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Novel pathogen introduction rapidly alters evolved movement strategies, restructuring animal societies

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Abstract

1

2 Animal sociality emerges from individual decisions on how to balance the costs and benefits of being
3 sociable. Movement strategies incorporating social information — the presence and status of neigh-
4 bours — can modulate spatial associations, helping animals avoid infection while benefiting from in-
5 direct information about their environment. When a novel pathogen is introduced into a population,
6 it should increase the costs of sociality, selecting against gregariousness. Yet current thinking about
7 novel pathogen introductions into wildlife neglects hosts' potential evolutionary responses. We built
8 an individual-based model that captures essential features of the repeated introduction, and subsequent
9 transmission of an infectious pathogen among social hosts. Examining movements in a foraging con-
10 text, widely shared by many species, we show how introducing a novel pathogen to a population pro-
11 vokes a rapid evolutionary transition to a dynamic social distancing movement strategy. This evolution-
12 ary shift triggers a disease-dominated ecological cascade of increased individual movement, decreased
13 resource harvesting, and fewer social encounters. Pathogen-risk adapted individuals form less clustered
14 social networks than their pathogen-risk naive ancestors, which reduces the spread of disease. The mix
15 of post-introduction social movement strategies is influenced by the usefulness of social information
16 and disease cost. Our work demonstrates that evolutionary adaptation to pathogen introductions and
17 re-introductions can be very rapid, comparable to ecological timescales. Our general modelling frame-
18 work shows why evolutionary dynamics should be considered in movement-disease models, and offers
19 initial predictions for the eco-evolutionary consequences of wildlife pathogen spillover scenarios.

Introduction

Animal sociality emerges from individual decisions that balance the benefits of associations against the costs of proximity or interactions with neighbours (Tanner and Jackson 2012; Gil et al. 2018; Webber and Vander Wal 2018; Webber et al. 2022). While such associations can inadvertently or deliberately yield useful social information about resource availability (Danchin et al. 2004; Dall et al. 2005; Gil et al. 2018), they also provide opportunities for the transmission of parasites and infectious pathogens among associating individuals (Weinstein et al. 2018; Romano et al. 2020; Albery et al. 2021; Cantor et al. 2021; Romano et al. 2021). Wildlife pathogen outbreaks affect most animal taxa, including mammals (Blehert et al. 2009; Fereidouni et al. 2019; Chandler et al. 2021; Kuchipudi et al. 2022), birds (Wille and Barr 2022), amphibians (Scheele et al. 2019), and social insects (Goulson et al. 2015). Weighing the potential risk of infection from social interactions against the benefits of social movements — where to move in relation to other individuals' positions — is thus a common behavioural context shared by many animal species. Movement strategies incorporating social information — the presence and status of neighbours — can facilitate or reduce spatial associations, and help animals balance the costs and benefits of sociality (Gil et al. 2018; Webber and Vander Wal 2018; Albery et al. 2021; Webber et al. 2022). Animals' social movements link landscape spatial structure, individual distributions, and the emergent structure of animal societies (Kurvers et al. 2014; Gil et al. 2018; Webber et al. 2022). Together, they influence the dynamics of disease outbreaks in animal populations (Keeling et al. 2001; White et al. 2018a; Romano et al. 2020; 2021), and such outbreaks may in turn have cascading effects on landscape structure and community ecology (Monk et al. 2022).

On ecological timescales, pathogen outbreaks often reduce social interactions among individuals. This is due to a combination of mortality-induced decreases in population density (e.g. Fereidouni et al. 2019; Monk et al. 2022), and adaptive behavioural responses by which animals reduce encounters between infected and healthy individuals (Stroeymeyt et al. 2018; Weinstein et al. 2018; Pusceddu et al. 2021; Stockmaier et al. 2021). The latter case includes self-isolating when infected, or avoiding potentially infectious individuals (Stroeymeyt et al. 2018; Weinstein et al. 2018; Pusceddu et al. 2021; Stockmaier et al. 2021). However, when pathogens are first introduced into a population, such as during novel cross-species spillover (Chandler et al. 2021; Kuchipudi et al. 2022), fine-tuned avoidance responses are less likely, as individuals may have no prior experience of cues that indicate infection (Weinstein et al. 2018; Stockmaier et al. 2021). Spreading through host-host contacts, pathogens causing chronic infections (Bastos et al. 2000; Vosloo et al. 2009; Jolles et al. 2021) may instead impose fitness costs, thus selecting against host social behaviour, and hence against social connectivity itself (Altizer et al. 2003; Cantor et al. 2021; Poulin and Fillion 2021; Romano et al. 2021; Ashby and Farine 2022).

Yet novel pathogen introductions are primarily studied for their immediate demographic (Fey et al. 2015), and potential medical (Levi et al. 2012; Chandler et al. 2021; Kuchipudi et al. 2022; Wille and

55 Barr 2022) and economic implications (Keeling et al. 2001; Goulson et al. 2015; Jolles et al. 2021), with
56 host evolutionary dynamics (and especially changes in sociality) mostly ignored. This is presumably be-
57 cause the evolution of pathogen host traits, and moreover complex behavioural traits such as sociality, is
58 expected to be slow and not immediately relevant. Since important aspects of animal ecology, including
59 the transmission of foraging tactics (Klump et al. 2021) and migration routes (Guttal and Couzin 2010;
60 Jesmer et al. 2018), depend on social interactions, it is necessary to understand the long-term conse-
61 quences of pathogen introductions for animal societies. Climate change is only expected to make novel
62 pathogen introductions more common (Sanderson and Alexander 2020; Carlson et al. 2022), making
63 such studies more urgent.

64 Theory suggests that animal sociality evolves to balance the value of social associations against the
65 risk of pathogen transmission (Bonds et al. 2005; Prado et al. 2009; Ashby and Farine 2022). How-
66 ever, analytical models often reduce animal sociality to single parameters, while it actually emerges
67 from individual decisions conditioned on multiple internal and external cues. Social decision-making
68 and movement often also vary among individuals (Tanner and Jackson 2012; Wolf and Weissing 2012;
69 Spiegel et al. 2017; Gartland et al. 2021), but analytical models are unable to include individual dif-
70 ferences in sociability. Epidemiological models based on contact networks can incorporate individual
71 variation in social behaviour by linking these differences to positions in a social network (White et al.
72 2017; Albery et al. 2020; 2021). Yet network models often cannot capture fine-scale feedbacks between
73 individuals' social and spatial positions (Albery et al. 2020; 2021), nor spatial variation in infection risk
74 (Albery et al. 2022), making such models sensitive to both the network formation process, and to sam-
75 pling biases in empirical data collection (White et al. 2017).

76 Mechanistic, individual-based simulation models (IBMs) suggest themselves as a natural solution;
77 they can incorporate substantial ecological detail, including explicit spatial settings (DeAngelis and
78 Diaz 2019), and detailed disease transmission (White et al. 2018*a,b*; Scherer et al. 2020; Lunn et al.
79 2021). Individual-based models hitherto have focused on immediate epidemiological outcomes, such
80 as infection persistence, and do not have an evolutionary component (White et al. 2018*b*; Scherer et al.
81 2020; Lunn et al. 2021). Incorporating an evolutionary component to movement-disease IBMs could
82 allow predictions on important feedbacks between the ecological outcomes of infectious disease and the
83 consequences for the evolution of host behaviour (Cantor et al. 2021). This could include the emergence
84 of tradeoffs in the costs and benefits of sociability (Gartland et al. 2021), with cascading ecological and
85 social effects (Tanner and Jackson 2012; Spiegel et al. 2017; Monk et al. 2022; Webber et al. 2022). The
86 range of animal taxa at risk from a wide array of pathogens and parasites (Sanderson and Alexander
87 2020; Carlson et al. 2022) makes it important to conceive of models that can capture the key features of
88 diverse host-pathogen dynamics and offer broad conceptual insights (White et al. 2018*a,b*).

