

# Chapter 2

## How Behaviour and the Environment Influence Transmission in Mobile Groups

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**Abstract** The movement of individuals living in groups leads to the formation of physical interaction networks over which signals such as information or disease can be transmitted. Direct contacts represent the most obvious opportunities for a signal to be transmitted. However, because signals that persist after being deposited into the environment may later be acquired by other group members, indirect environmentally-mediated transmission is also possible. To date, studies of signal transmission within groups have focused on direct physical interactions and ignored the role of indirect pathways. Here, we use an agent-based model to study how the movement of individuals and characteristics of the signal being transmitted modulate transmission. By analysing the dynamic interaction networks generated from these simulations, we show that the addition of indirect pathways speeds up signal transmission, while the addition of physically-realistic collisions between individuals in densely packed environments hampers it. Furthermore, the inclusion of spatial biases that induce the formation of individual territories, reveals the existence of a trade-off such that optimal signal transmission at the group level is only achieved when territories are of intermediate sizes. Our findings provide insight into the selective pressures guiding the evolution of behavioural traits in natural groups, and offer a means by which multi-agent systems can be engineered to achieve desired transmission capabilities.

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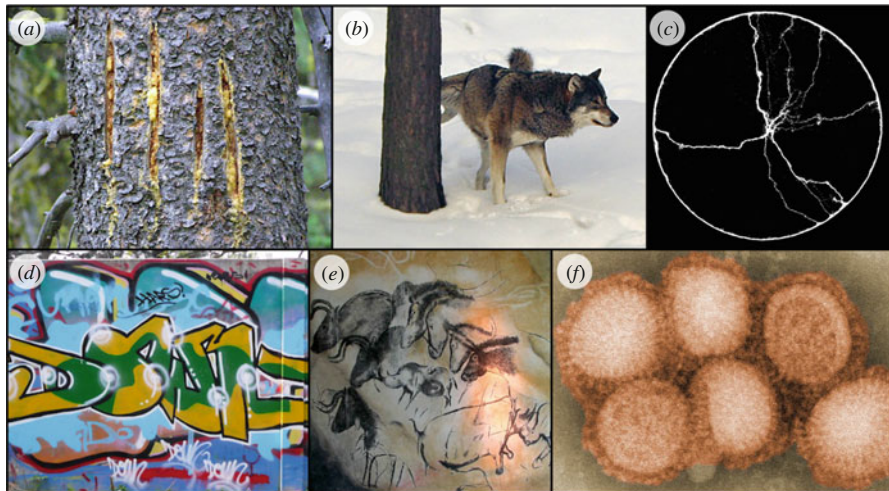
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## 2.1 Introduction

Animal societies consist of many individuals that must interact to coordinate their actions. The cohesion of such groups is typically achieved through a distributed network of short-range ‘direct’ interactions between neighbouring individuals. These serve to rapidly transmit information throughout the group [61, 92]. Short-range contacts also represent a channel for the transmission of harmful pathogens, with the potential for large-scale epidemics being closely linked to the structure of the group interaction network [72, 77, 80, 81, 89]. Whilst information often spreads via dedicated interactions that have evolved for the purpose of communication, diseases often ‘piggyback’ over a diverse range of different interaction types that have evolved for other purposes, such as sexual contacts [67, 86] or face-to-face conversations [96, 107]. Furthermore, while information and disease play different roles, they may both be viewed as ‘signals’ that can be transmitted across a group. Although direct interactions based on physical contact are the most obvious means by which such signals can spread, other forms of transmission are possible. For example, some signals remain viable after being deposited into the environment. If such a signal is still viable when that location is later visited by another individual, then the second individual could acquire the signal. As this pathway does not require the sender and receiver to be present at the same time, this is termed ‘indirect’ transmission [23, 38, 84].

Indirect communication is ubiquitous. It is found in species where individuals are generally solitary (Fig. 2.1a), as well as in highly cooperative species where individuals live together in tightly-knit societies (Fig. 2.1b–e). A commonly used example is that of pheromone trails in ant colonies where individual workers deposit chemical markers that recruit nestmates to rewarding food sources [30] (Fig. 2.1c). Disease can also exploit indirect pathways for transmission. Pathogens such as smallpox and influenza are able to remain intact outside a host for extended periods of time in ‘environmental reservoirs’ (Fig. 2.1f). These increase the number of opportunities for transmission and can lead to multiple waves of infection. Whilst researchers of animal behaviour have long appreciated that the shared environment can act as a substrate for indirect communication [29, 32, 46, 73, 98, 115], epidemiologists are only starting to quantify the important role that indirect transmission has during disease spread [2, 19, 28, 58, 87, 88, 103, 114, 116].

Over the last decade there has been an increasing number of studies focusing on the transmission properties of contact networks in humans [31, 56, 69, 95, 105–108] and other social animals [1, 14, 17, 17, 21, 75, 77, 94, 101]. Similarly, there has been a rapid growth in the effort devoted to understanding how adaptive collective behaviours such as swarming, flocking and shoaling [6, 22, 74, 92] emerge from the underlying peer-to-peer interactions. To date, these studies have exclusively focused on the role of direct interactions, without considering the potential for indirect transmission of materials or information. Although there are many species in which environmentally-mediated transmission is not viable because



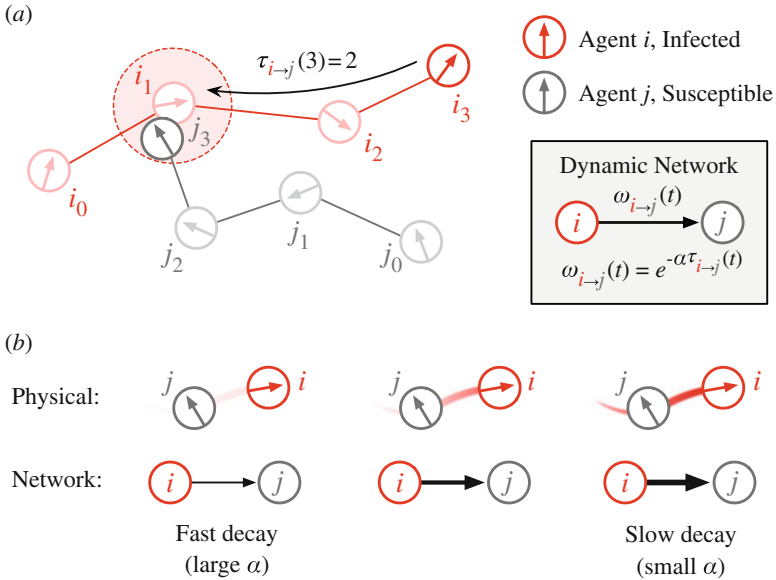
**Fig. 2.1** Indirect transmission of information and disease. (a) Territorial bear markings on a tree in Paradise Valley, Montana, USA (Image attribution: Suzanna Soileau, USGS). (b) Eurasian Wolf (*Canis lupus*) marking its territory. (c) Pheromone trails of Argentine ants (*Linepithema humile*) used to coordinate colony behaviours such as foraging [82]. (d) Modern graffiti. (e) Ancient human murals from the Chauvet cave. (f) Influenza A (H1N1) virus more commonly known as swine flu that can be transmitted indirectly through the air and infected surfaces (Image attribution: Cybercobra at English Wikipedia)

the environment cannot physically support such transmission (e.g. flocking birds), there are many species for which the potential is clear (e.g. those living within or upon the ground).

