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1 **Title:** It's all about connections: hubs and invasion in habitat networks

2 **Authors:** Thibaut Morel-Journel¹, Claire Rais Assa², Ludovic Mailleret^{2,3}, Elodie Vercken²

3 **Affiliations:**

4 ¹ Earth and Life Institute, Biodiversity Research Centre, Université Catholique de Louvain,
5 Louvain-la-Neuve, Belgium

6 ² Université Côte d'Azur, INRA, CNRS, ISA, 06900 Sophia Antipolis, France

7 ³ Université Côte d'Azur, Inria, INRA, CNRS, UPMC Univ. Paris 06, 06900 Sophia Antipolis,
8 France

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17 **Corresponding author:** Thibaut Morel-Journel (tel: +3210472759; e-mail:
18 thibaut.morel@uclouvain.be)

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23 results and the simulation code will be archived in Dryad and the data DOI will be included at
24 the end of the article.

1 **Abstract**

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

1 **Introduction**

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

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

26 another way: to investigate the establishment and spread of the invader, with a special focus on
27 small-population demographic processes.

28 Although few usages of networks are recorded in invasion biology, other fields provide
29 insight about the impact of the spatial structure of the introduction area on the first stages of an
30 invasion. Notably, several epidemiology studies correlate specific metrics with patterns of
31 spread. A prime example is the combination of networks with models derived from classical
32 Susceptible-Infected (SI) models (Kermack & McKendrick 1927), e.g. to study the spread of
33 nosocomial infections in hospitals (Ueno & Masuda 2008) or the transmission of pathogens
34 through grooming among macaques (Romano *et al.* 2016). These studies usually consider
35 networks made up of hosts (the vertices) connected by social interactions (the edges). Other
36 studies consider larger-scale networks, in which vertices are populations of hosts and edges are
37 host movements between populations (Arino & Van den Driessche 2006). Such a framework
38 has for example been used to explain the extent of plague during the fourteenth century (Gómez
39 & Verdú 2017) or to assess epidemic risks in the Japanese airline network (Tanaka *et al.* 2014).
40 The conceptual similarities between the spread of disease outbreaks and the spread of invading
41 organisms have led studies in invasion biology to use models originating from epidemiological
42 modelling. They notably have been used to study the impact of landscape structure on the risks
43 of invasive spread through forests (Ferrari & Lookingbill 2009) or across marinas via ballast
44 waters (Floerl *et al.* 2009). However, these studies fail to consider the internal dynamics of each
45 patch. Moreover, this impact of network structure on invasion remains to be experimentally
46 tested.

47 Networks are also often used in conservation biology, notably to map the structure of
48 existing metapopulations, in terrestrial (Bunn *et al.* 2000; Urban & Keitt 2001), marine (Trembl
49 *et al.* 2008; Almany *et al.* 2009) and riverine contexts (Schick & Lindley 2007). Network
50 metrics are notably used to describe the connectivity between sub-populations, to study the

51 mean lifetime of metapopulations (e.g. Bode *et al.* 2008; Drechsler 2009; Kininmonth *et al.*
52 2010; Shtilerman & Stone 2015) or the extinction risks of sub-populations (e.g. Gilarranz &
53 Bascompte 2012; Peck 2012; Webb & Padgham 2013). They are also used to identify essential
54 populations to maintain connectivity in a landscape (Bodin & Saura 2010; Baranyi *et al.* 2011;
55 Watson *et al.* 2011). Yet, those studies often focus on the dispersal capabilities of individuals
56 (Bodin & Saura 2010; Baranyi *et al.* 2011) or consider local population dynamics only through
57 global colonization and extinction probabilities, without considering explicitly the internal
58 dynamics of the sub-populations (Bode *et al.* 2008; Gilarranz & Bascompte 2012; Shtilerman
59 & Stone 2015). The studies considering the internal dynamics of populations more precisely
60 and its interaction with network features usually concern large, already established populations,
61 and their susceptibility to disturbances, biotic (Mari *et al.* 2014) or abiotic (Gilarranz *et al.*
62 2017). Conversely, the present study focuses on small populations during their growth phase,
63 and on specific associated mechanisms that can interact with the network structure of the
64 landscape.

65 Invaders usually experience multiple demographic bottlenecks, firstly at the initial
66 introduction and subsequently at each colonization event, which can strongly impact the success
67 of an invasion. Mechanisms associated with small population sizes are known to decrease
68 establishment rates if the number of individuals introduced is too low (Simberloff 2009) and to
69 block the spread of invaders to unoccupied patches (Keitt *et al.* 2001; Johnson *et al.* 2006).
70 Because of their easily tractable effect on population dynamics, Allee effects are classically
71 considered to account for small population dynamics (Courchamp *et al.* 2008). However, other
72 mechanisms affect small introduced populations. For instance, they have high risks of going
73 extinct at random, a phenomenon known as demographic stochasticity (Lande *et al.* 2003).
74 Their probability of not producing any individual able to successfully colonize other patches is
75 also greater, a phenomenon referred to as dispersal stochasticity (Morel-Journel *et al.* 2016a).

76 These demographic mechanisms have been shown to interact with dispersal and impact
77 establishment. Strong emigration notably tends to decrease the establishment rate of introduced
78 populations by decreasing individual density in the introduction site (Kean & Barlow 2000;
79 Robinet *et al.* 2008; Morel-Journel *et al.* 2016b). This study addresses the interplay between the
80 local dynamics of introduced populations and the network structure, during the first stages of
81 an invasion.