89 We built a model that seeks to capture the essential elements of pathogen (or parasite) transmission
90 among animals foraging on patchily distributed resources — this is a common behavioural context

91 shared by many potential host species (White et al. 2018a,b). We examined the eco-evolutionary con-
92 sequences of the introduction of a pathogen into a novel host population (such as during cross-species
93 spillover: Bastos et al. 2000; Blehert et al. 2009; Fereidouni et al. 2019; Scheele et al. 2019; Sanderson and
94 Alexander 2020; Carlson et al. 2022; Kuchipudi et al. 2022; Monk et al. 2022; Wille and Barr 2022). In
95 our evolutionary, spatial, individual-based simulation, we modelled the repeated introduction of an in-
96 fectious pathogen to populations that had already evolved foraging movement strategies in its absence.
97 Our model could be conceived as an abstract representation of, among others, spillovers of foot-and-
98 mouth disease from buffalo to impala (Bastos et al. 2000; Vosloo et al. 2009), or sarcoptic mange from
99 llamas to vicuñas (Monk et al. 2022), current and historic spread of avian influenza among sea- and
100 wading bird species (H5N8 and Related Influenza Viruses 2016; Wille and Barr 2022), or SARS-CoV-2
101 from humans to deer (Chandler et al. 2021; Kuchipudi et al. 2022).

102 We compared how social information was used in movement strategies evolved before and after
103 pathogen introduction, and the ecological outcomes for individual intake, movement, and associations
104 with other foragers. Using both IBMs and network epidemiological models (Bailey 1975; White et al.
105 2017; Stroeymeyt et al. 2018; Wilber et al. 2022), we examined whether pathogen-risk adapted popula-
106 tions were more resilient to the spread of infectious disease than their pathogen-risk naive ancestors.
107 We also investigated the effect of landscape productivity and the cost of infection, which are both ex-
108 pected to influence the selection imposed by pathogen transmission (Hutchings et al. 2000; Almberg
109 et al. 2015; Ezenwa et al. 2016). Overall, we provide a theoretical framework broadly applicable to novel
110 host-pathogen introduction scenarios, and demonstrate the importance of including evolutionary dy-
111 namics in movement-disease models.

112 **Methods**

113 We implemented an individual-based simulation model to represent foraging animals ('foragers') seek-
114 ing discrete, immobile, depleteable food items (see *SI Appendix Fig. S1 – S2*) (Spiegel et al. 2017; Gupte
115 et al. 2021). Food items are distributed over a two-dimensional, continuous-space resource landscape
116 with wrapped boundaries (a torus). Our model, similar to previous eco-evolutionary individual based
117 models (Getz et al. 2015; Gupte et al. 2021; Netz et al. 2021), has two distinct timescales: (1) an eco-
118 logical timescale comprising of T timesteps that make up one generation ($T = 100$ by default), and (2)
119 an evolutionary timescale consisting of 5,000 generations (G). At the ecological timescale, individuals
120 sense local counts of food items and competitors, move according to inherited movement strategies, and
121 forage for food. At the same timescale, individuals that carry an infectious, fitness-reducing pathogen,
122 may, when in close proximity with uninfected individuals, pass on the pathogen with a small prob-
123 ability (see *Pathogen Transmission and Disease Cost*). At the evolutionary timescale, individuals re-
124 produce and transmit their movement strategies (see *Starting Location and Inheritance of Movement*

125 *Rules*) to their offspring. The number of offspring is linked both to individuals' success in finding
126 and consuming food items, and to the duration that they were infected by the pathogen at the eco-
127 logical timescale. The model was implemented in R and C++ using Rcpp (Eddelbuettel 2013; R Core
128 Team 2020) and the *Boost.Geometry* library for spatial computations (www.boost.org); model code is at
129 github.com/pratikunterwegs/pathomove.

130 *Distribution of Food Items*

131 Our landscape of 60×60 units contains 1,800 discrete food items, which are clustered around 60 re-
132 source 'kernels', for a resource density of 0.5 items per unit² (see *SI Appendix Fig. S1 – S2*). This prevents
133 synchronicity in the availability and regeneration of food items. Each available food item can be sensed
134 and harvested by foraging individuals (see below). Once harvested, another food item is regenerated at
135 the same location after a fixed regeneration time R , which is set at 50 timesteps by default; alternative
136 values of 20 and 100 timesteps represent high and low productivity landscapes respectively. Food item
137 regeneration is delinked from population generations. Thus the actual number of available food items
138 is almost always in flux. In our figures and hereafter, we chose to represent R as the number of times a
139 food item would regenerate within the timesteps in a single generation T (default = 100), resulting in
140 R values of 1, 2, and 5 for regeneration times of 100, 50 (the default), and 20 timesteps. Items that are
141 not harvested remain on the landscape until they are picked up by a forager. Each food item must be
142 processed, or 'handled', by a forager for T_H timesteps (the handling time, default = 5 timesteps) before
143 it can be consumed (Ruxton et al. 1992; Gupte et al. 2021). The handling time dynamic is well known
144 from natural systems in which there is a lag between finding and consuming a food item (Ruxton et al.
145 1992).

146 *Individual Foraging and Movement*

147 *Foraging.* Individuals forage in a randomised order, harvesting the first available food item within
148 their movement and sensory range ($d_S = d_M$, a circle with a radius of 1 unit (see *SI Appendix Fig.*
149 *S1 – S2*). Once harvested, the item is no longer available to other individuals, leading to exploitation
150 competition among nearby foragers. Furthermore, the location of the item also yields no more cues to
151 other foragers that an item will reappear there, reducing direct cues by which foragers can navigate to
152 profitable clusters of food items. Individuals that harvest a food item must handle it for T_H timesteps
153 (default = 5 timesteps), while all individuals not handling a food item are considered idle (Ruxton et
154 al. 1992; Gupte et al. 2021). As handlers are immobilised at the location where they encountered food,
155 they may be good indirect indicators of the location of a resource cluster ('social information') (Danchin
156 et al. 2004; Romano et al. 2020; Gupte et al. 2021). Once individuals finish handling a food item, they

157 return to the non-handling, searching state.

158 *Movement.* Our model individuals move in small, discrete steps of fixed size ($d_M = 1$ unit). Each step
159 is chosen based on the individuals' assessment of local environmental cues, and this assessment is made
160 using evolved movement strategies (as in Gupte et al. 2021; Netz et al. 2021). First, individuals scan their
161 current location, and five equally spaced points around their position, at a distance of 1 unit for three
162 cues (d_S , see *SI Appendix Fig. S1 – S2*): the number of food items (F), the number of foragers handling
163 a food item ('handlers': H) and the number of idle foragers not handling a food item ('non-handlers':
164 N). Individuals assign a suitability (see Gupte et al. 2021; Netz et al. 2021) to their current position and
165 each of the five locations, using their inherited preferences for each of the cues: $S = s_F F + s_H H + s_N N$
166 $+ \epsilon$. The preferences s_F , s_H , and s_N for each of the three cues are heritable from parents to offspring,
167 while ϵ is a very small error term drawn for each location, to break ties among locations. The values of
168 each of the cue preferences *relative to each other* determine individuals' movement strategies (Gupte et
169 al. 2021). All individuals move simultaneously to the location to which they have assigned the highest
170 suitability ('step selection') (akin to step-selection; Fortin et al. 2005); this may be their current location,
171 in which case individuals are stationary for that timestep. Since individuals may differ in their inherited
172 preferences for each of the three cues, two individuals at the same location may make quite different
173 movement decisions based on the same local cues. Handlers, however, are considered immobile and
174 do not make any movement decisions.

