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A multi-agent model to study epidemic spreading and vaccination strategies in an urban-like environment

Matthieu Nadini[1], Lorenzo Zino[1,2], Alessandro Rizzo[3,4] and Maurizio Porfiri[1,5*]

[*]Correspondence:
mporfiri@nyu.edu
[1]Department of Mechanical and Aerospace Engineering, New York University Tandon School of Engineering, 11201 Brooklyn, USA
Full list of author information is available at the end of the article

Abstract

Worldwide urbanization demands for a deeper understanding of epidemic spreading within urban environments. Here, we tackle this problem through an agent-based model, in which agents move in a two-dimensional physical space and interact according to proximity criteria. The planar space comprises several locations, which represent bounded regions of the urban space. Based on empirical evidence, we consider locations of different density and place them in a core-periphery structure, with higher density in the central areas and lower density in the peripheral ones. Each agent is assigned to a base location, which represents where their home is. Through analytical tools and numerical techniques, we study the formation mechanism of the network of contacts, which is characterized by the emergence of heterogeneous interaction patterns. We put forward an extensive simulation campaign to analyze the onset and evolution of contagious diseases spreading in the urban environment. Interestingly, we find that, in the presence of a core-periphery structure, the disease spread is not affected by the time agents spend inside their base location before leaving it, but it is influenced by their motion outside their base location: a strong tendency to return to the base location favors the propagation of the disease. A simplified one-dimensional version of the model is examined to gain analytical insight into the spreading process and support our numerical findings. Finally, we investigate the effectiveness of vaccination campaigns, supporting the intuition that vaccination in central and dense areas should be prioritized.

Keywords: Agent-based model; core-periphery structure; epidemics; mobility; temporal network

1 Introduction

The number of people living in urban areas has already exceeded 4 billions and it is estimated to reach 7 billions by 2050 [1]. Global urbanization poses new challenges in different sectors, ranging from transportation to energy supply, to environmental degradation and healthcare [2]. Among these challenges, understanding how urban environments shape the evolution of epidemic outbreaks and how designing effective containment strategies have recently drawn a lot of attention from researchers and media. Paradigmatic are the examples of recent outbreaks, such as the 2003 SARS [3], 2012 MERS [4], and 2019–20 COVID-19 [5].

Analyzing how diseases spread within urban environments has been the topic of various experimental and theoretical studies [6, 7, 8, 9, 10, 11]. Experimental studies have offered a detailed analysis of urban environments [6, 7], suggesting specific preventive measures for both urban residents and travelers [8]. Theoretical studies have provided insights on how to contain outbreaks [9], as well on possible key drivers of contagion, such as the role of human mobility patterns [10] and socio-economical risk factors [11].

Despite the importance of urban environments in the global diffusion of diseases [12], how epidemic outbreaks unfold therein is yet to be fully elucidated. Some attempts to mathematically describe the diffusion of diseases within and among cities can be found in metapopulation models [13, 14, 15]. In these models, a fixed network of spatial localities is used to model the mobility patterns between cities, where homogeneously-mixed populations suffer from the epidemic process. While metapopulation models can be, at least partially, tackled through analytical methods [13, 14, 15], considerable experimental evidence challenges the assumption of homogeneously-mixed populations, which could yield misleading estimates of the extent of epidemic outbreaks [16].

On the other side of the spectrum of epidemic models, agent-based models [9, 17, 18] constitute a valuable framework to offer a realistic description of how diseases diffuse within urban environments. However, this advantageous feature is accompanied by some drawbacks, including the need of mobility data and models, the use of massive computational resources when the system size scales up, and the lack of analytical techniques for model characterizations. A viable approach to agent-based modeling is based on two-dimensional representations, where agents move and interact according to proximity criteria [19, 20, 21, 22, 23, 24, 25, 26]. As a first approximation, the motion of the agents can be described according to a random walk with sporadic long range jumps [19]. Building on this approximation, it is possible to include realistic features such as nonhomogeneous infection rates [20] and heterogeneous radii of interaction [22, 23]. Much work is needed, however, to fully capture and describe realistic patterns of human mobility, which are shaped by the complex structure of urban environments [27].

Here, we contribute to the field of agent-based modeling by presenting a two-dimensional model that is capable of reproducing a spatially inhomogeneous urban-like environment, in which a heterogeneous population follow realistic rules of mobility. Inspired by previous theoretical studies [22, 23], we assume that agents have a heterogeneous radius of interaction, which accounts for variations among individuals in their involvement in social behavior and activities.

