkins 1996; Levine & D'Antonio 2003). Biological invasions often reflect the flows of traffic within regional or global transportation networks. Infested locations

act as sources for subsequent spread of the organism to other locations in the network (Drake & Lodge 2004; Floerl & Inglis 2005). Such 'stepping stone' invasions have been observed for a variety of non-indigenous species (NIS) in aquatic and terrestrial ecosystems (Buchan & Padilla 1999; Suarez, Holway & Case 2001).

The role of transport hubs in determining the rate and spatial pattern of spread of invasive species is poorly understood. Although there is a variety of opinion about what constitutes a transport hub (O'Kelly & Miller 1994), we define them here

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as locations (or nodes) in a transportation network that are characterized by high traffic volume (frequency of vector movements) and high connectivity (movements of vectors to large numbers of other locations) to other transport nodes.

Non-indigenous marine species often establish first in nodes of shipping activity, such as ports and marinas, and are spread to other nodes within the shipping network (Carlton 1996; Minchin *et al.* 2006). Drake & Lodge (2004) recently modelled strategies for managing ballast water invasions in the global shipping network. Using a constant per-ship probability of initiating an invasion (p), they showed that reducing p was more effective in slowing the spread of invaders in ballast water than eliminating important ports from the network. However, p is also highly variable, as (in this case) it is the product of the chances of entrainment of the organism by the vessel, its density per unit volume of ballast water, the quantity of ballast water for a given number of ships, survival during transport and establishment after release (MacIsaac, Robbins & Lewis 2002; Verling *et al.* 2005). There is often large variation in the likelihood that individual vessels will carry NIS (Floerl & Inglis 2005; Floerl, Marsh & Inglis 2005; Verling *et al.* 2005). The frequency of vessel movements alone, therefore, may be a poor predictor of invasion risk.

We used an epidemiological model to simulate the spread of a hypothetical marine invader in New Zealand by hull fouling on recreational yachts. Hull fouling is a major pathway for the spread of NIS in marine environments and recreational yachts have been implicated in the introduction and spread of a number of well-known invaders in New Zealand and elsewhere (Minchin *et al.* 2006). Our model incorporates variation in the susceptibility of the yachts to colonization by the invader and is calibrated using empirical data on patterns of vessel movement and maintenance. Stochastic simulations were used to assess the importance of the initial site of establishment by the invader on the spatial pattern and rate of spread. In particular, we compared establishment in busy 'hub' locations and smaller regional nodes to determine the effect of initial location on subsequent spread within the network.

Materials and methods

MODEL CONCEPT

The model was based on Susceptible–Infected–Resistant (SIR) models that are used in disease epidemiology (Bjornstad, Finkenstadt & Grenfell 2002; Singh, Chandra & Shukla 2005). These divide hosts (individuals) in a population according to whether they are 'susceptible' (S) or 'resistant' (R) to a disease, or have become 'infected' (I) by it.

The spread of fouling species on vessel hulls is somewhat analogous to the epidemiology of disease. Toxic antifouling paints provide boat hulls with a temporary 'resistance' to fouling organisms by deterring or killing larvae that attempt to settle on them (Christie & Dalley 1987). This resistance diminishes over time, with a concomitant increase in the hulls' susceptibility to fouling. Yacht hulls, therefore, repeatedly progress through a series of states in which they are either: (i) protected by the antifouling paint (R), (ii) prone to becoming fouled (S), or, possibly, (iii) colonized by a particular

species of interest (I). Hulls that are manually cleaned of fouling without repainting remain susceptible to colonization ($S \rightarrow S$), but may have the NIS temporarily removed ($I \rightarrow S$) (Floerl, Marsh & Inglis 2005).

An incursion can occur when an infested yacht enters a port or marina. Organisms attached to the hull can either release propagules in the water column or become dislodged; the likelihood of either event increases with the time the vessel spends at the location (Minchin & Gollasch 2003). The main habitats available for fouling organisms in coastal marinas are floating pontoons and vertical pilings (Connell 2000; Floerl & Inglis 2005). Once an introduced species has become established on such structures, its population is likely to grow in size and, eventually, produce propagules that can colonize surrounding vessel hulls and become transported to further locations. The SIR component of our model determined the proportion of yachts that, at any time, were able to act as carriers of the species.

MODEL PARAMETERIZATION

Our model was parameterized using empirical data on the susceptibility of antifouling paints of different ages to colonization by marine fouling organisms, and by a questionnaire survey that quantified the maintenance schedule and frequency of travel of ~1300 yachts among New Zealand's coastal marinas. Our model was restricted to sailing yachts.