175 *Pathogen Transmission and Disease Cost*

176 We modelled circumstances that are expected to become increasingly common due to rapid global
177 changes; the population evolves for $3/5^{\text{th}}$ of the simulation (until $G = 3,000$; of 5,000) in the absence of
178 a pathogen, after which a pathogen is introduced in each generation until the end of the simulation (G
179 $= 5,000$). Our model captures some essential features of pathogen or parasite transmission among ani-
180 mals (White et al. 2017): the pathogen may transmit from infected host individuals to their susceptible
181 neighbours with a per-timestep probability p of 0.05. This transmission is only possible when the two
182 individuals are within a the transmission distance, d_β . For simplicity, we set d_β to be the movement
183 range (1 unit). Once transmitted, the pathogen is assumed to cause a chronic disease which reduces
184 host energy stores by a fixed amount called δE in every following timestep; δE is set to 0.25 by default
185 (alternative values: 0.1, 0.5). Since novel pathogen introductions can periodically re-occur in natural
186 environments (Bastos et al. 2000; Vosloo et al. 2009; Almberg et al. 2015; Goulson et al. 2015; Jolles
187 et al. 2021; Carlson et al. 2022; Wille and Barr 2022), we set up our model such that the pathogen was
188 introduced to 4% of individuals in each generation ($N = 20$; 'primary infections'). This is necessary to
189 kick-start the pathogen-movement eco-evolutionary feedback dynamics, and populations may indeed
190 repeatedly acquire novel pathogens (or strains) through external sources, such as infected individuals

191 of other spatially overlapping species (e.g. Bastos et al. 2000; Keeling et al. 2001; Vosloo et al. 2009;
192 Chandler et al. 2021; Carlson et al. 2022; Kuchipudi et al. 2022; Monk et al. 2022; Wille and Barr 2022).
193 For completeness, we also considered scenarios in which novel pathogen introductions only occur spo-
194 radically in the generations after the initial event, rather than in every generation (see *SI Appendix*).

195 *Starting Location and Inheritance of Movement Rules*

196 For simplicity, we considered a population of haploid individuals with discrete, non-overlapping gen-
197 erations, and asexual inheritance. At the end of the parental generation, the net lifetime energy of
198 each individual was determined as the difference of the total energy gained through food intake and
199 the energy lost through infection. In the *SI Appendix*, we also consider an alternative implementation
200 in which potential immune resistance against the pathogen requires a certain percentage of individual
201 intake, reducing the value of each food item. The parental population produces an offspring population
202 (of the same size) as follows: to each offspring, a parent is assigned at random by a weighted lottery,
203 with weights proportional to lifetime net energy (an algorithm following the replicator equation) (Hof-
204 bauer and Sigmund 1988; Hamblin 2013). This way, the expected number of offspring produced by a
205 parent is proportional to the parent's lifetime success (Hofbauer and Sigmund 1988). The movement
206 decision-making cue preferences s_F , s_H , and s_N are subject to independent random mutations with a
207 probability of 0.01. The mutational step size (either positive or negative) is drawn from a Cauchy dis-
208 tribution with a scale of 0.01 centred on zero. Thus, while the majority of mutations are small, there
209 can be a small number of very large mutations. As in real ecological systems, individuals in the new
210 generation are initialised around the location of their parent (within a standard deviation of 2.0), and
211 thus successful parents give rise to local clusters of offspring (see an alternative implementation in *SI*
212 *Appendix*).

213 *Model Output*

214 *Social Information Use.* To understand the evolution of movement strategies, and especially how indi-
215 viduals weighed social information, we recorded the population's evolved cue preferences in every sec-
216 ond generation, and interpreted them using the 'behavioural hypervolume' approach (Bastille-Rousseau
217 and Wittemyer 2019). We classified individuals based on how they used social information — the
218 presence and status of competing foragers — into four social movement classes: (1) agent avoiding,
219 if $s_H, s_N < 0$, (2) agent tracking, if both $s_H, s_N > 0$, (3) handler tracking, if $s_H > 0, s_N < 0$, and (4)
220 non-handler tracking, if $s_H < 0, s_N > 0$. We calculated the relative importance of social cues — H, N —
221 to each individual's movement strategy as $SI_{imp} = (|s_H| + |s_N|) / (|s_H| + |s_N| + |s_F|)$, with higher values
222 indicating a greater importance of social cues.

223 *Proximity-Based Social Network.* We created a proximity-based adjacency matrix by counting the num-
224 ber of times each individual was within the sensory and pathogen transmission distance d_β ($= d_S, d_M$
225 $= 1$ unit) of another individual (Whitehead 2008; Wilber et al. 2022). We transformed this matrix into
226 an undirected social network weighted by the number of pairwise encounters: in a pairwise encounter,
227 both individuals were considered to have associated with each other (White et al. 2017). The strength
228 of the connection between any pair was the number of times the pair were within d_β of each other over
229 their lifetime. We logged encounters and constructed social networks after every 10% of the total gener-
230 ations (i.e., every 500th generation), and at the end of the simulation, and omitted ephemeral pairwise
231 associations with a weight < 5 .

232 *Model Analysis*

233 We plotted the mix of social information-based movement strategies evolved across generations in each
234 parameter combination. Focusing on our default scenario ($\delta E = 0.25, R = 2$), we visualised the mean
235 per-capita distance moved, mean per-capita intake, and mean per-capita encounters with other for-
236 agers. We examined how the three main social movement strategies — agent avoidance, agent track-
237 ing, and handler tracking — changed in frequency over generations. We also examined differences
238 among strategies in the movement distance, associations with other agents, and frequency of infection,
239 after they had reached an eco-evolutionary equilibrium following pathogen introduction ($G > 3,500$).
240 We visualised the proximity based social networks of populations in a representative scenario ($\delta E =$
241 $0.25, R = 2$), focusing on the generations just before and after the pathogen introduction events begin
242 (pre-introduction: $G = 3,000$; post-introduction: $G = 3,500$). We plotted the numbers of individuals
243 infected in each generation after pathogen introduction to examine whether evolutionary changes in
244 movement strategies actually reduced infection spread. We also ran simple network epidemiological
245 models on the emergent individual networks in generations 3,000 and 3,500 (Bailey 1975; White et al.
246 2017; Stroeymeyt et al. 2018; Wilber et al. 2022), for robust comparisons of potential pathogen spread
247 in pathogen-risk naive and pathogen-risk adapted populations, respectively.

248 *Data and Code Availability*

249 The *Pathomove* simulation model code is available on Zenodo at <https://zenodo.org/record/6782640>,
250 and on Github at github.com/pratikunterwegs/pathomove. Code to run the simulations and analyse the
251 output is on Zenodo at <https://zenodo.org/record/6782665>, and on Github at: [github.com/pratikunterwegs/patho-](https://github.com/pratikunterwegs/pathomove-evol)
252 [move-evol](https://github.com/pratikunterwegs/pathomove-evol).