To address this shortcoming, we previously developed an analytical framework that combines both direct and indirect interactions within a single dynamic network representation [84]. Individuals are represented by nodes and weighted edges represent direct and indirect interactions (Fig. 2.2a). When a direct physical interaction occurs between two individuals, an edge with a weight of 1 is drawn between them. As both individuals are at the same place at the same time, they may both play the role of either sender or receiver, hence the edge is bidirectional. In contrast, indirect interactions occur when an individual  $j$  visits a location previously visited by another individual  $i$ . Assuming that  $i$  carries some signal (information or disease) that it deposits into the environment, that the signal decays at a rate  $\alpha$ , and that  $j$  visits the location at time  $t$ , this indirect interaction is represented by a directed edge from  $i$  to  $j$  with weight,

$$\omega_{i \rightarrow j}(t) = e^{-\alpha \tau_{i \rightarrow j}(t)}. \quad (2.1)$$

Here,  $\tau_{i \rightarrow j}(t)$  is the time delay between the visits of  $i$  and  $j$ , which is used to calculate the proportion of the signal that would remain viable given its environmental decay (Fig. 2.2b). This formalism allows any given time point to be represented as a static network ‘slice’ consisting of both strongly weighted direct interactions and more weakly weighted indirect interactions. Over time edges are created and



**Fig. 2.2** Extracting a dynamic network containing direct and indirect interactions from the paths of mobile agents. Example shows an indirect interaction between an infected (*red*) and susceptible (*grey*) agent. (a) Methodology for calculating edge weights for the dynamic interaction network. A susceptible agent  $j$  intersects a previously visited location of an infected agent  $i$  at  $t = 3$ . This gives an intersection delay of  $\tau_{i \rightarrow j}(3) = 2$  time steps. The *shaded circle* represents the maximum distance over which the signal can be transmitted. The edge weight is calculated using both the intersection delay  $\tau_{i \rightarrow j}$  and decay rate  $\alpha$  of the signal. Positions at time points 1 to 4 are denoted  $i_0, i_1, i_2$  and  $i_3$  for agent  $i$  and  $j_0, j_1$  and  $j_2, j_3$  for agent  $j$ , respectively. (b) The weight of an indirect interaction is modulated by the decay rate  $\alpha$  of the transmitted signal. Fast decay of a signal in the environment leads to weak trails and weakly weighted edges. Conversely, slow decay of a signal produces strong trails and highly weighted edges (Figure adapted from Ref. [84])

destroyed. Direct edges appear and disappear whenever two agents make and break contact. An indirect edge appears when one agent (the receiver) visits a location that was previously visited by another agent (the sender), and disappears when the receiving agent leaves that location. As such events are intermittent, the instantaneous static network slices are typically sparse. The overall network structure consists of a sequence of static slices, with direct interactions linking nodes within each slice, and with indirect edges linking nodes between slices. In the literature, such networks have been referred to as multi-slice, multiplex, dynamic, time-ordered, and temporal.

In our previous work [84], we used this approach to study the transmission properties of interactions within a colony of ants. Trajectories of individual worker ants were collected and used to create temporal networks where edges represented the direct and indirect interactions between the workers (referred to as ‘combined’ networks). To investigate the transmission properties of these temporal networks, we ran simulations inspired by susceptible-infected (SI) models of epidemiological

processes [52, 54]. In addition to simulating disease spread, SI models and their variants have been used to simulate transmission processes over a wide range of social and technological networks, including infrastructure networks [4], email and mobile phone call records [59, 62], and face-to-face conversation logs [56, 107]. In an SI model each individual may take one of two states; infected ( $I$ ) or susceptible ( $S$ ). Once an individual is infected it carries the signal of interest. The model is initiated with one node infected, and all others susceptible. To represent transmission between individuals, an infected individual  $i$  that interacts (either directly or indirectly) with a susceptible individual  $j$ , may result in  $j$  changing its state to infected. The probability that this state-change occurs is given by,

$$\Pr(j \rightarrow I \mid i \in I \wedge j \in S) = p_s \omega_{i \rightarrow j}(t), \quad (2.2)$$

where  $p_s$  is the transmission probability for direct interactions, and  $\omega_{i \rightarrow j}(t)$  is the edge weight defined above. Because the edge weight  $\omega_{i \rightarrow j}(t)$  decreases as a negative exponential function of the time delay  $\tau_{i \rightarrow j}(t)$ , direct interactions (which all have  $\tau_{i \rightarrow j}(t) = 0$ ) are much more likely to result in transmission than indirect interactions (which have  $\tau_{i \rightarrow j}(t) > 0$ ).

This SI model was applied to the ant colony interaction networks using the NetEvo software library [41, 42]. By systematically varying the transmission probability  $p_s$  and signal decay rate  $\alpha$ , we were able to analyse how the transmission properties of the ant interaction networks changed depending upon the characteristics of the signal being transmitted. We showed that both signal characteristics—the decay rate and transmission probability—significantly influenced the speed of signal transmission over the ant interaction networks. But do real-world signals exhibit such characteristics?

Signals that have evolved for the purpose of delimiting the borders of an animal territory or home-range, are expected to have a low environmental decay rate, particularly if the population is small or widely-spaced. In such cases, a small decay rate is essential, otherwise the territory-holder would need to allocate all of their time to re-marking. Whilst data concerning the environmental persistence of such signals is scarce, the prions that cause Chronic Wasting Disease (CWD) in deer can persist in the environment for several years [2], and the visual scratch marks used by some vertebrates to indicate territory borders (Fig. 2.1a) are essentially permanent (i.e.  $\alpha \approx 0$ ). Similarly, the scent- and scat-marks made by other vertebrates [25, 79] to indicate territory (Fig. 2.1b) and the pheromone marks used by invertebrates [57, 64] for communication (Fig. 2.1c), typically dissipate after a few days or weeks, and therefore correspond to signals with intermediate values of  $\alpha$ . At the other end of the scale, there are many examples of pathogens that decay so quickly that they cannot readily persist within the environment at all, and are therefore constrained to spreading by direct interactions (i.e.  $\alpha = \infty$ ). Most sexually-transmitted diseases fall into this category.