We consider a urban-like environment composed of multiple locations, each of them representing a well-defined region of the urban space (that is, a neighborhood of a city). Through this spatial organization, our model is able to encapsulate two key features of urban environments: first, it can reproduce typical core-periphery structures, where central regions are more densely populated than peripheral ones [28, 29]; and, second, it allows to mimic the inhomogeneity in movement patterns of humans, where people tend to spend most of their time in a few neighborhoods — for example, experimental studies suggest that individuals spend most time either at home or at work, while only sporadically visit other neighbors [30, 31, 32].

To reproduce realistic conditions for agents' mobility, we posit two different mobility schemes, applied within and outside the agents' base location (that is, where their home is). While the homogeneous mixing assumption seems reasonable within the agents' base location, we assume that agents tend to move outside of their base location following a gravity model and a biased random walk. Hence, agents are more likely to explore regions close to their base location rather than remotely-located regions [30, 31, 32]. From this mobility pattern, we construct a network of contacts, whose topology is examined in this study. Through some mathematical derivations an numerical simulations, we seek to identify analogies between the proposed agent-based model and existing temporal network approaches, where spatial mobility is lumped into nodal parameters [33, 34, 35, 36, 37].

We adopt the proposed framework to study how urban-like environments shape the diffusion of infectious diseases, using the illustrative epidemic progression models with the possibility of reinfection (susceptible–infected–susceptible, or SIS) or permanent removal (susceptible–infected–removed, or SIR) [38]. Our results confirm the intuition that agents' density plays a critical role on diffusion of both SIS and SIR processes. In the limit case where the entire urban area consists of one location, agents that move outside the location only seldom interact with other agents, thereby hindering the contagion process.

In the more realistic scenario of a core-periphery structure with multiple locations, we unexpectedly find that the time spent by agents in their base location does not influence the endemic prevalence in the SIS model and the epidemic size in the SIR model, which are measures of the overall fraction of population that is affected by the disease. A possible explanation for this counterintuitive phenomenon may be in the agents' mobility rules. In fact, commuting patterns that bring agents from central areas to peripheral ones may yield a reduction in the diffusion in the central areas. Contrarily, commuting patterns from peripheral to central areas lead to the opposite effects. To detail the inner working principles of this unexpected result, we present a minimalistic one-dimensional version of the model, which is amenable to a complete analytical treatment.

We also explore the interplay between the agents' radius of interaction and their positioning in the core-periphery structure. We find that when agents' with greater radii are assigned to the less dense and peripheral locations, then the endemic prevalence (in the SIS model) and the epidemic size (in the SIR model) strongly decrease with respect to a random assignment. Moreover, when agents' with greater radii are assigned to denser (and central) locations the fraction of population affected by the disease is not sensibly increased. In real cities, our results support the intuition that more central areas are the crossroads of individuals commuting in a city and are critical for the spread of diseases.

Finally, we numerically analyze the effect of targeted vaccination strategies, which consist of immunizing a portion of the population in a specific location, prior to the disease onset. Consistent with the intuition that central locations play a key role on the spread of epidemic diseases, we find that the best strategy is to prioritize the vaccination of agents belonging to central urban areas.

The rest of the manuscript is organized as follows. In Table 1, we summarize the notation and the nomenclature used throughout the paper. In Section 2, we introduce the model of agents' mobility. In Section 3, we describe and analyze the temporal network formation mechanism. In Section 4, we numerically study the spread of epidemic diseases and compare several vaccination strategies. In Section 5, we discuss our main findings and propose further research directions.

2 Model

We consider $N \geq 1$ agents (also called agents), labeled by positive integers $\mathcal{V} := \{1, \dots, N\}$, moving in a square planar space with side length $D > 0$ and with periodic boundary conditions, similar to [19]. The position of agent $i \in \mathcal{V}$ at the discrete time $t \in \mathbb{Z}_{\geq 0}$ in a Cartesian reference is denoted by $(x_i(t), y_i(t)) \in [0, D] \times [0, D]$.

2.1 Urban-like environment

We deploy the N agents over L locations, each of them representing a bounded portion of the square space. The set of all locations is $\mathcal{L} = \{1, \dots, L\}$ and each location $\ell \in \mathcal{L}$ occupies a convex region of the planar space $\Omega_\ell \subset [0, D] \times [0, D]$ with area A_ℓ. We assume that all the locations are mutually disjoint and we order them in ascending order according to their area, that is, $A_1 \leq \cdots \leq A_L$. We hypothesize that $A_L \ll D^2$, that is, each location is much smaller than the whole square space. Each agent is assigned a specific, base location (that is, their home) according to a map: $\beta : \mathcal{V} \longrightarrow \mathcal{L}$; we assume that each base locations is associated with the same number of agents, $n = N/L$ [1]. As a result, the density of agents assigned to location ℓ,

$$\rho_\ell := \frac{n}{A_\ell}\,, \tag{1}$$

varies with the location. Note that locations are sorted in descending order of density, that is $\rho_1 \leq \cdots \leq \rho_L$.