Results

253

254 In our model, individuals move and forage on a landscape with patchily distributed food items, and se-
255 lect where next to move in their vicinity, based on inherited preferences for environmental cues — food
256 items, and other individuals (see *SI Appendix Fig. S1*). Food items, once consumed, regenerate at a rate
257 R , and pathogen infection imposes a per-timestep cost δE . We classified individuals' social movement
258 strategies in our model using a simplified 'behavioural hypervolume' approach (Bastille-Rousseau and
259 Wittemyer 2019), based on the sign of their preferences for successful foragers handling a food item
260 ('handlers', preference s_H), and for unsuccessful foragers still searching for food ('non-handlers', pref-
261 erence s_N). In our default scenario, $R = 2$, food regenerates twice per generation, and $\delta E = 0.25$, i.e.,
262 consuming 1 food item offsets 4 timesteps of infection. Over the 3,000 generations before the intro-
263 duction of the pathogen, populations reached an eco-evolutionary equilibrium where the commonest
264 social movement strategy was to prefer moving towards both handlers and non-handlers ('agent track-
265 ing'; $s_H, s_N > 0$; but see below) (Fig. 1A).

Rapid Evolutionary Shift in Social Movement Strategies Following Pathogen

266

Introduction

267

268 Introducing an infectious pathogen to 4% ($n = 20$) of individuals in each generation (after $G = 3,000$),
269 leads to a remarkably rapid evolutionary shift — within only 25 generations of pathogen introduction
270 — in how social information is incorporated into individuals' movement strategies. There is a marked
271 increase in the frequency of individuals that track successful foragers, but avoid non-handlers ('handler
272 tracking'; $s_H > 0$, but $s_N < 0$) (Fig. 1A; $3,000 < G < 3,025$). Surprisingly, after a brief period (in
273 evolutionary terms) of handler tracking being the most common strategy, a third strategy also becomes
274 more common: avoiding both handlers and non-handlers ('agent avoiding'; $s_H, s_N < 0$). Within 250
275 generations after pathogen introduction, agent avoiding becomes as common as the handler tracking
276 strategy, and this appears to be a stable equilibrium that is maintained until the end of the simulation
277 (2,000 generations after pathogen introduction; Fig. 1A). The *SI Appendix* shows how the occurrence
278 of rapid evolutionary shifts is broadly robust to modelling assumptions; in brief, such shifts occur even
279 when individuals cannot benefit from evolved adaptation to local conditions (Badyaev and Uller 2009),
280 and when the pathogen saps a percentage, rather than an absolute value, from daily intake.

281 In addition to qualitative changes in social movement strategies, pathogen introduction also leads
282 to social information becoming more important to movement decisions. Prior to pathogen introduc-
283 tion ($G < 3,000$), individuals' handler- and non-handler preferences ($|s_H| + |s_N|$; taken together, social
284 information) barely influence their movement strategies (Fig. 1B). These are instead guided primar-
285 ily by the preference for food items (s_F ; see *Model and Analysis*; see also *Supplementary Material Fig.*

286 1). Social movement decisions are joint outcomes of individual preferences for social cues and the cue
287 value: consequently, in clustered populations (see below), even small positive values of s_H and s_N lead
288 to strong emergent sociality. After pathogen introduction, there is a substantial increase in the average
289 importance of individuals' preferences (or aversions) for the presence of other foragers (Fig. 1B). How-
290 ever, there is significant variation among individuals in the importance of social information to their
291 movement strategies, with distinct evolved polymorphisms that vary substantially between simulation
292 replicates (Fig. 1B).

293 *Disease-dominated Ecological Cascade Due to Evolutionary Shift in Movement* 294 *Strategies*

295 The evolutionary shift in social movement strategies causes a drastic change in ecological outcomes
296 (Fig. 1C – E; see *SI Appendix Fig. S3* for other scenarios). There is a sharp increase in mean distance
297 moved by individuals; while pre-introduction individuals moved 35% of their lifetimes on average (i.e.,
298 35 timesteps; handling for the remainder), post-introduction, individuals move for 80% of their life-
299 times (i.e., 80 timesteps; Fig. 1C). The handler tracking and agent avoiding strategies lead individuals
300 to move away from groups of individuals ('dynamic social distancing'; Pusceddu et al. 2021). Individu-
301 als being most likely to be found near resource clusters, this leads to movement away from productive
302 areas of the landscape. Consequently, there is a rapid, four-fold drop in mean per-capita intake after
303 pathogen introduction (Fig. 1D). The concurrent, near 100-fold drop in encounters between individu-
304 als after pathogen introduction (Fig. 1E) suggests that most encounters were likely taking place on or
305 near resource clusters. The reductions in intake observed are equivalent to those expected from halv-
306 ing landscape productivity (*SI Appendix Fig. S3*). Our model shows how even a non-fatal pathogen, by
307 influencing the evolution of movement strategies, can have substantial indirect ecological effects — a
308 disease dominated ecological cascade (Monk et al. 2022).

309 *Co-existence of Social Movement Strategies*

310 At eco-evolutionary equilibrium ($G > 3,500$) the relationship between movement and avoiding associ-
311 ations (and further, infection) is mediated by individual differences in how exactly social information
312 is incorporated into movement strategies. Individuals using the agent avoiding strategy move more
313 than handler tracking ones (Fig. 2A), about 85% of their lifetime (default scenario: $R = 2$; $\delta E = 0.25$).
314 At this limit, every step moved allows them to avoid approximately 2 encounters with other individu-
315 als. Handler tracking individuals move much less ($\sim 60\% - 80\%$), but are able to avoid approximately
316 20 encounters with other individuals with every extra step. These differences may explain why agent
317 avoiding and handler tracking individuals have similar mean infection rates, at $\sim 25\%$ and $\sim 33\%$ respec-

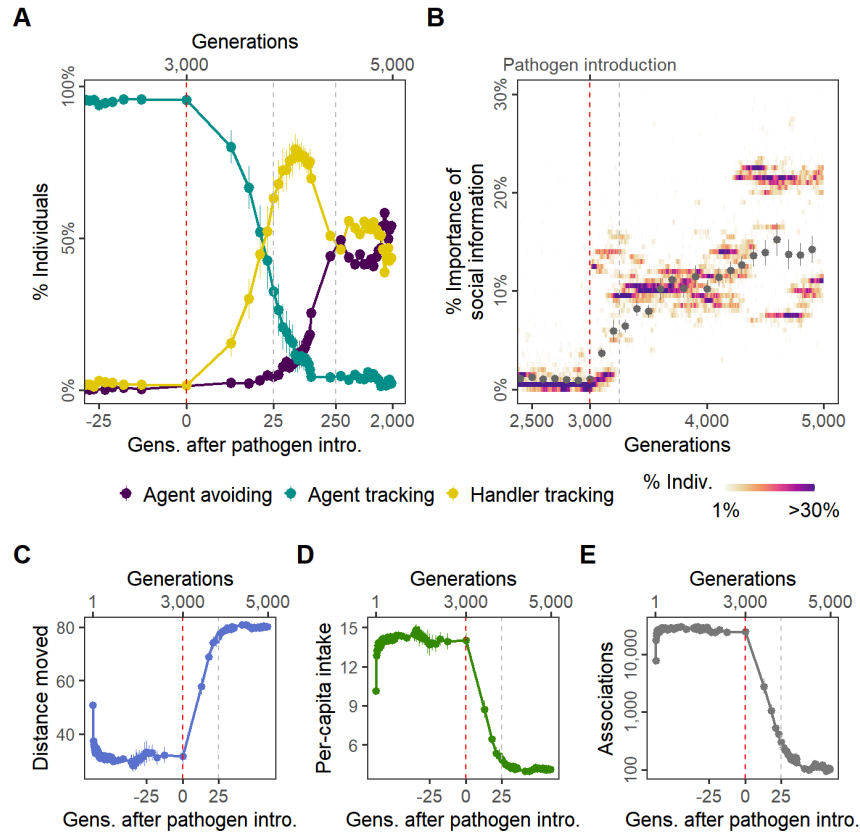


Figure 1: Pathogen introduction leads to rapid evolutionary changes in social information use, with cascading effects on population ecological outcomes. (A)