QUESTIONNAIRE SURVEY OF TRAVEL AND MAINTENANCE

A questionnaire was sent to 4476 yacht owners in New Zealand in 2002. Mailing addresses were sourced from a data base maintained by Yachting New Zealand, a national representative body for recreational and competitive sailing. The sample represents ~50% of the total number of permanently moored, ocean-going yachts in New Zealand (Yachting New Zealand, personal communication). Respondents were asked to provide information on: (i) the usual mooring location for their yacht; (ii) the current age and usual frequency of renewal of antifouling paint on hull; (iii) how often the yacht was manually cleaned of fouling between successive paint applications; (iv) the main use of the yachts (e.g. cruising distances, participation in races, etc); and (v) the total number of trips taken from the home port, the destinations visited, and time spent at each in the past 12 months.

These data were also obtained for an independent sample of 283 yachts that entered New Zealand from overseas in 2002 and 2003 (~40% of total international arrivals). Questionnaires were issued to the yachts by the New Zealand Customs Service prior to their departure from New Zealand. We were unable to obtain destination data from those international yachts that had entered, but not departed from New Zealand over the period of the study.

Data from the questionnaires were used to construct a probability matrix of yacht movements between 36 coastal locations around New Zealand. The locations included all New Zealand marinas and ~80–90% of available mooring facilities (New Zealand Marina Operators Association 2004). Embayments or harbours with several marinas or mooring facilities in close proximity were combined into a single location. Previous models on species invasions have used distance between locations as a measure of introduction likelihood (e.g. Buchan & Padilla 1999; Schneider, Ellis & Cummings 1997). Distance may be a correlate of movement likelihood for yachts around New Zealand, but the popularity of particular destinations

is also determined by their association with particular sights or racing events. Our movement probabilities were constructed from empirical data that inherently incorporated elements of distance and popularity (if any).

Information on the number of berths available in each location and estimates of their occupancy by domestic and international yachts were obtained from the operators of each facility. Based on these figures, the total number of domestic yachts in the model was set at 9702. The model also simulated arrival in New Zealand of 450 international yachts each year (New Zealand Customs Service, personal communication). The length of stay of international yachts was parameterized by a frequency distribution obtained from the survey of international yacht owners and ranged from 1 day to 3.7 years (median = 150 days). For simplicity, all international yachts were assumed to arrive at Opuia marina because > 90% of overseas yachts clear customs there (Floerl, Inglis & Hayden 2005).

HABITAT FOR FOULING ORGANISMS

We calculated the total amount of habitat available for fouling organisms in each of the 36 locations by multiplying the number of berths present in each by the average amount of artificial habitat present in an average berth. On average, each yacht berth was associated with approximately 17 m² of hard substratum (O. Floerl, unpublished data).

YACHT SUSCEPTIBILITY TO FOULING

The model repeatedly 'cycled' each yacht through states of being susceptible, infected and resistant to the invader. Newly painted yachts began as resistant (*R*), but became susceptible (*S*) to fouling over time. In our model, 'susceptibility' (*S_j*) had a continuous distribution between 0 (resistant) and 0.99 (99% likely to become colonized by the invader if propagules make contact with the hull), according to a logistic regression that described the empirical relationship between the age of the antifouling paint on 70 yachts surveyed by Floerl & Inglis (2005) and the presence of macro-fouling on them.

Depending on their travel and maintenance schedule, the yachts either became infected with the invader (*I*) or the antifouling paint was renewed (*R*) before infection could occur. The probability that a susceptible yacht *j* became infected at location *i* at time *t* was defined as the 'transmission probability' and derived as:

$$P(T)_{ij} = S_{ji} \times A_{it},$$

where *S_j* is the susceptibility of the yacht to fouling and *A_i* is the proportion of available habitat occupied by the invader in the location of interest on any given day (see below).

At *t* = 0, the age distribution of antifouling paints and associated susceptibility of all yachts was set at the frequency distribution of paint ages derived from the questionnaire responses. Yachts were randomly assigned antifouling paint renewal intervals from the frequency distribution derived from the boater survey, and this was repeated after each antifouling paint treatment. Renewal of the antifouling paint 'reset' susceptibility to zero (i.e. *R*). In addition, yachts were randomly assigned probabilities for manual hull cleaning based on a frequency distribution derived from the yacht survey. If an uninfected hull was manually cleaned, *S_j* did not change. If an infected hull was cleaned, the infection was removed and the hull was assigned an *S_j* based on its antifouling paint age.