For simplicity, in the numerical simulations implemented throughout this paper, the convex regions are taken as circles with nondecreasing radii $\Sigma_1 \leq \cdots \leq \Sigma_L$. Inspired by empirical and theoretical studies [28, 29, 39, 40], radii of the locations are extracted from a power law distribution so that $P[\Sigma_\ell = \Sigma] \sim \Sigma^{-\gamma}$ (where $P[\cdot]$ indicates probability), with bilateral cutoffs such that $\Sigma_\ell \in [\Sigma_{\min}, \Sigma_{\max}]$, for any $\ell \in \mathcal{L}$. The presence of cutoffs guarantees that all locations can be fit in the squared space and allows for setting an upper bound on the maximum density (consistently with real-world applications). Note that, since the radii are power law distributed with exponent $-\gamma$, also the areas of the locations are power law distributed with exponent -2γ with cutoffs such that $A_\ell \in [\pi\Sigma_{\min}^2, \pi\Sigma_{\max}^2]$.

Empirical studies on urban environments suggest that cities are constructed according to a core-periphery structure, whereby locations with smaller areas and denser population are located in their center, while locations with larger areas and sparser population pertain to peripheral areas [28, 29], as shown in Fig. 1 (a). We implement a heuristic algorithm to generate a locations' layout according to a core-periphery structure and qualitatively reproduce empirical results. Figure 1 (b) shows the output generated by our algorithm, whose structure is qualitatively consistent with the empirical observations reported in Fig. 1 (a). Details of the algorithm used to create such a core-periphery structure are presented in Appendix A.

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[1] We consider that N is a multiple of L.

In this paper, unless otherwise specified, we consider that the n members of each location are randomly chosen, independently of their radius of interaction. We also examine the cases in which there is a correlation (positive or negative) between the agents' radius of interaction and the density in their base location: a positive correlation means that agents with larger radius are assigned to denser (central) locations, while a negative correlation identifies the case in which agents with larger radius are placed in the less dense (peripheral) locations.

2.2 Law of motion

Agents' positions evolve in time according to a discrete-time dynamics. Hence, their positions are updated at each discrete time-step $t \in \mathbb{Z}_{\geq 0}$. The law of motion of the generic agent i depends on whether it is outside or inside its base location $\beta(i) \in \mathcal{L}$. If agent $i \in \mathcal{V}$ is outside its base location, that is, $(x_i(t), y_i(t)) \notin \Omega_{\beta(i)}$, it performs a biased random walk toward its base location[2]; on the contrary, if it is inside its base location, it can move to a random position (within its base location), or exit according to a probabilistic mechanism.

Specifically, if the agent is not in its base location, then

$$\begin{cases} x_i\,(t+1) = x_i(t) + v\cos\theta_i(t)\,, \\ y_i\,(t+1) = y_i(t) + v\sin\theta_i(t)\,. \end{cases} \tag{2}$$

Here, $v > 0$ is the (constant) speed and $\theta_i(t)$ is an angle, determined as follows:

$$\theta_i(t) := \Phi_i(t) + \alpha\Delta\theta_{it}\,, \tag{3}$$

where $\Phi_i(t)$ is the direction of the shortest path from $(x_i(t), y_i(t))$ to the region $\Omega_{\beta(i)}$; $\Delta\theta_{it}$ is a random variable with values uniformly distributed in $[-\pi, \pi)$, extracted at every time t and for every agent i independently of the others; and $\alpha \in [0, 1]$ is a randomness parameter that regulates how much the agents tend to deviate from the shortest path to return to the base location, when they are outside it. When $\alpha = 1$, the agent moves completely at random, while, when $\alpha = 0$, it moves along the shortest path toward its location. The direction $\Phi_i(t)$ is formally defined by introducing

$$(\bar{x}_i(t), \bar{y}_i(t)) := \operatorname*{arg\,min}_{(x,y)\in\Omega_{\beta(i)}} \left\{ \big(x_i(t) - x\big)^2 + \big(y_i(t) - y\big)^2 \right\}, \tag{4}$$

so that

$$\Phi(t) := \begin{cases} \arctan \dfrac{\bar{y}_i(t) - y_i(t)}{\bar{x}_i(t) - x_i(t)} & \text{if } \bar{x}_i(t) > x_i(t)\,, \\ \pi + \arctan \dfrac{\bar{y}_i(t) - y_i(t)}{\bar{x}_i(t) - x_i(t)} & \text{if } \bar{x}_i(t) < x_i(t)\,, \\ +\dfrac{\pi}{2} & \text{if } \bar{x}_i(t) = x_i(t) \text{ and } \bar{y}_i(t) > y_i(t)\,, \\ -\dfrac{\pi}{2} & \text{if } \bar{x}_i(t) = x_i(t) \text{ and } \bar{y}_i(t) < y_i(t)\,. \end{cases} \tag{5}$$

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[2] The distance between a point and a set is defined as the minimum Cartesian distance between the point and a generic point of the set.