Before pathogen introduction in the default scenario ($R = 2$, $\delta E = 0.25$), populations rapidly evolve a social movement strategy that tracks all other individuals (‘agent tracking’; $G \leq 3,000$) — however, their overall movement strategy is primarily guided by the presence of food items (B). Pathogen introduction leads to the rapid replacement, within 25 generations, of agent tracking with ‘handler tracking’ (preference for successful foragers; $3,000 < G < 3,025$). Within 250 generations, ‘agent avoidance’ (avoidance of both successful and unsuccessful foragers; $G > 3,250$) also becomes common, stably co-existing with the handler tracking strategy in an eco-evolutionary equilibrium. (B) After pathogen introduction ($G > 3,000$), the importance of social cues (the presence of other individuals; the sum of the absolute, normalised preferences sH , sN) increases substantially on average (grey points). Additionally, there is significant variation in the importance of social cues to individuals (shaded regions), which is not captured by the mean or standard error. At $G = 4,500$, for example, social information comprises $\approx 10\%$ of some individuals’ movement strategies, but some individuals have evolved a stronger weight for social cues ($> 20\%$). The rapid change in social movement strategies following pathogen introduction has cascading effects on ecological outcomes. Individuals, which have evolved strong aversions to at least some kinds of foragers (depending on their strategy), (C) move more on average, (D) have only 25% of the pre-pathogen average intake, and (E) have 100-fold fewer associations with other individuals. All panels show data averaged over 10 replicates, but shaded region in panel B shows only a single replicate for clarity.

318 tively (Fig. 2B). All other strategies, especially the agent tracking strategy common in pre-introduction
319 populations, are barely able to translate increased movement into fewer associations (Fig. 2A). These
320 strategies have a wide range of infection rates (Fig. 2B), potentially because they are very rare — these
321 likely represent mutants that do not give rise to persistent lineages.

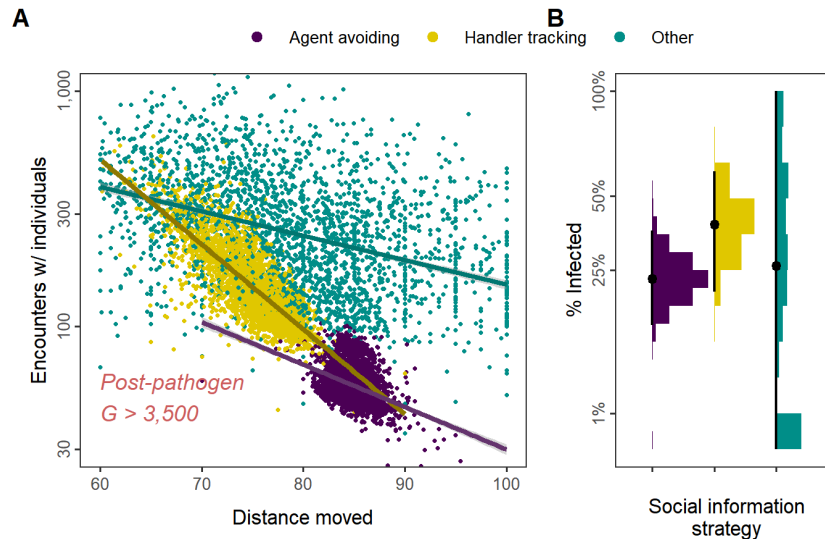


Figure 2: Social movement strategies trade movement for associations through dynamic social distancing, leading to differences in infection rates. In post-introduction populations at eco-evolutionary equilibrium ($G > 3,500$), (A) both agent avoiding and handler tracking individuals can reduce encounters with other individuals by moving to avoid other foragers (dynamic social distancing). Handler tracking individuals have many more encounters than agent avoiding individuals, but surprisingly, are better able to reduce encounters through increased movement. Individuals using other strategies (mostly agent tracking) have a wider range of movement distances, but cannot efficiently avoid other foragers by moving more. (B) Avoiding all other foragers leads to marginally lower infection rates than tracking successful foragers (and avoiding unsuccessful ones; handler tracking). Surprisingly, rare pre-introduction strategies such as following any nearby individuals (agent tracking) may also have low infection rates, potentially due to their rarity. Panel A shows linear model fits with a log scale Y-axis; panel B shows infection rates; all data represent generation- and replicate-specific means ($G > 3,500$; $R = 2$, $\delta E = 0.25$).

322

Reorganisation of Spatial-social Structure

323 Following pathogen introduction, the mixture of individual-level movement strategies elicits a substan-
324 tial re-organisation of emergent spatial and social structure at the population level. Pre-introduction
325 populations are strongly clustered in space (Fig. 3A), due to movement strategies that favour following
326 most other foragers. This spatial proximity means that most individuals encounter each other at least
327 once, leading to numerous unique partners (the ‘degree’) for each forager (Fig. 3 inset 1: blue). In con-

328 trast, the spread-out networks in pathogen-risk adapted populations suggest that most foragers move
329 substantially from their initial locations over their lifetime, associating only ephemerally with foragers
330 from all over the landscape (Fig. 3B). This reflects movement strategies which lead to near-perpetual
331 movement to avoid associations; a sort of dynamic social distancing seen in real animal societies under
332 risk of pathogen spread (Stroeymeyt et al. 2018; Weinstein et al. 2018; Pusceddu et al. 2021; Stockmaier
333 et al. 2021). This dispersed population structure means that most pathogen-risk adapted foragers en-
334 counter fewer than 10% of the population over their lifetime (Fig. 3 inset 1: red).

335 *Pathogen-risk adapted Movement Strategies Make Animal Societies More* 336 *Resilient to the Spread of Disease*

337 Nearly every individual in the generations just after pathogen introduction was infected. However,
338 tracking the evolutionary change in movement strategies, the number of infected individuals fell to just
339 about 50% within 25 generations (Fig. 3C). To examine potential pathogen spread in pre-introduction
340 populations, we ran a simple epidemiological model on the social networks emerging from individuals'
341 movements before and after pathogen introduction (pre-introduction: $G = 3,000$; post-introduction: G
342 $= 3,500$). We modelled two diseases, (i) first, a disease requiring one encounter, and (ii) second, a disease
343 requiring ten encounters between individuals for a potential transmission event (transmission rate $\beta =$
344 5.0 , recovery rate $\gamma = 1.0$).