SENSITIVITY ANALYSIS FOR INFECTION PARAMETER AND POPULATION GROWTH RATE

Model runs were initiated by 'seeding' one of the 36 locations with the invader. Establishment occurred when the species was transmitted from an infected yacht to an uninfected location (see below). Once the location was infected, other susceptible resident yachts could also become infected and transmit the species to additional locations. The probability of establishment by invasive species is often correlated with their rate of supply to a native location ('propagule pressure'; Verling *et al.* 2005). In our model, the probability of establishment was defined as the number of infected yachts that needed to reside in an uninfected location on any given day to result in a probability of 0.01 (i.e. 1%) of infecting the location on that day. This measure allows the probability of establishment to increase in accordance with the numbers of infected yachts that arrive in a location and/or with longer residency of infested yachts. Because there are no published data on the relationship among the frequency of ship visits, residency and the probability of local establishment of a NIS, we used a range of values for these parameters and conducted a sensitivity analysis to determine how choice of the parameters affected the rate of spread to new locations.

The probability of establishment was derived by first calculating an infection parameter, *M*:

$$M = (1 - 0.01)^{(1/N)},$$

where *N* is the number of infected yachts required to reside in an uninfected location for 1 day. The establishment probability of the NIS at location *i* at time *t* by yacht *j* was then calculated as:

$$P(E)_{ij} = 1 - M^{N(it)}$$

Three values for *N* were used in the sensitivity analysis: 1, 10 and 100 yachts (i.e. high, moderate and low establishment probability).

Once a population was established in a location, it underwent exponential growth (*g*) according to the formula:

$$g = \exp \ln(2)/(30 \times d),$$

where *d* represents the number of months taken for the population to double in area. Population size was expressed as a proportion of available habitat that the NIS occupied at any given time step (*t*) and was calculated as:

$$A(t) = A(t - 1) \times g.$$

Doubling times (*d*) of 6 months, 12 months and 24 months were simulated. Because single species never occupy all available habitat (Connell 2000; Floerl & Inglis 2005), we limited the maximum area the NIS was able to occupy in each infected location to 25% of the available habitat.

SIMULATING SPREAD FROM LOCATIONS OF VARYING VECTOR TRAFFIC AND CONNECTIVITY

To evaluate the relative importance of the site of initial establishment by an invader in a transportation network, we ran separate simulations in which we seeded an invasion in locations that had different amounts of vessel movements and connectivity to other locations. To choose the seeding locations for the simulations, we first sorted the 36 locations by the total number of annual yacht arrivals and

departures ('vector traffic') and by the number of other locations that yachts from that location travelled to, or from ('connectivity'). Within the data set, these two variables were correlated, with connectivity increasing as a logarithmic function of vector traffic ($\text{Connectivity} = 5.18 \times \ln(\text{vector traffic}) - 12.45$; $r^2 = 0.8251$). Three representative locations were then selected at random from each of the top, middle and lower thirds of the sorted list, giving a total of nine locations (Supplementary Material Fig. S1) – each an official point of entry for international craft to New Zealand.

Each of the nine locations was 'seeded' with the hypothetical invader, and the geographic spread of the species was simulated over a 10-year period using 100 iterations per location ($N = 900$ iterations). Only a single location was seeded during any simulation. The invader was then able to spread away from the seeding location and infect any of the remaining 35 model locations. We assumed that the identity of model locations infected early in the invasion sequence had an influence on which locations became infected subsequently and, therefore, we treated iterative model runs as statistically non-independent. For each model iteration, we recorded the number of locations infected with the invader after 2.5, 5, 7.5 and 10 years, and the time at which each location became infected. Differences in the rate of spread between invasions originating from locations with low, medium or high vector traffic and connectivity were determined by examining the averages, percentile ranges and 95% confidence intervals of the 100 replicate runs per modelled scenario. Variation in the rate of spread among the 100 replicate simulations per modelled scenario was quantified by calculating the coefficient of variation. To estimate the probability of infection of each model location in each simulated scenario, we also determined the proportion of runs in which each location in the network became infected with the invader.

Results

SENSITIVITY ANALYSIS FOR INFECTION PARAMETER AND POPULATION GROWTH RATE OF THE INVADER

The magnitude of the infection parameter (M) and the doubling time of the established invader population (d) used in the model had a large influence on the rate of spread of the invader over time (Fig. 1). Irrespective of the setting for d , a high infection parameter ($M = 1$ infected yacht required) consistently resulted in a significantly larger number of infected model locations at any time t than when M was medium (10 yachts) or low (100 yachts) (Fig. 1a–c), as shown by the non-overlapping 95% confidence intervals. Differences in the spreading rate of the invader between low, medium or high settings of M were most pronounced when d was set at 12 months or 6 months (Fig. 1b and c). Under these conditions, a high M resulted in the infection of almost all model locations after 10 years. In contrast, at the lowest value of d (24 months), less than half of the locations were affected even when M was high, and differences between different treatments of M were least pronounced (overlap of 95% confidence intervals, Fig. 1a). Variation in the combination of d and M also resulted in differences in the timing of the infection of new locations. For all values of d , the spread of the NIS away from the seeding location occurred soonest when M was high. For example, for a medium setting of d (12 months), the NIS established its first satellite population after an average