When the agent is in its base location, $(x_i(t), y_i(t)) \in \Omega_{\beta(i)}$, the law of motion is defined as follows. Given a parameter $p \in [0, 1]$ (constant in time and equal for all agents), with probability $1 - p$, the agent moves to a position chosen uniformly at random within its base location, so that its position is completely uncorrelated with the previous one. Otherwise, with probability p, the agent jumps outside its base location, ending in a position of the remaining space according to a distance decay law. In particular, we assume that the probability of jumping at a distance d from the border of the base location decays exponentially, that is,

$$P_{\text{jump}}(d) \sim e^{-cd}\,, \tag{6}$$

where the constant $c > 0$ is the decay rate. Specifically, the expected distance at which an agent jumps is equal to $1/c$. A sensible choice of the exponent in the law in Eq. (6) yields a typical behavior observed in many empirical studies [41, 42], whereby agents tend to gravitate within and around their base location, while sporadically initiating journeys toward further locations [43]. Two salient snapshots of agents' motion are illustrated in Figs. 2 (a) and 2 (c).

3 Temporal network of contacts

Upon the mobility model, we construct the network of contacts, which is the means through which the disease spreads. In this vein, agents create undirected temporal links based on proximity with other agents. Specifically, agent $i \in \mathcal{V}$ contacts all other agents located within a circle of radius σ_i centered in its current position $(x_i(t), y_i(t))$. We assume that agents have heterogeneously distributed radii, extracted from a power law $G(\sigma) \sim \sigma^{-\omega}$, with suitable cutoffs so that $\sigma \in [\sigma_{\min}, \sigma_{\max}]$.

An undirected temporal link between two agents i and j is created when the Euclidean distance at time t between the position of agent i, $(x_i(t), y_i(t))$, and the position of agent j, $(x_j(t), y_j(t))$, is less than or equal to the maximum of the two radii σ_i and σ_j, that is,

$$\sqrt{\big(x_i(t) - x_j(t)\big)^2 + \big(y_i(t) - y_j(t)\big)^2} \leq \max\{\sigma_i, \sigma_j\}\,. \tag{7}$$

Figures 2 (b) and 2 (d) show two consecutive instances of the network formation process. Toward modeling of epidemics in urban environments, our model allows agents inside a location to interact with agents outside the location, see, for example, agents 2 and 3 in Figs. 2 (a) and 2 (b).

The intricacy of the motion patterns and the nonsmooth process for generating the network of contacts hinder the analytical tractability of the model in its general formulation. However, for some cases it is possible to establish analytical insight on some model features. For example, it can be possible to analytically study the number of connections generated by the nodes, which represent potential paths of infection throughout the population. We examine two special cases: either in a free space, without any location ($L = 0$), or when the law of motion of the agents outside their base locations is deterministic ($\alpha = 0$) and the locations are uniformly distributed in the plane. The general case of a core-peripheral structure and stochasticity in the motion out of the location is treated through numerical simulations.

3.1 Analytical results on two special cases

We begin our analysis by considering the special case of a free space, that is, $L = 0$, where agents perform simple random walks with constant velocity in the plane. In this scenario, Eq. (3) should be intended without the component associated with the location $\Phi_i(t)$ and with $\alpha = 1$.

According to Eq. (7), at time t, agent i creates undirected interactions with the agents that are located within a circle of radius σ_i about its position. Since all the agents move according to an unbiased random walk, when the system is in its steady state, the expected fraction of agents within the circle is proportional to the ratio between the area of the circle and the whole planar space. We denote such a quantity as

$$\mathrm{E}\left[k_i^+\right] = \frac{\pi \sigma_i^2}{D^2} \left(N - 1\right), \tag{8}$$

where $\mathrm{E}[\cdot]$ indicates expectation.

