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LETTER

Its all about connections: hubs and invasion in habitat networks

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Abstract

During the early stages of invasion, the interaction between the features of the invaded landscape, notably its spatial structure, and the internal dynamics of an introduced population has a crucial impact on establishment and spread. By approximating introduction areas as networks of patches linked by dispersal, we characterised their spatial structure with specific metrics and tested their impact on two essential steps of the invasion process: establishment and spread. By combining simulations with experimental introductions of *Trichogramma chilonis* (Hymenoptera: *Trichogrammatidae*) in artificial laboratory microcosms, we demonstrated that spread was hindered by clusters and accelerated by hubs but was also affected by small-population mechanisms prevalent for invasions, such as Allee effects. Establishment was also affected by demographic mechanisms, in interaction with network metrics. These results highlight the importance of considering the demography of invaders as well as the structure of the invaded area to predict the outcome of invasions.

Keywords

Allee effect, connectivity, establishment, hub, individual-based model, invasion, microcosm. network, simulation, spread.

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INTRODUCTION

Managing invasions becomes increasingly costly and decreasingly efficient with the expansion of the invaded area (Simberloff *et al*. 2013). In invasion biology, focus is placed on the processes occurring during the first generations after the introduction of an exotic species, before its proliferation and spread besides the introduction site. The dispersal abilities of individuals during invasions and the way they evolve over the course of the range expansion have been documented theoretically (Travis *et al*. 2009; Burton *et al*. 2010) and experimentally, notably among protists (Fronhofer & Altermatt 2015) and arthropods (Ochocki & Miller 2017; Weiss-Lehman *et al*. 2017). Although the role of individual characteristics on the spread patterns is well known, the way individuals disperse across a landscape also depends on the interaction between individuals and the features of the environment (Calabrese & Fagan 2004), which can themselves affect the evolution of dispersal (Baguette & Van Dyck 2007). Therefore, understanding the structure of the invaded landscape is also essential to understand the patterns of colonisation observed during the first stages of an invasion.

Networks have gained popularity in the last decades as a method to represent the spatial structure of such landscapes (Urban & Keitt 2001; Minor & Urban 2007). They are used to represent habitat patches suitable for the species considered (the vertices of the network) and the way dispersal connects them (the edges of the network). Networks are a powerful tool associated with various metrics and used across a variety of scientific fields, but they are still seldom used in invasion biology. In this field, they are classically used to describe food webs, to study how they are impacted by non-native species or to assess the invasibility of a community based on its trophic structure (Romanuk *et al*. 2009; Lurgi *et al*. 2014). A few studies also represent the spatial structure of riverine systems as networks, to study the impact of the network structure on the composition of a community invading a new environment (Seymour & Altermatt 2014; Alther & Altermatt 2018). This study aims at using networks in another way to investigate the establishment and spread of the invader, with a special focus on small-population demographic processes.

Although few usages of networks are recorded in invasion biology, other fields provide insight about the impact of the spatial structure of the introduction area on the first stages of an invasion. Notably, several epidemiology studies correlate specific metrics with patterns of spread. A prime example is the combination of networks with models derived from classical Susceptible-Infected (SI) models (Kermack & McKendrick 1927), for example, to study the spread of nosocomial infections in hospitals (Ueno & Masuda 2008) or the transmission of pathogens through grooming among macaques (Romano *et al*. 2016). These studies usually consider networks made up of hosts (the vertices) connected by social interactions (the edges). Other studies consider larger scale networks, in which vertices are populations of hosts and edges are host movements between populations (Arino & Van den Driessche 2006). Such a framework has, for example, been used to explain the extent of plague during the 14th century (Gómez & Verdú 2017) or to assess epidemic risks in the Japanese airline network (Tanaka *et al*. 2014). The conceptual similarities

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between the spread of disease outbreaks and the spread of invading organisms have led studies in invasion biology to use models originating from epidemiological modelling. They notably have been used to study the impact of landscape structure on the risks of invasive spread through forests (Ferrari & Lookingbill 2009) or across marinas via ballast waters (Floerl *et al*. 2009). However, these studies fail to consider the internal dynamics of each patch. Moreover, this impact of network structure on invasion remains to be experimentally tested.