The second key characteristic of a signal is the transmission probability, which reflects the ability of a given signal to spread from one host to another when given the chance to do so. Diseases such as SARS which have evolved to have

a high virulence also have a very high probability of spreading during close-proximity interactions [76]. In terms of information transmission, unambiguous or high intensity signals such as elaborate courtship displays or eye-catching billboards (Fig. 2.1d, e) are unlikely to be missed or misinterpreted by the targeted recipient, and should therefore also have a high transmission probability. Interestingly, examples of signals with low transmission probabilities are scarce, probably because both information-bearing signals and pathogens have evolved to efficiently utilize any and all available transmission opportunities.

Whilst our previous study provided a mechanistic understanding of how the mixture of direct and indirect interactions produced by a real animal society determines how different types of signal may spread through it [84], we could not directly control the motion of the ants. Therefore, we were not able to provide a causal understanding of how individual-level behaviours determine the transmission properties of the overall interaction network. In this chapter, we overcome this limitation by defining an agent-based model in which the movement of virtual individuals (referred to as ‘agents’) is systematically varied. The motion of each agent takes the form of a parameterisable two-dimensional random walk. The environment within which these move also contains a spatially-explicit ‘signal field’ representing signals deposited by infected agents. This field is dynamically updated to reflect signal decay. By feeding the interaction data produced by this more physically realistic agent-based model into our previous network abstraction [84], we are able to establish a causal link between the behaviours of the individuals and the emergent group-level transmission properties.

## 2.2 Modelling Approaches

Numerous modelling approaches and formalisms have been developed to identify the key factors influencing the transmission of information and disease within populations of interacting individuals. In this section, we provide a brief overview of some of the most commonly used and discuss the benefits and limitations of each approach.

### 2.2.1 *Compartment Models*

Many of the earliest attempts to provide a quantitative description of spreading processes involve the use of compartment models. These employ deterministic or stochastic mathematical equations to describe contagious transmission of a signal (typically disease) through a population that is divided into compartments. In epidemiology, these compartments typically reflect an individual’s clinical status, for example, susceptible, exposed, infected or recovered [60, 102].

Compartment models remain popular today because their precise mathematical formulation can allow for the derivation of exact analytical solutions [55]. However, many simplifying assumptions are often made to ensure a tractable solution. For this reason, these models generally use deterministic differential equations to represent the flux of individuals between different states and assume that the population is infinite and well-mixed [66]. These simplifications are often not a good approximation for real-world systems where a finite number of individuals are non-uniformly distributed across space and behave stochastically rather than deterministically [7]. Furthermore, the physical laws governing real-world environments impose severe limits on how individuals can move and interact, yet compartment models assume no such constraints. Some attempts have been made to extend this approach to incorporate network structures that capture the heterogeneous mixing of populations, but generally such changes come at the cost of reduced analytical tractability.

## 2.2.2 *Network Models*

More recently, it has become popular to treat the spread of information or disease through spatially structured populations, as a contagious process propagating over a network. Nodes represent individuals and edges capture the interactions or contacts between them [15]. Although space is not explicitly modelled, the ability to constrain the interactions present between individuals allows for the effective description of the heterogeneous connectivity observed in many real-world systems (e.g. long-tailed degree distributions [9–11]). This has led to network-based approaches becoming common not only in epidemiology, but in a huge variety of different fields where contagious processes are observed, from the spread of rumours and gossip over social networks [68, 93], to cascading failures in power distribution networks [4, 20]. Attempts have also been made to combine the benefits of network models with other modelling approaches. For example, the GLocal Epidemic and Mobility (GLEaM) model makes use of structured metapopulation simulations that are linked to realistic networks capturing known mobility links (e.g. train and airline routes). Simulations using this combined model are able to accurately simulate the spatial propagation of disease pandemics at both regional and global scales [4, 5, 112].

The vast majority of network-based epidemic models investigate transmission using static networks where the structure is fixed over time. Doing so ignores the fact that in almost every real-world system the connectivity between individuals varies with time. For example, studies of the contact networks of both humans and other animals, have shown that interactions can be 'bursty' [9] or even cyclical [85], with large numbers of interactions concentrated in a short period of time. In cases where the dynamics of the epidemic process is much slower than the evolution of the network, this is not a problem as time-scale separation makes it possible to consider an 'annealed' network describing the averaged structure the process will encounter

[113]. However, if the process has a similar time-scale to the network dynamics, it is essential that both these aspects are modelled concurrently. Temporal networks are well suited to describing such intermittent fluctuations, as they represent the dynamics as a sequence of static network slices [15, 43, 53, 54]. Other approaches have also been developed to describe networks with dynamic topologies. Adaptive networks [49] and evolving dynamic networks [27, 43] make use of dynamical equations and stochastic rules to describe how the strength of each edge varies with time, whilst also allowing for changes in the size of the network through the birth and death of nodes. Although these more complex approaches can capture an even richer range of behaviours, they are a challenge to apply to real-world systems because a unified and accepted theory of time-varying networks is still emerging and new forms of analysis are often required [44].

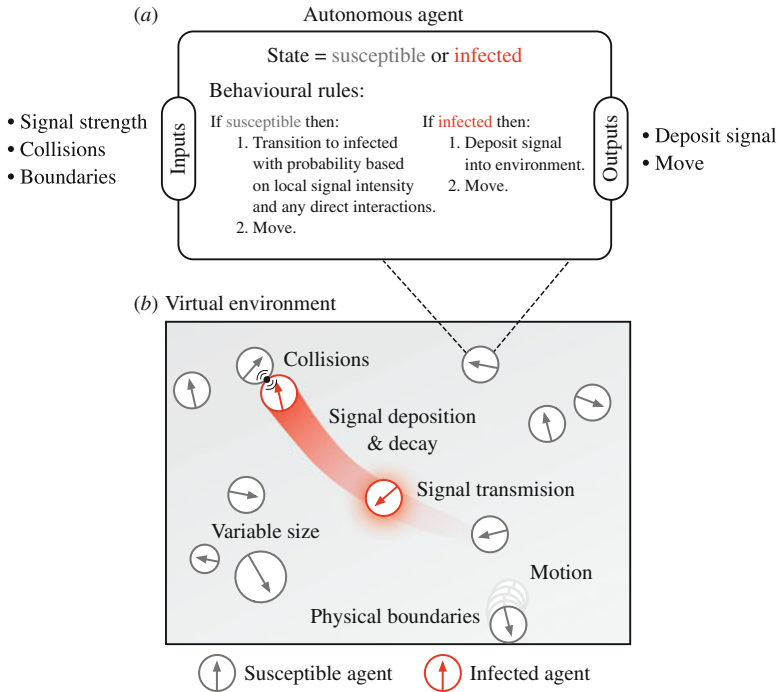
### 2.2.3 *Agent-Based Models*

Agent-based models attempt to simplify the description of real-world systems by modelling the behaviours of large numbers of autonomous ‘agents’ that move and interact within a virtual environment [40, 51] (Fig. 2.3). Unlike compartmental and network models, they provide a spatially- and physically-explicit representation of a population of mobile interacting individuals. As an agent-based model is essentially a physical representation of a given system, it is considerably more detailed than the compartmental and network abstractions described above. Agents can be used to represent any autonomous entity, from cells [45] to animals [110]. Each agent typically follows a prescribed set of rules controlling their behaviour and interactions with their peers. In a cellular context, these rules might represent the genetic circuits that control the expression of key genes in response to particular stimuli [71, 109], whereas in the context of shoals of fish or swarms of insects they would embody the behavioural responses each member makes in response to neighbouring individuals [22].