82 Network structure will be characterized using two metrics related to the distribution of
83 edges, i.e. the way patches are connected to one another: clustering and centralization.
84 Clustering indicates the presence of clusters, i.e. groups of vertices well connected to one
85 another (Watts & Strogatz 1998; Jordán *et al.* 2003). Clusters have notably been shown to
86 hinder the spread of pathogens (Badham & Stocker 2010), reduce the extinction risks of already
87 colonized patches (Kininmonth *et al.* 2010), and buffer the propagation of disturbances across
88 metapopulations (Gilarranz *et al.* 2017). In the context of invasion biology, they are expected
89 to slow down the spread by “trapping” invaders. Invasions in highly clustered landscape are
90 therefore expected to create fewer, larger populations, which could therefore also be less
91 vulnerable to extinction because of small population mechanisms. Centralization indicates the
92 presence of hubs, i.e. patches concentrating many dispersal flows across the landscape. Hubs
93 are usually central elements in metapopulations, increasing connectivity and the persistence of
94 remote populations otherwise isolated in fragmented landscapes (Watson *et al.* 2011). They
95 have been shown to increase the speed of infection spread (Ueno & Masuda 2008; Romano *et*
96 *al.* 2016) and to be more likely to generate epidemic outbreaks (Da Silva *et al.* 2012). In
97 invasion biology, hubs are expected to facilitate the rapid spread of invaders across the

98 landscape. Therefore, they may also increase extinction risks of small introduced populations
99 by decreasing the population density locally, especially if the hub is the introduction site itself.

100 We developed an individual-based model to simulate the first generations following
101 introduction, in landscapes whose structure was based on networks with known centralization
102 and clustering levels. In addition, we performed introductions of the parasitoid *Trichogramma*
103 *chilonis*, in artificial landscapes, and followed the invasion dynamics during ten generations.
104 We chose the spatial structures of these artificial landscapes among networks used in the
105 simulations to specifically test the impacts of centralization. We performed simulations in the
106 presence of Allee effects or with demographic stochasticity alone, to investigate the interaction
107 between landscape structure and small population mechanisms. *T. chilonis* does not suffer from
108 Allee effects (Morel-Journel *et al.* 2016a) but exhibits a strong stochasticity in reproduction and
109 in dispersal. Therefore, it provided a “null biological model” of an introduced population for
110 the experiment. The combination of simulations and experiment is a powerful tool to test
111 hypotheses, and this approach has been at the root of landmark results that have become classics
112 in ecology, e.g. the competitive exclusion principle (Gause 1934), chaotic population dynamics
113 (Cushing *et al.* 2002), stochasticity in spread rates (Melbourne & Hastings 2009) or population
114 extinction risks (Drake *et al.* 2011). Nevertheless, invasion biology – as well as epidemiology
115 and conservation biology, two other fields using spatial network – is heavily dominated by
116 theoretical development and lack experimental validations of classical theoretical predictions,
117 which are essential for the emergence of new hypotheses and the building of a comprehensive
118 ecological theory.

119 Our results provide the first experimental pieces of evidence of the impacts of landscape
120 structure on the spread of invasive species. The consistency between these results and others in
121 different fields demonstrates that the impacts of centralization and clustering on spreading
122 speed are robust to the ecological context. In addition, our simulations highlight the impact of

123 Allee effects, not only on the persistence of invasive populations, but also on the colonization
124 speed itself. Another interesting result is that high-density mechanisms, such as over-
125 competition, can operate even at an early stage and underpin invasion failures depending on the
126 network structure of the landscape. Besides highlighting the importance of landscape structure
127 to predict the outcome of invasions, our study demonstrates its interplay with internal
128 population dynamics.

1 **Methods**

2 *The network structure of the landscapes*

3 Using the *igraph* package (Csardi & Nepusz 2006) of the R software (R CoreTeam 2018), we
4 generated 112618 networks with a number of vertices $N_v = 10$ and a number of edges $N_e = 15$
5 (see Supporting information 1 about the generation and selection of these networks). This set
6 of networks represented all the possible different structure of connected, undirected and
7 unlabeled networks. We computed two metrics to characterize each network: their clustering
8 coefficient T indicating the presence of clusters, and their centralization level C indicating the
9 presence of hubs (see Supporting information 1 about the computation of these indices). The
10 value of C is based on two measures of centrality, which encompasses several, conceptually
11 distinct measures, such as the degree, the eigenvector centrality, the betweenness and the
12 closeness. Although all these measures were highly correlated in the set of networks considered
13 (see Supporting information 1), we chose to consider here the closeness and the betweenness.
14 Both were chosen because they carry information about the structure of the paths linking
15 vertices, the closeness accounting for the length of these paths, and the betweenness for their
16 tendency to lie on these paths (Freeman 1978). Because of the correlation between these
17 metrics, we based our measure of centralization on the product between betweenness and
18 closeness, rather than considering them separately. We also identified the most central vertex
19 of a network (called the hub thereafter) based on the product between its betweenness and its
20 closeness. If several vertices shared the highest centrality value, the hub was selected at random
21 among them.

22

23 *Simulations*

24 We developed an individual-based model to simulate invasions in landscape whose spatial
25 structure was described in the previous section (see Supporting information 2 about the

26 description of the model). This model was in discrete time, with two successive phases dispersal
27 between patches and local population growth. In this model, individuals had no intrinsic
28 dispersal propensity, and dispersal probability only depends on d_i (the degree of the vertex the
29 individual is in) and p_e (the dispersal rate of individuals in a vertex with $d_i = 1$). Local
30 population growth was itself divided in two phases. The first concerns mating, affected by m , a
31 density-independent mating probability, and a , a parameter describing potential mating Allee
32 effects. The second is a reproduction phase, affected by α , the intra-specific competition, β the
33 fecundity rate of individuals and s , the survival rate of juveniles.

34 Using this model, we simulated 20 time steps after a single initial introduction of 15
35 individuals in one patch of the landscape, for $p_e = 0.03$, $m = 0.8$, $\alpha = 0.008$, $\beta = 15$ and $s = 0.1$
36 (see Supporting information 4 for sensitivity analyses of these parameters). The initial
37 population size was chosen so that extinction because of low numbers was possible but not
38 systematic. We simulated 2x2 invasions for each network in our set, with either $a = 0$ (without
39 Allee effects) or $a = 2.5$ (with Allee effects), and with an introduction site which was either the
40 hub (the patch corresponding to the most central vertex) or another patch selected at random.

