Particle velocity controls phase transitions in contagion dynamics

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Interactions often require the proximity between particles. The movement of particles, thus, drives the change of the neighbours which are located in their proximity, leading to a sequence of interactions. In pathogenic contagion, infections occur through proximal interactions, but at the same time the movement facilitates the co-location of different strains. We analyse how the particle velocity impacts on the phase transitions on the contagion process of both a single infection and two cooperative infections. First, we identify an optimal velocity (around half of the interaction range) associated with the largest epidemic threshold, such that decreasing the velocity below the optimal value leads to larger outbreaks. Second, in the cooperative case, the system displays a continuous transition for low velocities, which become discontinuous for velocities of the order of twice the interaction range. Finally, we describe these characteristic regimes and explain the mechanisms driving the dynamics.

Keywords: Mobile particles, cooperative infections, phase transitions, temporal networks, SIR model

I. INTRODUCTION

Spreading processes, such as contagion or rumour transmission, have been modelled using agent-based approaches [1, 2]. These dynamics reach a higher level of complexity when, apart from the diffusive nature of the process, the particles move. For example, oviparous fauna can release sperm and eggs in aquatic environments and fertilization happens via mobility-driven encounters of these cells [3]. Mobility impacts biological contagion, such as the dispersal of fungi spores in plant fields [4], or the appearance of microcolonies through the attachment to the particles being carried inside the xylem for plants [5] or the veins for animals [6]. Pathogens do not spread only inside a single individual body, rather amongst different individuals too, such that proximity allows the contagion from one organism to another; this has led to a diversity of studies of epidemiology in systems of mobile fauna introducing, for example, the appearance of large outbreaks when a pathogen switches from spreading in one species to another [7], or the particularities of waterborne infections in marine environments, such as fishing or death-based transmission [8, 9]. Indeed, aquaculture represents a key industry for nutritional and financial security in developing countries, and can be strongly affected by infections spreading among the confined but mobile animals [10, 11]. This dynamics is also representative of socio-technological systems, where malware is transmitted through the connection of mobile agents’ devices to WiFi networks [12].

Hosts’ movement facilitated the spread of infections that, along the human history, have affected large populations [13, 14]. In fact, several infections that emerged in geographically distant locations may approach due to their hosts’ mobility, facilitating the interaction between different pathogens [15, 16], either cooperating or competing [17, 18]. The competition between two different infections can lead to cross-immunity, implying that the particles are immunized against other infections after a primary infection [19, 20], while the cooperation facilitates the appearance of secondary infections [21, 22]. In this article, we study contagion dynamics of either a single or two cooperative infections considering the particle velocity as a control parameter.

II. THE MODEL

We consider N particles randomly located in a two-dimensional space of size L × L with periodic boundary conditions. They move with velocity v, (x, y)(t + 1) = [(x, y)(t) + v(\cos \xi, \sin \xi)] mod (L, L), where the motion direction is represented by the angles \xi, uniformly distributed in [0, 2\pi) and set as initial condition. The Susceptible-Infected-Recovered (SIR) model describes the dynamics [23], where an infected particle transmits the infection with probability p to each node located at a distance smaller than d, and recovers after one time step, updating all the states synchronously. For two cooperative infections, A and B, the dynamics is similar to the SIR model [24], with primary infections from an infected neighbour happening with probability p, while secondary infections (those experienced by a previously infected particle) occur with probability q.

For a single infection, the initially infected population does not transmit the infection for probabilities below a critical value, p < p_c. However, for p > p_c, the infection affects a finite fraction of the system [25]. For two cooperative infections, the case q = p represents two independent contagion processes, while for q > p both infections have a cooperative interaction. Particularly, focusing on an infected particle, its neighbour i updates its state ac-
cording to the following rules (Fig. 1): a) if \( i \) is in state \( S \), it will be primarily infected with probability \( p \); b) if \( i \) is infected or recovered with/from the same infection as EGO, nothing will happen; c) if \( i \) is infected/recovered with/from the other infection, it will be secondarily infected with probability \( q \). In our simulations, we initially assign the state \( AB \) (I for single infection) to a randomly chosen particle and the state \( S \) to the rest.