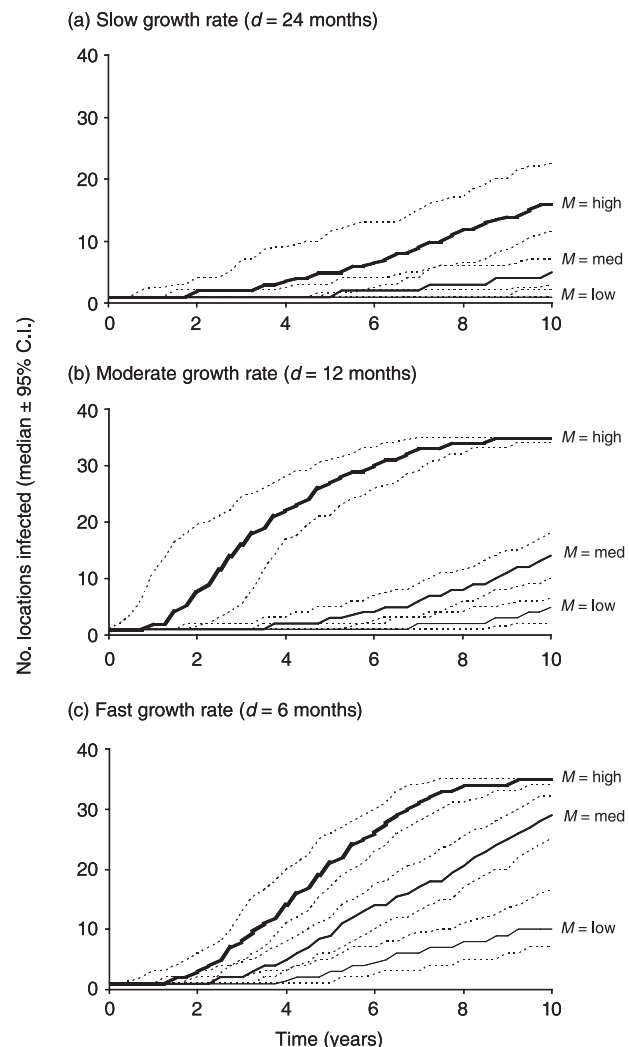


Fig. 1. Model sensitivity analysis on the influence of the infection parameter (M) and population doubling time (d) on the rate of spread. M was varied so that 1, 10 or 100 infected yachts (high, medium and low infection parameter, respectively) needed to be present in a location for 1 day to result in a probability of local establishment of 0.01 for that day. Dashed lines represent 95% confidence intervals.

of 10 months when M was high, after 3.25 years when M was moderate, and after 7.5 years when M was low (Fig. 1b).

Because of the considerable influence of population doubling time and infection parameter on the spreading rate of the invader, we kept these parameters constant and at a 'medium' setting during all following model runs that are discussed below. Accordingly, population doubling time was set at $d = 12$ months and the infection parameter M (number of resident infected yachts required for a daily infection probability of 0.01) was set at 10.

SPREAD FROM LOCATIONS WITH HIGH, MODERATE AND LOW VESSEL TRAFFIC AND CONNECTIVITY

The time taken for the invader to establish a new population away from the initial seeding location was shorter when

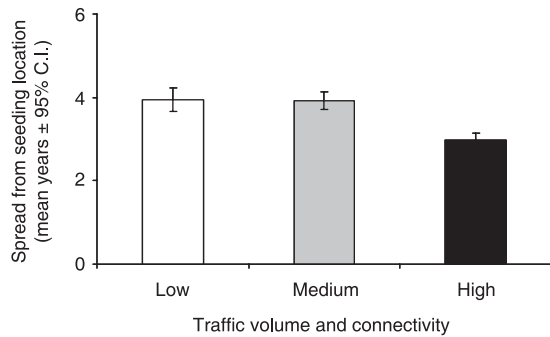


Fig. 2. Time (in years) taken for the hypothetical invader to establish in a new location away from the initial seeding location. Seeding locations were characterized by either low, medium or high levels of vector traffic and connectivity to other locations.