345 Both the single encounter and multiple encounter diseases would infect 75% – 80% of individuals
346 when spreading through the networks of pre-introduction populations (Fig. 3D) pathogen-risk adapted
347 populations' social networks are more resilient to both the single encounter and multiple encounter
348 disease, compared to their pre-introduction, pathogen-risk naive ancestors (Fig. 3D), as these social
349 networks are sparser and individuals are more weakly connected (Fig. 3D; see Fig. 3D). Less than 60%
350 of post-introduction populations were finally infected by the single encounter disease, compared with
351 $> 75\%$ of pre-introduction, pathogen-risk naive ancestors; in pathogen-risk adapted populations, the
352 spread of the multiple encounter disease was even slower (ever infected: $\approx 20\%$).

353 *Usefulness of Social Information and Infection Cost Influence Evolution of* 354 *Social Movement Strategies*

355 We further explored the effect of two ecological parameters, landscape productivity ($R \in 1, 2, 5$) and
356 infection cost per timestep ($\delta E \in 0.1, 0.25, 0.5$) on simulation outcomes. Before pathogen introduction,
357 landscape productivity alone determines the value of social information, and thus which social move-
358 ment strategies evolve (Fig. 4). On low-productivity landscapes ($R = 1$), social information is valuable

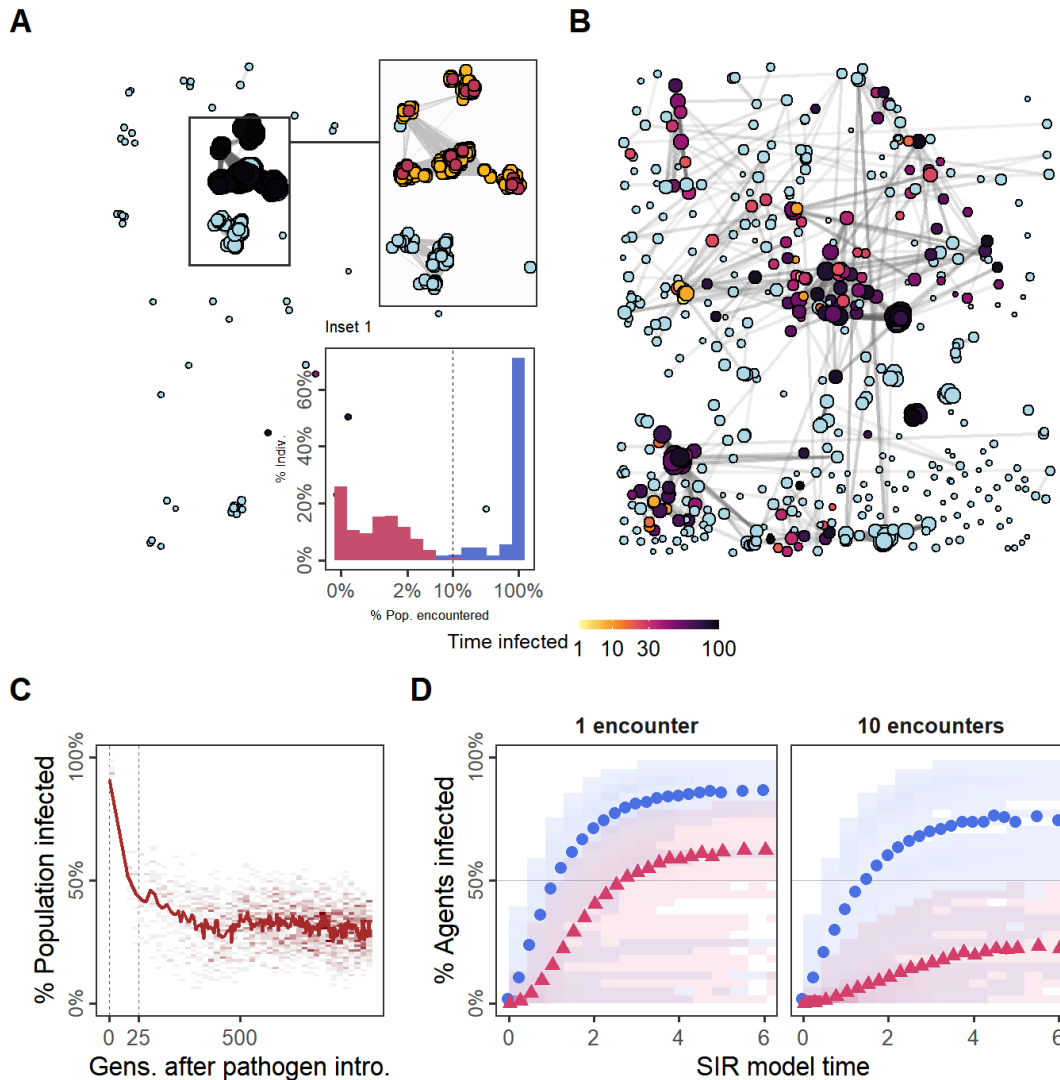


Figure 3: Reduced spatial-social clustering and disease transmission in populations adapted to the presence of an infectious pathogen. pathogen-risk naive populations (**A**; $G = 3,000$) are much more spatially clustered than pathogen-risk adapted populations (**B**; $G = 3,500$), and are thus rapidly infected (red: primary infections; yellow: secondary infections; blue: never infected). Pre-introduction individuals encounter many more unique neighbours (**inset 1**, blue) than pathogen-risk adapted individuals (**inset 1**; red). Dashed grey line represents 10% of individuals encountered ($N = 50$). Main panels show social networks from a single replicate of the default scenario ($R = 2$, $\delta E = 0.25$), insets show 10 replicates. Nodes represent individuals positioned at their final location. Connections represent pairwise encounters, and node size represents encounters (larger = more encounters). Darker node colours indicate longer infection (light blue = no infection). **(C)** In the first generations following pathogen introduction, nearly every single individual in the population is infected. However, within 25 generations, tracking the evolutionary shift towards movement strategies that avoid some or all other individuals, only about 50% of individuals are ever infected; this drops to a stable 30% within 500 generations after pathogen introduction. **(D)** The progression of two hypothetical diseases, requiring a single encounter, or 10 encounters for a potential transmission, on emergent social networks. The transmission of both diseases is reduced in populations with disease-adapted movement strategies (pre-introduction: $G = 3,000$, blue circles; post-introduction: $G = 3,500$, red triangles). Subfigures in panel D show means of 25 SIR model replicates (transmission rate $\beta = 5.0$, recovery rate $\gamma = 1.0$), run on emergent social network; both panels represent 10 simulation replicates the default scenario.