At each time step, a random geometric graph [24] describes the set of interactions. In that topology, the expected number of neighbours is \( \langle k \rangle = (N - 1) \pi d^2 \), displaying a percolation transition in 2D at \( \langle k \rangle_c \approx 4.52 \) [27]. Fixing \( N = 2^{12} \) and \( L = 1280 \) (same particle density as in Ref. [25]), we obtain \( d_c = L \sqrt{\frac{\langle k \rangle_c}{(N-1)\pi}} \approx 24 \). In contrast, the system is fully connected for \( d \geq d_{\text{max}} = \frac{L}{\sqrt{2}} \approx 905 \).

Recapitulating, our model has three control parameters \( (d, v, p) \). We will keep one of these parameters constant for studying the behaviour of the order parameter, which will be the density of (doubly) recovered particles \( \rho(\rho_{ab}) \) in the final absorbing configuration for single (two cooperative) infection(s).

III. SINGLE INFECTION

We analyse the case \( p = 1 \) in the \((d, v)\) space, observing four regimes (Fig. 2a): i) \( d < d_{\text{min}} \): the infection does not spread, independently of the velocity; ii) \( d_{\text{min}} < d < d_c \): there is a threshold velocity \( v_c \), such that for \( v > v_c \), the infection affects a finite number of particles; iii) \( d \gtrsim d_c \): the infection spreads in the static case \((v = 0)\), but the low velocities are detrimental for the contagion process, implying that increasing \( v \) leads to smaller \( r \). In this regime, for a given \( d \), there are two critical velocities, \( v_c^- \) and \( v_c^+ \), such that there are no macroscopic outbreaks for \( v_c^- < v < v_c^+ \). Additionally, if we set \( d = 30 \gtrsim d_c \) and vary \( p \), the epidemic threshold \( p_c \) has a non-monotonic behaviour with \( v \), approaching the limit \( p_c(\langle k \rangle = 1) \) as \( v \to \infty \) (Fig. 2b); iv) \( d \gg d_c \): the outbreak reaches all the system, independently of \( v \).

In the limit \( v \to \infty \), the time scale describing the particles motion is much shorter than the time scale associated with the contagion process. This limit, known as annealed network, corresponds to a sequence of uncorrelated realizations of the topology. In fact, after rescaling \( p \) dividing by \( \langle k \rangle^{-1} \), \( \rho \) describes a universal behaviour for all \( d \) (Fig. S1), following the mean-field solution for SIR dynamics, with \( p_c = 1/3 \), as expected for annealed networks [23]. This result is useful to obtain, imposing \( \langle k \rangle = 1 \), \( d_{\text{min}} = L \sqrt{\frac{1}{(N-1)\pi}} \approx 11 \). This estimation arises from \( \rho \) being a probability, \( 0 \leq p \leq 1 \), such that if \( \langle k \rangle < 1 \), \( p < p_c \) for all \( p \).

We focus on the regime showing non-monotonic behaviour of the outbreak size with \( v \). For simplicity, we explain the phenomenology observed for \( p = 1 \). For \( v = 0 \), the system is ordered, such that there are no interactions between recovered and susceptible particles because the infected particles are located between them, implying an expanding front (Fig. S2). In contrast, low velocities lead to local mixing, such that there is a front, but deformed. Finally, for higher velocities there is no expanding front. In fact, the average cluster size \( \langle S \rangle \) considering only the recovered particles, at any time, characterizes this phenomenology. For \( v = 0 \), \( \langle S \rangle = R \); \( \langle S \rangle \) scales sublinearly with \( R \) in the case of local mixing, while for higher velocities (global mixing), it grows slowly with \( R \) until a percolation transition is reached at \( R_c = 1 + \langle k \rangle , \frac{L}{\sqrt{2\pi}} \) (Fig. 3).