Further, agent i can form undirected interactions with other agents if it is located within their radii of interaction. To avoid double counting and exclude connections that are in Eq. (8), the radius of j should be greater than the one of i, and i should be in the circular crown centered in the location of j, between the circles of radius σ_i and σ_j. The probability of such an event is estimated as $\pi(\sigma_j^2 - \sigma_i^2)/D^2$. Let us introduce the set $\mathcal{C}_i$ of agents with radius of interaction greater than σ_i and let us define $\langle \sigma^2 \rangle_i = |\mathcal{C}_i|^{-1} \sum_{j \in \mathcal{C}_i} \sigma_j^2$ as their average square radius. The expected fraction of connections formed by agent i with other agents beyond those included in Eq. (8) is

$$\mathrm{E}\left[k_i^-\right] = \frac{1}{D^2} \pi \sum_{j \in \mathcal{C}_i} \left(\sigma_j^2 - \sigma_i^2\right) = \frac{\pi}{D^2} |\mathcal{C}_i| \left(\langle \sigma^2 \rangle_i - \sigma_i^2\right). \tag{9}$$

By summing Eqs. (8) and (9), we conclude that the average number of agents that an agent interacts with in a unit time, termed its average degree k_i, is equal to

$$\mathrm{E}\big[k_i\big] = \mathrm{E}\left[k_i^+\right] + \mathrm{E}\left[k_i^-\right] = \frac{\pi}{D^2} \Big((N-1)\, \sigma_i^2 + |\mathcal{C}_i| \big(\langle \sigma^2 \rangle_i - \sigma_i^2\big) \Big). \tag{10}$$

In the limit of large systems, $N \to \infty$, we approximate

$$|\mathcal{C}_i| \approx (N-1) \frac{\sigma_i^{1-\omega} - \sigma_{\mathrm{max}}^{1-\omega}}{\sigma_{\mathrm{min}}^{1-\omega} - \sigma_{\mathrm{max}}^{1-\omega}} \tag{11}$$

and

$$\langle \sigma^2 \rangle_i \approx \frac{(\omega - 1)(\sigma_{\mathrm{max}}^{3-\omega} - \sigma_i^{3-\omega})}{(3-\omega)(\sigma_i^{1-\omega} - \sigma_{\mathrm{max}}^{1-\omega})}. \tag{12}$$

Details on the derivation of Eqs. (11) and (12) can be found in Appendix B. Substituting these expressions in Eq. (10), the expected degree of node i in the limit of large systems reads

$$\mathrm{E}\big[k_i\big] \approx \frac{(N-1)\pi}{D^2}\left(\left(1-\frac{\sigma_i^{1-\omega}-\sigma_{\max}^{1-\omega}}{\sigma_{\min}^{1-\omega}-\sigma_{\max}^{1-\omega}}\right)\sigma_i^2+\frac{(\omega-1)(\sigma_{\max}^{3-\omega}-\sigma_i^{3-\omega})}{(3-\omega)(\sigma_{\min}^{1-\omega}-\sigma_{\max}^{1-\omega})}\right). \quad (13)$$

Note that when the agent radius is close to the minimum, that is, $\sigma_i \approx \sigma_{\min}$, Eq. (13) is dominated by the second term, while when the agent radius is close to the maximum, that is, $\sigma_i \approx \sigma_{\max}$, the right hand side of Eq. (13) scales as with σ_i^2.

Now, we consider the limit case in which agents move straight toward their base location, that is, $\alpha = 0$, and we assume that locations are uniformly distributed in the planar space. We observe that the system is ergodic [44] and the steady-state probability that a agent is inside its own base location is equal to

$$q_{\mathrm{in}} := \lim_{t\to\infty} P\big[(x_i(t), y_i(t)) \in \Omega_{\beta(i)}\big] = \frac{1}{1+\dfrac{pe^{cv}}{e^{cv}-1}} = \frac{e^{cv}-1}{e^{cv}\,(1+p)-1}\,. \quad (14)$$

The derivation of Eq. (14) is reported in Appendix B. When the system reaches its stationary state, the number of agents in location ℓ is equal to the sum of two contributions. The first one consists of agents whose base location is Ω_ℓ and are in that location, that is, on average, nq_{in}. The second one is due to agents whose base location is not Ω_ℓ, but are in Ω_ℓ. The second contribution is relatively small since locations are placed randomly in the entire space $D \times D$, and we discard it when the system is large.