Networks are also often used in conservation biology, notably to map the structure of metacommunities (Desjardins-Proulx & Gravel 2012; Ai *et al*. 2013; Gravel *et al*. 2014; Thompson *et al*. 2017) or metapopulations (Bunn *et al*. 2000; Urban & Keitt 2001; Treml *et al*. 2008; Almany *et al*. 2009). Network metrics are notably used to describe the connectivity between patches, to study speciation (Desjardins-Proulx & Gravel 2012) and species distribution across landscapes (Gravel *et al*. 2014; Thompson *et al*. 2017) as well as the mean lifetime of metapopulations (e.g. Bode *et al*. 2008; Drechsler 2009; Kininmonth *et al*. 2010; Shtilerman & Stone 2015) or the extinction risks of subpopulations (e.g. Gilarranz & Bascompte 2012; Peck 2012; Webb & Padgham 2013). They are also used to identify essential populations to maintain connectivity in a landscape (Bodin & Saura 2010; Baranyi *et al*. 2011; Watson *et al*. 2011). Yet, those studies often focus on the dispersal capabilities of individuals (Bodin & Saura 2010; Baranyi *et al*. 2011) or consider local population dynamics only through global colonisation and extinction probabilities, without considering explicitly the internal dynamics of the subpopulations (Bode *et al*. 2008; Gilarranz & Bascompte 2012; Shtilerman & Stone 2015). The studies considering the internal dynamics of populations more precisely and its interaction with network features usually concern large, already established populations, and their susceptibility to disturbances, biotic (Mari *et al*. 2014) or abiotic (Gilarranz *et al*. 2017). Conversely, the present study focuses on small-populations during their growth phase and on specific associated mechanisms that can interact with the network structure of the landscape.

Invaders usually experience multiple demographic bottlenecks, firstly at the initial introduction and subsequently at each colonisation event, which can strongly impact the success of an invasion. Mechanisms associated with small-population sizes are known to decrease establishment rates if the number of individuals introduced is too low (Simberloff 2009) and to block the spread of invaders to unoccupied patches (Keitt *et al*. 2001; Johnson *et al*. 2006). Because of their easily tractable effect on population dynamics, Allee effects are classically considered to account for small-population dynamics (Courchamp *et al*. 2008). However, other mechanisms affect small introduced populations. For instance, they have high risks of going extinct at random, a phenomenon known as demographic stochasticity (Lande *et al*. 2003). Their probability of not producing any individual able to successfully colonise other patches is also greater, a phenomenon referred to as dispersal stochasticity (Morel-Journel *et al*. 2016a). These demographic mechanisms have been shown to interact with dispersal and impact establishment. Strong emigration notably tends to decrease the establishment rate of introduced populations by decreasing individual density in the introduction site (Kean & Barlow 2000; Robinet *et al*. 2008; Morel-Journel *et al*. 2016b). This study addresses the interplay between the local dynamics of introduced populations and the network structure, during the first stages of an invasion.

Network structure will be characterised using two metrics related to the distribution of edges, that is, the way patches are connected to one another: clustering and centralisation. Clustering indicates the presence of clusters, that is, groups of vertices well connected to one another (Watts & Strogatz 1998; Jordán *et al*. 2003). Clusters have notably been shown to hinder the spread of pathogens (Badham & Stocker 2010), reduce the extinction risks of already colonised patches (Kininmonth *et al*. 2010) and buffer the propagation of disturbances across metapopulations (Gilarranz *et al*. 2017). In the context of invasion biology, they are expected to slow down the spread by 'trapping' invaders. Invasions in highly clustered landscape are therefore expected to create fewer, larger populations, which could therefore also be less vulnerable to extinction because of small-population mechanisms. Centralisation indicates the presence of hubs, that is, patches concentrating many dispersal flows across the landscape. Hubs are usually central elements in metapopulations, increasing connectivity and the persistence of remote populations otherwise isolated in fragmented landscapes (Watson *et al*. 2011). They have been shown to increase the speed of infection spread (Ueno & Masuda 2008; Romano *et al*. 2016) and to be more likely to generate epidemic outbreaks (Da Silva *et al*. 2012). In invasion biology, hubs are expected to facilitate the rapid spread of invaders across the landscape. Therefore, they may also increase extinction risks of small introduced populations by decreasing the population density locally, especially if the hub is the introduction site itself.