seeding had occurred in a location with high vector traffic and connectivity (mean \pm 95% confidence interval: 2.97 ± 0.16 years) than when traffic and connectivity were medium (3.92 ± 0.21 years) or low (3.95 ± 0.28) (Figs 2 and 3a). For the first 5 years of the simulated spread of the invader, there were no discernable differences in the number of new infected locations between seeding location categories (Fig. 3a and b). However, 7.5–10 years into the simulation, the average number of new infected locations was consistently larger for invasions originating from locations with high traffic and connectivity than for invasions that started in locations where traffic and connectivity were medium or low (Fig. 3c and d). There was considerable variation in the rate of spread from the three representative locations within each treatment category (L1–3, M1–3 and H1–3 in Fig. 3), with the greatest variation observed for seeding locations with low vector

traffic and connectivity. For example, when the simulation was seeded in Dunedin (L2 in Fig. 3), a larger average number of locations became infected over 10 years than when the invasion originated from L1 or L3, or from any of the three locations with medium traffic and connectivity (Fig. 3d). In contrast, seeding of the Hobson West Marina (L3) generally resulted in the spread of the invader to none or only few locations (Fig. 3d).

There were differences in the variability of the rate of spread of the hypothetical invader associated with seeding location treatment. For seeding locations characterized by low levels of vector traffic and connectivity, the coefficient of variation of the number of new infected locations increased over time. This indicated an increase in the variability of the spatial scale of the simulated invasion, that is, some simulations resulted in very limited spread and others in extensive spread (Fig. 4). In contrast, the coefficient of variation for the spread of the invader from seeding locations with high vector traffic and connectivity became progressively smaller over time, indicating reduced variability and a higher level of consistency in the spatial scale of the invasion (Fig. 4).

INFECTION PROBABILITY AND SEQUENCE OF INFESTATION

Over a period of 10 years, the 12 locations in the model that were characterized by high levels of vector traffic and connectivity (Supplementary Material Table S1, Fig. S1) were on average 2.4 and 4 times more likely to become infected with the invader, respectively, than were locations with medium or low traffic and connectivity (Fig. 5). The infection probability of model locations with high traffic and connectivity was further exacerbated (mean \pm 95% CI: 0.80 ± 0.09) when the

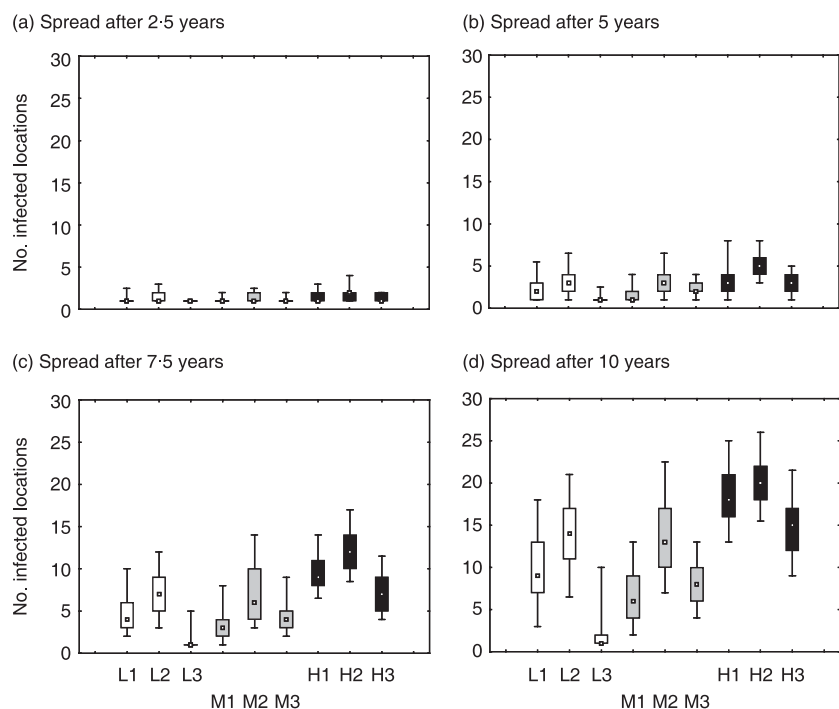


Fig. 3. Box plots of the number of locations infected after a simulated spread of the hypothetical NIS of (a) 2.5 years, (b) 5 years, (c) 7.5 years and (d) 10 years. Dots in the boxes show median values. Boxes represent the 25 and 75 percentiles, and whiskers represent the 5 and 95 percentiles. Nine separate simulations were run where the initial NIS incursion was seeded in three locations with low vector traffic and connectivity (L1–L3), moderate vector traffic and connectivity (M1–M3) and high vector traffic and connectivity (H1–H3). The data shown are based on 100 model iterations for each simulated scenario ($N = 900$).

