

Mutualists construct the ecological conditions that trigger the transition from parasitism

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Abstract

The evolution of mutualism between hosts and initially parasitic symbionts represents a major transition in evolution. Although vertical transmission of symbionts during host reproduction and partner control both favour the stability of mutualism, these mechanisms require specifically evolved features that may be absent in the first place. Therefore, the first steps of the transition from parasitism to mutualism may suffer from the cost of mutualism at the organismic level. We hypothesize that spatial structure can lead to the formation of higher selection levels favouring mutualism. This resembles the evolution of altruism, with the additional requirement that the offspring of mutualistic hosts and symbionts must co-occur often enough. Using a spatially explicit agent-based model we demonstrate that, starting from a parasitic system with global dispersal, the joint evolution of mutualistic effort and local dispersal of hosts and symbionts leads to a stable coexistence between parasites and mutualists. The evolution of local dispersal mimics vertical transmission and triggers the formation of mutualistic clusters, counteracting the organismic selection level of parasites that maintain global dispersal. The transition occurs when mutualistic symbionts increase the density of hosts, which strengthens competition between hosts and disfavors hosts inhabiting areas dominated by parasitic symbionts: mutualists construct the ecological conditions that allow their own spread. Therefore, the transition to mutualism may come from an eco-evolutionary feedback loop involving spatially structured population dynamics.

Keywords: mutualism, major transition, spatial structure, dispersal evolution, eco-evolutionary feedbacks, niche construction

Introduction

In their bestseller, [Szathmáry and Smith \(1995\)](#) explored several major transitions in biology, from the origin of chromosomes to the evolution of social groups. Most of these transitions resulted from the formation of a larger entity from smaller entities, smaller entities specializing within the larger ones. Following [Queller \(1997\)](#), [Szathmáry \(2015\)](#) suggested a dichotomy between fraternal transitions arising from a division of labour among closely related units (such as multicellularity) and egalitarian transitions, where phylogenetically distant units come together to complement their functions in a larger unit. Egalitarian transitions are generally achieved through mutualistic symbiosis¹ between a relatively large host and its symbiont ([Bronstein, 2015](#); [Drew et al., 2021](#)) and constitute one of the main sources of new lineages, underlying the origin of the eucaryotic cell and photosynthetic eucaryotes for instance ([Margulis and Sagan, 2002](#)). In many cases symbionts are unicellular microbes which are hosted by large eucaryotes, the whole corresponding to a holobiont ([Gilbert et al., 2012](#); [Bordenstein and Theis, 2015](#)); in other cases symbionts are multicellular organisms physically associated with their host at various degrees (e.g. plant-fungi, plant-ant, plant-seed eating pollinator). While symbionts depend on their host from the start, hosts often become dependent on the symbionts during later stages ([Roughgarden, 1975](#)), e.g. for reproduction or resource acquisition, eventually making the transition irreversible.

For a transition to occur and persist, evolutionary conflicts between the subentities must not overtake the whole's fate. In the case of fraternal transitions, this is prevented by the strong relatedness between subentities ([Hamilton, 1964b,a](#); [Queller, 2000](#); [Fisher et al., 2013](#)). However, in the case of egalitarian transitions, the subentities generally belong to different species. Thus, it can be advantageous for them to remain autonomous and exploit the other subentities. This parasitic behaviour occurs at the expense of the whole, as for the tragedy

Author contributions: SI, JG and LL originally formulated the project; all authors developed the model; LL and MR performed the numerical analyses; LL, SI and JG wrote the manuscript.

¹Symbiosis is used here in its etymological sense of "living together", encompassing parasitic and mutualistic symbiosis.

43 of the commons (Garrett, 1968; Hardin, 1998). For instance, a symbiont may remain parasitic rather than col-
44 laborate with its host (Drew et al., 2021). The resulting evolutionary conflict might be circumvented by vertical
45 transmission of the symbionts, which ensures that all subentities share a common fate (Wilson and Sober, 1994).
46 As a result, vertical transmission of symbionts indeed promotes the transition to mutualism (Smith, 1998; Herre
47 et al., 1999; Wilkinson and Sherratt, 2001; Ferdy and Godelle, 2005; Kerr and Nahum, 2011; Akçay, 2015; Es-
48 trela et al., 2016; Queller and Strassmann, 2016; Doebeli and Knowlton, 1998), although symbionts vertically
49 transmitted can persist without becoming mutualists (Saikkonen et al., 2002).

51 The importance of vertical transmission has been highlighted by experiments on microbial systems (Sachs
52 et al., 2011; Shapiro et al., 2016; King et al., 2016; Shapiro and Turner, 2018) as well as *in natura* observa-
53 tions of a Wolbachia-insect system (Weeks et al., 2007). However, in many mutualistic systems, the symbiont is
54 transmitted horizontally (Wilkinson and Sherratt, 2001), such as legume-rhizobium (Denison and Kiers, 2004),
55 squid-vibrio (McFall-Ngai, 2014), mycorrhizae (Allen, 1991), endophytes (Saikkonen et al., 2004) or plant-ants
56 (Bronstein et al., 2006; Rico-Gray and Oliveira, 2008). In such cases, several mechanisms such as partner choice,
57 sanction or fidelity can counteract the selection for selfishness (Genkai-Kato and Yamamura, 1999; Wilkinson and
58 Sherratt, 2001; Sachs et al., 2004; Foster and Wenseleers, 2006; Estrela et al., 2016; Akçay, 2017; Sachs et al.,
59 2010b). For instance, in legume-rhizobium, mycorrhizal and plant-ant associations, the plants can sanction the
60 less beneficial (or even detrimental) symbionts by allocating them fewer resources (West et al., 2002; Kiers et al.,
61 2003; Denison and Kiers, 2004; Edwards et al., 2006; Bever et al., 2009; Akçay, 2015). However, it is unclear
62 whether these mechanisms are present at the beginning of the transition to mutualism. Since they require the
63 evolution of complex and specific traits, they may occur in later stages, providing additional stability to the
64 system. In the absence of such traits, what mechanism could promote the transition in the first place? Using a
65 theoretical model, the present work aims to show that the joint evolution between mutualistic effort and local
66 dispersal of hosts and symbionts leads to a positive association between mutualistic hosts and symbionts and
67 subsequently triggers the formation of mutualistic clusters .

69 A similar issue exists with respect to the evolution of altruism², since partner choice and control mechanisms,
70 such as voluntary reciprocal altruism (Axelrod, 1981), may be restricted to higher animals or may appear during
71 later evolutionary stages. In line with the intuition of Darwin (1871), spatial structure has been recognized as
72 a general mechanism promoting the transition to altruism (Mitteldorf and Wilson, 2000; Lion and Van Baalen,
73 2007, 2008; Débarre et al., 2012). Spatial structure generally stems from local dispersal, which triggers the
74 formation of clusters dominated by altruistic organisms, while organisms with similar phenotypes are positively
75 assorted in space (Wilson and Dugatkin, 1997; Pepper, 2007). The balance between organismic-level selection
76 favouring cheaters and cluster-level selection favouring altruists ultimately determines the evolutionary outcome
77 (Van Baalen and Rand, 1998; Mitteldorf and Wilson, 2000). Moreover, the joint evolution of cooperation and
78 dispersal can allow the emergence of altruism, with spatial clusters of altruistic organisms promoting the persis-
79 tence and spread of altruistic phenotypes (Koella, 2000; Le Galliard et al., 2005; Hochberg et al., 2008; Purcell
80 et al., 2012; Mullon et al., 2018). Empirical evidence on the evolution of reduced virulence (Boots and Meador,
81 2007; Szilágyi et al., 2009), the evolution of altruism (Harcombe, 2010), and the evolution of restraint predation
82 (Kerr et al., 2006) also supports the crucial role of the spatial structure.

84 Similarly, spatial structure can allow mutualists to overcome non-mutualists (Yamamura et al., 2004; Akçay,
85 2017; Doebeli and Knowlton, 1998; Frank, 1994), and this can come along with the evolution of dispersal (Mack,
86 2012). However, this may not be sufficient to account for the transition from parasitism to mutualism, since
87 parasitic symbionts should discourage hosts from initiating the transition, whereas non-mutualists have a weaker
88 impact (Yamamura et al., 2004; Mack, 2012). In the case of holobionts, starting from free living bacteria,
89 Sachs et al. (2011) documented 27 transitions towards parasitism, 9 directly towards mutualism and 3 towards
90 commensalism, whereas the transition from parasitism to mutualism occurred only 3 times. This highlights that
91 the transition from parasitism to mutualism, although feasible, is relatively infrequent, and calls for a theoretical
92 understanding of the mechanisms involved. Moreover, in previous attempts mutualistic efforts were initially
93 polymorphic but were not subject to mutations (Mack, 2012). In that case, mutualistic clusters cannot be invaded
94 from inside through parasitic mutations, which favours mutualism. The present work therefore constitutes, to
95 our knowledge, the first spatially explicit eco-evolutionary model where the mutualistic efforts and dispersal
96 abilities of hosts and symbionts coevolve, beginning from a parasitic interaction. If some hosts and symbionts
97 simultaneously become mutualists and start dispersing locally, this may lead to the formation of mutualistic
98 host-symbiont clusters producing more offspring than in areas where hosts are mainly associated with parasitic
99 symbionts, thereby initiating the transition. Meanwhile, parasitic symbionts should continue dispersing globally
100 and invade the mutualistic clusters, which could homogenize the spatial structure and compromise the transition.
101 Also, densely populated mutualistic clusters might suffer from intraspecific competition between hosts, unless
102 competition acts on a large spatial scale. In sum, it is unclear whether mutualists will invade, whether mutualists
103 will replace parasites, or whether both strategies will coexist, as is often the case in nature (e.g. Després and
104 Jaeger, 1999; Borges, 2015; Saikkonen et al., 2004).

²An altruistic trait benefits conspecifics, at a cost to its bearer. In contrast, a mutualistic trait benefits heterospecifics.

105 The concept of major transitions also implies that the host and the symbiont become dependent upon each
 106 other (Szathmary and Smith, 1995; Szathmary, 2015), with each partner needing the other to perform essential
 107 functions like nutrient provisioning (Fisher et al., 2017). Dependence is often accompanied by gene loss and
 108 gene exchange, rendering the transition irreversible (Estrela et al., 2016). Most symbionts cannot live freely and
 109 therefore completely depend on their host, but most hosts can complete their life cycle without their symbiont
 110 (e.g., in plant-ant, plant-fungi or legume-rhizobium mutualisms) and several reverse pathways are possible from
 111 mutualism to parasitism (Sachs and Simms, 2006; Werner et al., 2018; Week and Nuismer, 2021). However in
 112 some cases hosts depend on their symbiont, for instance the intracellular bacterial symbiont *Buchnera aphidicola*
 113 provides essential amino acids to its aphid host (Akman Gunduz and Douglas, 2009; Bennett and Moran, 2015).
 114 Since the present work focuses on the transition and not on later stages, we will not assume that hosts depend on
 115 their symbionts for their physiology or development, which would render the transition irreversible *by construction*.
 116 Instead, hosts will always be able to produce offspring when alone. Nevertheless, the number of offspring produced
 117 by the hosts will depend on the mutualistic efforts of both species as well as on the population densities, which are
 118 expected to change during the transition. Under these altered ecological conditions, isolated hosts may exhibit a
 119 negative population growth rate, although they are physiologically able to produce offspring. This mechanism is
 120 hereafter called *ecological dependence*.

121 To sum up, we will tackle the following issues:

- 122 • **Main hypothesis:** In the absence of vertical transmission and partner control, we expect that the tran-
 123 sition from parasitism to mutualism can occur when the mutualistic efforts of both hosts and symbionts
 124 jointly evolve with local dispersal.
- 125 • **H1:** The formation of mutualistic clusters should be necessary for the initiation of the transition. The
 126 emergence of spatial structure should come along with the transition.
- 127 • **H2:** By maintaining global dispersal, non-mutualistic hosts and parasitic symbionts should be able to
 128 coexist with mutualists.
- 129 • **H3:** The transition to mutualism is due to the relatively higher fecundity of mutualistic clusters.
- 130 • **H4:** If competition between hosts is mostly local, this should hamper the formation of mutualistic clusters,
 131 thereby preventing the transition.
- 132 • **H5:** We expect that mutualistic hosts will become ecologically dependent on their symbiont.

133 To investigate these hypothesis, we built an agent-based model using a two-dimensional space lattice that
 134 supports an autonomous host and a host-dependent symbiont. Hosts compete for space and other resources, while
 135 symbionts compete for available hosts. This situation occurs in many biological systems, such as plant-fungi,
 136 plant-seed eating pollinator, plant-ant, and multicellular eucaryotes hosting bacteria. Less intimate associations
 137 like cleaning mutualisms or plant-pollinator interactions may also fit, provided that the animal is specialized
 138 and dependent on its host. To model the transition from parasitism, the symbiont is initially detrimental to
 139 the host, and the host provides it the minimal energy possible without any spontaneous mutualistic effort, as
 140 would be the case after an antagonistic evolutionary arms race. Moreover, the host-parasite system is ecologically
 141 viable even in the absence of any mutualistic agent in the landscape. At first, both species disperse globally;
 142 this situation corresponds to the most disadvantageous conditions for the emergence of mutualism. Through
 143 continuous mutations, mutualistic and locally dispersing symbionts and hosts can appear. The mutualistic effort
 144 encompass the provision of resources, shelter, immunity, anti-predator behaviours, digestive enzymes or any other
 145 type of benefit provided that this occurs at some cost. If mutualistic symbionts manage to persist for a while,
 146 they eventually change the population dynamics, triggering feedback on their own evolutionary dynamics. In
 147 addition to these general hypotheses, no assumptions specific to a particular biological system were required.

148 Model description

149 **Main rules** Our model considers two types of agents, hosts and symbionts, living on the same two-dimensional
 150 space lattice. The interaction between the two species occurs when they share the same cell. Each cell can
 151 assume three states: i) empty, ii) occupied by a solitary host, with only one host per cell), iii) occupied by a host-
 152 symbiont couple, with only one symbiont per host (but see Appendix A.7 for a relaxation of this assumption).
 153 Each organism bears two traits, an interaction trait α and a dispersal trait ε , which both influence fecundity. At
 154 every time point, agents undergo the following steps (see appendix A.1 and Figure A1 for more details):

- 155 • The host and symbiont die with fixed probability m .
- 156 • They produce offspring, possibly with mutations. The average offspring number of a parent depends on its
 157 traits and on its interactions with their cell-sharing partner, if any.
- 158 • The offspring are dispersed according to the parental trait ε .
- 159 • The host offspring may establish only in empty cells, while the symbiont offspring can only establish in
 160 cells already occupied by a solitary host. If several organisms come to implant in the same cell, a uniform
 161 lottery determines which one will implant, while the others die.

162 **Fecundity and mutualism/parasitism** Each agent produces offspring according to a Poisson distribution
 163 with parameter f , which corresponds to its fecundity. The fecundity defines the average number of offspring per
 164 agent. It results from an interaction fecundity positively dependent on the trait of its cell-sharing partner and a
 165 mutualistic cost negatively dependent on its trait.

Specifically, the fecundity of a symbiont f^s of trait α_s in interaction with a host of trait α_h is defined by:

$$f^s(\alpha_s, \alpha_h) = (1 - c_m \alpha_s)(f_{min}^s + (f_{max} - f_{min}^s)\alpha_h) \quad (1)$$

where c_m is the maximal mutualistic cost and f_{min}^s and f_{max} are the minimal and maximal interaction fecundity of symbionts. Similarly, the fecundity of a host f^h of trait α_h in interaction with a symbiont of trait α_s is defined by:

$$f^h(\alpha_h, \alpha_s) = (1 - c_m \alpha_h)(f_{min}^h + (f_{max} - f_{min}^h)\alpha_s^{\gamma_f}) \quad (2)$$

166 where f_{min}^h is the minimal interaction fecundity of hosts and γ_f describes the selection strength on the trait α_s
 167 – the selection strength on α_h is set to $\gamma_s = 1$.

Since hosts are autonomous, in absence of symbionts, their fecundity f^{ha} only depends on their trait α_h :

$$f^{ha}(\alpha_h) = (1 - c_m \alpha_h)f^a \quad (3)$$

168 where the fecundity alone f^a ranges between the minimal and maximal interaction fecundity: $f_{min}^h < f^a < f_{max}$.
 169 As a result, the establishment of a symbiont with a low interaction trait ($\alpha_s < \alpha_s^*$) reduces the fecundity of the
 170 host; the symbiont is parasitic. Instead, a symbiont with a large interaction trait ($\alpha_s > \alpha_s^*$) enhances the host's
 171 fecundity; the symbiont is mutualistic. The threshold α_s^* is defined by $f^h(\alpha_h, \alpha_s^*) = f^{ha}(\alpha_h)$ (see appendix A.1
 172 for mathematical derivation of the threshold). In the simulations, $\alpha_s^* = 0.475$ (Figure 1).