41

42 *Experiment*

43 To complement the simulations, we monitored artificial invasions of the egg parasitoid
44 *Trichogramma chilonis* (Hymenoptera: *Trichogrammatidae*) in laboratory microcosm
45 landscapes, for ten non-overlapping generations following an initial introduction of 15
46 individuals (see Supporting information 3 for details about the experimental setup). This model
47 species is especially suited for our experiment, because of its small size (approx. 1mm), its short
48 and regular developmental cycle (consistently 9 days to reach the adult stage), and its ability to
49 parasitize its laboratory host *Ephesia kuehniella* (Lepidoptera: *Pyralidae*). The latter allowed
50 us to focus on *T.chilonis* by suppressing the host's dynamic, and estimate population sizes

51 throughout the experiment by counting the parasitized at each generation (see Supporting
52 information 3). Moreover, we could control for impacts of the genetic makeup of the introduced
53 populations by introducing individuals from the same inbred line, founded by a single female
54 and maintained in the laboratory for over 100 generations.

55 All the microcosm landscapes were made up of 10 patches connected by 15 corridors,
56 and their spatial structure were selected at random in two subsets of the set of networks
57 previously established (see Supporting information 1): the “high-C” and “low-C” landscapes,
58 whose centralization values were respectively in the 10% highest and 10% lowest of all the
59 centralization values in the network set. We performed 63 artificial introductions equally
60 distributed across three treatments: (i) in a patch selected at random in a low-C landscape, (ii)
61 in a hub in a high-C landscape, (iii) in another patch than the hub in a high-C landscape. The
62 63 artificial landscapes were distributed equally across 7 experimental blocks, i.e. 3 replicates
63 of each treatment in each block.

64

65 *Analysis of the results*

66 We considered three response variables: the global extinction rate, the introduction site’s
67 extinction rate and the colonization speed. The global extinction rate was computed as the
68 proportion of invasions during which all the patches went extinct. The introduction site’s
69 extinction rate was computed as the proportion of invasions during which the introduction site
70 went extinct at least once. Both extinction rates followed binomial distributions and were
71 analyzed with logistic regressions. The colonization speed was computed as the ratio between
72 the maximal number of patches colonized and the number of generations to reach this maximal
73 extent. Considering the maximum extent allowed us to differentiate failed establishments from
74 the start from “boom-and-bust” invasions. The colonization speed had a continuous and positive
75 distribution clustered towards low values and was therefore analyzed with a gamma regression.

76 we considered a fourth response variable for the experiment: the size reached by a local
77 population at the generation preceding its extinction. These values were distributed as an
78 overdispersed Poisson distribution, that were analyzed using a negative binomial regression.

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

1 **Results**

2 *Simulations*

3 The global extinction rate was always negligible in the simulations when $a = 0$ (no Allee effect),
4 regardless of the introduction site or the landscape characteristics (Figure 1A, B). It was on
5 average higher when $a = 2.5$ but was independent from clustering whether the introduction was
6 performed in the hub ($\Delta_T = -1.02$) or elsewhere in the landscape ($\Delta_T = -1.87$). The
7 centralization level had an impact, but only when $a = 2.5$ and for an introduction in the hub
8 ($\Delta_C = 602.37$). In this case, the global extinction rate increased with C (Figure 1A).

9 The introduction site's extinction rate was also overall higher when $a = 2.5$ than when
10 $a = 0$ but was impacted by centralization regardless of Allee effects when individuals were
11 introduced in the hub (Table 1). In this case, centralization increased the extinction rate, with a
12 large majority of introduction sites going extinct at high C levels (Figure 1C). The impact of
13 clustering appeared only for introductions in the hub and $a = 2.5$ ($\Delta_T = 9.70$). In this case, it
14 decreased slightly the extinction rate of the introduction site (Figure 1D).

15 Centralization had a substantial positive impact on colonization speeds when individuals
16 were introduced in the hub itself (Figure 1E), whether $a = 0$ ($\Delta_C = 1578.84$) or $a = 2.5$
17 ($\Delta_C = 3250.45$). Its positive impact was weaker for introductions outside of the hub, although it
18 was still significant when $a = 0$ ($\Delta_C = 130.50$, Figure 1E). Clustering markedly decreased the
19 colonization speeds when $a = 0$, when introductions were performed in the hub ($\Delta_T = 865.15$)
20 or elsewhere ($\Delta_T = 648.80$). Its impact was however limited when $a = 2.5$ for introductions in
21 the hub ($\Delta_T = 29.20$) and negligible for introductions outside of the hub ($\Delta_T = -0.58$). Overall,
22 the colonization speeds were higher when $a = 0$ compared to $a = 2.5$.

23 The sensitivity analyses performed on the parameters of the model (see Supporting
24 information 4) showed that, although the parameters could have an impact on the values of the
25 response variables (the global extinction rate, the introduction site's extinction rate or the

26 colonization speed), they did not affect qualitatively the relationships between centralization or
27 clustering, and these response variables.

28

29 *Experiment*

30 We were not able to evidence differences between the three experimental treatments concerning
31 the extinction rates (Figure 2), neither at the level of the whole landscape (Likelihood Ratio
32 Test, $df = 2$; $p = 0.344$) nor at the level of the introduction site (Likelihood Ratio Test, $df = 2$;
33 $p = 0.747$). However, the sizes of the local populations going extinct during the invasions of
34 high-C landscapes after an introduction outside of the hub were significantly higher than those
35 in low-C landscapes (Wald test; $z = 2.325$, $p = 0.021$) and those in high-C landscapes with
36 introductions in the hub (Wald test; $z = 2.951$, $p = 0.004$).