A major benefit of using an agent-based approach, as opposed to considering averaged group behaviours, is that the system is modelled as a set of discrete elements. This makes it possible to directly include heterogeneity and stochasticity into the behaviour of agents. Moreover, agent-based models allow for environmental processes (e.g. diffusion of a chemical) to be more easily described and incorporated using known physical laws [43, 51]. Including such processes in other more abstract methodologies, such as network models, is difficult due to the simplifications that are made.

Agent-based models are also ideally suited to the study of how complex group-level features emerge from the behaviour of the individuals and their use of a shared environment. By varying the rules that agents follow and observing the changes in the group-level behaviours, it is possible to understand how these organisational levels are linked and the causal factors controlling them. It is important to note that by representing the discrete individuals in a system, we are able to capture features





**Fig. 2.3** Overview of an agent-based model. (a) Agent-based models contain a number of autonomous individuals that follow their own individual set of rules defining how they respond to different external stimuli. (b) The agents are placed in an environment in which they can interact with each other and other processes within the environment (e.g. deposit a signal at a specific location)

that averaging across the population would miss. For example, cells often exploit noise and stochastic effects to differentiate their behaviours. A famous example of this is the *lac* operon that encodes genes required for the transport and metabolism of lactose [78]. In this system, cells that are genetically homogeneous use stochastic noise to differentiate themselves into two separate populations: those that are active and strongly expressing the *lac* operon, and those that are not. The fraction of cells in an active state varies depending on the concentration of lactose. However, two separate populations are always maintained. If an average of *lac* operon expression was taken across the entire population, it would look as if each cell was tuning its expression to match the concentration. Only when the individual cells are modelled, as is done when using agent-based modelling, is an accurate understanding of the bistable structure of the system gained.

The need to capture the dynamics of each component of a system results in agent-based models being computationally expensive to run. While this limited their use initially, recent advances in high-performance computing have opened up the possibility to efficiently simulate large complex systems consisting of millions

of interacting components [40, 51]. In many cases this is sufficient to provide an accurate representation of real-world systems, and as a result, agent-based models are now commonplace in many different fields including social behaviour [3], ecology [48], microbiology [51] and economics [111]. The major difficulty that remains is the requirement for a detailed understanding of the individual-level rules, as such information is often unavailable.

## 2.3 Studying Signal Transmission Using an Agent-Based Model

In this section we describe the agent-based model we use to assess how high-level properties of the group, such as the transmission properties of the interaction network, emerge from the underlying behaviours of the individual agents. This model enables us to parameterise the random walk performed by each agent as well as environmental properties such as physical collisions and the decay rate of any signals deposited into the environment. We apply this model to a number of different scenarios to explore the role of these factors and link the observed changes in transmission to characteristics of the underlying interaction networks that are generated.

### 2.3.1 Agent-Based Model Definition

The agent-based model consists of a population of  $N$  agents, each performing a random walk in a two-dimensional environment with periodic boundary conditions. Each agent is defined by a set of four variables describing its current infection state  $s$  (either susceptible ‘S’ or infected ‘I’), its position within the environment  $\mathbf{x}$  stored as a two-dimensional vector, its two-dimensional heading vector  $\hat{\theta}$  (of unit length, arbitrary length units), and the time remaining  $t_r$  before a new heading is randomly selected. Each agent is represented as a circle of unit radius  $r = 1$ , unit mass (arbitrary mass units), and can propel itself with a constant force of 0.3 force units per time unit in the direction it is heading. Furthermore, agents display no inertia (i.e. the velocity of an agent will be zero when no force is applied) and no friction is present in the environment. Parameter values and variables for the model are shown in Table 2.1.

In order to allow indirect signal transmission via the shared environment, the model includes a ‘signal field’ that records the signal strength at every location within the environment. As an infected agent moves through the environment, it leaves a ‘trail’ of the signal behind it, which is modelled as a local increase in the signal field strength (Fig. 2.3b). At each time step  $t_s = 0.02$  time units, we begin by updating the field to simulate signal decay. Assuming the signal has decay rate  $\alpha$

**Table 2.1** Model features and parameters

Aspect	Parameter	Description	Value(s)	Units
Simulation	$t_s$	Simulation time step	0.02	Time unit
Environment	$N$	Number of agents	2–1500	–
	–	Environment dimensions	$100 \times 100, 25 \times 25$	Length unit <sup>2</sup>
	–	Boundary conditions	Periodic, Solid	–
	–	Physical collisions between agents	On, Off	–
Agent	$r$	Radius of agent	1	Length unit
	$\kappa$	Turn-angle distribution bias	–	–
	$s$	Infection state	$S, I$	–
	$\gamma$	Strength of attraction	0.001–0.2	–
Signal	$p_s$	Transmission probability	0.01–1.0	–
	$\alpha$	Decay rate	0.001–1.0	Time unit <sup>-1</sup>

per time unit, then the value of the field at each position has  $\alpha t_s F$  subtracted, where  $F$  is the current value of the field. Next, we cycle through each agent and if  $t_r > 0$  then their position is updated according to  $\mathbf{x} + 0.3\hat{\theta}t_s$  and  $t_s$  is subtracted from  $t_r$ . Otherwise, if  $t_r$  has reached zero, a new heading  $\hat{\theta}$  is chosen by generating a unit length vector in the direction of a randomly selected angle over the interval  $[-\pi, \pi]$ . In addition,  $t_r$  is set to a constant agent movement time (default is 1 time unit) and the agent is then moved as described above. Finally, if the agent is infected, we update the signal field to have a value of 1 for the entire space covered by the agent. Conversely, if the agent is susceptible, then a transition to an infected state occurs with probability  $p_s F$ , where  $p_s$  is the signals' probability of transmission through a direct contact, and  $F$  is the maximum value of the signal field for the space occupied by the agent. Because the path of each agent is determined by random sampling of the heading vectors, agents move independently of one another.