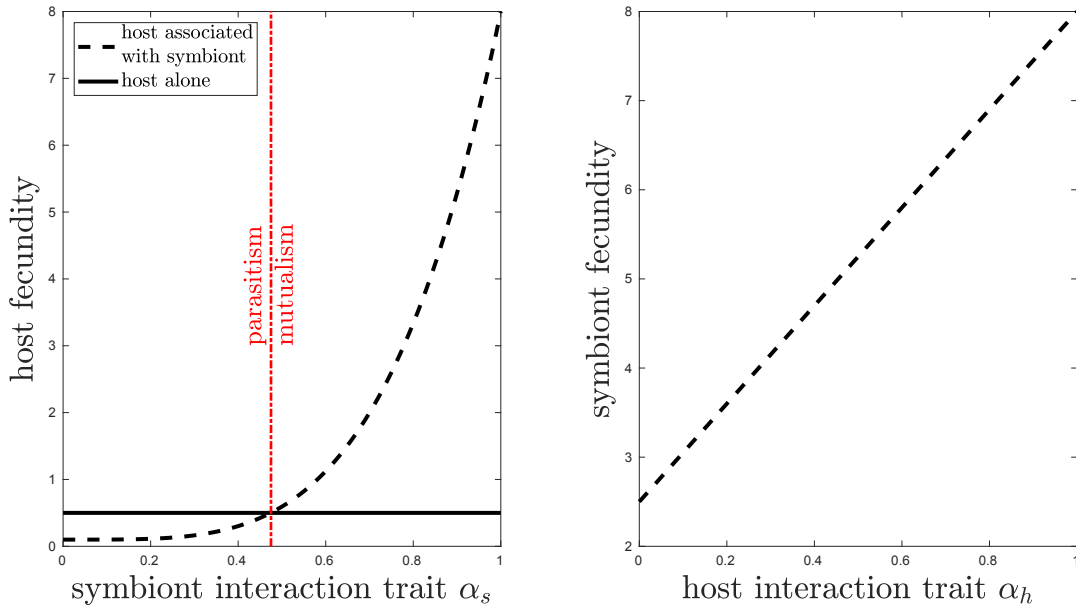


Figure 1: Fecundity of hosts f^h and symbionts f^s according to the interaction trait of their partners (dashed black lines). Plain black line corresponds to the fecundity of a solitary host f^a . The dashed red line corresponds to the threshold $\alpha_s^* = 0.475$ separates symbionts, which either reduce or enhance the fecundity or their host (appendix A.1).

173 **Mutation** Offspring inherit traits from their parents with variability due to mutations. The effects of muta-
 174 tions on each trait are independent. However, the distribution of mutation effects does depend on the trait of
 175 the parents. We use a Beta distribution with shape parameters (1, 3) to describe the amplitude of these effects,
 176 which could be either beneficial or detrimental. This mutation kernel allows for rare mutations with large effects.
 177 However, these effects can not exceed a maximal mutation size set to $\beta_{max} = 0.5$ (see Figure A3 in appendix A.1
 178 for details).

179 **Dispersal** The parents do not disperse, while their descendants disperse either locally in one of the 8 cells
 180 around the parent or globally, with a uniform distribution across the entire space (see Figure A5 for a sketch
 181 of the process). The dispersal trait ε is defined as the proportion of offspring dispersed globally, as in Kéfi
 182 et al. (2007, 2008). These two modes of dispersal correspond to a mixture of short and long distance dispersal

183 events. For instance, fleshy fruits may be dispersed either by small birds having a short-distance behaviour, or
 184 by mammals and large birds which disperse the seeds at long distances (Jordano et al., 2007). Fruits may also
 185 remain unconsumed and fall locally. Depending on the fruit's traits, its propensity to be consumed by either
 186 type of frugivores may vary among organisms, which is captured by the dispersal trait ε . Since the investment in
 187 global dispersal may reduce fecundity (Harada, 1999; Bonte et al., 2012), we assumed a linear trade-off between
 188 fecundity and dispersal: $f_e = (1 - d\varepsilon)f$, with f_e the effective fecundity and d the dispersal cost intensity, which
 189 is the same for both hosts and symbionts.

Competition Hosts compete for empty cells, especially if they disperse locally. Beside space, hosts may also
 compete with each other for resources like water, light or food. In order to test hypothesis H3 we introduced
 intraspecific density-dependent competition, acting either at the local or the global scale. For instance, compe-
 tition for light only involves the closest neighbors while competition for the water table might act at the entire
 space scale. The competition scale parameter w_h , ranging in $[0, 1]$, weights the effect of the local density ρ_h^{local}
 and the global density ρ_h^{global} of host on the competition. Competition reduces the establishment probability P_I
 of the offspring:

$$P_I = 1 - \left((1 - w_h)\rho_h^{local} + w_h\rho_h^{global} \right)^{\gamma_C} \quad (4)$$

191 The local host density ρ_h^{local} corresponds to the host density in the 8 neighbouring cells surrounding the offspring,
 192 while the global density ρ_h^{global} corresponds to the host density over the entire landscape (see Figure A2 for a
 193 schematic representation). The parameter γ_C corresponds to the competition strength. The competition is strong
 194 when $\gamma_C < 1$ (sub-linear function), while it is weak when $\gamma_C \geq 1$ (super-linear function).

Parameters		Reference values	Sensitivity analysis range
m	probability of mortality	0.06	[0.005; 0.15]
c_m	maximum mutualistic cost	0.3	[0; 1]
f_{max}	maximal host and symbiont interaction fecundity	8	fixed
f_{min}^h	minimal host interaction fecundity	0.1	fixed
f_{min}^s	minimal symbiont interaction fecundity	2.5	fixed
f^a	maximal solitary host fecundity	0.5	fixed
γ_f	selection strength on the symbiont interaction trait	4	fixed
β_{max}	maximum mutation size	0.5	[0.1; 1]
w_h	scale of host competition	1	0 or 1
γ_c	strength of host competition	0.2	[0.1; 2]
d	dispersal cost	0	[0; 1]

Table 1: List of parameters and their reference values used for the simulations. The parameters of host and symbiont fecundities are determined to ensure the viability of the antagonistic system, therefore they are fixed because they are constitutive of the model.

195 **Parasitic system and transition** To tackle the issue of transition to mutualism, we assume that the sys-
 196 tem is viable without mutualism (see appendix A.2 for details). More precisely, in the absence of mutation, the
 197 extinction probability of a population with parasitic global dispersers without dispersal cost (minimal interaction
 198 traits, $\alpha_s = \alpha_h = 0$, dispersal trait $\varepsilon = 1$ and dispersal cost $d = 0$) is given by $m/((1 - m)f^a)$ where m is the
 199 mortality rate and f^a the fecundity of a parasitic host without symbiont.. We choose parameters such that the ex-
 200 tinction probability is less than 1. In this case, the population stabilizes around a demographic equilibrium called
 201 the "parasitic system" where host density is around 0.15 and the symbiont density is around 0.1 with parameters
 202 value set in Table 1 (see Figure 2b-c and appendix A.2). From our perspective, this situation is the worst-case
 203 scenario because interactions are parasitic and dispersal cost is minimal. Then, mutualistic symbionts can appear
 204 by mutation, which generates approximately 2% of mutualistic symbionts in the population (see dashed purple
 205 curve in Figure 2c and Figure A8d). Natural selection eventually leads to a significant increase of the percentage
 206 of mutualistic symbionts, far above the 2% generated by mutations (Figure 2). Using an approximation model,
 207 we show that the extinction probability of mutualistic symbionts falls below 1 when the percentage of symbiont
 208 rises above 10% (appendix A.2 for details). In the simulations, a high density of mutualistic symbionts indeed
 209 persists in the long term when the percentage of mutualistic symbionts stands above 10% (Figure A9), which
 210 therefore characterizes the transition to mutualism. The transition time was defined as the time at which the
 211 percentage of mutualistic symbionts rises above this threshold.

213 **Assortment and aggregation indices** To investigate the spatial structure, which comes along with the
214 transition to mutualism, we compute assortment indices: intraspecific indices measuring the spatial autocorre-
215 lation among hosts and symbionts and an interspecific index quantifying the correlation between phenotypes of
216 host and symbiont sharing the same location. More specifically, the intraspecific indices compute the similarity
217 between the trait of an organism and the traits of its neighbors located in the 8 cells around it, and compare it
218 with the similarity between the organismic trait and the mean trait over the landscape (details in appendix A.1).
219 If the intraspecific index is positive (respectively negative), it means that on average the neighbors of any or-
220 ganism share similar (respectively dissimilar) traits. Similarly, the interspecific index is positive if hosts and
221 symbionts sharing the same cell have similar interaction traits. Spatial aggregation indices for hosts, mutualistic
222 symbionts and parasitic symbionts were also computed, measuring the formation of clusters (appendix A.1 for
223 details).

224 Results

225 In the following, the maximum cost of mutualism c_m is 30%, and the other parameters are set to satisfy the
226 viability of the parasitic system (Table 1 in appendix A.1 and appendix A.2 for a discussion of the effect of the
227 cost of mutualism).

228 **The transition from parasitism to mutualism** Our main objective was to investigate whether the tran-
229 sition to mutualism is possible starting from a viable parasitic system, without dispersal cost, which constitutes
230 the most stringent condition for the transition. In that case, the transition is more likely to occur under strong
231 ($\gamma_C = 0.2$) intraspecific host competition (with frequency 0.95) than under weak ($\gamma_C = 1$) competition (0.086).
232 Moreover, when the transition succeeds, it occurs more rapidly under strong competition (median transition time
233 around $2.5 \cdot 10^4$) than under weak competition (median transition time around $7 \cdot 10^4$, Figure 2a). When the cost
234 of dispersal is large ($d = 0.45$) the transition occurs systematically (with frequency 1) and the median transition
235 time is much lower (around $7 \cdot 10^2$), regardless of the strength of competition (Figure 2a). Dispersal cost was
236 therefore used as an instrumental tool to speed up the transition when necessary.

237
238 The transition begins with weakly mutualistic symbionts, which rapidly increase their mutualistic effort
239 toward 1 (Figure 2c). In contrast, the increase of the average host interaction trait is delayed in response to the
240 symbionts' transition (Figure 2b). Moreover, the transition does not occur at the expense of parasitic symbionts;
241 on the contrary their population density benefit from the increase in host density triggered by the mutualistic
242 symbionts (Figure 2c).

243 Since the symbiont population is monomorphic at the beginning of every simulation, the two distinct pheno-
244 typic clusters visible in Figure 3a indicate that both traits diverged, resulting in two classes of symbionts: parasitic
245 global dispersers ($\alpha_s \ll 1$ and $\varepsilon \sim 1$) and mutualistic local dispersers ($\alpha_s \sim 1$ and $\varepsilon \ll 1$). Furthermore, the
246 mutualistic and dispersal traits of symbionts evolve at the same time, during the transition (details not shown).
247 Conversely the host traits do not diverge; their joint evolution leads to a negative correlation between global
248 dispersal and mutualism intensity (Figure 3b, $R^2=0.102$). After the transition, most hosts provide a non-zero
249 mutualistic effort to the symbiont (most $\alpha_h > 0.2$).

250 The assortment indices indicate that after the transition to mutualism the organisms of both species are locally
251 similar. Moreover, hosts and symbionts sharing the same location also tend to have the same interaction behaviour
252 (Figure 4a). The intraspecific assortment is stronger than the interspecific assortment, which is not surprising
253 since the formation of the intraspecific spatial structure simply requires a sufficient proportion of local dispersal.
254 The aggregation indices (appendix A.1) behave similarly, after the transition the spatial aggregation of hosts,
255 parasitic symbionts and mutualistic symbionts all increase, and the parasitic and the mutualistic symbionts reach
256 the same level of aggregation (Figure A7). These results together indicate that the transition to mutualism comes
257 along with the emergence of a spatial structure, with clusters of mutualistic hosts and symbionts (Figure 4c).

258 **The effect of competition between hosts** Figure 2a shows that the host competition promotes the
259 transition to mutualism; we next investigate its quantitative effect on the percentage of mutualistic symbionts.
260 The following results were obtained using a large dispersal cost ($d = 0.45$) to reduce the mean time of transition
261 and thus save computational time.

262 The competition strength γ_C increases the percentage of mutualistic symbionts after the transition when
263 competition is global, i.e. when hosts compete with all the hosts present in the landscape (Figure 5a). However,
264 the transition can occur even in the absence of host competition, if the cost of mutualism is sufficiently low (e.g.,
265 a maximum cost of only 10% instead of 30% as in previous simulations, details not shown). When competition
266 is more local the percentage of mutualistic symbionts decreases drastically, until it drops below the transition
267 threshold (Figure 5b). In the absence of dispersal cost, when competition is reduced after the transition to
268 mutualism, the system switches back to the parasitic state (Figure 5c, see Figure A10 for details).

269
270 Another way to investigate the effect of competition is to reduce host density, through the eradication of
271 hosts in a region after a transition to mutualism. At first, the perturbed region is mainly recolonized by hosts

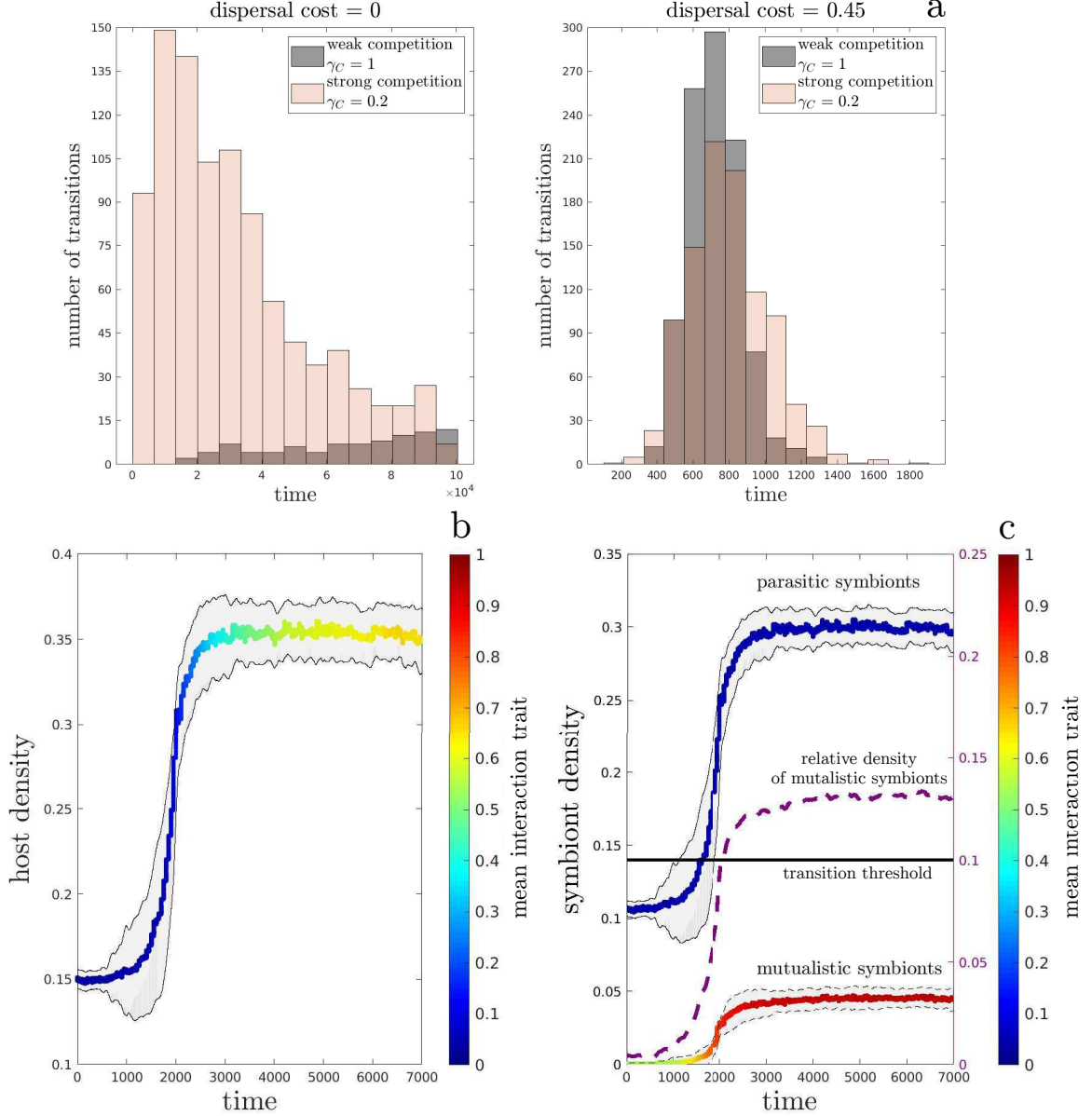


Figure 2: a) Histograms of the number of transitions over 1000 simulations as a function of time, with a maximum projection time of 10^5 . Without dispersal cost there are a total of 86 transitions when the competition is weak and 951 when the competition is strong. With dispersal cost there are 1000 transitions whether the competition is weak or strong. Panels b) and c) represent the host and symbiont densities over time averaged over 100 simulations (coloured plain curves) under strong competition $\gamma_C = 0.2$ and no dispersal cost $d = 0$ and with a maximum projection time of 10^4 steps. The densities correspond to the proportion of occupied cells. The time series are adjusted so that all simulations have a transition time $t = 2000$. The colour gradient corresponds to the mean interaction trait α , and shaded regions correspond to the standard deviation for densities. In panel c), the purple dotted line and the right y-axis show the relative density of mutualistic symbionts, and the black line indicates the 10% transition threshold. For all panels, other parameters are $m = 0.06$, $c_m = 0.3$, $w_h = 1$, $f_{min}^h = 0.1$, $f_{min}^s = 2.5$, $f_{max} = 8$, $f^a = 0.5$ and $\beta_{max} = 0.5$

272 and parasitic symbionts (Figure A11b), but mutualistic symbionts persist in the landscape. Due to the relaxation
 273 of global competition, the probability of host establishment is better, and the mutualistic clusters outside the
 274 perturbation zone gain in size, which explains why the proportion of mutualistic symbionts increases slightly de-
 275 spite the recolonization of the centre by parasites (Figure A11a). In the end, once recolonization is complete, the
 276 system returns to an equilibrium state whose trait distributions are close to distributions before the perturbation

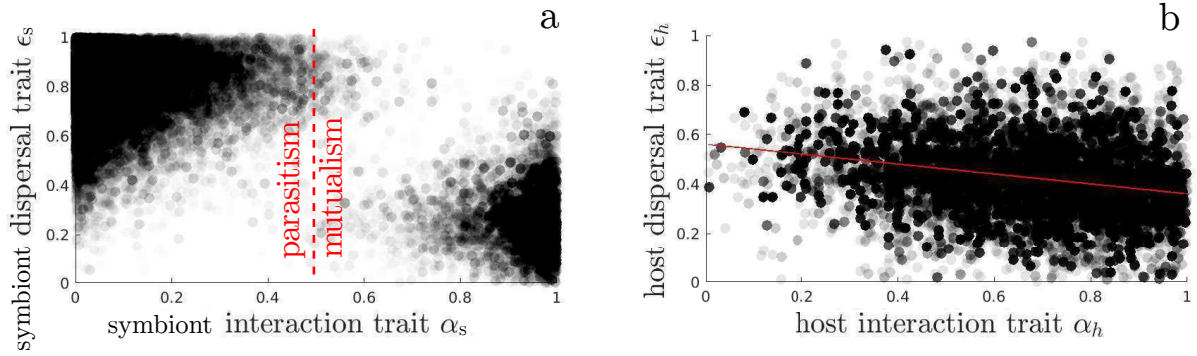


Figure 3: Post transition traits distribution (ϵ, α) of symbionts (panel a) and hosts (panel b). The dashed red line in panel a indicates the threshold $\alpha_s^* = 0.475$ above which a symbiont benefits its host. The plain red line in panel b shows the linear regression between host traits ($R^2=0.102$). Distributions corresponds to 100 simulations with strong competition $\gamma_C = 0.2$, no dispersal cost $d = 0$ and with a maximum projection time of 10^4 steps. Other parameters are $m = 0.06$, $c_m = 0.3$, $w_h = 1$, $f_{min}^h = 0.1$, $f_{min}^s = 2.5$, $f_{max} = 8$, $f^a = 0.5$ and $\beta_{max} = 0.5$.