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

1 **Discussion**

2 *Impacts of landscape structure on spread*

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

10 Centrality was repeatedly shown to facilitate the transmission of infections (e.g.
11 Christley *et al.* 2005; Ueno & Masuda 2008; Da Silva *et al.* 2012; Romano *et al.* 2016).
12 Epidemics starting in hubs were shown to reach greater sizes, thus underlining the influence of
13 the hub at the start of spread (Da Silva *et al.* 2012), which we confirm in the context of invasions
14 simulations and experiments. Other studies showed that the most central agents also have higher
15 chances of being infected (Christley *et al.* 2005; Romano *et al.* 2016). In studies in
16 epidemiology considering networks of populations, the most central vertices are susceptible to
17 re-infections, even after the initial epidemic outbreak. This phenomenon, likely behind some of
18 the greatest epidemics (e.g. Gómez & Verdú 2017), also underpins major challenges for the
19 control of invasive populations: the most central patches do not only increase the speed of
20 invasive spread but are also more likely to be invaded.

21 Our simulation results concerning clustering are also consistent with epidemiological
22 studies showing its role in limiting the speed and extent of infections (Keeling 2005; Miller
23 2009; Badham & Stocker 2010). Moreover, Gilarranz *et al.* (2017) provide experimental
24 evidence that modularity also prevents spread in a similar context: the propagation of
25 disturbances. Modularity, which is particularly used in trophic (Dormann & Strauss 2014;

26 Beckett 2016) and genetic networks (Fletcher Jr *et al.* 2013; Peterman *et al.* 2016), describes
27 the belonging of vertices to modules, i.e. subparts of the network highly connected (Newman
28 & Girvan 2004). In a metapopulation context, Gilarranz *et al.* (2017) show that disturbances
29 occurring in one module mostly affect other populations in this module, similarly to the way
30 clusters “trap” introduced individuals in our simulation results.

31 Although they did not dramatically change the impacts of centralization or clustering,
32 Allee effects reduced the colonization speed in the simulations. This result is consistent with
33 the theoretical predictions of Keitt *et al.* (2001), which suggest that Allee effects can act as a
34 supplementary hurdle to colonization, preventing the small populations at the margin of the
35 invaded area to produce enough dispersing individuals to successfully colonizing new patches.
36 This impact of population dynamics on colonization speed echoes theoretical results on the
37 variation of speed rates with density-dependent growth (Sullivan *et al.* 2017) and on pushed
38 invasion (Lewis & Kareiva 1993; Roques *et al.* 2012). The low colonization speeds observed
39 during our microcosm invasions indicated that colonization failures also occurred during the
40 experiment, creating a “pushed-like” invasion pattern likely caused by dispersal stochasticity,
41 as evidenced in Morel-Journel *et al.* (2016a) in the same experimental system.

42

43 *Impacts of landscape structure on establishment*

44 In addition to affecting the spread of invaders, the network structure of the introduction area
45 also impacted the dynamics of the introduced populations. While the results concerning spread
46 suggest that the same mechanisms were at play in the experiment and the simulations,
47 extinction appears to be underpinned by different causes. In the simulations, the centralization
48 of the landscape increased the introduction site’s extinction rate when individuals were
49 introduced in the hub. This result highlights a tradeoff between colonization and local
50 persistence at the beginning of invasions, which is consistent with previous works in invasion

51 biology. Theoretical (Lewis & Kareiva 1993; Kanarek *et al.* 2013) and empirical (Robinet *et*
52 *al.* 2008; Vercken *et al.* 2011) studies underline the negative impact of dispersal soon after the
53 introduction on the persistence of introduced populations. However, the mechanism invoked
54 by these studies to explain these extinctions is the Allee effect, while our simulation results
55 show that the extinction of the introduction site can occur because of demographic stochasticity
56 alone.

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

67 Complete establishment failures did not only require the extinction of the introduction
68 site, but also that individuals fail to colonize other patches in the landscape. While we recorded
69 extinctions of the introduction site regardless of Allee effects in the simulations, most of them
70 did not lead to global extinctions for $a = 0$. Interestingly, the most important impact of Allee
71 effects on establishment was to prevent the colonization of new patches in the landscape.
72 During the experiment, colonization failures resulted from an extinction of the introduction site,

73 either because of demographic stochasticity or over-competition, combined with colonization
74 failures because of low dispersal rate and dispersal stochasticity.

75

76 *Impact of the size of the landscape*

77 The number of vertices of the landscapes used in this study ($N_v = 10$) was not only smaller than
78 the values used for epidemiology studies, but also smaller than the sizes used for network
79 describing entire landscapes. As we aimed at describing invasions dynamics just after the
80 introduction, we created landscapes corresponding to the direct surroundings of the introduction
81 site only. To test the validity of the results presented in larger landscapes, we performed
82 additional simulations, with networks with a larger number of edges (N_e varying between 20
83 and 40) and with a larger number of vertices (N_v varying between 20 and 100) (see Supporting
84 information 5). The results indicate that the effects of clustering were robust to variations in the
85 number of vertices, while the effects of centralization became weaker, although they remained
86 qualitatively the same. The effects of these metrics were also robust to variations in the number
87 of edges, up to a point. For high values of N_e , such network-level metrics become irrelevant, as
88 almost every patch can be considered a hub and belonging to a cluster.

89

90 *Impact of dispersal behavior of the individuals*

91 In our model, the dispersal of individuals was treated as a diffusion process. Hence, individuals
92 had no intrinsic dispersal propensity, and dispersed only depending on the way the introduction
93 site was connected to the rest of the landscape. Previous results by Morel-Journel *et al.* (2016b)
94 demonstrated that this approximation is valid to describe the dispersal of *T. chilonis* across our
95 experimental system. Yet, we completed our results by considering emigration rates dependent
96 on the choice of individuals, rather than the structure of the landscape (see Supporting
97 information 6). Dispersal choice based on environmental quality would not have been relevant

98 to our study, as the sameness of habitat quality across all patches of the landscape is a central
99 aspect of the study. We rather focused on the impact of the presence of conspecifics, i.e. density-
100 dependent dispersal. The results we obtained were qualitatively similar to those previously
101 obtained on spread and on the extinction rate of the introduction site, with little impact of the
102 strength of the density-dependence of dispersal. These additional simulations therefore suggest
103 that the patterns observed in our simulations are robust to variations in the dispersal propensity
104 of individuals. However, we did not test the potential role of the patch quality on this propensity,
105 as all the patches in all our landscapes were considered having the same quality.