In addition to agents following simple random walks, we also extended the model to allow each agent to have its own point of attraction, which enabled the formation of territories in space. When an agent is close to its point of attraction it moves randomly, but as the distance from the point of attraction increases, the heading distribution (which in the basic model is uniformly distributed around the circle), becomes increasingly biased towards the point of attraction. This allowed us to constrain the motion of each individual to a limited region of space. To implement this behaviour we adapted the basic model such that new headings for agents were sampled using a circular normal (von Mises) distribution over the range  $[-\pi, \pi]$ . The probability density function was given by,

$$f(x | \mu, \kappa) = \frac{e^{\kappa \cos(x-\mu)}}{2\pi I_0(\kappa)}, \quad (2.3)$$

where  $\mu$  is the mean,  $\kappa$  is the bias (analogous to the variance of a normal distribution), and  $I_0(\kappa)$  is the modified Bessel function of order zero [70]. This

distribution was orientated such that its mean value  $\mu$  pointed from the agent towards its' point of attraction. The bias of the distribution around the mean was then given by  $\kappa = d_a\gamma$ , where  $d_a$  is the distance between the agent and the point of attraction, and  $\gamma$  is a parameter governing the strength of attraction (Table 2.1). Hence, the further away the agent is from its point of attraction, the more strongly its heading will be biased towards it.

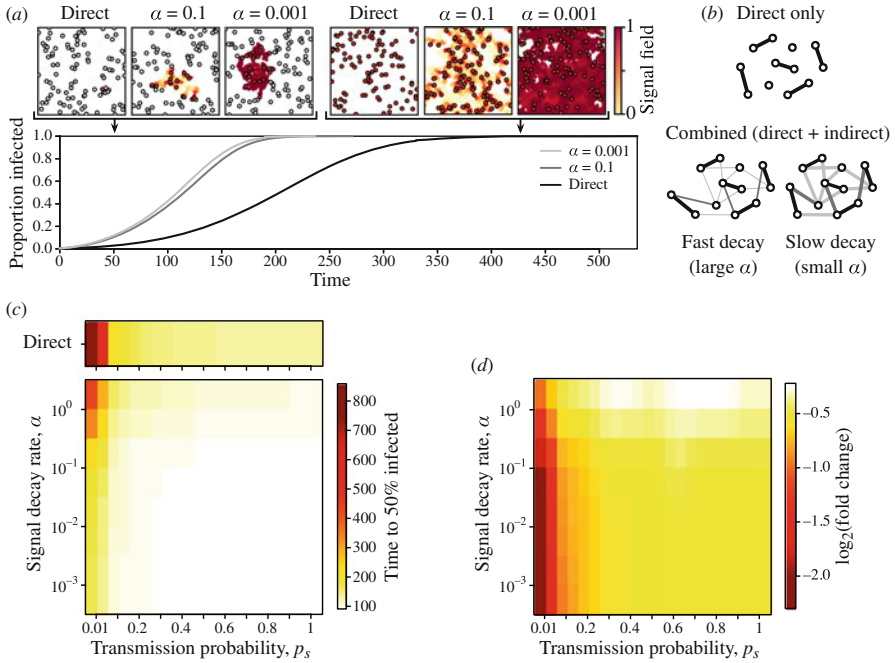
Although in the model described above, each agent occupies a non-zero area in space, agents do not physically interact with one another. Hence, two agents are allowed to occupy the same position at the same time. Clearly this is not realistic, as packing constraints impose an upper limit on real world animal population densities. Therefore, we optionally allow for the inclusion of physical collisions between agents. To implement this feature a reaction force was applied whenever two agents' boundaries touched one another. For a distance  $d$  between the boundaries of two agents, a reaction force of  $R_f = 25d \times \log(d/2r)$  was applied to both agents in opposing directions. For the chosen parameters of our simulations (Table 2.1) this enabled some limited overlap of agents, but prevented agents passing through one another.

We initiated each simulation by randomly-selecting a single 'seed' individual whose state was set to infected. We then followed the propagation of the infection across the rest of the population. As transmission was stochastic, 1000 simulations were run for each parameter combination and the average of these runs was used to calculate the proportion of the population infected over time.

### 2.3.2 *Role of Indirect Transmission Pathways*

Interaction networks composed of both direct and indirect interactions (referred to as 'combined' networks) have been shown to have fundamentally different transmission characteristics from those composed solely of direct interactions (referred to as 'direct-only' networks) [84]. To confirm that the addition of indirect interactions affects the transmission properties of the group, we measured the progression of signals spreading across the combined interaction networks generated by the agent based model. This was then compared to signals spreading across groups where the agents could only interact by direct physical contacts. Signal progression over both network types was quantified by simulating SI transmission of a wide range of signals that varied in both their transmission probability  $p_s$ , and their decay rate  $\alpha$ .

For both network types and for all combinations of transmission probability  $p_s$ , and decay rate  $\alpha$  of a signal, the SI model produced sigmoid shaped infection curves (Fig. 2.4a). Sigmoidal curves are a hallmark of density-dependent processes, such as the spread of information or disease through a finite population. The early explosive growth is due to the exponential nature of the SI dynamics where contact between one infected and one susceptible agent can result in two infected agents. The later slowing of spread arises from the gradual depletion of the pool of uninfected agents. To compare the transmission among different types of signals, it is necessary to



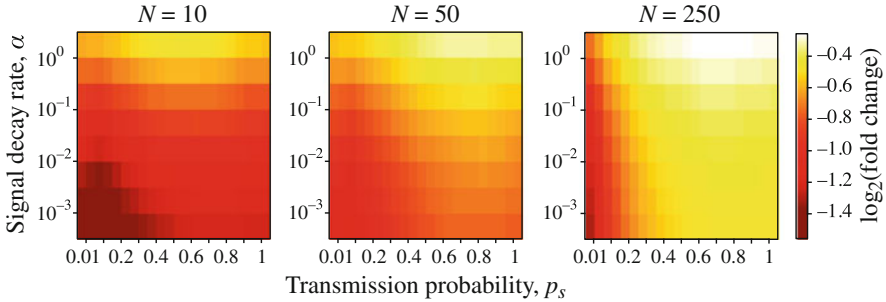
**Fig. 2.4** Influence of indirect pathways on transmission rate. **(a)** The spread of three different signals are shown. In one, the signal decays immediately (i.e.  $\alpha = \infty$ ) and so infections can only spread via direct interactions. In the other two, the signals have decay rates of  $\alpha = 0.1$  and 0.001, respectively, making indirect transmission possible. All three signals have a transmission probability  $p_s = 0.1$ . *Small panels above* show a time point from the agent-based model for a representative simulation. Agents are shown by *circles*, which are grey if susceptible and red if infected. The *background colour* denotes the strength of the environmental signal field; *dark red* = 1 to *white* = 0. **(b)** Illustration of the static network 'slice' for a single point in time. *Circles* denote agents, *thick black edges* denote direct interactions and *thinner grey edges* denote indirect interactions. *Line thickness* corresponds to edge strength. **(c)** Average time to reach 50% of the population infected  $t_c$  for a range of signal decay rates  $\alpha$  and transmission probabilities  $p_s$ . **(d)** Comparison of the  $\log_2$  fold change difference in  $t_c$  between simulations using only direct interactions and those incorporating both direct and indirect interactions. Simulations for all panels contained 250 agents and are the average of 1000 simulations

define a feature of each growth curve that reflects the overall transmission speed. We chose to use the time taken for 50% of the population to become infected to define the characteristic time of infection  $t_c$ . Larger  $t_c$  values correspond to slower transmission through a population. In order to more easily assess the size of changes in transmission, we normalized (divided) the  $t_c$  for the combined networks by the respective  $t_c$  times for the direct-only networks. This provided a fold change in the  $t_c$  value that could then be compared for all signal decay rates  $\alpha$  and transmission probabilities  $p_s$  (Fig. 2.4d). Positive log fold changes would indicate that collisions lead to a slower transmission, whereas negative values correspond to an accelerated transmission rate.