We developed an individual-based model to simulate the first generations following introduction, in landscapes whose structure was based on networks with known centralisation and clustering levels. In addition, we performed introductions of the parasitoid *Trichogramma chilonis*, in artificial landscapes, and followed the invasion dynamics during 10 generations. We chose the spatial structures of these artificial landscapes among networks used in the simulations to specifically test the impacts of centralisation. We performed simulations in the presence of Allee effects or with demographic stochasticity alone, to investigate the interaction between landscape structure and small-population mechanisms. *T. chilonis* does not suffer from Allee effects (Morel-Journel *et al*. 2016a) but exhibits a strong stochasticity in reproduction and in dispersal. Therefore, it provided a 'null biological model' of an introduced population for the experiment. The combination of simulations and experiment is a powerful tool to test hypotheses, and this approach has been at the root of landmark results that have become classics in ecology, for example, the competitive exclusion principle (Gause 1934), chaotic population dynamics (Cushing *et al*. 2002), stochasticity in spread rates (Melbourne & Hastings 2009) or population extinction risks (Drake *et al*. 2011). Nevertheless, invasion biology – as well as epidemiology and conservation biology, two other fields using spatial network – is heavily dominated

by theoretical development and lack experimental validations of classical theoretical predictions, which are essential for the emergence of new hypotheses and the building of a comprehensive ecological theory.

Our results provide the first experimental pieces of evidence of the impacts of landscape structure on the spread of invasive species. The consistency between these results and others in different fields demonstrates that the impacts of centralisation and clustering on spreading speed are robust to the ecological context. In addition, our simulations highlight the impact of Allee effects not only on the persistence of invasive populations but also on the colonisation speed itself. Another interesting result is that high-density mechanisms, such as over-competition, can operate even at an early stage and underpin invasion failures depending on the network structure of the landscape. Besides highlighting the importance of landscape structure to predict the outcome of invasions, our study demonstrates its interplay with internal population dynamics.

METHODS

The network structure of the landscapes

Using the *igraph* package (Csardi & Nepusz 2006) of the R software (R CoreTeam 2018), we generated 112618 networks with a number of vertices $N_v = 10$ and a number of edges $N_e = 15$ (see Supporting information 1 about the generation and selection of these networks). This set of networks represented all the possible different structures of connected, undirected and unlabelled networks. We computed two metrics to characterise each network: their clustering coefficient T indicating the presence of clusters, and their centralisation level C indicating the presence of hubs (see Supporting information 1 about the computation of these indices). We chose to use closeness centrality as a basis for computing C and to identify the most central vertex of each network (called the hub thereafter). This metric accounts for the length of each path linking a given node to the rest of the network (Freeman 1978). If several vertices shared the highest closeness value, the hub was selected at random among them.

Simulations

We developed an individual-based model to simulate invasions in landscape whose spatial structure was described in the previous section (see Supporting information 2 about the description of the model). This model was in discrete time, with two successive phases dispersal between patches and local population growth. In this model, individuals had no intrinsic dispersal propensity, and dispersal probability only depends on d_i (the degree of the vertex the individual is in) and p_e (the dispersal rate of individuals in a vertex with $d_i = 1$). Additional simulations including density-dependent dispersal were performed in order to understand how the way dispersal was modelled influenced the impact of C and T on the invasion dynamics (see Supporting information 4). Dispersal was then defined by γ (a minimal dispersal rate) and δ (describing the increase in dispersal propensity with local density). Local population growth was itself divided into two phases. The first concerns mating, affected by m, a density-independent mating probability, and a, a parameter describing potential mating Allee effects. The second is a reproduction phase, affected by α, the intraspecific competition; β, the fecundity rate of individuals; and s, the survival rate of juveniles.

Using this model, we simulated 20 time steps after a single introduction of 15 individuals in one patch of the landscape, for $p_e = 0.03$, $m = 0.8$, $\alpha = 0.008$, $\beta = 15$ and $s = 0.1$ (see Supporting information 5 for sensitivity analyses of these parameters). The initial population size was chosen so that extinction because of low numbers was possible but not systematic. We simulated 2×2 invasions for each network in our set, with either $a = 0$ (without Allee effects) or $a = 2.5$ (with Allee effects) and with an introduction site which was either the hub (the patch corresponding to the most central vertex) or another patch selected at random.

Experiment

To complement the simulations, we monitored artificial invasions of the egg parasitoid *Trichogramma chilonis* (Hymenoptera: *Trichogrammatidae*) in laboratory microcosm landscapes, for 10 non-overlapping generations following an initial introduction of 15 individuals (see Supporting information 3 for details about the experimental setup). This model species is especially suited for our experiment, because of its small size (*c.* 1 mm), its short and regular developmental cycle (consistently 9 days to reach the adult stage) and its ability to parasitise its laboratory host *Ephestia kuehniella* (Lepidoptera: *Pyralidae*). The latter allowed us to focus on *T. chilonis* by suppressing the host's dynamic and estimate population sizes throughout the experiment by counting the parasitised at each generation (see Supporting information 3). Moreover, we could control for impacts of the genetic makeup of the introduced populations by introducing individuals from the same inbred line, founded by a single female and maintained in the laboratory for over 100 generations.