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

116

117 *Conclusion*

118 Predicting the fate of introduced species remains a central objective of invasion biology. This
119 study is a first demonstration of the use of network theory in this context to characterize the
120 structure of landscapes and predict their invasibility. We built upon previous results in
121 epidemiology or conservation to investigate the robustness of the influence of network structure
122 on spread patterns. In addition, our study highlights interactions between the two network

123 metrics studied – centralization and clustering – and small population dynamics characteristic
124 of early stages of invasion. Among the involved small population mechanisms, Allee effects
125 often provide a simple and elegant way to describe the positive density dependence occurring
126 in small introduced populations, but they lack generality and empirical support (Kramer *et al.*
127 2009; Gregory *et al.* 2010). Our results demonstrate that demographic and dispersal
128 stochasticities can create similar patterns, by bringing small, well connected populations to
129 extinction, or by preventing colonization. Moreover, negative density-dependence and
130 competitive interactions can also interact with landscape structure and affect the outcome of
131 potential invasions.

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1 References

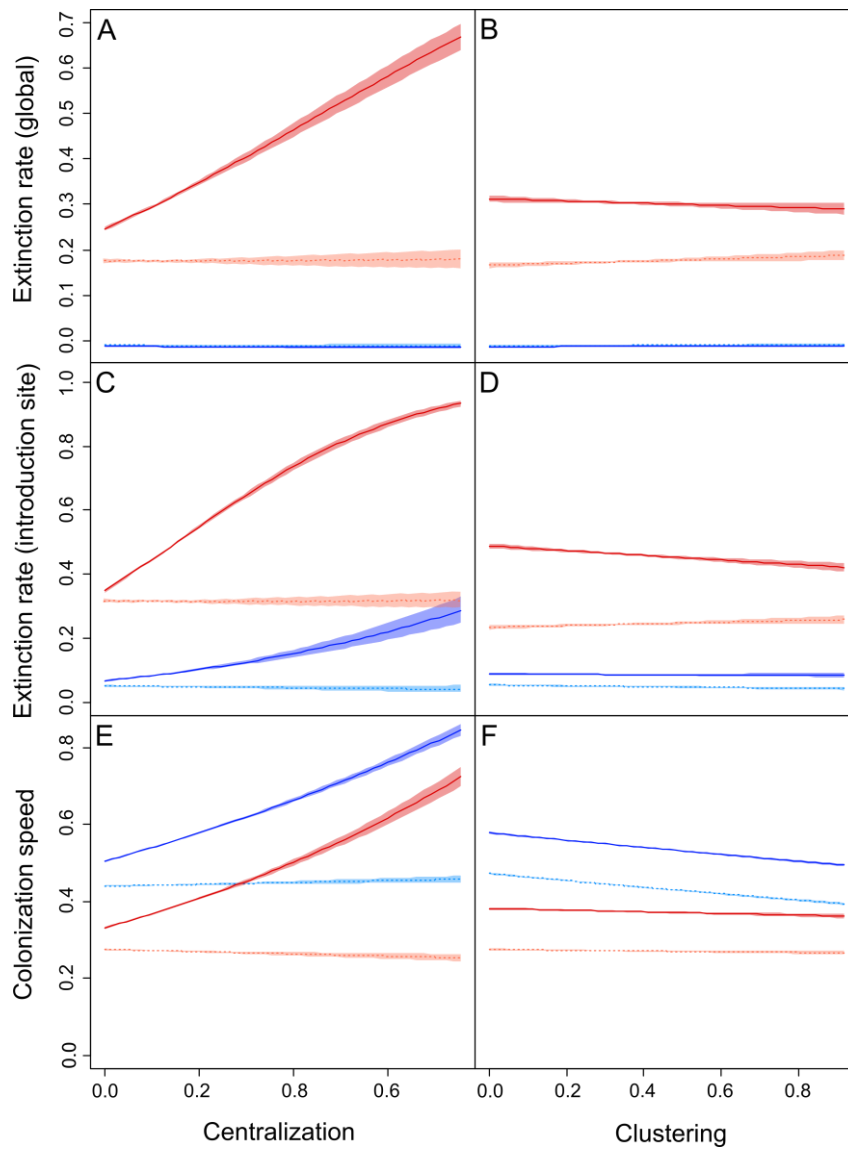
- 2 Alther, R. & Altermatt, F. (2018). Fluvial network topology shapes communities of native and
3 non-native amphipods. *Ecosphere*, 9.
- 4 Arino, J. & Van den Driessche, P. (2006). Disease spread in metapopulations. *Nonlinear*
5 *dynamics and evolution equations*, 48, 1–13.
- 6 Badham, J. & Stocker, R. (2010). The impact of network clustering and assortativity on
7 epidemic behaviour. *Theoretical population biology*, 77, 71–75.
- 8 Baguette, M. & Van Dyck, H. (2007). Landscape connectivity and animal behavior: functional
9 grain as a key determinant for dispersal. *Landscape ecology*, 22, 1117–1129.
- 10 Baranyi, G., Saura, S., Podani, J. & Jordán, F. (2011). Contribution of habitat patches to
11 network connectivity: redundancy and uniqueness of topological indices. *Ecological*
12 *Indicators*, 11, 1301–1310.
- 13 Beckett, S.J. (2016). Improved community detection in weighted bipartite networks. *Royal*
14 *Society open science*, 3, 140536.
- 15 Bode, M., Burrage, K. & Possingham, H.P. (2008). Using complex network metrics to predict
16 the persistence of metapopulations with asymmetric connectivity patterns. *ecological*
17 *modelling*, 214, 201–209.
- 18 Bodin, Ö. & Saura, S. (2010). Ranking individual habitat patches as connectivity providers:
19 integrating network analysis and patch removal experiments. *Ecological Modelling*,
20 221, 2393–2405.
- 21 Burnham, K.P. & Anderson, D.R. (2003). *Model selection and multimodel inference: a*
22 *practical information-theoretic approach*. Springer Science & Business Media.
- 23 Christley, R., Pinchbeck, G., Bowers, R., Clancy, D., French, N., Bennett, R., *et al.* (2005).
24 Infection in Social Networks: Using Network Analysis to Identify High-Risk
25 Individuals. *Am J Epidemiol*, 162, 1024–1031.
- 26 Courchamp, F., Berec, L. & Gascoigne, J. (2008). *Allee effects in ecology and conservation*.
27 Oxford University Press.
- 28 Csardi, G. & Nepusz, T. (2006). The igraph software package for complex network research.
29 *InterJournal, Complex Systems*, 1695, 1–9.
- 30 Cushing, J.M., Costantino, R.F., Dennis, B., Desharnais, R. & Henson, S.M. (2002). *Chaos in*
31 *ecology: experimental nonlinear dynamics*. Elsevier.
- 32 Da Silva, R.A.P., Viana, M.P. & da Fontoura Costa, L. (2012). Predicting epidemic outbreak
33 from individual features of the spreaders. *Journal of Statistical Mechanics: Theory and*
34 *Experiment*, 2012, P07005.
- 35 Dormann, C.F. & Strauss, R. (2014). A method for detecting modules in quantitative bipartite
36 networks. *Methods in Ecology and Evolution*, 5, 90–98.
- 37 Drake, J.M., Shapiro, J. & Griffen, B.D. (2011). Experimental demonstration of a two-phase
38 population extinction hazard. *Journal of the Royal Society Interface*, 8, 1472–1479.
- 39 Drechsler, M. (2009). Predicting metapopulation lifetime from macroscopic network
40 properties. *Mathematical biosciences*, 218, 59–71.
- 41 Ferrari, J.R. & Lookingbill, T.R. (2009). Initial conditions and their effect on invasion velocity
42 across heterogeneous landscapes. *Biological Invasions*, 11, 1247–1258.
- 43 Fletcher Jr, R.J., Revell, A., Reichert, B.E., Kitchens, W.M., Dixon, J.D. & Austin, J.D. (2013).
44 Network modularity reveals critical scales for connectivity in ecology and evolution.
45 *Nature communications*, 4, 2572.
- 46 Floerl, O., Inglis, G., Dey, K. & Smith, A. (2009). The importance of transport hubs in stepping-
47 stone invasions. *Journal of Applied Ecology*, 46, 37–45.
- 48 Freeman, L.C. (1978). Centrality in social networks conceptual clarification. *Social networks*,
49 1, 215–239.