The steady-state density in location ℓ can be approximated by considering only the agents assigned to it. Hence, the expected number of connections of agent i within its base location is approximated by

$$\mathrm{E}\left[k_{\mathrm{in},i}\right] \approx \frac{q_{\mathrm{in}}}{\pi\Sigma_\ell^2}\pi\Big((n-1)\,q_{\mathrm{in}}\sigma_i^2 + |\mathcal{C}_{i,\ell}|q_{\mathrm{in}}\big(\langle\sigma^2\rangle_{i,\ell}-\sigma_i^2\big)\Big)\,, \quad (15)$$

where $\mathcal{C}_{i,\ell}$ and $\langle\sigma^2\rangle_{i,\ell}$ are the set of nodes with radius greater than σ_i in location ℓ and their average square radius, respectively. In the limit of large systems and assuming the distribution of the radii of interaction to be independent of the agents' base locations, then $|\mathcal{C}_{i,\ell}| \approx \frac{n-1}{N-1}\mathcal{C}_i$ and $\langle\sigma^2\rangle_{i,\ell} \approx \langle\sigma^2\rangle_i$. Combining Eqs. (11) and (12) into Eq. (15), we obtain

$$\mathrm{E}\left[k_{\mathrm{in},i}\right] \approx \frac{q_{\mathrm{in}}^2}{\pi\Sigma_\ell^2}\,(n-1)\pi\left(\sigma_i^2+\frac{\sigma_i^{1-\omega}-\sigma_{\max}^{1-\omega}}{\sigma_{\min}^{1-\omega}-\sigma_{\max}^{1-\omega}}\left(\frac{(\omega-1)(\sigma_{\max}^{3-\omega}-\sigma_i^{3-\omega})}{(3-\omega)(\sigma_i^{1-\omega}-\sigma_{\max}^{1-\omega})}-\sigma_i^2\right)\right). \quad (16)$$

When a core-periphery structure is present, as in Fig. 1, locations are not uniformly distributed in space and often are close to each other. For instance, a central location ℓ is surrounded by other locations and interactions generated by agents whose base location is not Ω_ℓ cannot be neglected. This case is discussed in the following, by means of numerical simulations.

3.2 Numerical results

We conclude this section with some numerical simulations to validate our analytical findings and explore the more general case of multiple locations arranged in a core-periphery structure. In our numerics, we estimate the agents' average degree by averaging all the interactions recorded over a sufficiently long time window ($T \gg 1$, where T is the duration of the observation).

In Fig. 3 (a), we consider the case without locations. Our numerical results are consistent with our analytical prediction in Eq. (13), that is, agents with a greater radius of interaction have a greater average degree. In Fig. 3 (b), we examine the case of multiple locations uniformly distributed in a vast space. From Eq. (16), we observe that $\mathrm{E}[k_{\mathrm{in},i}]$ is inversely proportional to the square of the radius of location $\beta(i)$. In Fig. 3 (b), we multiply the numerical estimation of each agents' average degree by the corresponding location's squared radius, to allow a graphical representation of the comparison between numerical estimations and analytical predictions. Once again numerical results are in close agreement with analytical findings from Eq. (16).

In order to offer insight into the influence that a core-periphery structure has on the agents' average degree, we analyze three different scenarios. First, we study the case in which agents are strongly tied to their base location, such that they have low probability of jumping outside their base location (small p) and low probability of deviating from the shortest path to return to the base location, when they are outside (small randomness α), in Fig. 4 (a). Second, we examine the case in which the probability of jumping outside their base location and the agents' randomness in the random walk are intermediate, in Fig. 4 (b). Finally, we investigate the case in which agents tend to spend most of their time outside their base location (large p and α), in Fig. 4 (c).

In general, we observe that agents with larger radius of interaction have a larger average degree. More specifically, we note that, among the agents with small radius of interaction, the agents that are assigned to central locations have a larger average degree than agents with small radius of interaction that are assigned to peripheral locations. This result is independent of the time spent outside their base location (that is, independent of p and α). Interestingly, the same argument does not apply when agents have a large radius of interaction. In this situation, agents assigned to peripheral locations may have a greater degree than agents assigned to central ones because their high radius of interaction allows a multitude of interactions, independently from the position of their base location.

Further, we comment that time spent outside the base location (regulated by p and α) is inversely proportional to the dispersion of the agents' degree. In fact, the largest dispersion in agents' degree is registered when the probability of jumping outside the base location and the agent's randomness are small, in Fig. 4 (a). Dispersion in agents' degree decreases as the probability of jumping outside the base location and the agent's randomness increase, in Fig. 4 (b) and in Fig. 4 (c). A possible explanation of this phenomenon can be based on the following argument. The more agents spend time inside their base location, the more they remain isolated from other agents in the system. On the contrary, agents' isolation is reduced when they spend more time outside their base location: they are able to interact with all the agents in the system, and, as a consequence, the dispersion in their degree decreases.

4 Epidemic processes

Here, we investigate on the spreading of epidemics over spatially-distributed populations that behave according to the presented model. We focus on the impact of three salient model's characteristics on the SIS and SIR epidemic processes. Namely, (i) the random exploration of the space governed by the parameter α, (ii) the probability of jumping outside the base location p, and (iii) the presence of a core-periphery structure. Interestingly, when a core-periphery structure is present, the time spent outside the base location does not play an important role in the evolution of the contagion process.