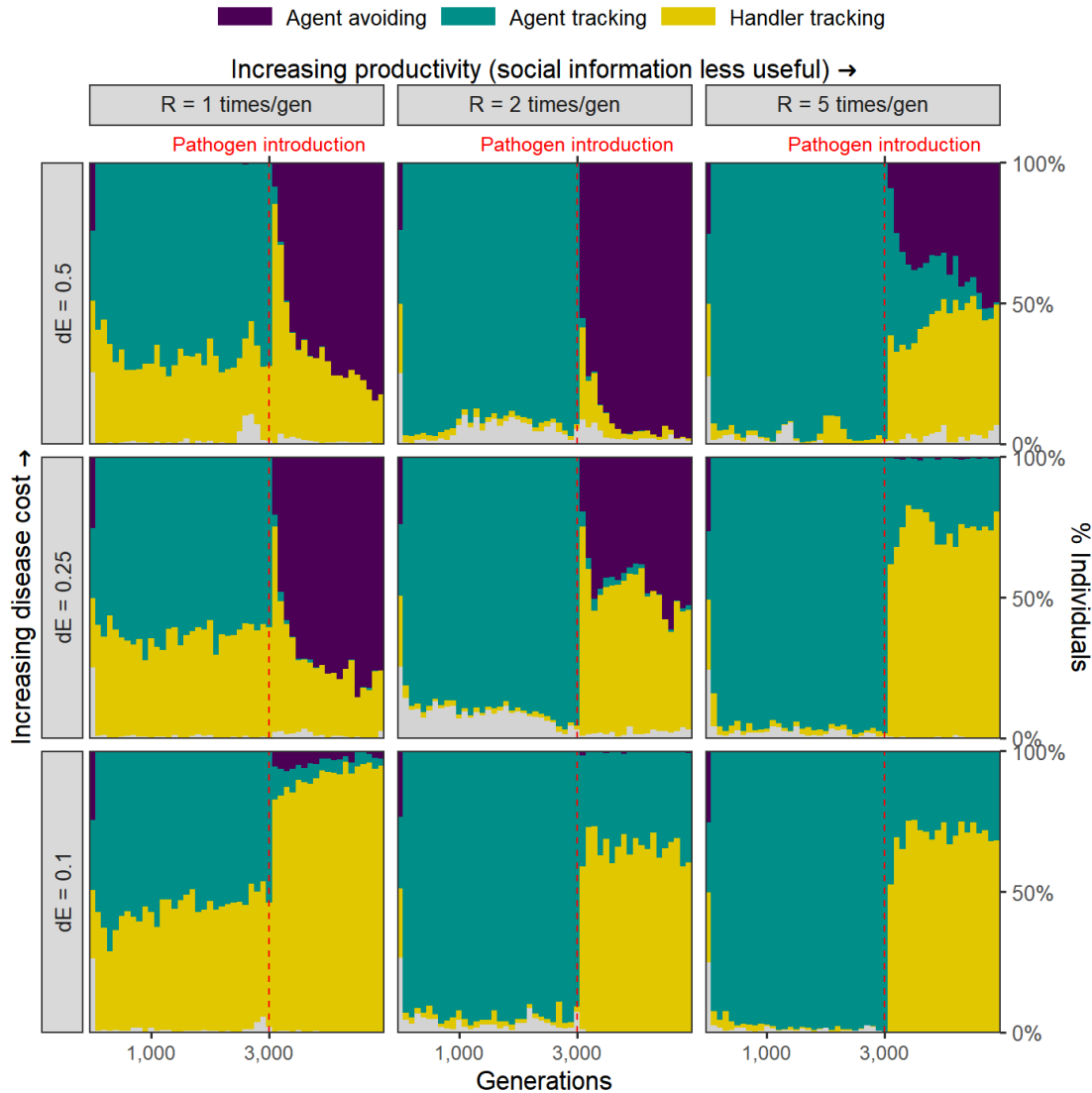


Figure 4: The balance of infection cost and the usefulness of social information together shape the rapid evolutionary change in movement strategies triggered by pathogen introduction.

Pre-introduction ($G = 3,000$; dashed line) populations contain a mix of individuals that either track all foragers (agent tracking), or only successful foragers (handler tracking). Handler tracking is more common on low-productivity landscapes ($R = 1$), where social information is more useful to find patchily distributed resources. After pathogen introduction, handler tracking rapidly becomes the most common strategy when the apparent usefulness of social information is greater than the cost of infection. This occurs both when productivity is low ($R = 1$) and infection costs are low ($\delta E = 0.1$), but also when productivity is high ($R = 5$) with intermediate infection costs ($\delta E = 0.25$). When the cost of infection outweighs the apparent usefulness of social information, the agent avoidance (avoiding both successful and unsuccessful foragers) emerges and rapidly becomes a common strategy ($\delta E = 0.5$; $\delta E = 0.25$, $R = 1$). In scenarios of high landscape productivity combined with low infection costs (e.g. $R = 5$, $\delta E = 0.1$), the agent tracking strategy persists in a large proportion after pathogen introduction, as these individuals can balance disease costs with intake alone. All panels show mean frequencies over 10 replicate simulations in 100 generation bins; frequencies are stacked. Grey areas show the relatively uncommon ‘non-handler’ tracking strategy.

359 as direct resource cues are scarce; here, the handler-tracking strategy persists. On high-productivity
360 landscapes ($R \in 2, 5$), social information is less valuable as individuals can directly detect food items
361 more often; here, the agent tracking strategy is most common. Across parameter combinations, the in-
362 troduction of the infectious pathogen leads to a rapid evolutionary shift in social movement strategies.
363 The benefits of social information, and infection cost jointly determine how pathogen introduction al-
364 ters the mix of social movement strategies, but populations generally shift away from indiscriminate
365 agent tracking, as that strategy is associated with higher infection risk (see Fig. 3A).

366 When the benefit of social information is equivalent to the cost of infection, the handler tracking
367 strategy is common ($R = 1, \delta E = 0.1$; $R = 5, \delta E = 0.25$). When apparent social information bene-
368 fits are lower than infection costs (e.g. $\delta E = 0.5$), the agent avoiding strategy is common. The effect
369 of landscape productivity in obviating a sensitivity to social information cues (especially, conspecific
370 status) is also eroded by pathogen introduction. On high-productivity landscapes where individuals
371 were indiscriminately social, ($R \in 2, 5, \delta E = 0.1$), the handler tracking strategy becomes common, as
372 individuals prioritise higher-quality social information (handlers, which indicate a resource cluster).
373 However, high landscape productivity can also compensate for the cost of infection, as evidenced by
374 the agent tracking strategy remaining prevalent: this is only possible if these individuals can consume
375 sufficient resources to overcome disease costs.

376 Discussion

377 Our general model captures important features of infectious pathogen (or parasite) transmission among
378 host animals in a (foraging) context that is relevant to most species. The combination of ecological, evo-
379 lutionary, and epidemiological dynamics in a spatial setting is unprecedented for movement-disease
380 models, and extends current understanding of animal spatial and social ecology (Kurvers et al. 2014;
381 Webber and Vander Wal 2018; Romano et al. 2020; Albery et al. 2021; Romano et al. 2021; Webber
382 et al. 2022). Presently, most movement-disease models are non-evolutionary (White et al. 2017; 2018b;
383 Scherer et al. 2020; Lunn et al. 2021), presumably because evolution is expected to be too slow to impact
384 epidemiological-ecological outcomes (Monk et al. 2022). We demonstrate the pitfalls of this assump-
385 tion: evolutionary transitions in sociality occur over fewer generations than required for the develop-
386 ment of key aspects of animal ecology, such as migration routes (Jesmer et al. 2018; Cantor et al. 2021).
387 We also demonstrate the tension inherent to sociality under the risk of an infectious pathogen, in an ex-
388 plicitly spatial context. Our work shows how qualitatively and quantitatively different social movement
389 strategies — making different trade-offs between social information and infection risk — can co-exist
390 in a single population (Wolf and Weissing 2012; Webber and Vander Wal 2018; Gartland et al. 2021;
391 Webber et al. 2022).