- 50 Fronhofer, E.A. & Altermatt, F. (2015). Eco-evolutionary feedbacks during experimental range
51 expansions. *Nature communications*, 6, 6844.
- 52 Gause, G.F. (1934). *The Struggle for Existence*. Williams & Wilkins, Baltimore.
- 53 Gilarranz, L.J. & Bascompte, J. (2012). Spatial network structure and metapopulation
54 persistence. *Journal of Theoretical Biology*, 297, 11–16.
- 55 Gilarranz, L.J., Rayfield, B., Liñán-Cembrano, G., Bascompte, J. & Gonzalez, A. (2017).
56 Effects of network modularity on the spread of perturbation impact in experimental
57 metapopulations. *Science*, 357, 199–201.
- 58 Gómez, J.M. & Verdú, M. (2017). Network theory may explain the vulnerability of medieval
59 human settlements to the Black Death pandemic. *Scientific reports*, 7, 43467.
- 60 Gregory, S.D., Bradshaw, C.J., Brook, B.W. & Courchamp, F. (2010). Limited evidence for the
61 demographic Allee effect from numerous species across taxa. *Ecology*, 91, 2151–2161.
- 62 Johnson, D.M., Liebhold, A.M., Tobin, P.C. & Bjørnstad, O.N. (2006). Allee effects and pulsed
63 invasion by the gypsy moth. *Nature*, 444, 361.
- 64 Jordán, F., Báldi, A., Orci, K.-M., Racz, I. & Varga, Z. (2003). Characterizing the importance
65 of habitat patches and corridors in maintaining the landscape connectivity of a
66 *Pholidoptera transsylvanica* (Orthoptera) metapopulation. *Landscape Ecology*, 18, 83–
67 92.
- 68 Kanarek, A.R., Webb, C.T., Barfield, M. & Holt, R.D. (2013). Allee effects, aggregation, and
69 invasion success. *Theoretical ecology*, 6, 153–164.
- 70 Kean, J. & Barlow, N. (2000). Effects of dispersal on local population increase. *Ecology letters*,
71 3, 479–482.
- 72 Keeling, M. (2005). The implications of network structure for epidemic dynamics. *Theoretical*
73 *population biology*, 67, 1–8.
- 74 Keitt, T.H., Lewis, M.A. & Holt, R.D. (2001). Allee effects, invasion pinning, and species’
75 borders. *The American Naturalist*, 157, 203–216.
- 76 Kermack, W.O. & McKendrick, A.G. (1927). A Contribution to the Mathematical Theory of
77 Epidemics. *Proceedings of the Royal Society of London A: Mathematical, Physical and*
78 *Engineering Sciences*, 115, 700–721.
- 79 Kininmonth, S., Drechsler, M., Johst, K. & Possingham, H. (2010). Metapopulation mean life
80 time within complex networks. *Marine Ecology Progress Series*, 417, 139–149.
- 81 Kramer, A.M., Dennis, B., Liebhold, A.M. & Drake, J.M. (2009). The evidence for Allee
82 effects. *Population Ecology*, 51, 341.
- 83 Lande, R., Engen, S. & Saether, B. (2003). *Stochastic population models in ecology and*
84 *conservation*. Oxford University Press, Oxford.
- 85 Lewis, M. & Kareiva, P. (1993). Allee dynamics and the spread of invading organisms.
86 *Theoretical Population Biology*, 43, 141–158.
- 87 Lurgi, M., Galiana, N., López, B.C., Joppa, L.N. & Montoya, J.M. (2014). Network complexity
88 and species traits mediate the effects of biological invasions on dynamic food webs.
89 *Frontiers in Ecology and Evolution*, 2, 36.
- 90 Melbourne, B.A. & Hastings, A. (2009). Highly variable spread rates in replicated biological
91 invasions: fundamental limits to predictability. *Science*, 325, 1536–1539.
- 92 Miller, J.C. (2009). Spread of infectious disease through clustered populations. *Journal of the*
93 *Royal Society Interface*, rsif-2008.
- 94 Morel-Journel, T., Girod, P., Mailleret, L., Auguste, A., Blin, A. & Vercken, E. (2016a). The
95 highs and lows of dispersal: how connectivity and initial population size jointly shape
96 establishment dynamics in discrete landscapes. *Oikos*, 125, 769–777.
- 97 Morel-Journel, T., Piponiot, C., Vercken, E. & Mailleret, L. (2016b). Evidence for an optimal
98 level of connectivity for establishment and colonization. *Biol Lett*, 12.