All the microcosm landscapes were made up of 10 patches connected by 15 corridors, and their spatial structure was selected at random in two subsets of the set of networks previously established (see Supporting information 1): the 'high-C' and 'low-C' landscapes, whose centralisation values were, respectively, in the 10% highest and 10% lowest of all the centralisation values in the network set. We performed 63 artificial introductions equally distributed across three treatments: (1) in a patch selected at random in a low-C landscape, (2) in a hub in a high-C landscape, (3) in another patch than the hub in a high-C landscape. The 63 artificial landscapes were distributed equally across three experimental blocks, that is, three replicates of each treatment in each block.

Analysis of the results

We considered three response variables: the global extinction rate, the introduction site's extinction rate and the colonisation speed. The global extinction rate was computed as the proportion of invasions during which all the patches went extinct. The introduction site's extinction rate was computed

as the proportion of invasions during which the introduction site went extinct at least once. Both extinction rates followed binomial distributions and were analysed with logistic regressions. The colonisation speed was computed as the ratio between the maximal number of patches colonised and the number of generations to reach this maximal extent. Considering the maximum extent allowed us to differentiate failed establishments from the start from 'boom-and-bust' invasions. The colonisation speed had a continuous and positive distribution clustered towards low values and was therefore analysed with a gamma regression. We considered a fourth response variable for the experiment: the size reached by a local population at the generation preceding its extinction. These values were distributed as an overdispersed Poisson distribution that were analysed using a negative binomial regression.

We used AIC comparisons to assess the respective importance of C and T as explanatory variables in the simulations. We considered a model including both variables (CT model), one with only C (C model) and one with only T (T model). We computed Δ_C as the difference between the AIC of the CT model and the T model, and Δ_T as the difference between the AIC of the CT model and the C model. We considered that values of Δ_T or Δ_C greater than 10 indicated that the support for the CT model was unequivocally higher than for the other model (Burnham & Anderson 2003). To analyse the experimental results, we used generalised linear mixed models, to account for potential differences created by the experimental blocks. The experimental block was treated as a crossed random effect, as the treatments were distributed equally across all blocks.

RESULTS

Simulations

The global extinction rate was always negligible in the simulations when $a = 0$ (no Allee effect), regardless of the introduction site or the landscape characteristics (Fig. 1a,b). It was on average higher when $a = 2.5$ but was independent from clustering whether the introduction was performed in the hub ($\Delta_T = -0.66$) or elsewhere in the landscape ($\Delta_T = -0.95$). The centralisation level had an impact, but only when $a = 2.5$ and for an introduction in the hub ($\Delta_C = 605.44$). In this case, the global extinction rate increased with C (Fig. 1a).

The introduction site's extinction rate was also overall higher when $a = 2.5$ than when $a = 0$ but was impacted by centralisation regardless of Allee effects when individuals were introduced in the hub (Table 1). In this case, centralisation increased the extinction rate, with a large majority of introduction sites going extinct at high C levels (Fig. 1c). The impact of clustering appeared only for introductions in the hub and $a = 2.5$ ($\Delta_T = 9.70$). In this case, it decreased slightly the extinction rate of the introduction site (Fig. 1d).

Centralisation had a substantial positive impact on colonisation speeds when individuals were introduced in the hub itself (Fig. 1e), whether a = 0 ($\Delta_C = 1549.51$) or $a = 2.5$ ($\Delta_C = 3576.94$). Its positive impact was weaker for introductions outside of the hub, although it was still significant when $a = 0$ ($\Delta_C = 55.18$, Fig. 1e). Clustering markedly decreased the colonisation speeds when $a = 0$, when introductions were performed in the hub ($\Delta_T = 865.15$) or elsewhere ($\Delta_T = 648.80$). Its impact was, however, limited when $a = 2.5$ for introductions in the hub ($\Delta_T = 29.20$) and negligible for introductions outside of the hub ($\Delta_T = -0.58$). Overall, the colonisation speeds were higher when $a = 0$ compared to $a = 2.5$.