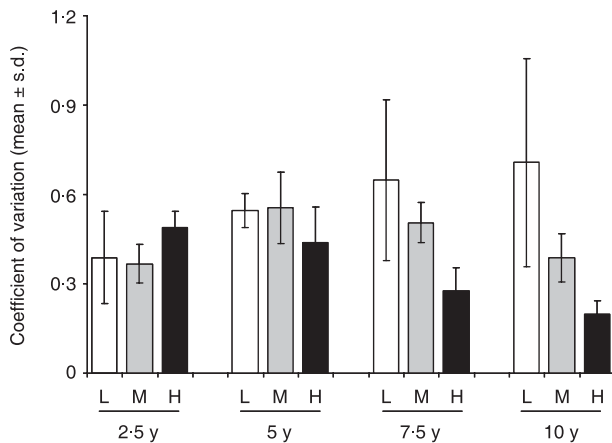


Fig. 4. Mean coefficient of variation (\pm standard deviation) of the rate of spread of the hypothetical invader from seeding locations of low, medium and high vector traffic and connectivity (L, M, H, respectively, with $n = 3$ locations per treatment). The coefficient of variation illustrates the dispersion of the data for each treatment and is shown for the rate of spread after 2.5, 5, 7.5 and 10 years.

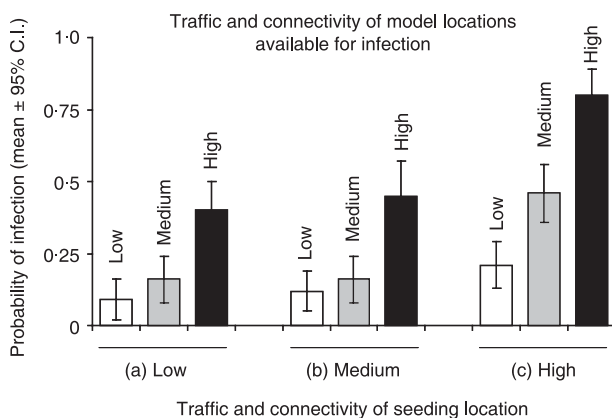
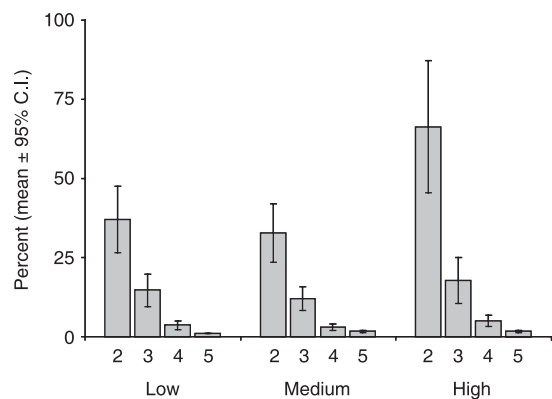


Fig. 5. Infection probabilities of model locations over a 10-year period. The bars are arranged along the x-axis according to whether the invader was originally seeded in a location with (a) low, (b) medium or (c) high vector traffic and connectivity to other locations. The white, grey and black bars represent the mean infection probability (\pm 95% confidence intervals) of the *other* model locations (of low, medium and high vector traffic and connectivity, respectively) over the 10-year period.

invader had originated from an initial seeding location with similar characteristics (Fig. 5c). Lowest overall infection probabilities were recorded for locations with low vector traffic and connectivity, particularly when the invasion had initially been seeded in a similar location (Fig. 5a).

The sequence of spread of the invader among the 36 locations was highly variable (Fig. 6a). None of the 100 iterations run for each seeding location resulted in an identical sequence. The proportion of simulations where the first two infected locations were identical was largest when seeding had occurred in a location with high traffic and connectivity ($66.3 \pm 20.8\%$; Fig. 6a). However, this trend disappeared as

(a) Proportion of identical invasion sequences



(b) Rank of infection of model locations

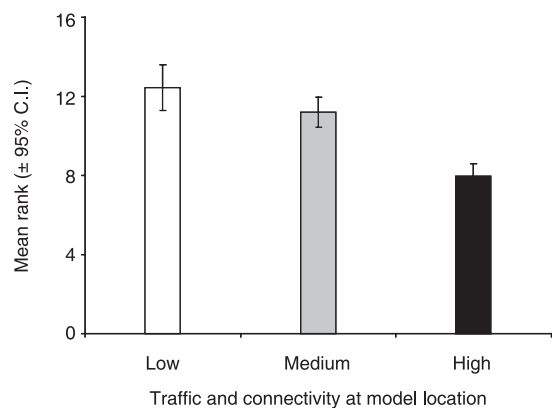


Fig. 6. (a) Proportion (%) of 10-year simulations in which the first 2, 3, 4, or 5 infected locations were identical. Data are presented separately according to whether the seeding location of the invader was characterized by low, medium or high vector traffic and connectivity. (b) Average rank of infection of model locations over a 10-year period. White, grey and black bars represent mean infection ranks for locations with low, medium or high vector traffic and connectivity, respectively.

the spread of the NIS progressed in space and time; identical sequences of the first four infected locations occurred for less than 5% of simulations under any seeding conditions.