Indeed, the dynamics reaches an absorbing configuration when none of the infected particles has a link with a susceptible particle. Hence, the comparison between the number of IS and IR links, connecting an infected particle with, respectively, a susceptible or a recovered, illustrates the likelihood of reaching an absorbing configuration. For \( v = 0 \), after an initial transient, the ratio \( IS/(IS+IR) \) reaches a stationary value around 0.5 (Fig. 3b). This stationary value decreases with \( v \), implying a reduction in the number of susceptible neighbours that an infected particle has and, as \( d \gtrsim d_c \), a dynamical behaviour similar to that observed for \( d < d_c \). This mechanism is illustrated with a similar set-up in 1D systems with maximum infection probability \( p = 1 \) and low velocities, leading to three possibilities (Fig. 3b inset): i) the susceptible and the infected particles are approaching: the susceptible particle will get infected, but at next time step it will be surrounded by many recovered par-

![FIG. 1. Contagion of B from EGO to its neighbours (particles at a distance smaller than d). Labels on the links indicate the infection probabilities.](image)
FIG. 3. a, Average connected component size ⟨S⟩ considering just the recovered particles, as a function of the number of recovered particles R. The black vertical line represents the critical value -validator for percolation in a random geometric graph. b, Time evolution of the ratio between the number of links IS and the sum of IS and IR. Inset: the three possible scenarios for a mobile system in 1D, with susceptible and infected particles depicted, respectively, in white and red colours. Velocity is changed from v = 0 (blue) to v = d (cyan) with Δv = 0.2d, and from v = d to v = 5d (red) with Δv = 0.4d, for d = 30 ≥ d_c and p = 1.

ticles; ii) they are getting away: if initially they are separated by d_0, at next time step their distance will be d_1 = d_0 + 2v such that, if v = 0.5, d_1 > d (optimal velocity); iii) they move in the same direction, leading to the same microscopic behaviour as in the static case. In conclusion, this non-monotonic behaviour is explained through the cases i) and ii), that, for an expanding front, imply a detrimental effect of low velocities on the contagion. When the velocity is further increased, the system does not display an expanding front, approaching asymptotically the mean-field behaviour that describes the infinite velocity regime.

IV. TWO COOPERATIVE INFECTIONS (q = 1)

Focusing on the static case, there are no finite outbreaks for d < d_c. For connected systems with short-range interactions (d ≥ d_c), the epidemic threshold p_c is non-linear with ⟨k⟩^{-1} (Fig. S3). While for d ≥ d_c the transition is continuous (Fig. S4), long-range interactions appear as d is increased, facilitating the presence of discontinuous transitions [29], with the critical point shifting from p_c(k) = 1 to p_c(k) = 0.5 (Fig. S3). However, in the case d ≫ d_c, p_c has a linear growth with the inverse average degree ⟨k⟩^{-1} [30], and the transition is continuous (Fig. S5), due to the immediate secondary infection of the particles after a primary infection (q ≥ p_c); hence, the dynamics is driven by primary infections, which lead to continuous transitions. However, if we decrease q for high d ≫ d_c, p_c grows, shifting from 0.5 to 1, and having a discontinuous transition (Figs. S5, S6, S7).

The opposite case to the static scenario is the infinite velocity limit. We find a universal shape for all d, when p and q are normalized dividing by ⟨k⟩^{-1}. For non-interacting infections (q = p), there is a continuous transition at p(k) = 1. However, as q(k) is increased, a discontinuous transition appears, with a characteristic gap that grows for higher values of q (Figs. S8, S9). This behaviour is qualitatively similar with that already reported for Erdős-Rényi networks [24], with the same p_c for low q(k), but leading to a change in the nature of the transition when q is varied. In fact, for q = 1, the behaviour displayed by these annealed networks is understood taking into account the results on mean-field (Fig. S10) [22]. Specifically, for d_{min} < d < d_c, the system displays a continuous transition at p_c(k) = 1; in fact, q = 1 is not higher enough than p_c for observing a discontinuous transition. As d is increased (d ≥ d_c), a discontinuous transition appears (Fig. S11) and p_c(k) shifts towards lower values. Finally, for d ≥ d_c, p_c(k) ≈ 0.5, exhibiting a continuous transition, in agreement with the results found for the static case with d ≫ d_c, as the random redistribution of the particles, with ⟨k⟩ ~ N, does not imply large changes on the topology.

Finally, we vary v to analyse the evolution from the static scenario (Fig. S3) to the infinite velocity case (Fig. S10). We focus on the case d = 30 ≥ d_c with q = 1, which has a discontinuous transition for v → ∞, while the static case lead to a continuous transition, and corresponds to the regime associated with the non-monotonic behaviour of ρ with v for a single infection (Fig. 2a).