Even though the complexity of the mobility mechanism and of the presence of a geographical structure hinders the general mathematical treatment of the epidemics, some mathematical insight can be obtained by studying a simplified, one-dimensional version of our model, as reported in Appendix C. Then, we study whether some regions of our core-periphery structure are more susceptible from the presence of agents with greater radius. To this end, we consider the effect of having agents with greater radius of interaction in either the more central or more peripheral locations, thereby discovering that central locations are important for sustaining the overall diffusion. Finally, we analyze the outcome of vaccination strategies, finding that the highest beneficial effect for the entire population is registered when the vaccination of agents in central locations is prioritized.

We consider an infectious disease with the possibility of re-infection (SIS model) or immunization (SIR model), after the contraction of the infection. In the SIS model, agents can be either susceptible to the disease or infected [38]. Two mechanisms characterize the epidemic dynamics: infection propagation and recovery process. The former occurs when an infected agent contacts a susceptible one, who may become infected with a probability λ, independently of the others. The latter consists of the spontaneous transition from the infected state to the susceptible one and occurs with probability μ at each unit time, independently of the others. In the SIR model, instead, individuals who recover cannot be infected again and transition from the infected state to a removed state with probability μ per unit time [38].

In the SIS model, we examine the endemic prevalence (that is, the total number of cases in the long-term), which has typically two possible outcomes: either it quickly dies out and tends to zero, or it fluctuates around a quantity greater than zero for a nonnegligible amount of time, denoted by i^*. For the SIR model, instead, the fraction of infected individuals in the system always dies out in the long-run. However, the total fraction of individuals who have been infected may vary, depending on the model parameters. The SIR epidemic size, denoted as r^∞, is defined as the fraction of recovered individuals at the end of the epidemic process.

4.1 Impact of key parameters

We start our analysis by exploring the case of a space containing one location, that is, $L = 1$, which is the base for all the agents. Our results reveal that increasing either α or p reduces the impact of the epidemic disease, both in the case of possible reinfection (SIS), as shown in Fig. 5 (a), and in the case of immunization after recovery (SIR), as illustrated in Fig. 5 (b). Specifically, in the SIS process, the endemic prevalence, i^*, is high when α and p are low because agents spend more time inside the location, which is the densest region of the entire space, thus favoring interactions between agents. On the contrary, when agents spend more time outside the location (by increasing either α or p [3]), interactions become more sporadic, and so the likelihood that the disease spreads is lower. From our numerical simulations, we observe that there is a threshold for α (in this case, $\overline{\alpha}$ is close to 0.5), beyond which the spread of diseases is halted. Simulations with different values of the parameters show a similar behavior, with varying values of the threshold $\overline{\alpha}$. As a result, in the SIS dynamics, the disease is not able to spread and the endemic prevalence tends to zero, in Fig. 5 (a); a similar behavior is observed for the SIR process. Consistent results are found for the one-dimensional approximation in Appendix C.

Next, we consider the case in which multiple locations are present. We investigate a scenario with $L = 100$ locations, as reported in Fig. 6. Our numerical results suggest that that increasing the agent's randomness, α, still reduces the endemic prevalence (in the SIS model) and the epidemic size (in the SIR model), i^* and r^∞, similar to the case of a single location. Numerical results in Figs. 6 (a) and 6 (c), however, seem to display a nonmonotonic behavior of the fraction of population affected by the disease, whereby small values of α may favor the epidemic outbreak instead of hindering its inception. We record the existence of a threshold for α (in our simulations, this is close to 0.5) at which a sharp transitions takes place for both the endemic prevalence (in the SIS model) and the epidemic size (in the SIR model). According to Eq. (3), by increasing α, agents' randomness is increased and, as a consequence, agents are less likely to interact with each other and support the spread of the disease.

Surprisingly, we observe that the probability of jumping outside the base location, p, seems to have a negligible effect on the outcome of the SIS and SIR disease processes. A reason for this phenomenon may be found in the following intuition. The core-periphery structure analyzed in our work, in Fig. 1, allows two contrasting effect to simultaneously occurs. On the one hand, agents moving outside the central areas are likely to end in peripheral ones, decreasing the agents' density in the central regions and increasing the density in the peripheral ones. On the other hand, agents moving outside the peripheral areas are likely to end in the central ones, thereby increasing the density in the central regions and decreasing the density in the peripheral ones. Overall, these two opposite effects tend to balance each other. Similar evidence is gathered in the one-dimensional version of the model in Appendix C.