The sensitivity analyses performed on the parameters of the model (see Supporting information 4) showed that, although the parameters could have an impact on the values of the response variables (the global extinction rate, the introduction site's extinction rate or the colonisation speed), they did not affect qualitatively the relationships between centralisation or clustering and these response variables.

Experiment

We were not able to evidence differences between the three experimental treatments concerning the extinction rates (Fig. 2), neither at the level of the whole landscape (Likelihood Ratio Test, df = 2; $P = 0.344$) nor at the level of the introduction site (Likelihood Ratio Test, df = 2; $P = 0.747$). However, the sizes of the local populations going extinct during the invasions of high-C landscapes after an introduction outside of the hub were significantly higher than those in low-C landscapes (Wald test; $z = 2.325$, $P = 0.021$) and those in high-C landscapes with introductions in the hub (Wald test; $z = 2.951$, $P = 0.004$).

The colonisation speeds recorded during the experiment were overall low (way less than one patch per generation on average), but they were significantly higher in the high-C landscapes compared to the low-C landscapes (Wald test; $z = 2.980$, $P = 0.0014$), but only when the hub was the introduction site. Otherwise, there were no discernable differences in the colonisation speeds between high-C and low-C landscapes (Wald test $z = 0.594$; $P = 0.552$).

DISCUSSION

Impacts of landscape structure on spread

Both the simulations and the experiments evidenced an impact of landscape structure on spread. The colonisation of new patches by invaders was slowed down by the presence of clusters and accelerated by the presence of hubs. The effect of centralisation was dominant when individuals were initially introduced in the hub itself, although it was still significant when the introduction site was chosen at random in the simulations. Our results are consistent with other studies in other fields, and thus confirm the effect of the network structure itself on spread, regardless of context.

Centrality was repeatedly shown to facilitate the transmission of infections (e.g. Christley *et al.* 2005; Ueno & Masuda 2008; Da Silva *et al.* 2012; Romano *et al.* 2016). Epidemics starting in hubs were shown to reach greater sizes, thus underlining the influence of the hub at the start of spread (Da Silva *et al.* 2012), which we confirm in the context of invasions using simulations and experiments. Other studies showed that the most central agents also have higher chances of being