A comparison of the  $t_c$  times between the direct-only and combined networks revealed that transmission was considerably slower in the direct-only networks for signals with identical transmission probabilities (Fig. 2.4c). The addition of indirect interactions reduced the  $t_c$  time by up to 80% for signals with a low transmission probability and small decay rate (Fig. 2.4d;  $p_s = 0.01$ ,  $\alpha = 0.001$ ). In contrast, for signals with high transmission probability and rapid decay rate, the addition of indirect interactions had a much weaker effect, reducing the  $t_c$  times by only 15% (Fig. 2.4d;  $p_s = 1$ ,  $\alpha = 1$ ). Therefore, the addition of indirect transmission opportunities greatly increases the speed of transmission between group members.

This accelerated transmission derives from the impact of indirect interactions upon the connectivity (i.e. the degree) of the agents. When direct interactions are possible, the transmission rate is fully dependent on the rate of physical contacts generated by the random walks performed by each member of the population to transmit the signal. Moreover, this rate scales linearly as the number of infected individuals grows. In contrast, when indirect transmission becomes possible because an infected agent deposits a signal into the environment, the effective area for indirect transmission grows. It is as though the size of the agent grows over time, and has the effect of increasing the number of interactions, and therefore the connectivity, of infected agents. For fast decaying signals, this effect will be constrained as the signal trail will be of a limited length (see Fig. 2.4a,  $\alpha = 0.1$  top panels). However, if the decay rate of a signal is sufficiently small, the trail will continue to grow enabling the entire environment to quickly become a potential transmission pathway, even though the infected agents only take up a small fraction of the total area (see Fig. 2.4a,  $\alpha = 0.001$  top panels). In all cases, the increased connectivity that is gained by the indirect pathways will lower the overall diameter of the interaction network and help accelerate transmission [84].

Another major factor influencing the interaction rate between individuals in a group is their density. To assess how the density of individuals affects the rate of transmission, we produced combined networks from the agent based simulations as before, but with 10, 50 and 250 agents. As the size of the environment was fixed, the agent density varied over an order of magnitude (0.3–8% of the total area). For all agent densities, we again found that the transmission speed over combined networks was increased relative to networks composed of direct edges; signals with a low transmission probability and small decay rate saw the greatest enhancement in transmission for the combined networks (Fig. 2.5). Furthermore, the agent density also modulated the magnitude of the transmission enhancement brought about by the addition of indirect edges; at low agent densities, the strength of the enhancement was maximal (Fig. 2.5,  $N = 10$ ), whereas at high agent densities the enhancement was weaker (Fig. 2.5,  $N = 250$ ). In the case of a slow decaying signal with high transmission probability ( $\alpha = 0.001$ ,  $p_s = 1.0$ ), the difference in  $t_c$  between direct and combined networks rose from 1.4-fold to 2.3-fold for 250 to 10 agents, respectively. This suggests that the beneficial role of indirect pathways has a greater impact when agents are sparse, raising the interesting possibility that natural populations broadly distributed in space will more heavily rely on indirect transmission pathways [25].



**Fig. 2.5** Influence of agent density on transmission rate. Comparison of the  $\log_2$  fold change difference in  $t_c$  between simulations using only direct interactions and those incorporating both direct and indirect interactions, when 10, 50, and 250 agents are present (densities of 0.3%, 1.6% and 8% of the total area, respectively). All values are the average of 1000 simulations

The reason for this density dependence derives from the presence of indirect edges. At low agent densities infected agents will have a low rate of physical contacts, whereas they may frequently visit locations that were previously visited by other agents. Hence, at low agent densities indirect interactions are the primary channel for transmission. As the density of the agents increases, so does the contact rate. Therefore, transmission at high agent densities will occur primarily via the strongly-weighted direct interactions.

### 2.3.3 Role of Territories in Space

Studies of the mobility patterns of humans and other animals have shown that individuals often revisit specific locations over time [18] and maintain specific areas as territories. In humans, examples include homes, workplaces, restaurants, and the transit routes that connect them [108]. In other animals these locations take the form of watering holes, foraging patches, leks, valuable resources that must be defended, or nesting areas where there are brood that must be regularly provisioned. Depending upon the system and context, biases towards particular locations have been given many different names, including recurrence [18, 39, 104], recursion [8, 12, 13, 33], site tenacity [50], site allegiance [26], site recognition [97], site fidelity [36, 37, 65, 83, 99], spatial fidelity [100], ortstreue [91] and route fidelity [90].

The pervasive nature of this phenomenon across so many different animal species was the motivation for the first extension of the basic random walk model, to allow for the formation of territories. The aim was to investigate how individual-level spatial preferences influence transmission of a signal across the population. We hypothesized that compared to a population in which all agents perform random walks, a population of agents that exhibit spatial fidelity should display

slower transmission over the group-level interaction network. This is because a system composed of spatially constrained agents should intuitively be dominated by short-range local interactions. Put another way, when each agent interacts with only a small set of nearest neighbours, transmission should be slower than when agents move freely as then all agents can potentially interact with all other agents.