277 (details not shown). A similar experiment with a perturbation causing the death of 50% of uniformly occupied
 278 cells leads to the same results.

279

280 **Host dependency** Under favourable conditions leading to the transition to mutualism, the population of
 281 mutualistic hosts always persists in the absence of mutualistic symbionts, which excludes any absolute dependency
 282 of hosts for symbionts. However, *ecological dependency* may occur, where isolated hosts may have a negative
 283 growth rate because of intraspecific competition, although they would be able to form stable populations at
 284 lower densities. The transition to mutualism co-occurs with an increase in host density and thus an increase
 285 in intraspecific competition. If this increase in competition is sustainable only in the presence of mutualistic
 286 symbionts, the hosts are *ecologically dependent* on the symbionts. In order to determine the occurrence of
 287 ecological dependency, the intensity of intraspecific competition between hosts was measured in a system at
 288 equilibrium after the transition to mutualism (e.g., at the end of Figure 2c), and subsequently used as a fixed
 289 parameter to test if the host population can now survive in the absence of mutualistic symbionts and mutation.
 290 We found that ecological dependency occurs in the hatched area of Figure 6, when the system evolves toward a
 291 mutualistic system in which the percentage of mutualistic symbionts is sufficiently large.

292 Figure 6 further shows that both dispersal cost and mortality promote mutualism. For the parameter pair
 293 in the area indicated by the grey star, where dispersal cost is zero, Figure 2a showed that the probability of
 294 transition during the 10^5 time steps is only 0.086, with a mean transition time of 7.10^4 . This explains why no
 295 transition occurred in Figure 6, where 50 simulations per parameter combination were performed, with only 10^4
 296 time steps. Finally, Figure 6 also shows that for some parameter combination, mutualism evolves even though
 297 the parasitic system is initially unviable. The viability of the parasitic system was assessed by simulations of
 298 5000 time steps, without evolution. This implies that in a relatively short period of time in comparison to the
 299 transition times shown in Figure 2 for other parameter values, transitions can occur quickly enough and prevent
 300 the extinction of a parasitic system otherwise unviable. However this occurs rarely; Figure A14 shows that for
 301 some parameter combinations up to 90% of the simulations go extinct, the remaining being able to persist thanks
 302 to the evolution of mutualism. In those cases the mean percentage of mutualistic symbionts is much higher,
 303 ranging from 35 to 60%.

304 Discussion

305 The mechanisms underlying the transition to mutualism.

306 In line with our main hypothesis, our results indicate that the transition from parasitism to mutualism occurs
 307 when mutualistic efforts evolve together with dispersal, despite the absence of vertical transmission or partner
 308 control. The following paragraphs review the mechanisms which contribute to the transition, and related them
 309 with the hypothesis formulated earlier.

310 **The formation of clusters.** Before the transition, the host performs better when alone; therefore, it
 311 has no interest in increasing its mutualistic effort and natural selection keeps it as low as possible. In contrast
 312 the symbiont population is limited by the number of available hosts, which increases when the symbiont becomes

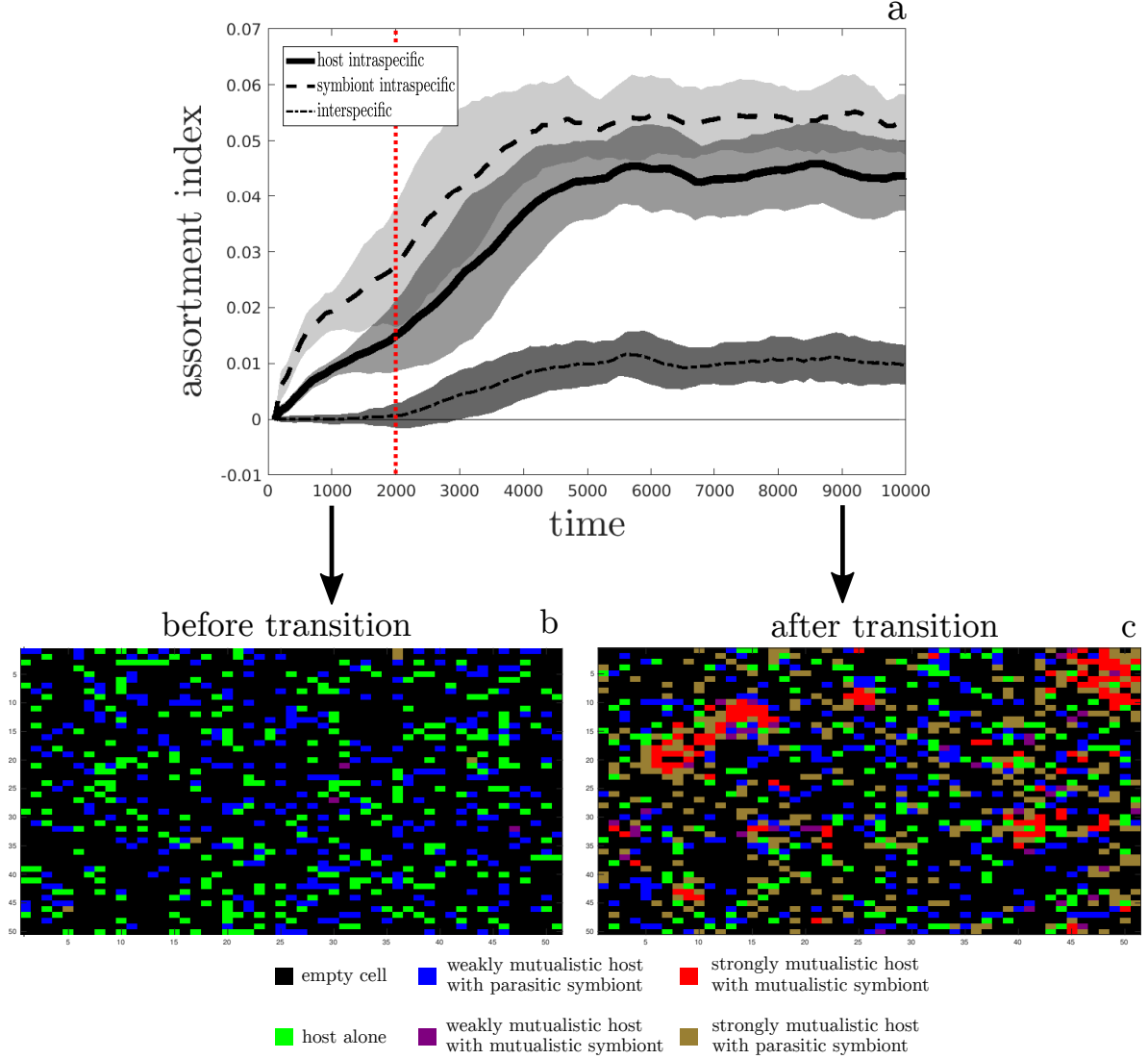


Figure 4: a) Spatial structures are described by the assortment index measuring the intraspecific assortment between hosts (plain line), the intraspecific assortment between symbionts (dashed line) and the interspecific assortment between hosts and symbionts (dash-dot line). Results are averaged over 100 simulations with strong competition $\gamma_C = 0.2$ and no dispersal cost $d = 0$. The time series are adjusted so that all simulations have a transition time $t = 2000$ (red dotted line). Grey areas show the standard deviation. The threshold separating mutualistic and antagonistic symbionts is as in Figures 1 and 3. b)-c) Snapshots of a region of 40x40 cells before (panel b) and after (panel c) the transition to mutualism. For the sake of the figure, a host is considered weakly mutualistic if its interaction trait is less than 0.5 and strongly mutualistic if it is greater than 0.5. Other parameters are $m = 0.06$, $c_m = 0.3$, $w_h = 1$, $f_{min}^h = 0.1$, $f_{min}^s = 2.5$, $f_{max} = 8$, $f^a = 0.5$ and $\beta_{max} = 0.5$

313 mutualistic. Mutualistic symbionts, which help globally dispersing hosts, would be counter-selected. However,
314 in spatially structured populations, rare mutants can interact with each other (Lion and Van Baalen, 2008), so
315 if by chance mutations produce a mutualistic symbiont dispersing locally and interacting with a host dispersing
316 locally as well, its offspring will benefit from the increased density of hosts in their neighbourhood and will form
317 a mutualistic cluster (in line with hypothesis H1, Figures 4c) and A7). The cluster can then be invaded by
318 parasitic symbionts dispersing globally, resulting in a dynamic equilibrium between mutualism and parasitism
319 (in line with hypothesis H2, Figure 2c). Parasitic symbionts become themselves aggregated (Figure A7) since
320 they develop around the mutualistic clusters, at their expense (Figure 4c). Joint evolution between mutualistic
321 effort and dispersal results in a negative correlation between mutualism intensity and global dispersal (80% of
322 mutualists disperses locally, Figure 3), which mirrors the link between altruistic behaviour and local dispersal
323 (Koella, 2000; Le Galliard et al., 2005; Hochberg et al., 2008; Purcell et al., 2012; Mullon et al., 2018) as well as

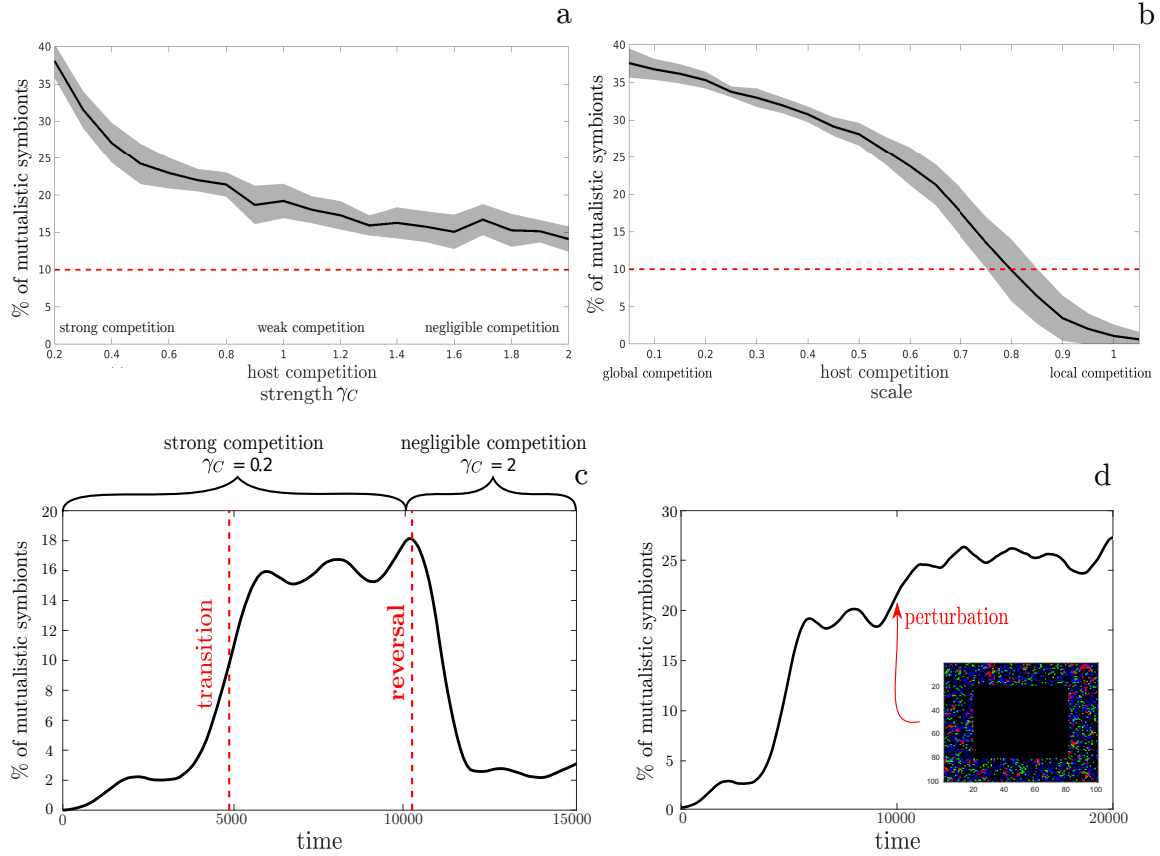


Figure 5: The role of host competition in the transition to mutualism. Effects of competition strength γ_c (panel a) and spatial scale w_h (panel b) on the percentage of mutualistic symbionts with dispersal cost $d = 0.45$ and after 10^4 time steps and over 50 replicates. Black curves is the median and shaded regions are 95% confident intervals. Dashed red line is the transition threshold of 10%. In panel a) competition is global ($w_h = 1$) and in panel b) competition is strong $\gamma_C = 0.2$. Panel c) represents the effect of competition on the transition and on the maintenance of mutualism (simulation without dispersal cost $d = 0$). Panel d) presents the effect of a reduction in competition caused by a perturbation eradicating all organisms in a large square. The perturbation occurs around $t = 10^4$, which is 5000 time steps after the transition. Other parameters are $m = 0.06$, $c_m = 0.3$, $f_{min}^h = 0.1$, $f_{min}^s = 2.5$, $f_{max} = 8$, $f^a = 0.5$ and $\beta_{max} = 0.5$.

324 the relationship between local interactions and avirulence evolution (Boots and Meador, 2007).

325

326 **The key role of intraspecific competition** The invasion of a mutualistic cluster by parasites may
 327 cause its extinction and hinder the transition. We postulated that the higher fecundity of mutualistic clusters
 328 could compensate for their susceptibility to parasites (hypothesis H3). We instead found that, in the absence
 329 of dispersal cost, an eco-evolutionary feedback involving intraspecific competition between hosts was necessary
 330 for the transition. Indeed when competition between hosts is weak, the transition to mutualism rarely occurs
 331 (Figure 2a), and when it does, the percentage of mutualistic symbionts remains low (Figure 5a). Conversely,
 332 when hosts strongly compete for resources, the ecological conditions change dramatically. The formation of
 333 mutualistic clusters (Figure 4a) increases population densities (Figures 2b and 2c), which enhances competition
 334 between hosts. Areas dominated by hosts associated with parasitic symbionts were initially viable, but their
 335 population growth rate becomes negative following the increase in competition. This creates empty space that
 336 can be colonized by mutualists, which still disperse globally from time to time. By lowering the abundance of
 337 parasitic symbionts, this also reduces the frequency at which mutualistic clusters are invaded by parasites. The
 338 transition needs some time to occur (Figure 2a) because several obstacles must be overcome (simultaneity of
 339 the mutations, demographic stochasticity, possible invasions by parasites) before the mutualists are numerous
 340 enough to induce the shift in host competition. Therefore, contrary to hypothesis H3, the transition is not
 341 directly caused by the higher fecundity of mutualistic pairs (which would fit soft selection, Wallace, 1975) but
 342 only indirectly by the increase in host competition, which renders areas dominated by parasites unviable (hard

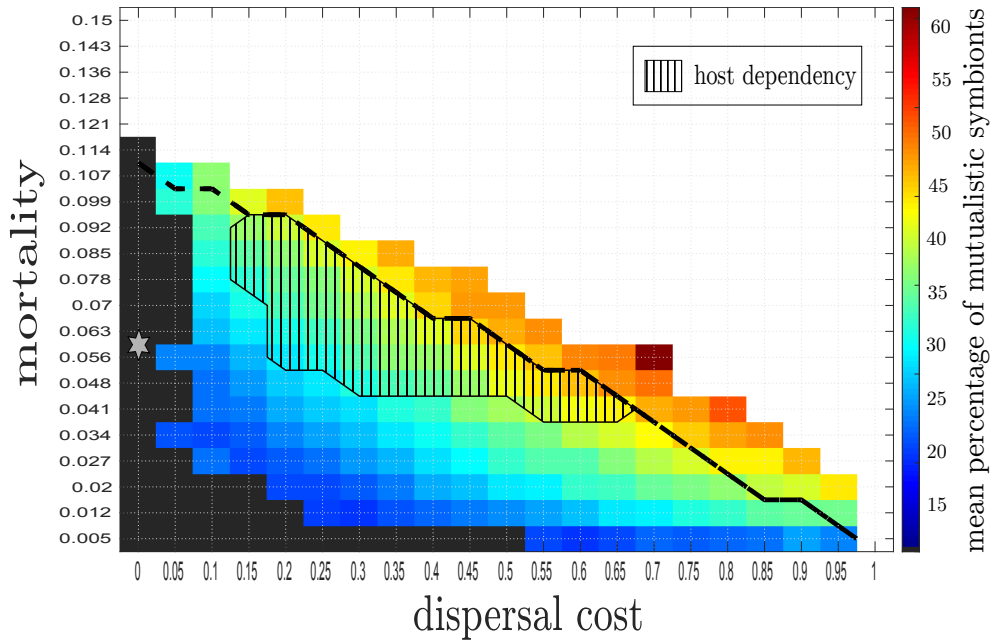


Figure 6: Percentage of mutualistic symbionts as a function of intrinsic mortality m and dispersal cost d . We run 50 simulations per parameter combination, with strong global competition $\gamma_C = 0.2$ and $w_h = 1$ with a maximum projection time of 10^4 steps. The percentages are averaged over the simulations leading to transition, if any occur. Above the black dotted line, the parasitic system is not viable, although the evolution of mutualism can occur above this line (evolutionary rescue). White cells correspond to the nonviability domain for the whole system, even with evolution. In the dark grey area, none of the simulations gave birth to transitions. The evolution of host ecological dependency occurs in the hatched area, where an average isolated host has a negative growth rate because of intraspecific competition. The grey star corresponds to the restrictive conditions of Figure 2. Other parameters are $c_m = 0.3$, $f_{min}^h = 0.1$, $f_{min}^s = 2.5$, $f_{max} = 8$, $f^a = 0.5$ and $\beta_{max} = 0.5$.