Locations with a high level of traffic and connectivity became infected significantly earlier in the invasion sequence (mean rank \pm 95% CI: 7.9 ± 0.6) than locations with medium (11.2 ± 0.8) or low (12.5 ± 1.2) traffic and connectivity (Fig. 6b).

Discussion

Human-assisted spread of NIS is contingent upon: (i) the availability of suitable transport vectors; (ii) the frequency of vector movements between infected and uninfected locations; (iii) the number of propagules released in individual inoculation events; (iv) the timing of any inoculation event relative to the species' life history; and (v) the availability of limiting resources in uninfected locations (Buchan & Padilla 1999;

Davis, Grime & Thompson 2000; Verling *et al.* 2005). The confluence of all these factors makes the spread of NIS a highly stochastic and often unpredictable process (Heger & Trepl 2003). Our model incorporated parameters for processes (i), (ii), (iii) and (v). The model did not consider reproductive seasonality of the invader because of the large variability in the reproductive physiology of marine organisms (Santelices 1990; Levin & Bridges 1995). Initial pilot runs indicated that while inclusion of reproductive seasonality may reduce overall spreading rates, it would do so for all simulated scenarios equally and, therefore, would not have influenced the patterns we describe here.

Our results reinforce the stochasticity of the invasion process. The order in which the 36 model locations were invaded was not repeated in any of the 900 iterations. The spatial invasion pattern was moderately consistent (32–63% of cases) for the first two secondary infestations but became highly unpredictable subsequently. On average, invasions originating from locations that are highly connected within the transport network and have a high volume of vessel movements resulted in a higher number of secondary infestations than invasions originating from less busy locations. Locations characterized by high connectivity and traffic were up to four times more likely to become invaded than locations of lesser traffic and connectivity. Locations with high vector traffic and connectivity were generally infected earlier than less frequented locations.

The frequency of vector movements and the connectivity of locations have been shown to affect spreading patterns of known NIS. For example, the spread of the root pathogen *Phytophthora lateralis* by vehicle traffic is enhanced by an increase in connectivity between invasible sites. The presence and abundance of roads connecting infested with uninfested sites influences the risk of infection and establishment of satellite populations (Jules *et al.* 2002). Similarly, development of freshwater reservoirs has been shown to attract increased numbers of recreational users that facilitate rapid spread of NIS established in these reservoirs to connected locations (Havel, Lee & Vander Zanden 2005). 'Promiscuous' behaviour of vectors originating from infested locations can accelerate the spread of NIS (Johnson, Ricciardi & Carlton 2001).

There was considerable variation in the rate at which new locations in the model became infected. We offer two explanations: (i) variation in the susceptibility of vectors and the likelihood that they carry the NIS, and (ii) the sequence in which the locations in the model became infected over time. Variability in spreading rate was highest when the NIS was seeded in locations with low vector traffic and connectivity. At times, spread from these locations occurred rapidly. In each of these cases, one or several locations with high vector traffic and connectivity had become infected early in the invasion sequence, causing accelerated spread. If no busy locations were infected during the first 5 years of the simulation, the invasion tended to proceed more slowly. The rate at which the invader spread among locations was generally more consistent when the initial incursion occurred in a location with high vector traffic and connectivity. Spread from such

locations was also associated with a relatively short lag period following the seeding of the invader. Management authorities may have more time to respond to an incursion in a location with low levels of vector traffic and connectivity than to one that occurs in a busy transport hub (Leung *et al.* 2005).

Previous studies have reported relationships between rates of invasion and volume of vector traffic, and highlighted the influence of hub locations on spreading rates (Muirhead & MacIsaac 2005). In the North American Great Lakes, the number of ship-vectored NIS recorded per decade is correlated with the intensity of vessel traffic (Ricciardi 2001, 2006). A recent model developed by Drake & Lodge (2004) shows that the rate of spread of marine NIS among global ports is influenced by the likelihood of NIS establishment per inoculation event and the rate of ship arrivals at a port. Drake & Lodge (2004) found that a reduction of the per-ship-visit chance of causing an invasion has a greater relative influence on spreading rate than the removal of important locations in the network. However, in their model, all vessels had a constant probability of transporting and successfully introducing the invader at any given time. In our model, variation in the likelihood that any given yacht carried the invader on its hull [$P(T_i)$] translated into variation in establishment probability for any given inoculation event [$P(E_i)$]. Our model thus incorporated a stochastic element for a range of stages in the invasion process (propagule engagement with vector, transport by vector, establishment at recipient location) (Floerl & Inglis 2005; Verling *et al.* 2005). Our preliminary sensitivity analysis showed that both establishment probability and growth rate of the NIS significantly influenced the spread of the species to new model locations over time. Thus, a species' life-history and physiological traits have an important bearing on the course of an invasion.