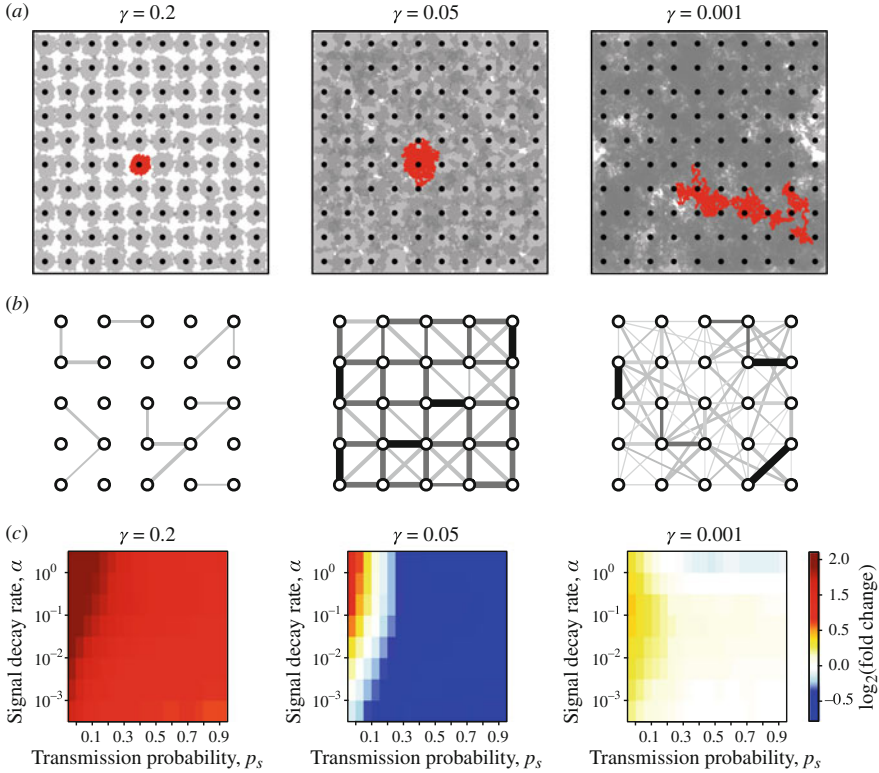
We used a simple model of territory formation where each agent performs a random walk biased towards a single point of attraction (method described above). A unique point of attraction was used for each of the 100 agents, and the points of attraction were arranged as a uniformly spaced grid within an environment with periodic boundary conditions. Simulations were then run for various territory sizes by varying the strength of attraction  $\gamma$ . This resulted in a range of agent movement, from distinct territories formed by agents that remained very close to their point of attraction ( $\gamma = 0.2$ ), to nearly non-existent territories formed by agents that were only slightly attracted to their point of attraction, and which therefore moved much like a random walker ( $\gamma = 0.001$ ). Examples of a representative simulation output and the paths taken by each agent are shown in Fig. 2.6a.

To measure the effect of territories upon signal transmission, we compared the  $t_c$  time of the combined networks with territories to those of the original agent-based model in which agents performed an unbiased random walk. For high strengths of attraction ( $\gamma = 0.2$ ), we observed a more than 2-fold increase in  $t_c$  for all signal decay rates and transmission probabilities (Fig. 2.6c). This confirmed our hypothesis that spatially structured populations (i.e. those where an agent's movement is spatially restricted) also experience reduced transmission speeds. We also found that this increase was greater for signals with lower transmission probabilities and larger decay rates. Under these conditions, agents were so highly constrained to the vicinity of their point of attraction that they only had very limited connectivity to their nearest neighbours (Fig. 2.6a). This led to interaction networks that were highly compartmentalised with multiple sparsely connected components (Fig. 2.6b). These features reduce mixing of the agents and hamper the ability for a signal to propagate throughout the entire population [95] (Fig. 2.6c).

For low strengths of attraction ( $\gamma = 0.001$ ), the  $t_c$  times were very similar to the original agent-based model in which agents performed unbiased random walks (Fig. 2.6c). This is to be expected given that as the strength of attraction decreases, a movement similar to an unbiased random walk is produced. Interestingly, for intermediate strengths of attraction ( $\gamma = 0.05$ ), both decreases and increases in transmission speed were observed, depending on the signal transmission and decay characteristics (Fig. 2.6c). Slower transmission was found when signals had large decay rates ( $\alpha = 1$ ) and low transmission probabilities ( $p_s = 0.01$ ). Such signals will find it difficult to exploit indirect pathways. Faster transmission occurred for signals with low decay rates, as such signals are better able to use indirect pathways. Furthermore, the speed of transmission also increased for higher transmission probabilities, even when the signal decay rate was high.

The presence of both enhancement and inhibition of transmission at intermediate attraction strengths can be explained by considering the relation between the average





**Fig. 2.6** Influence of agent territories on transmission rate. **(a)** Agent-based simulations shown at  $t = 500$  time units for 100 agents with uniformly spaced points of attraction and attraction strengths  $\gamma = 0.2, 0.05$  and  $0.001$ . *Black circles* denote the starting point of the agent, which also acts as the point of attraction. *Gray lines* show the path of each agent with *darker gray regions* denoting an overlap between multiple agents. *Red line* in each simulation shows the path of a single agent. **(b)** Illustrative time averaged interaction networks generated by the agent-based simulations. *Circles* denote agents, *thick black edges* denote direct interactions and *gray edges* denote indirect interactions. *Line darkness and thickness* corresponds to edge strength, with *darker and thicker* edges corresponding to stronger edges. **(c)** Comparison of the  $\log_2$  fold change difference in  $t_c$  between simulations where the territory is present and those where agents perform random walks

number of interaction partners of each agent (their degree), and the average strength of their interactions (edge weights). At low attraction strengths ( $\gamma = 0.001$ ), agents can move freely throughout the entire environment (Fig. 2.6a, right panel). This allows them to interact with potentially all other agents, but on average it results in weak interactions between any given pair of agents. This produces interaction networks in which agents have a high degree with many long-range links (i.e. links between agents with points of attraction very far from each other), but where the majority of these links are weak (Fig. 2.6c, right panel). In contrast, intermediate attraction strengths ( $\gamma = 0.05$ ) lead to the formation of territories that significantly

overlap with all nearest neighbours, and potentially more distant agents (Fig. 2.6a, middle panel). In this scenario, an agent's movement is restricted to a much smaller area, resulting in a greater number of repeated interactions with neighbours over shorter periods of time. This generates an interaction network where each agent is connected by strongly weighted edges to its neighbours (Fig. 2.6c, middle panel). Varying the attraction strength alters the size of the territory and so also the number of neighbours it includes. However, as the attraction strength is reduced the potential area for movement also increases. This reduces the interaction rate between the agents and thus leads to an interaction network containing weaker edge strengths. Optimal transmission rates are achieved by trading-off these factors to generate a network in which the weighted diameter is minimised, which occurs when the shortest paths between agent pairs typically consist of a small number of strongly weighted edges.

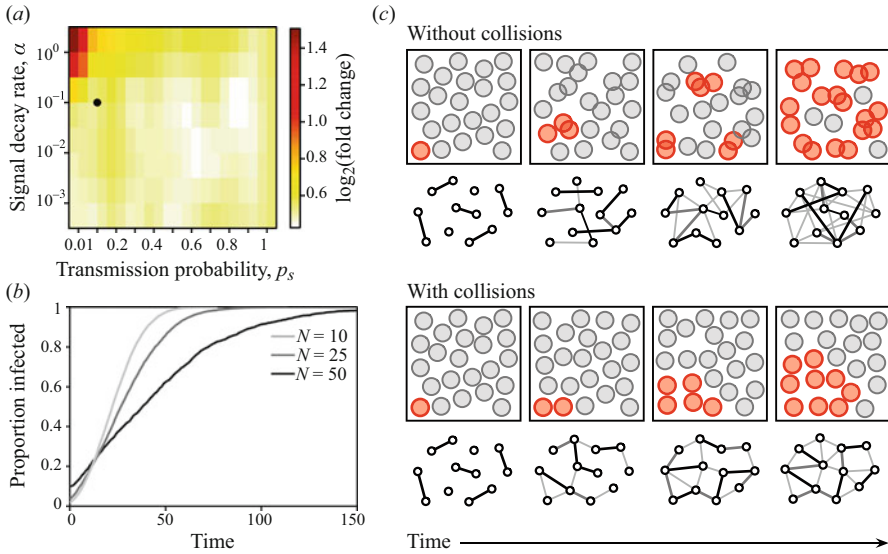
### 2.3.4 *Role of Physical Collisions Between Agents*

Across the natural world, there are numerous examples of populations in which the individuals are so crowded that movement becomes difficult. For example, in bacterial colonies, nests of social insects [47], penguin huddles [117], and human crowds [16], the individuals may be so densely-packed together that the group itself becomes 'jammed' with some individuals unable to move. Most models of transmission over animal contact networks have ignored such physical considerations [63].