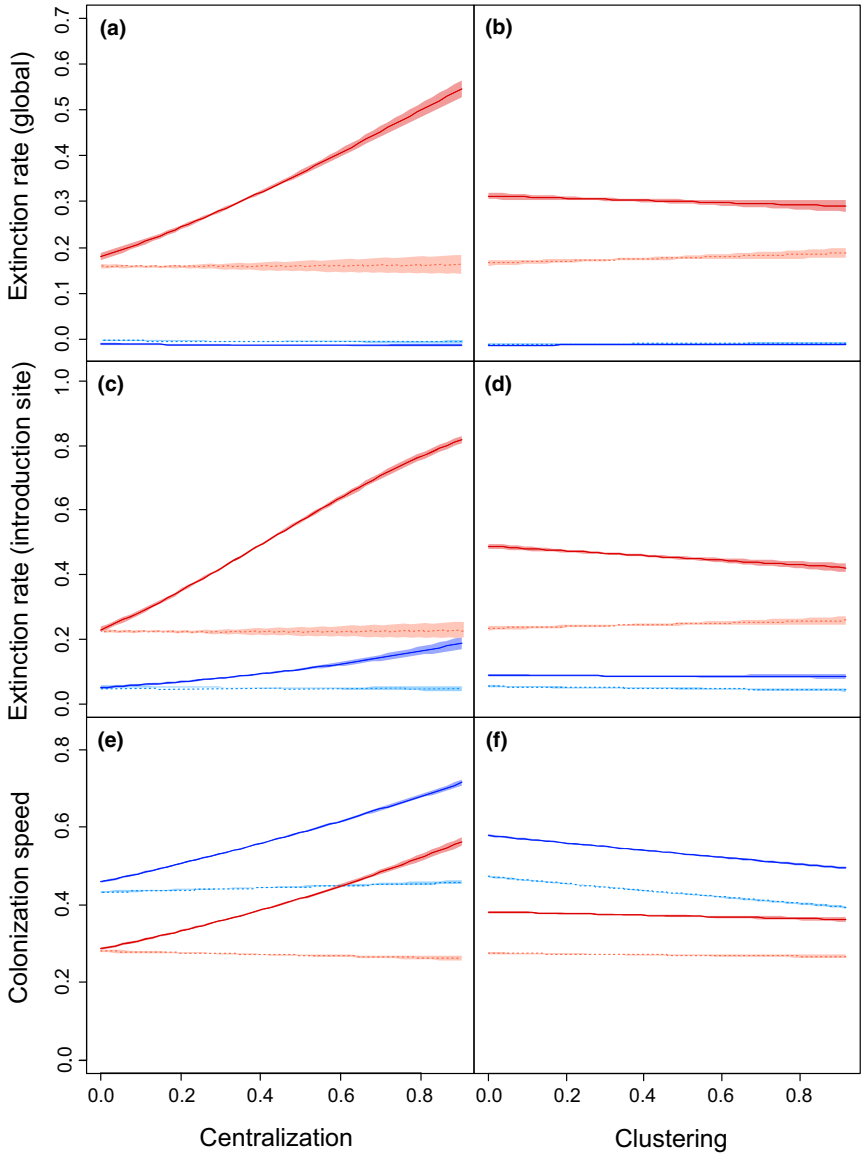

Figure 1 Estimated impacts of centralisation C (a, c, e) and clustering T (b, d, f) on the global extinction rate (a, b), introduction site's extinction rate (d, e) and colonisation speeds (e, f), with their 95% confidence intervals), for $a = 0$ (blue) and $a = 2.5$ (red) and for an introduction in the hub (full lines, dark) and outside the hub (dashed lines, light).

infected (Christley *et al.* 2005; Romano *et al.* 2016). In studies in epidemiology considering networks of populations, the most central vertices are susceptible to re-infections, even after the initial epidemic outbreak. This phenomenon, likely behind some of the greatest epidemics (e.g. Gómez & Verdú 2017), also underpins major challenges for the control of invasive populations; the most central patches do not only increase the speed of invasive spread but are also more likely to be invaded.

Our simulation results concerning clustering are also consistent with epidemiological studies showing its role in limiting the speed and extent of infections (Keeling 2005; Miller 2009; Badham & Stocker 2010). Moreover, Gilarranz *et al.* (2017) provide experimental evidence that modularity also prevents spread in a similar context: the propagation of disturbances. Modularity, which is particularly used in trophic (Dormann & Strauss 2014; Beckett 2016) and genetic networks (Fletcher *et al.* 2013; Peterman *et al.* 2016), describes the belonging of vertices to modules, that is, subparts of the network highly connected (Newman & Girvan 2004). In a metapopulation context, Gilarranz *et al.* (2017) show that disturbances occurring in one module mostly affect other populations in this module, similar to the way clusters "trap" introduced individuals in our simulation results.

Although they did not dramatically change the impacts of centralisation or clustering, Allee effects reduced the colonisation speed in the simulations. This result is consistent with the theoretical predictions of Keitt *et al.* (2001), which suggest that Allee effects can act as a supplementary hurdle to colonisation, preventing the small-populations at the margin of the invaded area to produce enough dispersing individuals to successfully colonising new patches. This impact of population

Table 1 Differences in AIC between the *T*-model (Δ_C) or the *C* model (Δ_T) and the *CT* model for the simulation results. All the values greater than 10 (indicating unequivocal higher support for the *CT* model compared to the other one) are in bold

Response variable	Allee effect	Intro. site	Δ_C	Δ_T
Global extinction rate	$a = 0$	In the hub	−1.90	−1.49
	$a = 0$	Outside	5.44	0.79
	$a = 2.5$	In the hub	**605.44**	−0.66
	$a = 2.5$	Outside	2.47	−0.95
Introduction site extinction rate	$a = 0$	In the hub	**233.61**	−0.11
	$a = 0$	Outside	−0.56	0.38
	$a = 2.5$	In the hub	**2219.53**	9.70
	$a = 2.5$	Outside	2.65	−1.95
Colonisation speed	$a = 0$	In the hub	**3576.94**	**865.15**
	$a = 0$	Outside	**55.18**	**648.80**
	$a = 2.5$	In the hub	**1549.51**	**29.20**
	$a = 2.5$	Outside	1.27	−0.58

dynamics on colonisation speed echoes theoretical results on the variation in speed rates with density-dependent growth (Sullivan *et al.* 2017) and on pushed invasion (Lewis & Kareiva 1993; Roques *et al.* 2012). The low colonisation speeds observed during our microcosm invasions indicated that colonisation failures also occurred during the experiment, creating a 'pushed-like' invasion pattern likely caused by dispersal stochasticity, as evidenced in Morel-Journel *et al.* (2016a) in the same experimental system.