There is a continuous transition for low velocities, with p_c increasing with v (Fig. 4a,b). However, in the region associated with global mixing effects, the transition point p_c does not vary too much, but the lines of constant ρ_{ab} are shifting to lower values of p as v is increased, leading to the appearance of a discontinuous transition with a gap which grows with the velocity (Figs. 4a,c). This behaviour is explained taking into account previously reported mechanisms for discontinuous transitions under this dynamics [23]. The appearance of a gap in ρ_{ab} at p_c represents the presence or not of avalanches of secondary infections in our system. The avalanches occur when the two infections A and B meet after following different relatively long paths in the network. As q = 1, when this meeting event occurs, an avalanche of secondary contagion events spreads over the paths that the infections
followed independently previously. The requirements for the occurrence of this mechanism are that the network describing the interactions has loops, such that this will not happen in trees, and that the infections do not meet after short paths, implying that network has a low level of local clustering. Specifically, in static networks, this dynamics was leading to continuous transitions in low-dimensional lattices (1D and 2D), and to discontinuous transitions for higher dimensions (4D lattices and Erdös-Rényi networks) [23, 29]. In our system, the mobility allows that the infections spread to a higher (and located further) fraction of particles and also decreases the effect of the local clustering, as the movement hinders the meeting events after a short path. Then, the two infections follow different and long paths before meeting: when \( p = p_c \), if the two infections meet, there will be a high fraction of doubly recovered particles \( p_{ab} \) in the final absorbing configuration; otherwise, the fraction of singly recovered particles will belong to a continuously growing branch, but the fraction of doubly recovered particles will tend to zero. Hence, there will be a discontinuous transition with two branches, with the probability of being in the upper branch representing the probability of the two infections meeting after a long path of independent contagion events.

V. CONCLUSIONS

The dynamics of a single infection illustrated how the epidemic threshold can be controlled with the particle velocity, leading to a non-monotonic behaviour with the particle velocity. This implied that, for low velocities and \( d \gtrsim d_c \), the mobile system with low velocities was leading to higher epidemic thresholds than in the static case. Specifically, we have described the mechanism leading to a dynamical fragmentation, which has already been reported in the context of disease spreading in temporal networks [31], but also in other dynamics such as the coevolving voter model [32], in contrast to the later, where the evolution of the topology depends on the dynamical configuration, our approach leads to a dynamical fragmentation even when the particles’ movement is independent of their states. In the infinite velocity limit, we found the scaling relationship between the epidemic threshold and the topological parameters, imposing \( p_c(k) = 1 \), leading to \( p_c = \frac{k^2}{(N-1)kd^2} \). These results suggest that blood or sap velocity inside different species may have evolved in order to minimize the risk of the microcolonizations by pathogens in their environment.

For the case of two cooperative infections, not only the epidemic threshold, but also the order of the phase transition varied with the particle velocity. Previous work studying this dynamics reported that the nature of the phase transitions was influenced both by the topology and the level of cooperation [23, 29]. Specifically, intermediate levels of cooperation in mean-field approximations were leading to abrupt transitions [22]. However, although a qualitatively similar behaviour was observed for two-dimensional lattices with long-range interactions, the phase transitions were continuous with short-range interactions. We analysed the interplay between this dynamics and the mobility focusing on the case \( d \gtrsim d_c \).

While in the case of low velocities there was a continuous transition, the global mixing effects dominated for higher velocities, making the system evolve towards discontinuous transitions. This behaviour may arise from the dynamical fragmentation of the short-term loops due to the high particle velocity: in contrast to the static case, where the short loops are relatively abundant, the dynamical short-term loops do not appear in the high velocity regime. This leads to the nucleation, which happens only when the population of singly recovered \( (a,b) \) has grown enough such that the proximal interaction between one infected \( A \) (or one recovered \( b \) (or) is more likely, leading to a cascade of secondary infection events.

We anticipate that the mechanism leading to a non-monotonic behaviour with the particle velocity, arising due to the differences between front dynamics and global
mixing, may appear for different dynamics on systems of mobile particles, such as synchronization or evolutionary game theory [28, 33–35]. Additionally, future research will explore other movement approaches, such as the Vicsek model [36], which leads to collective motion through the coupling of the movement direction between proximal particles, heterogeneity in the particle velocities, or the coupling between the particle velocity and its dynamical state.