How do our simulations compare to the real world? New Zealand's best-documented marine NIS is the kelp *Undaria pinnatifida*, which established in Wellington in the 1980s and can now be found around most of New Zealand (Forrest *et al.* 2000). Our simulated invader colonized the upper South Island (Picton and Nelson, Supplementary Material Fig. S1) within a period of approximately 4 years, which is consistent with *Undaria*. However, our modelled invader took 7 years to reach Dunedin (Supplementary Material Fig. S1), while *Undaria* occurred there less than 3 years following its initial discovery in Wellington. Similar discrepancies occur for the North Island's Auckland region (modelled arrival, 7 years; actual discovery, 18 years, Forrest 2007). We offer two explanations that are likely to be transferable to other species. First, our model incorporated a single vector – recreational yachts. The spread of *Undaria* occurs via hull fouling on yachts, commercial ships, towed barges, via domestic transfers of ballast water and via translocation of aquaculture equipment (Forrest 2007). Secondly, our invasions occurred from a single seeding location. There is evidence that the current distribution of at least two NIS in New Zealand – *Undaria* and *Styela clava* – is the result of multiple independent introductions, each with their own associated pattern of spread (Forrest 2007; S. Goldstien personal communication). The discrepancy

between our model outputs and observed spreading patterns do not affect the validity of our conclusions but provides an additional reminder of the complexity of biotic invasions and their underlying processes.

A common recommendation for biotic invasions is to manage spread to geographically separated sites, because large numbers of 'satellite' populations can greatly increase the overall rate of spread (Moody & Mack 1988). Our study shows that, for invasions within human transport networks, locations with a high degree of vector traffic and connectivity to other nodes are likely to give rise to a larger number of new satellite populations faster than an incursion in locations that are less central. Containing incursions in these important nodes can significantly reduce the likelihood of secondary spread. Regular surveillance of busy transport nodes can increase the chances of early detection, but requires significant resources if it is to be effective (Hayes *et al.* 2005).

The transmission rate and spread of a disease are positively correlated with the density of suitable hosts. For example, the spread of furunculosis (a disease of farmed salmon) and measles occurs faster when the average susceptibility of hosts to infection is higher, and/or when large numbers of susceptible hosts are present in an area (Grenfell, Bjornstad & Kappey 2001). The locations with high vector traffic and connectivity in our study are generally large marina facilities that are frequented by a large number of yachts from a range of local, regional or distant locations. They also have a larger habitat available for infestation, and greater numbers of resident vessels. Consequently, compared to the 'quiet' locations in the model, busy 'hub' locations were associated with a high density of susceptible vectors, a large area of available habitat and had a high likelihood of receiving infested vectors from other locations. Once a NIS population has established, and is growing, in a location with high traffic and connectivity ('transport hubs'), the relatively large number of susceptible vectors are at a steadily increasing risk of becoming infected with the NIS and transporting it to a range of secondary locations. The principal distinction between transport hubs and comparatively quiet locations may be the relative difference in the transmission of the NIS from infested surfaces to resident susceptible vessels, and the subsequent potential for transport to secondary locations where satellite populations may establish.

The rapid geographic spread of some of the most notorious invaders, e.g. the zebra mussel *Dreissena polymorpha* and Argentine ant *Linepithema humile*, has been accomplished by long-distance transport events via human-mediated vectors (Buchan & Padilla 1999; Suarez, Holway & Case 2001). Our results underline the stochasticity and unpredictability associated with biotic invasions: given the right conditions, rapid spread of a NIS can occur from both busy and from quiet nodes in a transport network. However, the lower variation in spreading rates from busy nodes, and the high likelihood of colonization of these locations, suggest that they should be considered a priority for the allocation of preventative and management efforts, such as regular baseline or target surveys and the development of incursion response plans that minimize the risk of spread within the transport network.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Fig. S1. Yachting marinas and coastal locations included in the model.

Table S1. The 36 locations used in the model, sorted by total number of connected locations (departures and arrivals) and numbers of movements of domestic and international yachts over a 12-month period

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