343 selection). However, this only occurs when competition between hosts is partly global (Figure 5b); if it is purely
 344 local, mutualistic clusters cannot influence the viability of parasitic regions and will suffer from kin competition.
 345 In line with hypothesis H4, local competition between hosts for resources thereby prevents the transition to
 346 mutualism. Local competition between hosts for available space also occurs when hosts disperse locally, but this
 347 does not jeopardize the transition.

348 Empirical work has shown that the outcome of interactions between hosts and symbionts depends not only on
 349 the traits of the protagonists, but also on the surrounding ecological conditions (Bronstein, 1994). For instance,
 350 plants take advantage of seed-eating pollinators in the absence of alternative pollinators but not in their presence
 351 (Thompson and Cunningham, 2002). Mycorrhizae are beneficial for plants when soil resources are scarce while
 352 they are detrimental when resources are abundant (Johnson et al., 1997). In the above cases, the outcome of the
 353 interaction depends on both biotic and abiotic factors that are external to the host-symbiont system. Our model
 354 showed that the association with symbionts remains parasitic when host competition is low, while it evolves
 355 towards mutualism when host competition increases. In that case, the outcome of the interaction depends on
 356 intrinsic features of the interactions that are constructed by the eco-evolutionary dynamics of the system, as the
 357 emergence of mutualists increases host density.

358

359 **The impact of dispersal cost and mortality** As expected, dispersal cost speeds up the transition
 360 (Figure 2a and 6) because it induces a selection pressure at the organismic level in favour with local dispersal,
 361 which increases the likelihood of the formation of mutualistic clusters. Mortality also enhances the probability of
 362 transition (Figure 6), but with another mechanism. We have stressed that competition between hosts creates an
 363 eco-evolutionary feedback loop, where the evolution of mutualism increases global densities, which strengthens
 364 competition and therefore turns the growth rate of the parasitic system negative. Given that mortality pushes
 365 the parasitic system towards its viability boundary, high mortality enhances the ability of competition to launch
 366 the transition. Although the transition occurs in a wide range of parameters where the parasitic system is viable,
 367 it is more likely when the parasitic system is close to extinction (Figure 6). However, mortality cannot itself
 368 trigger the transition since the parasitic system is unviable from the start when mortality is too high. Finally,
 369 mortality may also facilitate the transition through the reduction of global densities, which decreases the threat

370 of parasites invading mutualistic clusters. The facilitation of mutualistic symbiosis in harsh environmental conditions has also been observed in previous empirical (Callaway et al., 2002; Maestre et al., 2003; Werner et al., 371 2015) and theoretical (Travis et al., 2006) works. However in the context of altruism the opposite relationship 372 was found (Taylor and Irwin, 2000).
373
374

375 **Evolutionary rescue** As evidenced by Figure 6, the evolution of mutualism can prevent the extinction of
376 the parasitic system for parameter combinations that are just above the upper limit of its viability domain. This
377 echoes the concept of evolutionary rescue (Ferriere and Legendre, 2013; Gomulkiewicz and Holt, 1995), according
378 to which the persistence time of a population is longer with than without evolution. In the present case, instead
379 of a single population, the populations of two distinct species are rescued by evolution. More generally, the
380 parasitic system benefits from the evolution of mutualism even when it is initially viable, through an increase in
381 population densities (Figure 2).

382 **The evolution of mutualistic hosts** So far, only the mechanisms responsible for the evolution of
383 mutualistic symbionts have been elucidated, but not those involved in the evolution of mutualistic hosts. Surpris-
384 ingly, mutualistic hosts evolve after the transition (Figure 2c). Following the transition, the density of mutualistic
385 symbionts is much higher, so that mutualistic hosts tend to be associated with mutualistic symbionts (Figure 4c),
386 which disperse locally (Figure 3a). In that case, mutualistic hosts will increase the local density of mutualistic
387 symbionts in the following generations, which will benefit their offspring provided that they disperse locally as
388 well (Figure 3b). Symbionts may become less abundant for instance because of additional intraspecific competi-
389 tion between them, as in Appendix A.5. As a result, more hosts remain non-mutualistic because they are less
390 often associated with a symbiont (Figure A13), which further highlights that the evolution of mutualistic hosts
391 relies on high symbiont densities.

392 **The role of quasi-vertical transmission** Although mutualistic symbionts are environmentally ac-
393 quired, when both hosts and symbionts disperse locally this produces a similar effect as vertical transmission (as
394 for mycorrhizae, Wilkinson, 1997), which we term "quasi-vertical" transmission. However, local dispersal (even
395 100%) is not equivalent to vertical transmission because host and symbiont offspring can disperse to any of the
396 8 neighbouring cells, so vertical transmission due to specific reproductive and physiological adaptations would
397 have produced transitions to mutualism more easily. Moreover, the colonization of empty space by a mutualistic
398 pair requires that both species disperse to the same remote place by chance, whereas in the case of vertical trans-
399 mission this always occurs. Nevertheless, since hosts need to colonize empty space a significant fraction of hosts
400 with mutualistic phenotypes also dispersed globally ($\sim 40\%$, Figure 3), which partly counteracts the necessity
401 of quasi-vertical transmission. As well as hosts, mutualistic symbionts may also suffer from limited dispersal
402 when they need to percolate in a landscape of non-adjacent hosts, which explains why they maintain $\sim 20\%$ of
403 global dispersal (Figure 3). On the other hand, parasitic symbionts also evolve towards an intermediate dispersal
404 strategy, although they tend to disperse globally much more often ($\sim 80\%$, Figure 3). In purely parasitic systems
405 it has been shown that some degree of vertical transmission, which is close to local dispersal in our case, is nec-
406 essary for persistence in fragmented landscapes (Su et al., 2019; Schinazi, 2000). In those cases as well as here,
407 the parasitic population needs some degree of local dispersal in order to exploit a patch of hosts, once it has been
408 "found" by global dispersers. Intermediate dispersal strategies have been found to favor persistence of a variety
409 of systems. For instance, frequent short-distance and rare long-distance dispersal together favor metacommunity
410 persistence in fragmented habitats (Huth et al., 2015) and intermediate migration rate is required for the spread
411 of cooperative strategies in spatial prisoner's dilemma games (Vainstein et al., 2007).

412 Colonization ability comes along with the avoidance of local overpopulation generated by mutualism. This
413 is in line with both the evolution of altruism, which can be limited by kin competition (Wilson et al., 1992;
414 Alizon and Taylor, 2008), and the evolution of dispersal which is in part due to the reduction of kin competition
415 (Hamilton and May, 1977; Poethke et al., 2007; Harada, 1999). A mixed strategy combining both dispersal
416 modes takes advantage of kin selection and simultaneously maintains the opportunity to escape kin competition.
417 Figure 5b shows that purely local competition between hosts prevents the transition to mutualism because kin
418 competition overcomes kin selection. Similarly, the evolution of cooperation by group selection can be hindered
419 if competition between groups is local (Akdeniz and Van Veelen, 2020). In nature, global competition between
420 hosts may arise when plants compete for water present in the same groundwater (Lejeune et al., 1999; Rietkerk
421 et al., 2002), while competition for light is more local. Thus, the evolution of mutualism may depend on the
422 dominant form of competition for resources between hosts.

423 Assumptions, limitations and generality of the model

424 Our results rely on several hypothesis which have contrasting effects on the likelihood of the transition to mutu-
425 alism.

426 **No vertical transmission** We excluded the possibility of vertical transmission because it is a complex
427 feature involving many traits, which more likely evolve some time after the transition once the mutualistic
428 relationship is well established. For this reason an alternative mechanism is needed, and our results demonstrate
429 that the coevolution of mutualistic effort and limited dispersal in both species can mimic vertical transmission,
430 as argued above. However, in some parasitic systems (e.g. birds displacing parasitic flatworms, ticks carried
431 on large vertebrates) vertical transmission may be a passive feature, present from the start. In such cases the
432 evolution of mutualism is theoretically possible even if hosts keep dispersing globally, provided that mutations
433 turning the parasites into mutualists exist.

434 **Benefits and costs** Benefits only depend on the interaction trait of the partner. In turn, costs depend
435 on the interaction trait of the focal organism as well as on the benefits provided by the partner (Appendix A.1).
436 This would correspond for instance to the development of organs like plant domatia (Szilágyi et al., 2009): if
437 the symbiotic ants are mutualistic, the plant can grow bigger, thereby producing more domatia, which is more
438 costly in absolute terms. An alternative would be to assume that the costs do not increase with the mutualistic
439 benefit; this would in any case be favourable to the evolution of mutualism. Moreover, in the model some cost
440 is paid even if the partner is parasitic or if the host is free of symbiont. For instance, domatia or extrafloral
441 nectaries are unconditionally produced (Bronstein, 1998), even though domatia size can be plastic (Kokolo et al.,
442 2020). Also, plants produce costly floral displays even in the absence of pollinators. Finally, another alternative
443 arises when partners interact repeatedly, for instance during their growth. Using an iterated prisoners' dilemma
444 model, Doebeli and Knowlton (1998) assumed that large received benefits trigger higher investment in the
445 relationship. The interaction traits therefore become subject to phenotypic plasticity, in function of the partner's
446 trait. This assumption favors the transition to mutualism since mutualists benefit more from being associated
447 with mutualists. In contrast, our set of assumptions is more conservative.

448 **Antagonistic coevolution of the parasitic system** The evolutionary dynamics of the parasitic
449 system have been ignored here, although they might affect the probability of transition. In the model the hosts
450 cannot become resistant against the parasitic symbiont, which fits with the "superpathogen" of the gene-for-gene
451 model (Salathé et al., 2008). This can be interpreted as a monomorphic long-term result of Red Queen dynamics,
452 some constrain preventing the appearance of new resistant and virulent alleles. However, if the host-parasite
453 interaction is instead ruled by a matching allele model (Salathé et al., 2008), dispersal and the associated spatial
454 structure is likely to maintain polymorphism (Sasaki et al., 2002). During the early stages of the transition,
455 formerly parasitic symbionts turned mutualistic will inherit this matching genetic system and will need to find
456 compatible hosts. This adds another requirement, rendering the transition less likely.

457 **Asexual reproduction** Many models of (co)evolutionary dynamics assume asexual reproduction (e.g.
458 Kéfi et al., 2008; Loeuille and Loreau, 2005), especially within the framework of Adaptive Dynamics (e.g. Dieck-
459 mann et al., 1995; Loeuille and Loreau, 2005). In the case of sexual reproduction, recombination may soften the
460 correlation between dispersal and interaction traits, which is nevertheless essential to the transition. However,
461 the work of Dieckmann and Doebeli (1999) on the coevolution between a niche and a mating trait showed that
462 linkage disequilibrium can itself evolve, thereby preserving the correlation between traits. In the present case,
463 we speculate that sexual reproduction would lengthen the waiting time until a successful transition, without
464 hindering the transition in the long term.

465 **Symbiont competition within hosts** We previously assumed that only a single symbiont could infect
466 a host, however several strains may compete within the same (Bongrand and Ruby, 2019; Zytynska and Weisser,
467 2016; Alizon et al., 2013). The host may be able to prevent the proliferation of parasitic strains (Sachs et al.,
468 2010b), but parasitic strain may also overcome the others, which could prevent the evolution of mutualism (Jones
469 et al., 2012). An extension of the model, presented in Appendix A.7, includes superinfections where mutualistic
470 symbionts can be dislodged by parasites reaching the same host. When superinfection probability rises above
471 50%, the transition is prevented, otherwise mutualistic symbionts can persist, although at lower densities than
472 without superinfections (Figure A15). Thus, although superinfections are clearly detrimental to the transition,
473 mechanisms favouring the evolution of mutualism in our present model can resist some degree of competitive
474 exclusion by parasites.

475 **The evolution of cheating** Our main interest was to understand how mutualism can evolve from a
476 parasitic relationship (Roughgarden, 1975; Drew et al., 2021) but mutualism may also have evolved in the first
477 place, the classic evolutionary problem in this case being how can it resist to the invasion of "cheaters" (e.g.
478 Sachs et al., 2010a; Jones et al., 2015; Ferriere et al., 2002). According to Jones et al. (2015), cheating "(1)
479 increases the fitness of the actor above average fitness in the population and (2) decreases the fitness of the
480 partner below average fitness in the partner population". The latter condition is always satisfied by parasitic
481 symbionts, but the former remains to be checked. Simulations starting with mutualistic symbionts only are rapidly
482 invaded by parasites, leading to an evolutionary equilibrium identical to the one reached by Figure 2c (details
483 not shown). The population-level fitness (sensu Metz et al., 1992) of parasites is therefore positive when they

484 are rare, thereby satisfying condition (1), and it gradually decreases to zero until the evolutionary equilibrium is
485 reached. Hence, our model also accounts for the invasion by cheaters of an initially mutualistic system, leading
486 to a coexistence of both strategies. Mutualism may also evolve from a competitive interaction, if two competitors
487 start exchanging resources, each being a better exploiter of the resource it provides, and limited by the resource
488 it receives (De Mazancourt and Schwartz, 2010). However it is unknown to what extent this kind of mutualism
489 is sensitive to cheating; spatial effects similar to those studied here might stabilize it.

490 The interplay between several levels of selection

491 Although the first models of group selection relied on well-defined groups (e.g. Wilson, 1975; Smith, 1964),
492 multilevel selection theory has since been extended to fuzzy group boundaries and more complex landscapes
493 (e.g. Lion and Van Baalen, 2008; Nunney, 1985; Tekwa et al., 2015) like in the present case. Earlier in the
494 discussion, intermediate dispersal has been interpreted as the result of a balance between two components of
495 inclusive fitness, kin selection and kin competition, which have been recognized as particular cases of multilevel
496 selection (Goodnight, 2005; Lion and Van Baalen, 2008; Queller, 1992; Sober and Wilson, 1999). Although our
497 model is too complex for an analytical derivation of inclusive fitness, this should be possible in principle, as
498 it has been done for simpler models of the evolution of altruism (Hamilton and Fox, 1975; Lion et al., 2011;
499 Lehmann et al., 2007; Marshall, 2011; Wade, 1980). However, the levels-of-selection problem is more a question
500 about the level at which there is a causal link between character and fitness (Okasha, 2006, 2016; Sober, 1984;
501 Sober and Lewontin, 1982), rather than the level at which a mathematical formulation of fitness can be derived
502 ("bookkeeping" in the words of S. J. Gould 2002, p. 619). Following Sober (1984), we will consider that selection
503 at a given level of organization occurs if the different entities belonging to this level are variable with respect
504 to some property causally involved in the survival or reproduction of the organisms forming the entities. Since
505 Sober's formulation has been originally framed in the context of group selection, we first discuss how the levels-
506 of-selection problem for mutualism can be related to the group selection debate in the context of altruism. The
507 mechanism by which parasitic symbionts and hosts can invade mutualistic clusters is a two-species version of the
508 tragedy of the commons (Garrett, 1968; Feeny et al., 1990; Hardin, 1998). In the case of altruism, the tragedy of
509 the commons can be bypassed by local dispersal which triggers the formation of cooperative clusters (Mitteldorf
510 and Wilson, 2000; Le Galliard et al., 2003), as in the present case. The evolution of altruism results from
511 the conflict between two levels of selection, the organismic-level favouring cheaters and the group-level favoring
512 altruism (Van Baalen and Rand, 1998; Simon et al., 2013; Wilson and Sober, 1989). At a given time step,
513 neighbouring altruistic organisms help each other, which favors their fecundity. Shortly after the local density of
514 altruists increases, which is beneficial for their offspring's fecundity as well. Since the transition to mutualism is
515 egalitarian whereas the transition to altruism is fraternal, it is unclear if the evolution of mutualism involves the
516 same levels of selection as for altruism. Sure enough, mutualism is also counter-selected at the organismic level,
517 since mutualism is costly to both hosts and symbionts. However, differences between altruism and mutualism
518 may arise at higher organization levels because at a given time step mutualists help their heterospecific partners
519 but not their neighbouring conspecifics. In the present model the evolution of mutualism involves selection at
520 the level of the host-symbiont pair, since at a given time step the reproduction of each of its organisms depends
521 on the properties of the pair (the interaction traits α_h and α_s). This resembles the tit-for-tat strategy where
522 cooperators are selected at the pair level (Wilson, 2004; Sober and Wilson, 1999). The mutualistic host-symbiont
523 holobiont therefore emerges as a new unit of selection (Roughgarden et al., 2018; Drew et al., 2021).