Impacts of landscape structure on establishment

In addition to affecting the spread of invaders, the network structure of the introduction area also impacted the dynamics of the introduced populations. While the results concerning spread suggest that the same mechanisms were at play in the experiment and the simulations, extinction appears to be underpinned by different causes. In the simulations, the centralisation of the landscape increased the introduction site's extinction rate when individuals were introduced in the hub. This result highlights a trade-off between colonisation and local persistence at the beginning of invasions, which is consistent with previous works in invasion biology. Theoretical (Lewis & Kareiva 1993; Kanarek *et al.* 2013) and empirical (Robinet *et al.* 2008; Vercken *et al.* 2011) studies underline the negative impact of dispersal soon after the introduction on the persistence of introduced populations. However, the mechanism invoked by these studies to explain these extinctions is the Allee effect, while our simulation results show that

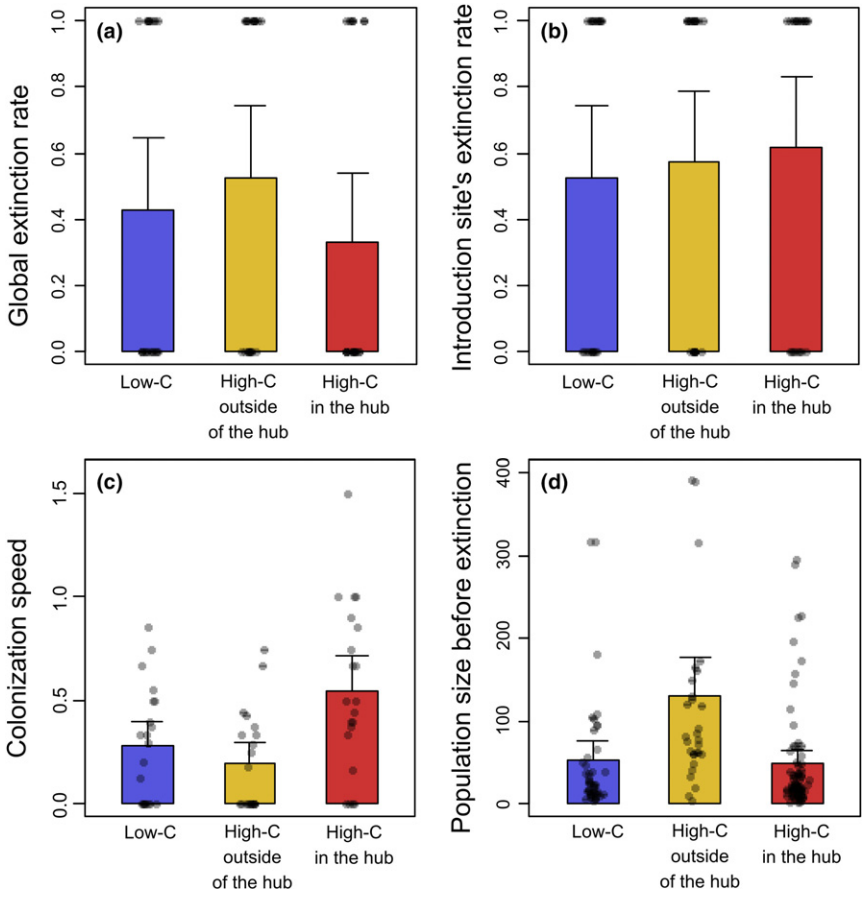

Figure 2 Mean experimental values for global extinction rates (a), the introduction site's extinction rates (b), colonisation speeds (c) and populations size before their extinction (d), for the experimental introductions in low-C landscapes (blue), high-C landscapes outside of the hub (yellow) and high-C landscapes in the hub (red). The error bars represent two times the standard deviation from the mean value and the dots represent the raw values.

the extinction of the introduction site can occur because of demographic stochasticity alone.

During the experiment, local extinctions occurred regardless of the treatment considered. If some extinctions occurring at small-population sizes can be attributed to demographic stochasticity, other populations went extinct while they were close to the carrying capacity, which rather suggests that they suffered from over-competition. This would be consistent with the biology of *T. chilonis*, which can be subject to strong over-competition through superparasitism (Suzuki *et al.* 1984). This phenomenon occurred most often for introductions outside of the hub in high-C landscapes, that is, in poorly connected patches. Because of their low dispersal rate, individuals mostly remained in the isolated introduction site, where they rapidly suffered from superparasitism and eventually went extinct.