To assess how physical collisions between agents might influence signal transmission in mobile groups, we considered a simulated environment of  $25 \times 25$  length units<sup>2</sup> with solid boundary conditions to allow for higher agent densities than in previous simulations. We ran three sets of simulations containing 10, 25 and 50 agents (densities of 5%, 12.5% and 25% of the total area, respectively) and measured  $t_c$  for each set. We hypothesized that the introduction of collisions would reduce transmission speeds via the constraint it places on an agent's movement, and that this slowing-down would be exaggerated at high agent densities.

As expected, we found that collisions slowed transmission for all types of signal (Fig. 2.7a). The magnitude of this effect was greater at high agent densities (Fig. 2.7b), and showed a non-linear dependence upon the signal characteristics (Fig. 2.7a). Specifically, for signals with a low transmission probability and high decay rate ( $p_s = 0.01$ ,  $\alpha = 1$ ), a more than 2-fold increase in the  $t_c$  time was observed.

An obvious explanation for the slower transmission when agents can collide is that the dense packing severely reduces individual movement, and constrains both direct and indirect interactions towards the immediate neighbours (Fig. 2.7c). This is in stark contrast to when collisions are absent, where infected agents can move



**Fig. 2.7** Role of physical collisions between agents on transmission rate. (a) Comparison of the  $\log_2$  fold change difference in  $t_c$  between simulations where collisions between agents are present and those where collisions are absent. (b) Infection time course for a signal with decay rate  $\alpha = 0.1$  and transmission probability  $p_s = 0.1$  (highlighted as *black point* in *panel above*) where simulations contained 10, 25 and 50 agents. (c) Illustration of simulations with physical collisions between agents absent and present. Hypothetical simulations are shown over time with *circles* representing agents that are susceptible if *grey* or infected if *red*. When collisions are absent, agents can overlap with one another. Networks below each simulation illustrate the general structural features over time. *Circles* denote agents, *thick black edges* denote direct interactions and *grey edges* denote indirect interactions. *Line darkness and thickness* corresponds to edge strength, with *darker and thicker edges* corresponding to stronger edges

throughout the environment to generate direct and indirect interactions with all other members of the population. As described previously, such increased mixing greatly reduces the overall diameter of the network and increases transmission speeds [84]. Long-range interactions also allow for a signal to be seeded at many different locations within the environment, leading to a rapid transmission across the entire population (Fig. 2.7c). Conversely, when collisions are present individuals become confined to a local area due to physical exclusion by nearby agents, hence an infected individual can only infect its nearest neighbours (Fig. 2.7c). At the highest densities, the interaction network is essentially a direct reflection of the agents' spatial locations, with the all edges reflecting short-range interactions between immediate neighbours. Such networks only allow transmission in the form of a moving infection 'front' passing through the densely-packed population. This limits the role of indirect transmission in 'seeding' infections in new areas and results in a slowing-down of transmission.

## 2.4 Conclusions and Future Directions

To understand how the behaviour of an individual within a group can affect the transmission of a signal, it is essential to establish causal relationships between these two organizational levels. In previous work, we applied a dynamic network methodology to an empirical dataset describing the movement and interactions of individual ants living within a communal nest [84]. However, because the behaviour of individuals could only be observed and not controlled, it was impossible to establish a direct link between individual-level behaviours and group-level transmission properties. Here, we used an agent-based model to simulate populations of individuals whose behaviour can be precisely controlled. This allowed for direct links to be made between an individual's behaviour (e.g. movement), environmental factors (e.g. signal decay rate, agent density and physical collisions), and transmission properties of the system.

Our simulations have revealed that indirect transmission pathways can play a significant role in shaping the spread of infections with differing environmental decay rates; even signals with a very low probability of transmission can when deposited into the environment propagate quickly throughout an entire group, if they are able to remain viable for some period of time. Both individual behaviour and group density also play an important role in the transmission capabilities of the group. Higher agent densities led to the faster spread of an infection. However, at very high densities, if physical collisions are considered then the movement of each individual becomes sufficiently impaired to reduce the ability for long-range interactions to form and slows transmission. More complex behaviours such as territory formation also modulated transmission rate, with a trade-off observed between maintaining stronger but shorter range interactions for smaller territories, or weaker but longer range interactions when an individual is free to diffuse throughout the entire environment. Optimal transmission was found when individuals maintained territories of intermediate size.

The focus of this work has been to simulate the transmission of a contagious signal across a group of mobile agents whose behavioural rule remains fixed over time. This idealised 'toy' model is deliberately simple to allow for an easier interpretation of the results and to provide clearer links between the behaviours of individuals and high-level collective transmission properties. Nevertheless, it is important to recognise that these simplifications ignore the fact that individuals in many real-world animal populations do exhibit strong behavioural responses to the presence of disease [35] and that these changes can significantly alter the transmission properties of the group [34]. Indeed, many animal species exhibit strong aversive behaviour to others that appear to be disease-carriers [24], and in highly social species, responses to the presence of disease may also be implemented by coordinated group-level responses. For examples, in human societies quarantines and curfews are used to reduce the potential for interactions between susceptible and infected individuals. Incorporating some form of adaptive behaviour would offer an interesting future direction for this work that the methodology is ideally suited to tackle.

This work was made possible by the availability of high-performance computing resources that enable large-scale simulations to be performed. A challenge often faced when using this type of approach is that the complexity of the underlying model makes interpretation of the results difficult. Here, we have exploited the wealth of knowledge in network theory to better understand how the structural features of the dynamic interaction networks generated by simulations influence the general transmission properties of the system. Such a combined approach offers a powerful means to bring together both numerical methods and proven mathematical theories to understand the role of direct and indirect pathways in natural systems, and provides a means to engineer new distributed systems with desired transmission capabilities.

**Acknowledgements** T.E.G. was supported by an EPSRC Institutional Sponsorship award from the University of Bristol (EP/P511298/1), and BrisSynBio, a BBSRC/EPSRC Synthetic Biology Research Centre (BB/L01386X/1). T.O.R is supported by an EU Marie Curie Actions Intra-European Fellowship, ‘Mapping spatial interaction networks in honeybee colonies’ (project number 30114). Simulations and analyses were carried out using the computational facilities of the Advanced Computing Research Centre, University of Bristol, UK.

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