524 Considering several times steps in a row, another level of selection appears. Since mutualists also disperse
525 locally (Figure 3), after some time a mutualistic pair may trigger the formation of a mutualistic cluster (Figure 4c).
526 Neighbouring mutualistic pairs do not help each other directly at a given time step, but indirectly by increasing
527 the likelihood that their offspring will encounter mutualistic partners in the subsequent time steps. Although
528 only hosts and symbionts reproduce in the traditional sense of organismic reproduction, the association between
529 mutualistic hosts and symbionts is also re-produced (Doolittle and Inkpen, 2018; Griesemer, 2001) via local
530 dispersal and cluster formation. Selection at the cluster level therefore occurs, since clusters dominated by
531 mutualistic pairs will favour the reproduction of organisms and the re-production of mutualistic pairs. The
532 re-production of pairs constitutes a another mechanism of inheritance, different from the one occurring during
533 organismic reproduction. It is therefore hard to match Hull's (1980) categorization of replicators (here, hosts and
534 symbionts) and interactors (here, pairs), since during the transition mutualistic pairs also acquire a replicative
535 power via the evolution of local dispersal. This also emphasizes that Sober's (1984) formulation of group selection
536 needs to be generalized for the present context, since the properties of clusters favor not only the reproduction of
537 organisms but also the transmission of higher-level properties. Mutualistic clusters are self-perpetuating systems
538 (Lenton et al., 2021), some of their properties being homeostatic (Ibanez, 2020). However, we believe this is not
539 enough to qualify to evolutionary individuality (sensu Godfrey-Smith et al., 2013) since conflicts are still vivid
540 (Queller and Strassmann, 2016); mutualistic clusters being prone to the invasion by parasites (Figure 4c).

541 Lastly, in the absence of dispersal cost mutualism rarely invades when host competition is weak (Figure 2a),
542 despite the occasional formation of mutualistic pairs. Without dispersal cost, competition between hosts at the
543 global scale is necessary for the transition to mutualism (Figure 5b). The global scale therefore constitutes an-
544 other level of organization involved in the transition to mutualism. Global competition between hosts acts as an

545 environmental factor mitigating selection at the different organization levels discussed above. This environmental
546 factor is not fixed by a parameter but instead determined by the evolutionary dynamics of the whole system, it
547 is at the same time subject and object of evolution (Lewontin, 1982, 1983).
548

549 Host dependency and irreversibility of the transition

550 Major transitions in evolution are characterized by their irreversibility and by the interdependence between the
551 agents (Szathmáry and Smith, 1995; Estrela et al., 2016). The model does not include any physiological or
552 developmental dependence of the host on its symbiont, or any loss of functions in the host due to gene transfers,
553 because we assumed that this generally occurs during later stages of the evolution of mutualism. Instead,
554 dependence has been defined from a population dynamics perspective: the host is *ecologically dependent* when its
555 population growth rate is negative in the absence of the symbiont. In that case the host can produce offspring,
556 although not enough to compensate for mortality. In line with hypothesis H5, we found that mutualistic hosts
557 deprived of their symbiont exhibit a negative growth rate when the host density after the transition to mutualism
558 becomes sufficiently large (Figure 6). This ecological dependency resulted from the density-dependent competition
559 between hosts and the assumption that mutualism is costly for the host, even when its symbiont is absent (as
560 discussed above). However, ecological dependency is not absolute: once the density of hosts becomes sufficiently
561 low, the mutualistic hosts alone are viable. Dependency may become absolute for a sufficiently high cost of
562 mutualism, but in these conditions the transition to mutualism will not occur.

563 If host competition strength decreases permanently, for instance following the continuous supply of extra
564 resources, the reverse transition back to parasitism occurs (Figure 5c). This has been documented in nature
565 as well (Pellmyr and Leebens-Mack, 2000; Kawakita et al., 2015; Sachs et al., 2011), although the mechanisms
566 involved may well be different. Reversal towards parasitism occurs because ecological dependency relies on
567 host competition, which change with host densities, highlighting that mutualistic symbiosis may be sensitive to
568 environmental change (Drew et al., 2021). However, if host competition decreases punctually, e.g., following a
569 perturbation of a fraction of the landscape, then mutualism persists (Figure A11) because mutualistic clusters
570 take advantage of the reduction of global host competition to colonize free cells around them. This leads to
571 an increase in host competition; in that case mutualism can restore the ecological conditions allowing its own
572 persistence, as in a niche construction process (Lewontin, 1982, 1983; Odling-Smee et al., 2013; Laland et al.,
573 2016). Niche construction is generally understood as the improvement of abiotic conditions (e.g. Arnoldi et al.,
574 2020). In the context of mutualism, it is due to the improvement of host densities, which induces an increase in
575 host competition. This also occurs at the beginning of the transition, when the first mutualistic clusters trigger
576 an increase in global host density. Although this has not been tested formally, the reversion is also very likely to
577 occur if host competition for resources shifts from global to local, since it is apparent from Figure 5b that local
578 competition completely prevents mutualism, even in the presence of dispersal cost.

579 Conclusion

580 In the present paper, we aim to understand the mechanisms promoting the transition from parasitism to mutu-
581 alism. To tackle this issue, we develop an agent based model on a lattice. In our general model, we only assume
582 that the mutualistic interactions influence the fecundity of both partners and that hosts face density-dependent
583 competition. In addition, we ensure that the antagonistic system is stable in absence of mutations. We show that
584 in the absence of vertical transmission or partner control mechanisms, the joint evolution between mutualistic
585 effort and local dispersal can trigger the transition from parasitism to mutualism, provided that intraspecific
586 competition between host is sufficiently global and that either dispersal cost or competition strength is large
587 enough.

588 Unexpectedly, we found that mutualistic clusters invade the antagonistic system thanks to their ability to
589 increase the population densities of both partners, thereby triggering global competition between hosts and
590 rendering regions where hosts are mainly associated with parasitic symbionts unsuitable. In contrast, the higher
591 fecundity of mutualists is not advantageous enough to compensate for the ability of parasites to invade mutualistic
592 clusters. Thus, our results suggest that the eco-evolutionary feedback involving competition between hosts might
593 promote the transition from parasitism to mutualism in a wide range of biological systems, such as plant-fungi,
594 plant-ant and plant-seed-eating pollinator interactions.

595 Acknowledgements

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597 Massol and four anonymous reviewers for their thoughtful comments and discussion on this work.

598 Data and code accessibility

599 All the codes used to compute the outcomes of our model and the figures of the paper are available on the
600 following github repository: <https://github.com/leoledru/PCI-Ledru-et-al.-2021->.

601 A Appendix

602 A.1 Mathematical and numerical details of the model

603 We present here the mathematical underpinnings of the model as well as some details of the numerical compu-
604 tation.

605 **Rules of the individual based model description** Our model follows the cycle presented in Figure A1:

- 606 • Host and symbiont die with fixed probability $m \in (0, 1)$.
- 607 • They produce offspring, possibly with different traits from them due to mutation. The fecundity of the
608 parents depends on their two traits $(\alpha, \varepsilon) \in [0, 1]^2$ and on their interactions with their possibly cell-sharing
609 partner.
- 610 • The offspring are dispersed according to the parents' dispersal traits ε .
- 611 • The offspring of the hosts may establish only in empty cells, while the offspring of the symbionts can only
612 establish in cells already occupied by a solitary host. If several organisms arrive in the same cell, a lottery
613 determines which one will establish, while the others die.

614 In our numerical computations, mutations occurred only after the descendant was successfully established in a
615 cell. This procedure saves computational time and did not influence our results because offspring dispersal and
616 establishment do not depend on their traits but only on their parent traits. Furthermore, the mortality process
617 was applied to both types of agents simultaneously, while the reproduction and dispersal processes were applied
618 consecutively to the hosts and then to the symbionts. We confirmed that the order of the algorithm did not
619 qualitatively affect our results.

620
621 **Fecundity and the average offspring number** The fecundity f of an agent depends on its mutualistic
622 interaction trait α as well as the interaction trait of its cell-sharing partner. This continuous trait ranging between
623 0 and 1 determines the intensity of the agent investment in the mutualistic relationship.

We assumed a positive interaction trait dependence between agents. A mutualistic agent tends to increase
the fecundity of its cell-sharing partner. The interaction fecundity $f_I^i(\alpha_j)$ of an organism of type $i \in \{h, s\}$, ($h =$
host, $s =$ symbiont) interacting with an organism of type $j \in \{s, h\}$ with trait α_j was defined by

$$\begin{aligned} f_I^h(\alpha_s) &= f_{min}^h + (f_{max} - f_{min}^h)\alpha_s^{\gamma_f} \\ f_I^s(\alpha_h) &= f_{min}^s + (f_{max} - f_{min}^s)\alpha_h \end{aligned} \quad (5)$$

624
625 The coefficient γ_f corresponds to the selection strength on the interaction trait α_s . Using a coefficient $\gamma_f > 1$, we
626 create a convex function allowing a transition from parasitism to mutualism for a central value of the symbiont
627 interaction trait. However, note that modifying the shape of this fecundity curve (from concave to convex via
628 linear) does not qualitatively change our results.

629
On the other hand, a mutualistic agent has an intrinsic cost reducing its fecundity. The mutualism cost
 $C_m(\alpha_i)$ of an organism of type $i \in \{h, s\}$ ($h =$ host, $s =$ symbiont) ranges between 0 and 1, and it increases with
interaction trait α_i of the agent. It is defined by

$$C_m^i(\alpha_i) = 1 - c_m \alpha_i \quad (6)$$

630 where c_m is the maximal cost of mutualism.

Thus, for the host as for the symbiont, the fecundity f_i of an organism i interacting with an organism j is
the product of the interaction fecundity $f_I(\alpha_j)$ defined by (5) and the cost of mutualism $C_m(\alpha_i)$ defined by (6).

$$\begin{aligned} f^h &= C_m^h(\alpha_h) f_I^h(\alpha_s) \\ f^s &= C_m^s(\alpha_s) f_I^s(\alpha_h) \end{aligned} \quad (7)$$

When a host agent is alone in a cell, its fecundity is defined by its intrinsic host fecundity f^a weighted by its
mutualism cost $C_m(\alpha_h)$: Fecundity of the solitary host:

$$f^{ha} = C_m^h(\alpha_h) f^a. \quad (8)$$

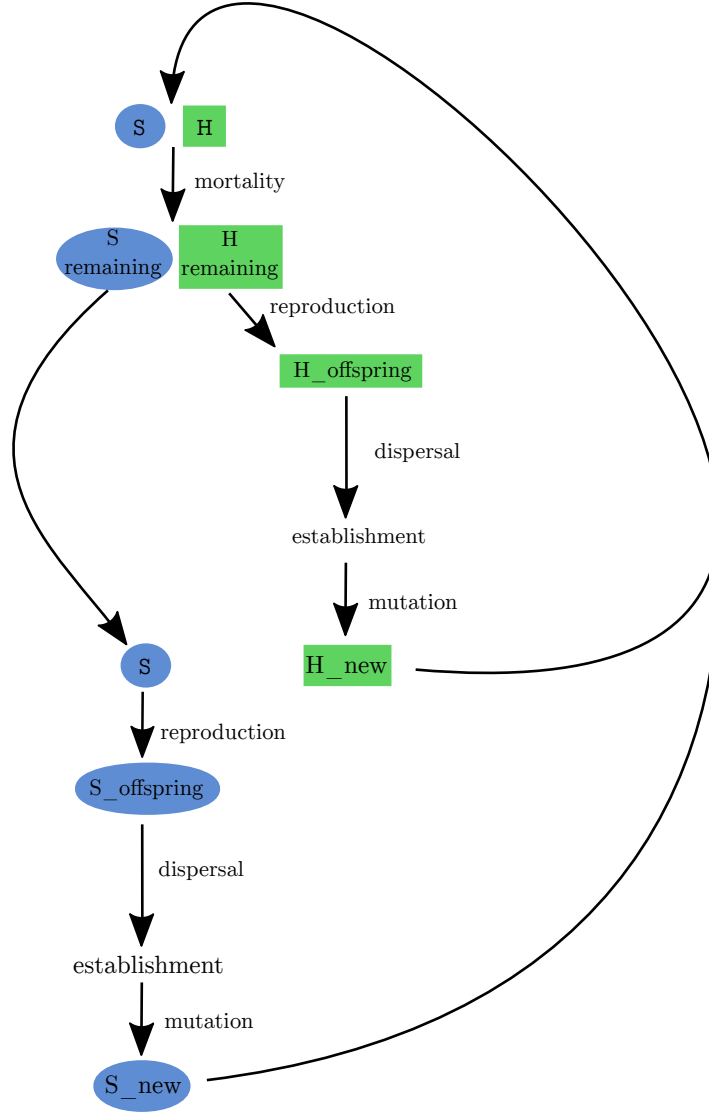


Figure A1: Sketch representation of the individual based model. The host population (H) and the symbiont population (S) undergo intrinsic mortality, then reproduction, dispersal, establishment, and finally mutation. The mortality step is simultaneous for the host and the symbiont, while the other steps occur first for the host and then for the symbiont.

631 We assume that the cost of mutualism is paid regardless of whether the interaction is realized.

632

633 In general, the average offspring number is not integer, yet the number of offspring in our model can only
 634 be represented by an integer. Thus, in the numerical algorithm, the fecundity was used as the λ parameter of
 635 a Poisson distribution. If the value drawn from the distribution was greater than the maximum fecundity f_{max} ,
 636 then it was set back to the maximum fecundity.

Mutualism/parasitism threshold In our model, the presence of a host always produces a net benefit for the symbiont. However, the presence of the symbiont might be detrimental for the host. Indeed, the fecundity of a host h interacting with a symbiont s is $f^h(\alpha_h, \alpha_s) = f_I^h(\alpha_s)C_m(\alpha_h)$, while the fecundity of the same host h without a symbiont is $f^{ha}(\alpha_h) = f^a C_m(\alpha_h)$. Thus, the host has net benefit only if its fecundity in association with a symbiont is larger than its fecundity alone. Therefore, mutualism only occurs when $f_I^h(\alpha_s) > f^a$. Otherwise, the interaction is parasitic. This criterion does not depend on the host mutualism trait α_h because hosts always pay the same mutualism cost. Thus, we can define the mutualism/parasitism threshold α_s^* such that $f_I^h(\alpha_s^*) = f^a$; thus, we obtain

$$\alpha_s^* = \left(\frac{f^a - f_{min}^h}{f_{max} - f_{min}^h} \right)^{1/\gamma_f} \approx 0.475 \quad (9)$$

637 with the parameters set in Table 1.

Competition To test the effect of the spatial scale of the competition, we introduced a scale parameter $w_h \in [0, 1]$ that weighs the effect of local ρ_h^{local} and global ρ_h^{global} host density on the competition. The establishment probability thus satisfies

$$P_I = 1 - \left((1 - w_h) \rho_h^{local} + w_h \rho_h^{global} \right)^{\gamma_C} \quad (10)$$

638 The local host density ρ_h^{local} corresponds to the host density in the 8 neighbouring cells surrounding the implan-
 639 tation cell of the host, while the global density ρ_h^{global} corresponds to the host density all over the landscape (see Figure A2 for a schematic representation).

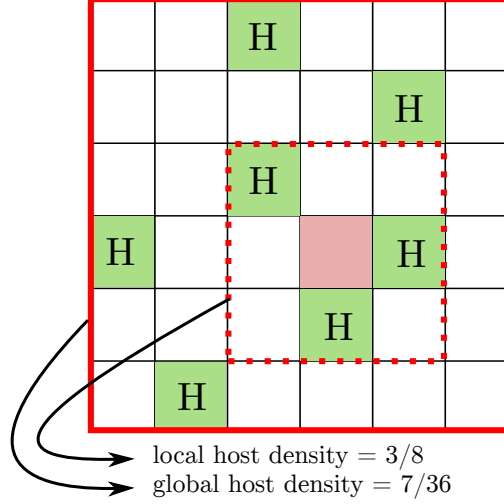


Figure A2: Local and global host densities influence the probability of establishment in the focus cell (pink filled square). The global density corresponds to the host density in the whole 36-cell landscape. The local density corresponds to the density in the eight cells vicinity around the focus cell.