Complete establishment failures not only required the extinction of the introduction site but also that individuals fail to colonise other patches. While we recorded extinctions of the introduction site regardless of Allee effects in the simulations, most of them did not lead to global extinctions for $a = 0$. Interestingly, the most important impact of Allee effects on establishment was to prevent the colonisation of new patches in the landscape. During the experiment, colonisation failures resulted from an extinction of the introduction site, either because of demographic stochasticity or over-competition, combined with colonisation failures because of low dispersal rate and dispersal stochasticity.

Impact of dispersal behaviour of the individuals

Since the dispersal was treated as a diffusion process in our model, individuals had no intrinsic dispersal propensity and dispersed only depending on the connections between patches. Although previous results by Morel-Journel *et al.* (2016b) showed the validity of this approximation to describe *T. chilonis* in our experimental system, we performed additional simulations in which dispersal was impacted by local density, rather than the structure of the landscape (Supporting information 4). The colonisation patterns observed remained similar to those observed without density-dependent dispersal (a positive impact of centralisation and a negative impact of clustering), but these effects were independent from the location of the introduction site. The effects of landscape structure on extinction were also slightly modified, as the increase in the extinction rate with centralisation was partly buffered by density-dependent dispersal, and clustering tended to decrease the extinction rates. Indeed, centralisation consistently increased the extinction rate of the introduction site for high enough dispersal rates, but it had no impact on the global extinction rate, regardless of Allee effects. Clustering decreased extinction rates in the presence of Allee effects, both globally and in the introduction site, especially with strong density dependence of dispersal. Therefore, the persistence of metapopulations was overall higher when dispersal was density dependent.

Besides external factors such as local density, dispersal can also depend on intrinsic differences between individuals. During spread, dispersal is not only impacted by the genetic background of the introduced individuals but also affects the spatial distribution of genotypes across space. This feedback loop was not considered in our model because all the individuals were considered identical, but such evolution during range expansion has been documented experimentally (Fronhofer & Altermatt 2015; Ochocki & Miller 2017; Weiss-Lehman *et al.* 2017). Yet, we expected the impact of the genetic background of individuals or potential variations between populations to be minimal during our experiment, because of the very low genetic variability in the populations initially introduced (15 individuals from the same inbred line).

Impact of the size of the landscape

The number of vertices of the landscapes used in this study ($N_v = 10$) was not only smaller than the values used for epidemiology studies but also smaller than the sizes used for network describing entire landscapes. As we aimed at describing invasions dynamics just after the introduction, we created landscapes corresponding to the direct surroundings of the introduction site only. To test the validity of the results presented in larger landscapes, we performed additional simulations, with networks with a larger number of edges (N_e varying between 20 and 40) and with a larger number of vertices (N_v varying between 20 and 100) (see Supporting information 6). The results indicate that the effects of clustering were robust to variations in the number of vertices, while the effects of centralisation became weaker, although they remained qualitatively the same. The effects of these metrics were also robust to variations in the number of edges, up to a point. For high values of N_e, such network-level metrics become irrelevant, as almost every patch can be considered a hub and belonging to a cluster.

CONCLUSION

Predicting the fate of introduced species remains a central objective of invasion biology. This study is a first demonstration of the use of network theory in this context to characterise the structure of landscapes and predict their invasibility. We built upon previous results in epidemiology or conservation to investigate the robustness of the influence of network structure on spread patterns. In addition, our study highlights interactions between the two network metrics studied – centralisation and clustering – and small-population dynamics characteristic of early stages of invasion. Among the involved small-population mechanisms, Allee effects often provide a simple and elegant way to describe the positive density dependence occurring in small introduced populations, but they lack generality and empirical support (Kramer *et al.* 2009; Gregory *et al.* 2010). Our results demonstrate that demographic and dispersal stochasticities can create similar patterns, by bringing small, well-connected populations to extinction, or by preventing colonisation. Moreover, negative density dependence and competitive interactions can also interact with landscape structure and affect the outcome of potential invasions.

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AUTHORSHIP

TMJ, LM and EV designed the model and experiments; TMJ and CRA carried out the simulations, experiments and data analyses; all authors participated in the writing of the manuscript and gave their final approval for publication.

DATA ACCESSIBILITY STATEMENT

R simulation code and experimental data: are available from the Dryad Digital Repository: http://doi.org/10.5061/dryad.1g3gp03.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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