- 99 Newman, M.E. & Girvan, M. (2004). Finding and evaluating community structure in networks.
100 *Physical review E*, 69, 026113.
- 101 Ochocki, B.M. & Miller, T.E. (2017). Rapid evolution of dispersal ability makes biological
102 invasions faster and more variable. *Nature communications*, 8, 14315.
- 103 Peck, S.L. (2012). Networks of habitat patches in tsetse fly control: Implications of
104 metapopulation structure on assessing local extinction probabilities. *Ecological*
105 *modelling*, 246, 99–102.
- 106 Peterman, W.E., Ousterhout, B.H., Anderson, T.L., Drake, D.L., Semlitsch, R.D. & Eggert,
107 L.S. (2016). Assessing modularity in genetic networks to manage spatially structured
108 metapopulations. *Ecosphere*, 7, e01231.
- 109 R CoreTeam. (2018). *R: A language and environment for statistical computing*. R Foundation
110 for Statistical Computing, Vienna, Austria. 2018.
- 111 Robinet, C., Lance, D.R., Thorpe, K.W., Onufrieva, K.S., Tobin, P.C. & Liebhold, A.M. (2008).
112 Dispersion in time and space affect mating success and Allee effects in invading gypsy
113 moth populations. *Journal of Animal Ecology*, 77, 966–973.
- 114 Romano, V., Duboscq, J., Sarabian, C., Thomas, E., Sueur, C. & MacIntosh, A.J. (2016).
115 Modeling infection transmission in primate networks to predict centrality-based risk.
116 *American journal of primatology*, 78, 767–779.
- 117 Romanuk, T.N., Zhou, Y., Brose, U., Berlow, E.L., Williams, R.J. & Martinez, N.D. (2009).
118 Predicting invasion success in complex ecological networks. *Philosophical*
119 *Transactions of the Royal Society B: Biological Sciences*, 364, 1743–1754.
- 120 Roques, L., Garnier, J., Hamel, F. & Klein, E.K. (2012). Allee effect promotes diversity in
121 traveling waves of colonization. *Proceedings of the National Academy of Sciences*, 109,
122 8828–8833.
- 123 Schick, R.S. & Lindley, S.T. (2007). Directed connectivity among fish populations in a riverine
124 network. *Journal of applied ecology*, 44, 1116–1126.
- 125 Seymour, M. & Altermatt, F. (2014). Active colonization dynamics and diversity patterns are
126 influenced by dendritic network connectivity and species interactions. *Ecology and*
127 *evolution*, 4, 1243–1254.
- 128 Shtilerman, E. & Stone, L. (2015). The effects of connectivity on metapopulation persistence:
129 network symmetry and degree correlations. *Proc. R. Soc. B*, 282, 20150203.
- 130 Simberloff, D. (2009). The role of propagule pressure in biological invasions. *Annual Review*
131 *of Ecology, Evolution, and Systematics*, 40, 81–102.
- 132 Simberloff, D., Martin, J.-L., Genovesi, P., Maris, V., Wardle, D.A., Aronson, J., *et al.* (2013).
133 Impacts of biological invasions: what’s what and the way forward. *Trends in ecology &*
134 *evolution*, 28, 58–66.
- 135 Sullivan, L.L., Li, B., Miller, T.E., Neubert, M.G. & Shaw, A.K. (2017). Density dependence
136 in demography and dispersal generates fluctuating invasion speeds. *Proceedings of the*
137 *National Academy of Sciences*, 114, 5053–5058.
- 138 Suzuki, Y., Tsuji, H. & Sasakawa, M. (1984). Sex allocation and effects of superparasitism on
139 secondary sex ratios in the gregarious parasitoid, *Trichogramma chilonis*
140 (Hymenoptera: Trichogrammatidae). *Animal Behaviour*, 32, 478–484.
- 141 Tanaka, G., Urabe, C. & Aihara, K. (2014). Random and targeted interventions for epidemic
142 control in metapopulation models. *Scientific reports*, 4, 5522.
- 143 Ueno, T. & Masuda, N. (2008). Controlling nosocomial infection based on structure of hospital
144 social networks. *Journal of Theoretical Biology*, 254, 655–666.
- 145 Vercken, E., Kramer, A., Tobin, P. & Drake, J. (2011). Critical patch size generated by Allee
146 effect in gypsy moth, *Lymantria dispar* (L.). *Ecology letters*, 14, 179–186.