640 These competition scales may have various ecological explanations. For instance, plants sharing the same
 641 water table face global competition for this resource. Conversely, the competition for light between plants is
 642 an example of local competition. Thus, our competition scale model allows us to describe the competition for
 643 several different resources that may appear at different scales. Following our previous examples, if the water
 644 supply represents 90% of the competition and light supply represents only 10%, then the competition scale w_h is
 645 $w_h = 0.9$ (90% global competition and 10% local competition).
 646
 647

Distribution of mutation effects During reproduction, organisms generate offspring with traits that can deviate from their traits due to mutation. The effects of mutation on each trait are independent. However, the mutation effect does depends on the trait of the parent. For instance, an organism with trait α will give birth to an organism of trait $\alpha + \beta$ where β is drawn from a distribution with probability distribution function given by $K(\beta|\alpha)$, which depends on the trait of the parent α (Figure A3). In our model, we use a modified Beta distribution with shape parameters (1, 3) to describe the effects of the mutation. More precisely, for a parent of trait α , the effect of mutaion is a random variable β defined by

$$\beta = (\beta_{max} \xi B) \mathbf{1}_{0 \leq \alpha + \beta_{max} \xi B \leq 1}$$

where B is a random variable, which follows a Beta distribution, ξ is a random variable independednt of B , which follows a Bernoulli distribution ($\mathbf{P}(\xi = 1) = \mathbf{P}(\xi = -1) = 1/2$). In other word, the random variable β follows the probability distribution function $K(\beta|\alpha)$, with $\alpha \in [0, 1]$:

$$K(\beta|\alpha) = \frac{3}{2\beta_{max}^3} (\beta_{max} - |\beta|)^2 \mathbf{1}_{|\beta| \leq \beta_{max}} \mathbf{1}_{0 \leq \alpha + \beta \leq 1} + K_0(\alpha) \delta_{\beta=0} \quad (11)$$

where $\mathbf{1}$ is the indicators function, δ is the Dirac mass and the function $K_0(\alpha)$ is defined by

$$K_0(\alpha) = \begin{cases} \frac{1}{2\beta_{max}^3} (\beta_{max} - \alpha)^3 & \text{if } \alpha \leq \beta_{max} \\ 0 & \text{if } \beta_{max} \leq \alpha \leq 1 - \beta_{max} \\ \frac{1}{2\beta_{max}^3} (\beta_{max} - 1 - \alpha)^3 & \text{if } \alpha \geq 1 - \beta_{max} \end{cases} \quad (12)$$

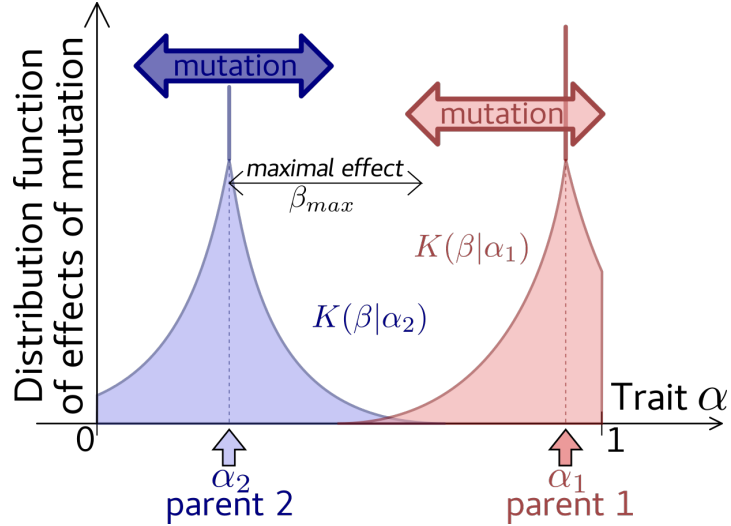


Figure A3: Distribution of mutational effects $K(\beta|\alpha)$. Each parent of trait, e.g. α_1 or α_2 , produce offspring with trait $\alpha_i + \beta$ where β has the density $K(\beta|\alpha_i)$ depending on its parent traits (red and blue curves for α_1 and α_2 , respectively).

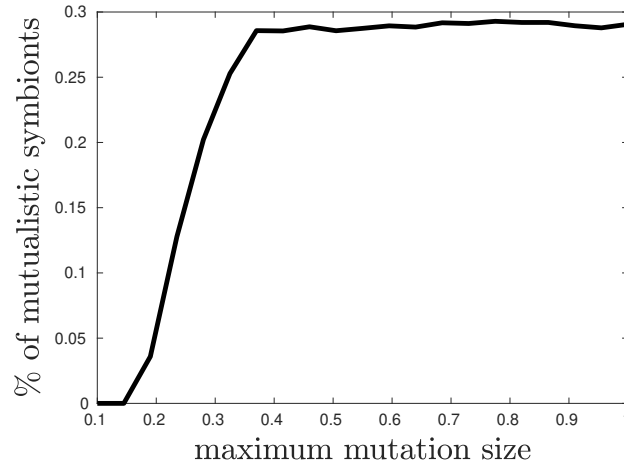


Figure A4: Effect of the average mutation effect (parameter β_{max} of distribution kernel of mutation effect) on the proportion of mutualistic symbionts.

648

649 Moreover, we investigate the effect of the maximal effects of mutation β_{max} on the proportion of mutualistic
650 symbionts. From our formula, we know that the mean effect of mutation depends on the trait of the parent α
651 but it is proportional to β_{max} , and it ranges between $3\beta_{max}/8$ for parents with intermediate trait ($\alpha \sim 0.5$) and
652 $3\beta_{max}/4$ for parents with trait either close to 1 or 0. We show in Figure A4 that increasing the mean effect of
653 mutation increases the proportion of mutualistic symbionts in the population. Thus large effects of mutation
654 favour the emergence of mutualism. In our simulations we fix the maximal effect of mutation to $\beta_{max} = 0.5$.

655 **Dispersal** At each time step, hosts and symbionts produce offspring which can disperse over the landscape
656 either locally or globally. For each agent, the proportion of its offspring dispersing globally is given by the
657 dispersal trait ε . The location of offspring dispersed locally is chosen randomly uniformly over the 8 neighbors of
658 its parents, while the location of those dispersed globally is chosen uniformly over the entire landscape expected
659 the location of the parent (Figure A5 for the description of the local and global scale). In particular, a globally
660 dispersed organism can arrive in the local neighbor of parents as the locally dispersed one. Moreover, the offspring
661 are dispersed independently from each other and their location is chosen independently of the current landscape.
662 In particular, offspring can arrive at an already occupied location and symbionts' offspring are not only dispersed
663 in location where there is already a host. For instance if a host disperse $2/3$ of its offspring at large distance
664 from it, its dispersal trait satisfies $\varepsilon = 2/3$. Then the $2/3$ of its offspring are dispersed randomly uniformly in

665 the entire landscape (red stars in Figure A5) while the remaining 1/3 is dispersed locally around it (red circles
 666 in Figure A5).

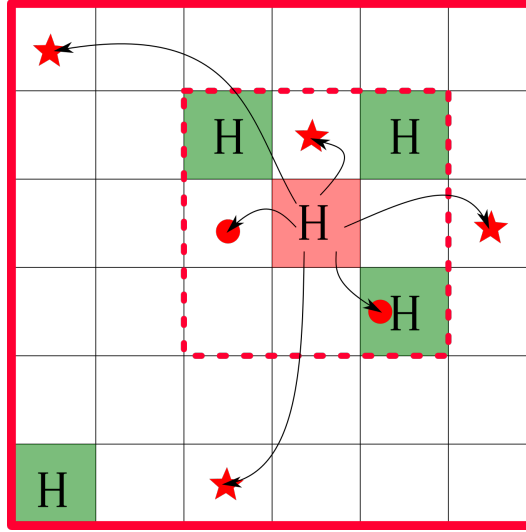


Figure A5: Local and global dispersal of offspring from the host in the red cell over the landscape. Local dispersal (red circles) occurs only within the neighborhood of the host (red dashed square) while global dispersal (red stars) occurs over the entire landscape (red plain square). The host has a dispersal trait $\varepsilon = 2/3$ and it disperses 6 offspring: 4 globally (red stars) and 2 locally (red circles).

667 **Assortment index** To compute the assortment index, we measured the similarity between spatially neighbouring phenotypes for the spatial repartition resulting from the transition to mutualism and for the same spatial
 668 repartition but with phenotypes randomly redistributed among organisms. The assortment index corresponds to
 669 the difference between the measurement made on the space resulting from the transition to mutualism and the
 670 measurement on the randomly rearranged space. If the index shifts positively (resp. negatively) from zero, it
 671 means that similar phenotypes are closer (resp. more distant) than different phenotypes compared to random
 672 spatial distribution. This methodology is similar to that used in [Pepper and Smuts \(2002\)](#) and [Pepper \(2007\)](#).
 673

674

Intraspecific assortment index. More precisely, for the intraspecific assortment index we use the following similarity index for host and symbiont. For each simulation and time t , we compute the similarity indices S_h and S_s respectively among hosts and symbionts, as follows

$$S_h(t) = 1 - \frac{1}{N_h} \sum_{h=1}^{N_h} |\alpha_h - \bar{\alpha}_h| \quad (13)$$

where α_h is the trait of the host h and N_h is the total number of host in the landscape at time t . The quantity $\bar{\alpha}_h$ is the average trait in the neighborhood V_h of the host h . The neighborhood V_h of a host h is the 8 closest cells surrounding it (figure A2). It is defined by

$$\bar{\alpha}_h = \frac{1}{|V_h|} \sum_{i \in V_h} \alpha_i.$$

675 The similarity index among symbionts S_s is computed similarly.

676 Then for each time, we reshuffle the traits among the location occupied by hosts and symbionts and we
 677 compute the associated similarity indices using equation (13). We average those indices over 1000 replicates to
 678 compute the similarity indice S_{r_h} and S_{r_s} corresponding to a random spatial distribution.

Finally, We build the assortment index A_h as the difference between the similarity index of host S_h observed and the similarity index S_{r_h} of host when we randomly assigned trait of the host over the landscape,

$$A_h(t) = S_h(t) - S_{r_h}(t). \quad (14)$$

679 We also compare our assortment index with the spatial autocorrelation Moran index for the host and symbiont.
 680 The two indices show the same pattern. A positive spatial autocorrelation is observed after the transition occurred
 681 (Figure A6).
 682

Interspecific assortment index. For the assortment index between host and symbiont, we also use a measure of similarity between the host and symbiont trait at each location of the couple. More precisely, we define for each simulation and each time t the similarity index S_{sh} between host and symbiont sharing the same location as follows

$$S_{sh}(t) = 1 - \frac{1}{N_s} \sum_{s=1}^{N_s} |\alpha_s - \alpha_h| \quad (15)$$

where N_s is the number of symbiont, which is also the number of host–symbiont couple. As before, we compare this observed index with the random index S_{rsh} defined by randomly rearranging pairs of symbiont and host and taking average over 1000 replicates. The assortment index A_{sh} is thus given by

$$A_{sh}(t) = S_{sh}(t) - S_{rsh}(t). \quad (16)$$

683 We also compare our index with the correlation coefficient between the interaction traits of hosts and symbionts. We find a positive correlation between trait in a same location (Figure A6).

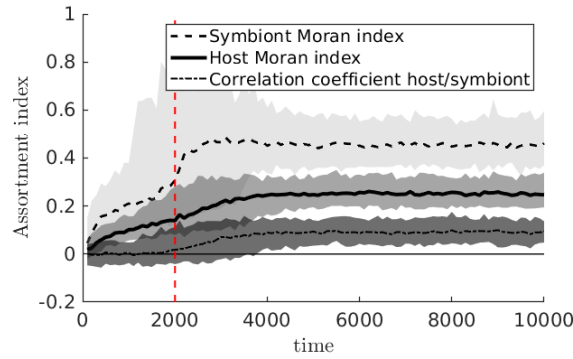


Figure A6: Spatial autocorrelation among hosts (plain curve) and symbionts (dashed curve) are described by the Moran index. The spatial correlation between the host and symbionts are described by the correlation coefficient (dash-dotted curve). The shadow regions corresponds to the 95% confident interval and curves corresponds to the median over 100 replicates. The parameters are similar as Figure 4.

684

Aggregation index From the assortment index analysis, we show that the symbionts and hosts are spatially assorted according to their trait. Now we aim to investigate how they are aggregated in space. We use a relative aggregation index \mathcal{A} based on a measure of the number of pair of neighbors P where a neighbor of an organism is its 8 closest cells. For instance, Figure A2 provides a schematic representation of a host spatial configuration and the dashed square represents the neighborhood of the red organism. The number of pair of the red organism is 3 in this example. Then for any spatial configuration with n organisms, we can define the maximal number of possible pair of organism which is given by $P_{max} = 4n - [6\sqrt{n}]$ (Harary and Harborth, 1976). Thus, we define the aggregation index \mathcal{A} as the ratio between P and P_{max} :

$$\mathcal{A} = \frac{P}{P_{max}}.$$

685 We compute the aggregation index over time for the hosts, the parasitic symbionts ($\alpha_s < \alpha_s^*$) and the mutualistic
686 symbionts ($\alpha_s \geq \alpha_s^*$) (Figure A7).

687 Hosts are always more aggregated than symbionts. Moreover, after the transition occurred, mutualistic and
688 parasitic symbionts have the same spatial signature in terms of aggregation. This pattern was already observed
689 in Figure 4 where we see mutualistic clusters surrounded by parasitic clusters.

690 A.2 Mathematical approximations

691 In order to provide some heuristics about our stochastic model, we develop some deterministic approximation.
692 This mathematical analysis also provides some quantitative insights on our choice of parameters and the threshold
693 used to describe our outcomes (mutualistic vs parasitic symbionts, emergence of mutualists). We first investigate
694 a simple model with hosts and parasitic symbionts and then we investigate a model with hosts and both parasitic
695 and mutualistic symbionts.

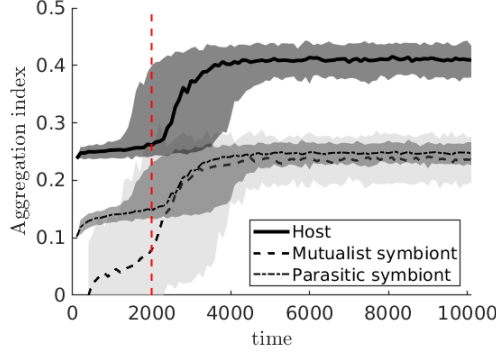


Figure A7: Aggregation index of the spatial distribution of hosts (plain curve), parasitic symbionts (dash-dotted curve) and mutualistic symbionts (dashed curve) over time. The shadow regions corresponds to the 95% confident interval and curves corresponds to the median over 100 replicates. The parameters are similar as Figure 4.

An approximation of the parasitic system. First, we aim to describe the expected proportion of sites occupied by hosts and parasitic symbionts at equilibrium. We assume no mutations of interaction or dispersal traits and hosts and symbionts disperse globally randomly over the landscape composed of N sites. According to our model, the dynamics of the proportion of sites occupied by the host alone ρ_{ha} or host with symbionts ρ_{hs} is given by

$$\begin{aligned}\rho_{ha}(t+1) &= (1-m)\rho_{ha}(t) \left[(1-f^s(\alpha_h, \alpha_s)\rho_{hs}(t)) + f^{ha}(\alpha_h)(1-\rho_h(t)^{\gamma_C}) \right] \\ &\quad + (1-m)\rho_{hs}(t)f^h(\alpha_h, \alpha_s)(1-\rho_h(t)^{\gamma_C}) \\ \rho_{hs}(t+1) &= (1-m)\rho_{hs}(t) \left(1 + f^s(\alpha_h, \alpha_s)\rho_{ha}(t) \right)\end{aligned}\tag{17}$$

696 where $\rho_h = \rho_{ha} + \rho_{hs}$ is the total proportion of hosts and α_h and α_s are interactions trait of host and symbionts
697 respectively. In this model, the traits are fixed – if $\alpha_s < \alpha_s^*$ symbionts are parasitic while there are mutualistic
698 if $\alpha_s \geq \alpha_s^*$. Since the symbionts need host to survive, the proportion of sites occupied by symbionts is ρ_{hs} . Even
699 if hosts and symbiont does not share the same mortality rate m , the model holds true by multiplying the terms
700 f^s by $(1-m_s)$ the survival rate of symbionts instead of $(1-m)$. We can check that the following qualitative
701 properties holds true with a different mortality rate. However, it will modify the quantitative outcome of the
702 model.

703 For any given pair of interaction traits, we can compute the equilibria of this dynamical system.

Extinction. The extinction equilibrium, which corresponds to $\rho_{ha} = \rho_{hs} = 0$, always exists but it is unstable if

$$\frac{1}{f^{ha}(\alpha_h)} \frac{m}{(1-m)} = \frac{1}{(1-c_m\alpha_h)f^a} \frac{m}{(1-m)} < 1$$

704 We have picked parameters, which fulfill this criterion (Table 1). In particular, we can see from this formula that
705 increasing the mutualism cost c_m can lead to non viability of more mutualistic host. In our simulations, we fix
706 this value to $c_m = 0.3$.

Absence of symbionts. We first look at the case where the symbionts are absent, $\rho_{hs} = 0$. Then we have $\rho_{ha} = \rho_h$ which is given by

$$\rho_h = \rho_{ha} = \left(1 - \frac{1}{f^{ha}(\alpha_h)} \frac{m}{(1-m)} \right)^{1/\gamma_C} = \left(1 - \frac{1}{(1-c_m\alpha_h)f^a} \frac{m}{(1-m)} \right)^{1/\gamma_C}\tag{18}$$

707 This equilibrium should be positive for any host trait α_h . Our fixed parameters stated in Table 1 satisfy this
708 assumption and the proportion of host ρ_h alone without symbionts ranges between 0.36 and 0.5.

709 Without symbiont, the proportion of host converges to $\rho_h = \rho_{ha} = 50, 5\%$ ($\gamma_C = 0.2$ and $\alpha_h = 0$). Thus in
710 absence of any symbionts, the host survives. Its intrinsic growth rate is given by $(1-m)(1+f^{ha}(\alpha_h))$. It is
711 decreasing with α_h and it ranges between 1.41 ($\alpha_h = 0$) and 1.27 ($\alpha_h = 1$). Thus in absence of symbionts, the
712 hosts always survives.

713 However, this equilibrium is unstable in our parameters range – the eigenvalue of the jacobian around this
714 equilibrium has an eigenvalue with modulus greater than 1. This suggests that a third equilibrium exists and
715 may be stable.

Coexistence of symbionts and host. We also have the coexistence equilibrium which is given by the following formulae

$$\begin{aligned}\rho_{ha} &= \frac{1}{f^s(\alpha_h, \alpha_s)} \frac{m}{(1-m)} \\ 0 &= \rho_h \left(f^h(\alpha_h, \alpha_s)(1 - \rho_h^{\gamma_C}) - m \right) + (1-m)\rho_{ha} (f^{ha}(\alpha_h) - f_h(\alpha_h, \alpha_s))(1 - \rho_h^{\gamma_C})\end{aligned}\quad (19)$$

In our parameters range, this equilibrium always exists and it is always stable and attractive for any values of the interactions traits (α_h, α_s) .