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Appendix A: Single infection with infinite velocity

![Figure S1](image1)

**FIG. S1.** Universal behaviour of the density of recovered particles $\rho$ in the final absorbing configuration, for different interaction radii $d$ (from $d = 20$ (blue) to $d = 100$ (red), with colours varying continuously between these limits with $\Delta d = 10$), when the control parameter is rescaled to $p(k)$. The solid line represents the mean-field solution, given by $\rho = 1 - e^{p(k)p}$.

Appendix B: Single infection with static agents

![Figure S2](image2)

**FIG. S2.** Visualization of the contagion process at $t = 10$ and $t = 20$ with $p = 1$, $d = 30$ and $v = 0$. Recovered agents (blue symbols) are connected only to whether other recovered or infected agents (red symbols), and not with susceptible agents (black symbols).

Appendix C: Cooperative contagion with static particles

![Figure S3](image3)

**FIG. S3.** Density of doubly recovered particles $\rho_{ab}$ in the final absorbing configuration as a function of the normalized primary infection probability $p(k)$ and the interaction radius $d$, for $q = 1$. The white horizontal line represents $d = d_c$, and the vertical lines indicate the cases $p(k) = 0.5$ and $p(k) = 1$. 
FIG. S4. Time evolution of the number of infected particles $I$ for a system of static particles with $d = 30$ and $q = 1$. The plotted values of $p(k)$ are, from the black to the grey curve, of 1.7, 1.9, 2.1, 2.3, 2.5, 2.7 and 2.9.

FIG. S5. Time evolution of the number of infected particles $I$ for a system of static particles with $d = 700$ and $q = 1$. The plotted values of $p(k)$ are, from the black to the magenta curve, of 0.5, 0.6, 0.7, 0.8, 0.9, 1.0, 1.1, 1.2, 1.3 and 1.4.

FIG. S6. Density of doubly recovered particles in the final absorbing configuration, as a function of $p$ and $q$, for $d = 700 \gg d_c$. The upper and lower plots represent, respectively, the limit cases $q = 1$ and $q = p$, and the white line in the central plot indicates the limit $p(k) = 1$.

FIG. S7. Time evolution of the number of infected particles $I$ for a system of static particles with $d = 700$ and $q(k) = 10$. The plotted values of $p(k)$ are, from the black to the magenta curve, of 0.1, 0.2, 0.3, 0.4, 0.5, 0.6, 0.7, 0.8, 0.9 and 1.0.
Appendix D: Cooperative contagion with infinite velocity

FIG. S8. Cooperative contagion processes on systems of mobile particles with infinite velocity. The system has a phase transition at $p_c \langle k \rangle = 1$, which becomes discontinuous as $q$ is increased. Symbols denote different values of $q$: $q = p$ (squares), $q(k) = 2$ (diamonds) and $q(k) = 3$ (circles). Colours represent different interaction radii.

FIG. S9. Time evolution of the number of infected particles for $d = 30$, infinite velocity and $q(k) = 3$. The plotted values of $p(k)$ are, from the black to the violet curve, of 0.5, 0.6, 0.7, 0.8, 0.9, 1.0, 1.1, and 1.2.

FIG. S10. Cooperative contagion on systems of particles with infinite velocity and $q = 1$. As $d$ is increased ($d_c < d \ll d_{\text{max}}$), a continuous phase transition becomes discontinuous, coming back to a continuous phase transition for high interaction ranges, $d \sim L$. The white horizontal line represents $d = d_c$ and the vertical lines indicate the cases $p(k) = 0.5$ and $p(k) = 1$.

FIG. S11. Distribution of the number of recovered particles $m = 1 - S$ in the final absorbing configuration, for $d = 30$, infinite velocity, $p(k) = 1$, and $q = 1$, and different system sizes $N$, keeping the density $\sigma = N/L^2$ constant. There is a gap between the low prevalence configuration, leading to a fraction of recovered nodes which decays as a power-law, and the high prevalence configuration, which leads to a fraction of recovered nodes which scales linearly with the system size.
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