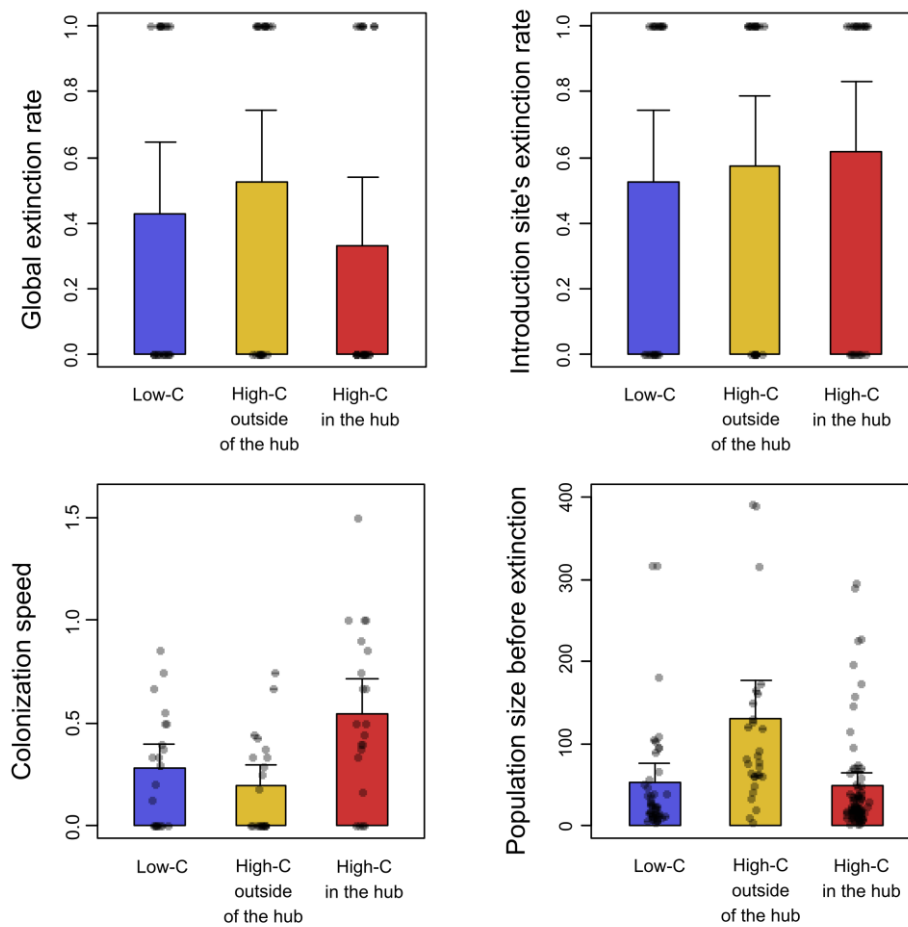
- 147 Watson, J.R., Siegel, D.A., Kendall, B.E., Mitarai, S., Rassweiller, A. & Gaines, S.D. (2011).
148 Identifying critical regions in small-world marine metapopulations. *Proceedings of the*
149 *National Academy of Sciences*, 108, E907–E913.
- 150 Watts, D.J. & Strogatz, S.H. (1998). Collective dynamics of “small-world” networks. *nature*,
151 393, 440.
- 152 Webb, J.A. & Padgham, M. (2013). How does network structure and complexity in river
153 systems affect population abundance and persistence? *Limnologica-Ecology and*
154 *Management of Inland Waters*, 43, 399–403.
- 155 Weiss-Lehman, C., Hufbauer, R.A. & Melbourne, B.A. (2017). Rapid trait evolution drives
156 increased speed and variance in experimental range expansions. *Nature*
157 *communications*, 8, 14303.

1 Figures



2

3 **Figure 1:** Estimated impacts of centralization (A, C, E) and modularity (B, D, F) on the global
4 extinction rate (A, B), introduction site's extinction rate (D, E) and colonization speeds (E, F),
5 with their 95% confidence intervals), for $a = 0$ (blue) and $a = 2.5$ (red) and for an introduction
6 in the hub (full lines, dark) and outside the hub (dashed lines, light).



1

2 **Figure 2:** Mean experimental values for global extinction rates (A), the introduction site's
 3 extinction rates (B), colonization speeds (C) and populations size before their extinction (D),
 4 for the experimental introductions in low-C landscapes (blue), high-C landscapes outside of the
 5 hub (yellow), and high-C landscapes in the hub (red). The error bars represent 2 times the
 6 standard deviation from the mean value and the dots represent the raw values

1 **Tables**

2 **Table 1:** Differences in AIC between the M-model (Δ_C) or the C-model (Δ_M) and the CM-model
 3 for the simulation results. All the values greater than 10 (indicating unequivocal higher support
 4 for the CM- model compared to the other one) are in bold.

5

Response variable	Allee effect	Intro. site	Δ_C	Δ_M
Global extinction rate	$a = 0$	In the hub	2.02	-1.49
	$a = 0$	Outside	-1.99	0.79
	$a = 2.5$	In the hub	602.37	-0.66
	$a = 2.5$	Outside	-1.82	-0.95
Introduction site extinction rate	$a = 0$	In the hub	173.45	-0.11
	$a = 0$	Outside	-1.31	0.38
	$a = 2.5$	In the hub	1806.50	9.70
	$a = 2.5$	Outside	-0.77	-1.95
Colonization speed	$a = 0$	In the hub	3250.45	865.15
	$a = 0$	Outside	130.50	648.80
	$a = 2.5$	In the hub	1578.84	29.20
	$a = 2.5$	Outside	-1.97	-0.58

6