So, in the presence of a parasitic symbiont ($\alpha_s = 0$), the proportion of hosts converges to $\rho_h = 12,5\%$ and the proportion of symbionts to $\rho_{hs} = 10\%$ which is in accordance with our simulation at initial time $t = 0$ (Figure 2 b and c).

In addition, when hosts ($\alpha_h = 0$) are associated with mutualistic symbionts ($\alpha_s = 1$), the proportion of hosts rises to $\rho_h = 96\%$ and the proportion of symbionts to $\rho_{hs} = 92,5\%$. Thus the gain of cohabiting with mutualistic symbionts is indeed huge.

An approximation of the mutualistic/parasitic system without mutations. We now investigate the outcome of the competition between mutualistic symbionts and parasitic symbionts on a host. In this case, our previous model (17) extends to the following form

$$\begin{aligned}\rho_{ha}(t+1) &= (1-m)\rho_{ha}(t) \left[\left(1 - \frac{f^s(\alpha_h, \alpha_{sp})\rho_{sp}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)} - \frac{f^s(\alpha_h, \alpha_{sm})\rho_{sm}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right) \right. \\ &\quad \left. + f^{ha}(\alpha_h)(1 - \rho_h(t)^{\gamma_C}) \right] \\ &\quad + (1-m)\rho_{hs}(t)(1 - \rho_h(t)^{\gamma_C}) \left(\frac{f^h(\alpha_h, \alpha_{sp})\rho_{sp}(t)}{\rho_{sp}(t) + \rho_{sm}(t)} + \frac{f^h(\alpha_h, \alpha_{sm})\rho_{sm}(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right) \\ \rho_{hs}(t+1) &= (1-m)\rho_{hs}(t) + (1-m)\rho_{ha}(t) \left(\frac{f^s(\alpha_h, \alpha_{sp})\rho_{sp}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)} + \frac{f^s(\alpha_h, \alpha_{sm})\rho_{sm}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right) \\ \rho_{sp}(t+1) &= (1-m)\rho_{sp}(t) \left(1 + \frac{f^s(\alpha_h, \alpha_{sp})\rho_{ha}(t)\rho_{sp}(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right) \\ \rho_{sm}(t+1) &= (1-m)\rho_{sm}(t) \left(1 + \frac{f^s(\alpha_h, \alpha_{sm})\rho_{ha}(t)\rho_{sm}(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right)\end{aligned}\quad (20)$$

where ρ_{sp} and ρ_{sm} represent respectively, the proportion of parasitic and mutualistic symbionts. The nature of the symbiont only depends on the interaction trait α_s : parasitic symbiont ($\alpha_{sp} < \alpha_s^*$), mutualistic symbiont ($\alpha_{sm} > \alpha_s^*$) where α_s^* is the mutualistic/parasitic threshold defined by (9).

For this model, we still recover the three equilibria described in the previous section: extinction $(0, 0, 0, 0)$, host alone $(\rho_h, 0, 0, 0)$ (equation (18)), host with one symbiont $(\rho_{ha}, \rho_{hs}, \rho_{sp}, 0)$ or $(\rho_{ha}, \rho_{hs}, 0, \rho_{sm})$ (equation (19)). However, another equilibrium may exist: host with two coexisting symbionts $(\rho_{ha}, \rho_{hs}, \rho_{sp}, \rho_{sm})$. In the following section, we will investigate the stability of this coexisting equilibrium.

Competitive exclusion or coexistence of symbionts? The outcome of this model crucially depends on the initial proportion of parasitic and mutualistic symbionts.

The coexistence of parasitic and mutualistic symbionts with a host may occur when the initial relative proportion of mutualistic symbiont is exactly the proportion at coexistence equilibrium, which is given by

$$p^* = \frac{f^s(\alpha_h, \alpha_{sp})}{f^s(\alpha_h, \alpha_{sp}) + f^s(\alpha_h, \alpha_{sm})}\quad (21)$$

However, when the proportion of mutualistic symbionts is initially larger than the critical threshold p^* , the mutualistic symbionts can win the competition by excluding the parasitic symbiont. While if the parasitic symbionts are more present with a proportion larger than $1 - p^*$, they drive the mutualistic symbionts toward extinction.

As a consequence, the coexistence of mutualistic and parasitic symbiont seems unlikely to occur without mutations among symbionts because of competitive exclusion. Moreover, this mechanism should create segregation if we use a spatially explicit model.

An approximation of the mutualistic/parasitic system with mutations. In our stochastic model, the interaction trait may change from one symbiont to its offspring. So now we assume that during reproduction, symbionts can mutate from one type to the other at a rate $U = 0.02$. In this situation, the previous model

extends to

$$\begin{aligned}
\rho_{ha}(t+1) &= (1-m)\rho_{ha}(t) \left[\left(1 - \frac{f^s(\alpha_h, \alpha_{sp})\rho_{sp}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)} - \frac{f^s(\alpha_h, \alpha_{sm})\rho_{sm}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right) \right. \\
&\quad \left. + f^{ha}(\alpha_h) \left(1 - \rho_h(t)^{\gamma_C} \right) \right] \\
&\quad + (1-m)\rho_{hs}(t)(1-\rho_h(t)^{\gamma_C}) \left(\frac{f^h(\alpha_h, \alpha_{sp})\rho_{sp}(t)}{\rho_{sp}(t) + \rho_{sm}(t)} + \frac{f^h(\alpha_h, \alpha_{sm})\rho_{sm}(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right) \\
\rho_{hs}(t+1) &= (1-m)\rho_{hs}(t) + (1-m)\rho_{ha}(t) \left(\frac{f^s(\alpha_h, \alpha_{sp})\rho_{sp}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)} + \frac{f^s(\alpha_h, \alpha_{sm})\rho_{sm}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right) \\
\rho_{sp}(t+1) &= (1-m)\rho_{sp}(t) \left(1 + (1-U) \frac{f^s(\alpha_h, \alpha_{sp})\rho_{ha}(t)\rho_{sp}(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right) + U(1-m) \frac{f^s(\alpha_h, \alpha_{sm})\rho_{ha}(t)\rho_{sm}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \\
\rho_{sm}(t+1) &= (1-m)\rho_{sm}(t) \left(1 + (1-U) \frac{f^s(\alpha_h, \alpha_{sm})\rho_{ha}(t)\rho_{sm}(t)}{\rho_{sp}(t) + \rho_{sm}(t)} \right) + U(1-m) \frac{f^s(\alpha_h, \alpha_{sp})\rho_{ha}(t)\rho_{sp}^2(t)}{\rho_{sp}(t) + \rho_{sm}(t)}
\end{aligned} \tag{22}$$

740 In this case, the competitive exclusion process is damped by the mutation process and the coexistence always
741 occurs. However, the proportion of mutualistic truly varies and depends on its initial proportion. If the initial
742 proportion of mutualistic symbiont is above p^* stated in (21), then its proportion stabilizes around an equilibrium
743 value $\rho_{sm}^* \approx 0.02$ (this value coincides with the initial value of our simulation, Figure A8d and Figure 2b-c). Thus
744 this last model provides an accurate approximation of the antagonistic system used as initial condition of our
745 stochastic model.

746 **Transition from parasitic system to mutualistic/parasitic system.** Our model also allows us to
747 quantify the transition between a parasitic system with few mutualistic symbiont ($\rho_{sm} \sim 0.02$) and a system
748 with a larger proportion of mutualistic symbiont. From the previous analysis, we capture the threshold p^* such
749 that the system becomes mainly mutualistic. However, in our stochastic simulations, we do not observe this huge
750 transition (Figure A9). When the percentage of mutualistic symbiont evolves, it stabilizes around 12.5% and the
751 host density converges around 0.35.

From our deterministic model with mutation, we know that the density of host alone in the parasitic system,
is around $\rho_{ha}^* = 0.1$. Moreover, the intrinsic growth rate of the mutualistic symbiont R_s is given from (22) by

$$R_s = (1-m) \left(1 + (1-U) f^s(\alpha_h, \alpha_{sm}) \rho_{ha} p_{sm} \right) + U(1-m) f^s(\alpha_h, \alpha_{sp}) \rho_{ha} \frac{(1-p_{sm})^2}{p_{sm}} \tag{23}$$

752 where p_{sm} represents the proportion of mutualistic symbionts in the population. The mutualistic symbiont
753 population increases if its intrinsic growth rate R_s is larger than 1, $R_s > 1$. In the parasitic system with the host
754 density alone $\rho_{ha} = \rho_{ha}^* \approx 0.1$, the growth rate is larger than 1 if the proportion of mutualistic symbiont p_{sm} rises
755 above $p_{sm}^* \approx 0.1$, where p_{sm}^* is such that $R_s(p_{sm}^*) = 1$. So in the parasitic system, if the proportion of mutualistic
756 symbiont rises above 10%, its intrinsic growth rate becomes greater than 1 and its extinction probability falls
757 below 1. As consequences, we can assume that if the proportion of mutualistic symbiont rises above the threshold
758 of 10%, their percentage will remain truly above the 2% observed in the parasitic system, which characterizes a
759 transition from parasitism to mutualism.

760 A.3 Competition strength, perturbation and mutualism persistence

761 **Competition strength determines mutualism persistence** In the main text, we show that competi-
762 tion is essential for the transition to mutualism, but it is also important for its persistence, as shown here.
763 In this section, we explored the effect of a sudden variation in competition strength γ_C on the persistence of
764 mutualism. We started with a strong competition $\gamma_C = 0.2$. As expected from our previous results, a transition
765 to mutualism occurred (Figure A10a, c) and d)). Then, around $t = 10^4$, we suddenly switched the competition
766 strength to $\gamma_C = 2$, corresponding to negligible competition. We observed reversal of mutualism due to the
767 proportion of mutualistic symbionts decreasing from 20% to less than 5% (Figure A10a) and e)). We observed
768 that the reversal of mutualism due to a weakening of the competition corresponded with an increase in host and
769 symbiont densities. This increase is due to the reduction of competition, which determines densities more than
770 the presence or absence of mutualism does.

771 **Density perturbation does not affect mutualism persistence** Next, we tested how mutualism
772 responds to a decrease in competition due to eradication of hosts and symbionts in a large homogeneous region of
773 space (Figure A11). While previously we demonstrated that mutualism regresses when competition is set to be
774 weak, we show here that mutualism persists in the face of decreased competition due to decreased host density.

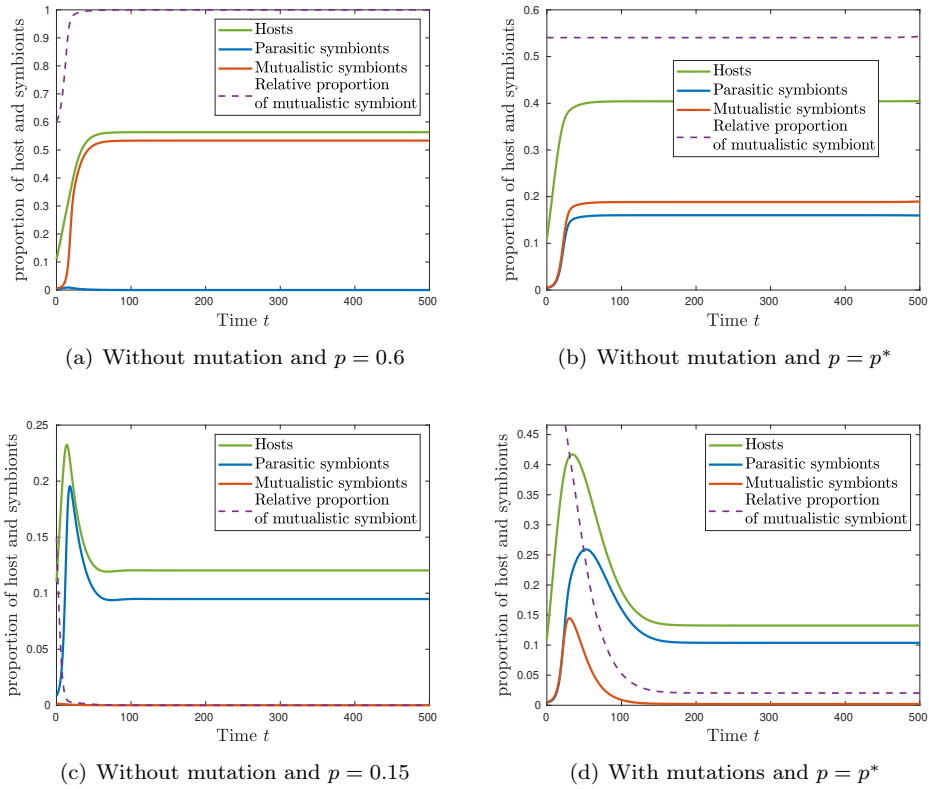


Figure A8: Evolution over time of the host-symbiont model (20) (panel a-c) and (22) (panel d) with two types of symbionts: parasitic symbionts ($\alpha_s = 0$) and mutualistic symbionts ($\alpha_s = 1$). We present the model (20) without mutation for various initial relative proportion of mutualistic symbiont p : (a) $p = 0.6$, (b) $p = p^* \approx 0.45$ and (c) $p = 0.15$. The model (22) with mutation is presented in (d) with initial proportion of mutualistic symbiont $p = p^*$.

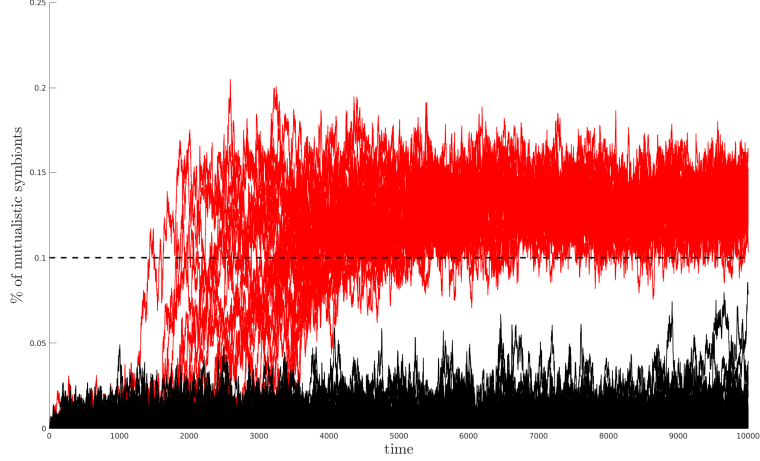


Figure A9: Evolution of the percentage of mutualistic symbionts in the population over 129 simulations. Red curves corresponds to replicates such that the percentage of mutualistic symbionts remains greater than the threshold of 10% – transition to mutualism. Black curves corresponds to replicates where the percentage remains below the 10% threshold – no transition.

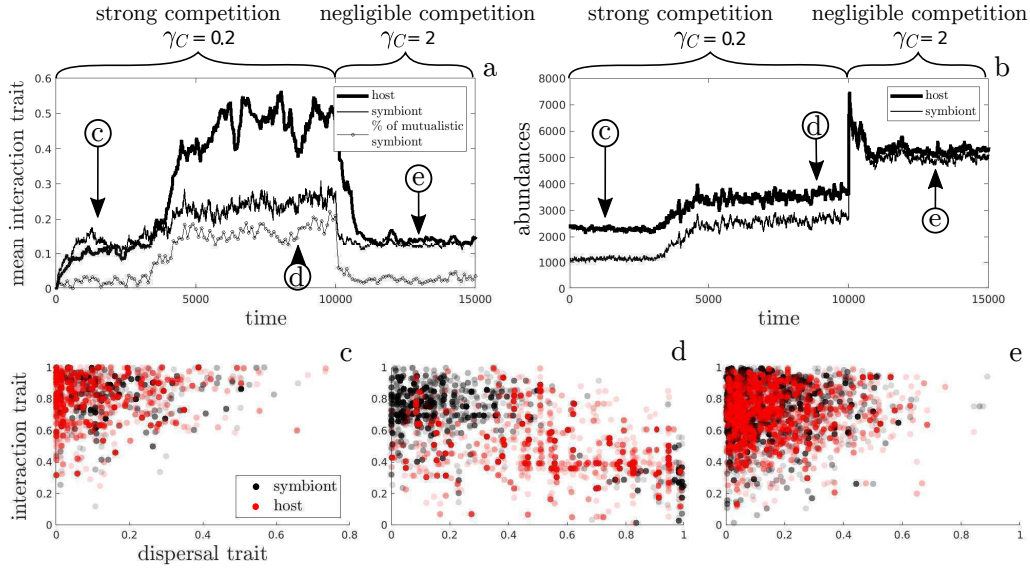


Figure A10: The transition to mutualism and collapse of mutualism depending on competition. a) Host and symbiont average interaction traits and the percentage of mutualistic symbionts over time. b) Host and symbiont abundance. There is strong competition from time $t = 0$ to $t = 10^4$ and negligible competition from $t = 10^4$ to the end. c) Dispersal and interaction traits distribution before the transition to mutualism. d) Dispersal and interaction traits distribution during mutualism persistence. e) Dispersal and interaction traits distribution after mutualism collapse. Parameters are $m = 0.06$, $c_m = 0.3$, $w_h = 1$, $f_{min}^h = 0.1$, $f_{min}^s = 2.5$, $f_{max} = 8$, $f^a = 0.5$, $\beta_{max} = 0.5$ and $d = 0$.

775 A.4 Mortality and dispersal cost can induce host dependency in emerging 776 mutualistic systems.

777 In the main text, we focused on the effect of mortality m and dispersal cost d on the transition to mutualism
778 and host dependency. Here, we present in more detail the effect of dispersal cost on the distribution of hosts and
779 symbionts in trait space for three values of dispersal cost and fixed mortality rate (Figure A12). In addition, the
780 table A1 shows the features of the clusters in the trait distribution.