392 Prior to pathogen introduction, the value of social information influenced which social movement

393 strategies were evolved. Individuals initialised ('born') near their parent's final location may benefit
394 from 'ecological inheritance' (Badyaev and Uller 2009) of their parent's favourable position near re-
395 source clusters (see *SI Appendix Fig. S2, S4*). Avoiding potential competitors (and kin) thus correlates
396 with avoiding profitable areas, and this leads to the persistence of the indiscriminately social agent
397 tracking strategy, despite the evident costs of exploitation competition. In an alternative implemen-
398 tation with large-scale natal dispersal, handler tracking is the commonest strategy prior to pathogen
399 introduction (see *SI Appendix*). Following pathogen introduction, the agent tracking strategy of our
400 default scenario allows the disease to spread very easily among entire lineages of social individuals (see
401 Fig. 3A) (Kurvers et al. 2014). This neatly demonstrates why the risk of infection or parasitism could be
402 among the mechanisms underlying density dependence in natal dispersal decisions (Travis et al. 1999).

403 Following pathogen introduction, the evolutionary shift in social movement strategies is much more
404 rapid than the timescales usually associated with the evolution of complex traits such as sociality (about
405 25 generations). Avoiding potentially infectious individuals is a key component of navigating the 'land-
406 scape of disgust' (Weinstein et al. 2018). Our results show that sensitivity to cues of high pathogen
407 transmission risk can rapidly evolve following the introduction of a novel pathogen, with a complete
408 replacement of the hitherto dominant social strategy. The emergence of qualitative individual variation
409 in social movement strategies, and especially the trade-off between movement, associations, and infec-
410 tion risk also demonstrates the evolution of 'sociability as a personality trait' (Gartland et al. 2021). We
411 also find substantial individual variation in the quantitative importance of social cues overall, which
412 is a key component of the evolution of large-scale collective behaviours, such as migration (Guttal and
413 Couzin 2010). Our work suggests how, by leading to the necessary diversity in social movement strate-
414 gies, a novel pathogen may actually lay the groundwork for the evolution of more complex collective
415 behaviour. Nonetheless, the rapid decreases in social interactions should primarily prompt concern that
416 the evolutionary consequences of pathogen introduction could slow the transmission of, and erode, an-
417 imal culture (Cantor et al. 2021) — including foraging (Klump et al. 2021) and migration behaviours
418 (Guttal and Couzin 2010; Jesmer et al. 2018).

419 In our model, landscape productivity (R), is a proxy for the usefulness of sociality overall, as social
420 information is less useful when direct resource cues are abundant (high R). Social information bene-
421 fits in disease models often have no mechanistic relationship with the subject of the information (e.g.
422 food or predators) (Ashby and Farine 2022). In contrast, social information benefits in our model are
423 emergent outcomes of animal movement and foraging behaviour. Our predictions may help explain
424 intra- and inter-specific diversity in social systems across gradients of infection risk and the usefulness
425 of social information (Altizer et al. 2003; Sah et al. 2018), and studies tracking social movements and po-
426 tential for disease spread could form initial tests of our basic predictions (Wilber et al. 2022). While our
427 individuals do not die, the evolved pathogen-risk adapted, dynamic social distancing strategies (Stock-
428 maier et al. 2021) lead to a significant worsening (equivalent to a halving) of individuals' intake. In
429 real systems, this could increase populations' susceptibility to extreme climate change related mortal-

430 ity events (Fey et al. 2015).

431 More positively, animals may be able to adapt relatively quickly to the spillover and eventual persis-
432 tence of infectious pathogens, even when they cannot specifically detect and avoid infected individuals
433 (Altizer et al. 2003; Stroeymeyt et al. 2018; Pusceddu et al. 2021; Stockmaier et al. 2021). While the most
434 noticeable effect of pathogen outbreaks is mass mortality (Fey et al. 2015), even quite serious pathogens
435 — Sarcoptic mange (Almberg et al. 2015), foot-and-mouth disease (Bastos et al. 2000; Vosloo et al. 2009;
436 Jolles et al. 2021), SARS-CoV-2 (Chandler et al. 2021; Kuchipudi et al. 2022), and avian influenza (H5N8
437 and Related Influenza Viruses 2016; Wille and Barr 2022) among others — appear to spread at sub-lethal
438 levels for many years between lethal outbreaks. Our model shows how disease-dominated ecological
439 cascades (Monk et al. 2022) could occur even without mortality effects, due to evolutionary shifts in
440 sociality alone. The altered ecological state (here, less resource consumption, as in Monk et al. 2022)
441 may be maintained long after — and indeed because — a population has adapted to be less social in
442 the presence of a pathogen. Our work suggests that decreased sociality resulting from adaptation to
443 a novel pathogen could slow the transmission of future novel pathogens. While decreased sociality
444 could also reduce the prevalence of previously endemic pathogens adapted to a more social host, it may
445 also degrade ‘social immunity’ through reduced sharing of beneficial commensal microbes, or of low,
446 immunising doses of pathogens (Almberg et al. 2015; Ezenwa et al. 2016).

447 Our model results are contingent upon sustained introduction of the pathogen (or its novel strains)
448 to host populations. More sporadic introductions (once every few generations) apparently do not cause
449 evolutionary shifts in social movement (*SI Appendix*). Yet repeated pathogen and parasite introductions
450 among susceptible populations appear to be quite common (Bastos et al. 2000; Vosloo et al. 2009; Levi et
451 al. 2012; H5N8 and Related Influenza Viruses 2016; Scherer et al. 2020; Jolles et al. 2021; Wille and Barr
452 2022). Such introductions are often detected only among easily observed groups such as birds (Wille
453 and Barr 2022), or after evident mass mortality events (Fey et al. 2015; Fereidouni et al. 2019). Seasonal
454 host-pathogen dynamics could and do keep pathogens circulating in reservoir hosts, with regular pulses
455 in primary infections similar to our model (e.g. due to new calves in African buffalo hosting foot-and-
456 mouth disease: Jolles et al. 2021, or winter peaks in mange among wolves: Almberg et al. 2015). Existing
457 host-pathogen dynamics, and potential pathogen range expansions, could thus provide more frequent
458 opportunities for novel transmissions to overlapping species than previously guessed. Our model shows
459 how this provides a powerful selective force in favour of detecting and avoiding infection risk cues
460 (Weinstein et al. 2018).

461 In order to be widely applicable to diverse novel host-pathogen introduction scenarios, our model
462 is necessarily quite general. A wide diversity of pathogens and their dynamics remains to be accurately
463 represented in individual-based models (White et al. 2017; 2018b; Scherer et al. 2020; Lunn et al. 2021).
464 Our framework can be expanded and specifically tailored to real-world situations in which populations
465 are repeatedly exposed to novel pathogens (or strains) (Bastos et al. 2000; Scherer et al. 2020; Chandler

466 et al. 2021; Jolles et al. 2021; Kuchipudi et al. 2022; Wille and Barr 2022). Such detailed implemen-
467 tations could include aspects of the pathogen life-cycle (White et al. 2017; 2018a), account for social-
468 ity as a counter to infection costs (Almberg et al. 2015; Ezenwa et al. 2016), or model host-pathogen
469 sociality-virulence co-evolution (Bonds et al. 2005; Prado et al. 2009; Ashby and Farine 2022). Future
470 work would ideally combine wildlife monitoring and movement tracking across gradients of pathogen
471 prevalence, to detect novel cross-species spillovers (Chandler et al. 2021; Kuchipudi et al. 2022) and
472 study the spatial and epidemiological consequences of animal movement strategies (Bastille-Rousseau
473 and Wittemyer 2019; Monk et al. 2022; Wilber et al. 2022). Our model shows why it is important to con-
474 sider evolutionary responses in movement-disease studies, and provides a general framework to further
475 the integration of evolutionary approaches in wildlife spatial epidemiology.

476

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