781 We demonstrated that the dispersal cost favours the transition to mutualism. Moreover, even when the cost
782 was high, the features of the clusters revealed that parasitic symbionts maintained a more global dispersal than

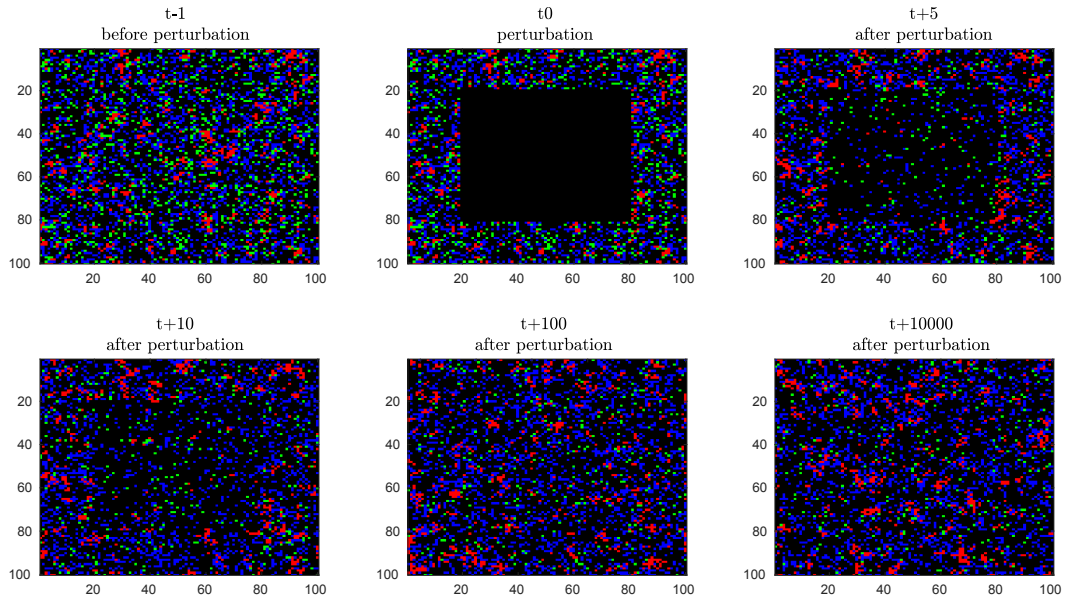


Figure A11: Maintenance of mutualism in the face of a reduction in competition caused by a perturbation creating a large square of free cells. Snapshot at several times: $t - 1$ is the eco-evolutionary equilibrium with mutualism just before the perturbation, then at t_0 the perturbation, and then $t + 5$, $t + 10$, $t + 100$ and $t + 10000$ after the perturbation. In black, the free cells; in green, the hosts alone; in blue, the couples with parasitic symbionts; and in red, the couples with mutualistic symbionts. Parameters are $m = 0.06$, $c_m = 0.3$, $w_h = 1$, $\gamma_C = 0.2$, $f_{min}^h = 0.1$, $f_{min}^s = 2.5$, $f_{max} = 8$, $f^a = 0.5$, $\beta_{max} = 0.5$ and $d = 0$.

783 mutualistic symbionts.

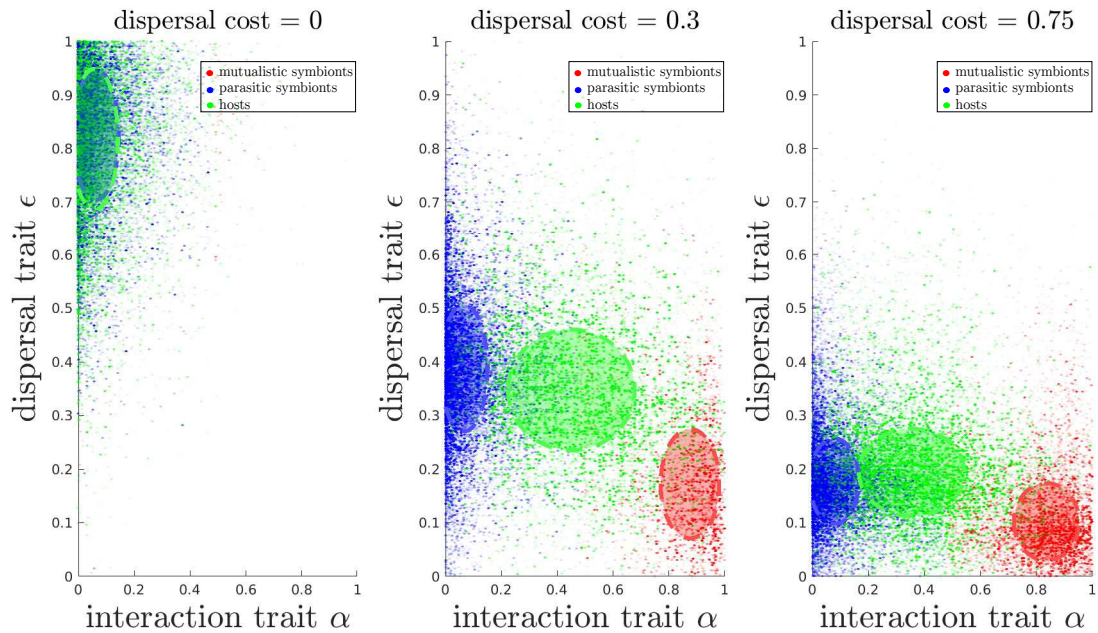


Figure A12: Joint distribution of the host (green), the parasitic symbiont (blue) and the mutualistic symbiont (red) populations in the traits domain for mortality $m = 0.03$ and an increasing dispersal cost $d \in \{0, 0.3, 0.75\}$. The ellipses correspond to the standard deviation. The 48 runs averaged in Figure 6 are plotted together. Other parameters are $c_m = 0.3$, $w_h = 1$, $\gamma_C = 0.2$, $f_{min}^h = 0.1$, $f_{min}^s = 2.5$, $f_{max} = 8$, $f^a = 0.5$ and $\beta_{max} = 0.5$.

dispersal cost	species	mean interaction trait	mean dispersal trait
$d = 0$	hosts	0.074	0.81
	parasitic symbionts	0.06	0.82
	<i>mutualistic symbionts</i>	* (<i>density < 2%</i>)	* (<i>density < 2%</i>)
$d = 0.3$	hosts	0.45	0.35
	parasitic symbionts	0.07	0.39
	mutualistic symbionts	0.88	0.17
$d = 0.75$	hosts	0.36	0.19
	parasitic symbionts	0.08	0.18
	mutualistic symbionts	0.84	0.10

Table A1: Features of the clusters in the traits domain

784 A.5 Density-dependent competition between symbionts

785 In the main text, symbionts compete for free hosts, which is a form of density-dependent competition. Other
786 ecological factors may also lead to density-dependent competition between symbionts, for instance if symbionts
787 compete for resources that are not provided by the hosts. Figure A13b shows that density-dependent competition
788 between symbionts reduces symbiont density, as expected. Hosts are therefore free of symbionts more often, which
789 selects for non-mutualistic hosts (Figure A13c, to be compared with Figure 3b).

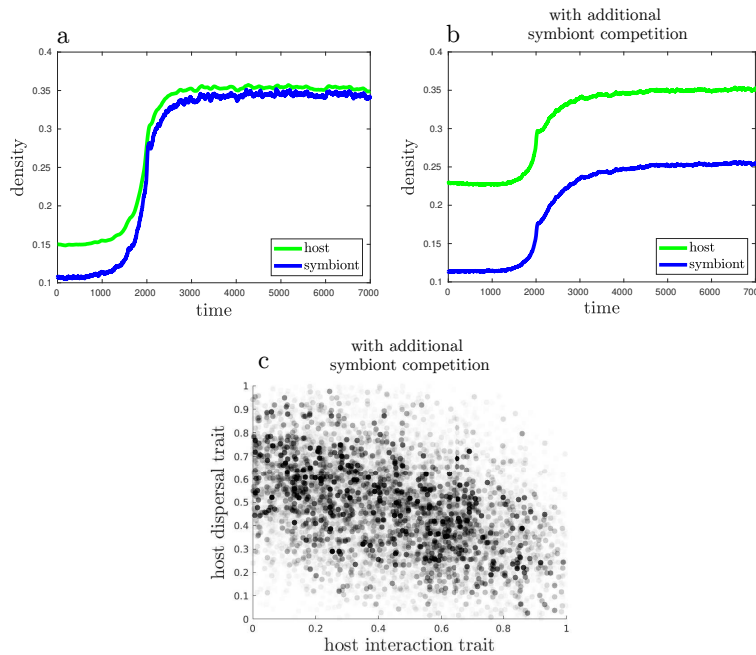


Figure A13: Effect of density-dependent competition between symbionts on host evolution. a) Symbiont and host densities after and before the transition, without density-dependent competition between symbionts. b) Symbiont and host densities after and before the transition, with density-dependent competition between symbionts. c) Distribution of host traits after the transition, with density-dependent competition between symbionts. To be compared with Figure 3b.

790 A.6 Evolutionary rescue

791 Figure 6 provides evidence for evolutionary rescue, as discussed in the main text. Figure A14 shows that this
792 occurs only in a fraction of the simulations, when mutualists arise soon enough to rescue the whole system.

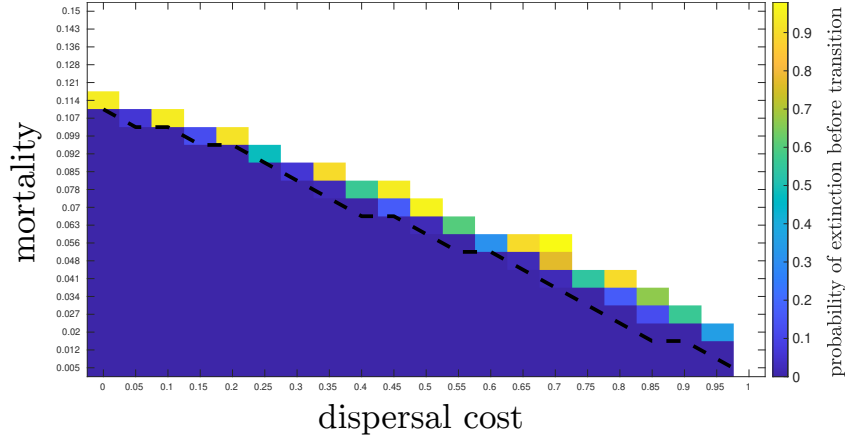


Figure A14: Frequency at which evolutionary rescue occurs. This figure is identical to Figure 6, except that it maps the probability of extinction before the transition, instead of the mean percentage of mutualistic symbionts. The dotted black line indicates the upper boundary of viability for the parasitic system, without evolution. Above the dotted black line, in some cases the evolution of mutualism rescued the whole system, although the parasitic system is unviable alone. In the white region, the systems goes extinct, even with evolution

793 A.7 Symbionts competition within hosts

794 In our current model, a host can be colonised only by one symbiont and once the symbiont is established on a
 795 host, it cannot be replaced by another symbiont. Furthermore, when several symbionts arrive at the same time
 796 on an available host, the symbiont, which establishes, is chosen randomly uniformly among the contenders. Here,
 797 we relax these assumptions in order to model symbionts' competition within a host, or "superinfection". We
 798 assume that within a host, the most parasitic symbiont, with the lowest interaction trait, is the most competitive
 799 symbiont. Thus, it will be more efficient to establish in a host or dislodge a symbionts from the host.

Establishment of symbionts on a host. Specifically, when N symbionts, with trait $\{\alpha_1, \dots, \alpha_N\}$ arrive on a host, the establishment probability P_e^i of the symbiont i is given by :

$$P_e^i = \frac{p_i}{\sum_{i=1}^N p_i} \quad \text{with} \quad p_i = \min \left(\max \left(\frac{1}{N} - (\alpha_i - \bar{\alpha}) S_{max}, 0 \right), 1 \right), \quad \text{and} \quad \bar{\alpha} = \frac{1}{N} \sum_{j=1}^N \alpha_j \quad (24)$$

800 where S_{max} measures the superinfections' intensity, which corresponds to the maximal competitive advantage of
 801 a symbiont. For instance, when a truly parasitic symbiont $\alpha_1 = 0$ tries to establish with a truly mutualistic
 802 symbiont $\alpha_2 = 1$, its establishment probability is $P_e^1 = (1 + S_{max})/2 \geq 1/2$. The establishment probability of the
 803 mutualistic symbiont is $P_e^2 = (1 - S_{max})/2 \leq 1/2$. If $S_{max} = 0$, they have the same probability of establishment,
 804 while if $S_{max} = 1$, the parasitic symbiont always over-competes the mutualistic symbiont.

Replacement of a resident symbiont When N symbionts with trait $\{\alpha_1, \dots, \alpha_N\}$ arrive in a host already occupied by a resident symbiont with trait α_s , they may dislodge the resident. Specifically, the probability of the resident symbiont to persist P_p is given by

$$P_p = \min (1 - (\alpha_s - \bar{\alpha}) S_{max}, 1) \quad \text{with} \quad \bar{\alpha} = \frac{1}{N} \sum_{i=1}^N \alpha_i. \quad (25)$$

805 In particular, if the resident has a trait α_s lower than the mean trait of the invaders $\bar{\alpha}$, then the resident always
 806 persists. Otherwise, the resident may be dislodged with a probability smaller than S_{max} . Then if the resident is
 807 dislodged, the establishment probability of the N invader symbionts is given by the previous formula (24).

808 Figure A15 shows the effect of the superinfection intensity S_{max} on the percentage of mutualistic symbionts.
 809 We show that despite the competitive advantage of parasitic symbionts when competing for a host, the transition
 810 to mutualism is possible when the superinfection intensity is not too large (if $S_{max} < 1/2$, transition occurs, that
 811 is the percentage of mutualistic symbionts stays above 10%). Moreover, when $S_{max} < 1/2$, the trait distribution
 812 of symbionts is bimodal.

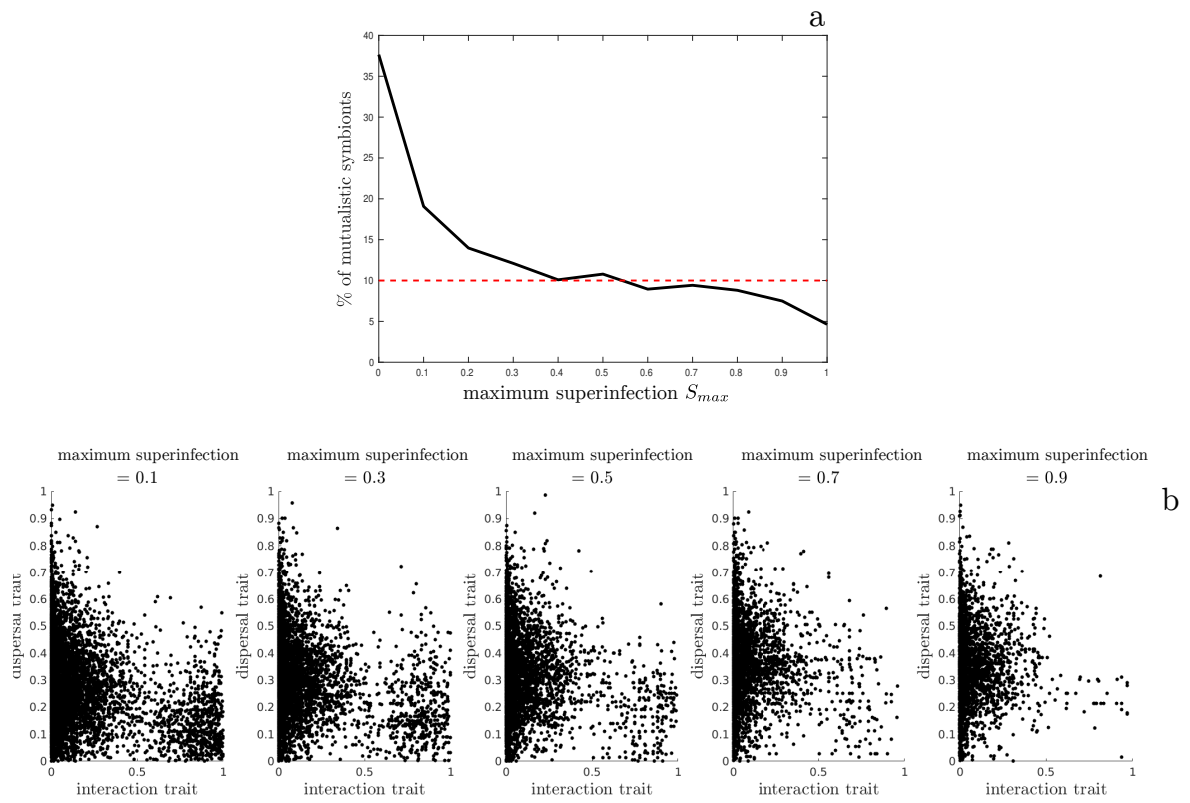


Figure A15: a) Percentage of mutualistic symbionts in function of the maximum superinfection advantage S_{max} averaged over 20 simulations per parameter values. b) Distributions of symbionts population in traits domain according to five intensity of superinfection advantage S_{max} . Distributions corresponds to 20 simulations for each parameter values. These results are obtained with a maximum time projection of 5000 time steps, a strong and global competition ($\gamma_C = 0.2$) and a dispersal cost ($d = 0$). Others parameters are $m = 0.06$, $c_m = 0.3$, $f_{min}^h = 0.1$, $f_{min}^s = 2.5$, $f_{max} = 8$, $f^a = 0.5$ and $\beta_{max} = 0.5$.

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