4.2 Impact of correlation between agents' radius and locations' density

Here, we study the impact of the correlation between the radius of interaction of agent i, σ_i, and the density of its base location, $\rho_{\beta(i)}$. We compare the uncorrelated case (analyzed earlier in Figs. 6 (a) and 6 (c)), where agents are randomly assigned to a location, with the cases of either positive or negative correlation between the two variables. In the case of positive correlation, agents with larger radius are assigned to denser (and central) locations. In the case of negative correlation, agents with

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[3] In order to allow to agents to exit from the base location, the probability to jump outside should be $p > 0$.

larger radius belong to the less dense (and peripheral) locations. We consider a scenario with $L = 100$ locations, whose results are illustrated in Fig. 7.

Both the endemic prevalence, i^*, and the epidemic size, r^∞, are marginally affected by a positive correlation, while they strongly diminish if the radii and locations are negatively correlated, as shown in Figs. 7 (a) and 7 (b), respectively. In both the positive-correlated and uncorrelated cases, agents with larger radii occupy the central locations, thereby sustaining the diffusion of the disease. On the other hand, if agents with large radii are relegated to peripheral and sparser areas, it would be more difficult for them to create connections and fuel the diffusion process.

4.3 Vaccination strategies

Finally, we study the effect of different vaccination strategy applied to our population. Specifically, we consider a purely randomized strategy and two targeted vaccination policies. In the three strategies, we assume that a fraction of the population is vaccinated and is thus immune to the disease. In the “Random” vaccination mechanism, we vaccinate a fraction of the population, sampled uniformly at random. In the “Center” targeted mechanism, we select such a fraction starting from the agents assigned to the most central locations. In the “Peripheral” targeted mechanism, we choose such a fraction starting from the agents assigned to the most peripheral locations.

From Fig. 8, we observe that prioritizing the vaccination of agents assigned to the most central locations has the most beneficial effect for the prevention of the diffusion of the epidemic disease, while the worst strategy focuses vaccination in peripheral areas. In fact, agents whose base locations are in the center can easily reach all portions of the environment, thereby contacting the majority of the agents. By focusing the vaccination on central areas, the contacts generated by these agents do not contribute to the spread, thereby significantly reducing the contagion.

5 Discussion and conclusion

In this paper, we studied a class of agent-based models [19], in which agents move in a two-dimensional space and interact according to proximity criteria. We extended such class of models by encapsulating a core-periphery structure, typical of urban environments [28, 29], where central areas are more densely populated than peripheral ones. Our urban-like environment is partitioned in several locations, each of them representing a restricted portion of the space where a fraction of agents tend to spend most of their time (that is, the neighborhood where the agents have their home). Agents’ law of motion depends on whether they are inside or outside their base location. When agents are inside their base location, they take a random position within the base location at every time-step. When outside, they tend to move back to their base location by following a biased random walk. We also introduced a simplified, one-dimensional version of the model, whose analysis allows for gaining analytical insight into the process.

The contribution of the study is fourfold. First, we analytically and numerically studied the temporal network formation mechanism, demonstrating that heterogeneously distributed radii of interaction in the population generate heterogeneity in the degree distribution of the temporal network of contacts, similar to what is observed in many real-world systems [45, 46, 47].

Second, we investigated the role of the urban-like environment on the spread of epidemic outbreaks. Specifically, we considered epidemic prevalence in the susceptible–infected–susceptible (SIS) model and epidemic size in the susceptible–infected–recovered (SIR) model. We found that both these quantities, which measure the fraction of the system that is affected by the disease, are hindered by increasing the randomness of the agents' law of motion. In fact, increasing agents' randomness increases the chances that agents randomly explore areas without other agents, thereby diminishing the contagions. Interestingly, we discovered that the endemic prevalence and epidemic size have nontrivial relationships with the probability of jumping outside the base location. When the entire urban environment is modeled as a unique location, larger probabilities of jumping outside hinder the epidemic diffusion. In fact, outside the location, interactions between agents are rare, slowing down the disease spread. Instead, when multiple locations are arranged in a core-peripheral structure, our numerical results suggest that epidemic prevalence and size are independent of the probability of jumping outside the base location. A possible explanation for this phenomenon might be that, when agents in central locations jump outside them, they are likely to end in peripheral locations, diminishing the fraction of agents in central areas. This event is compensated by agents from peripheral locations that jump in central ones. Our numerical results are in agreement with the theoretical findings in the simplified, one-dimensional, version of our agent-based model.

Third, we found that central locations play a key role on the diffusion of epidemic diseases. In particular, we studied the influence of the correlation between agents' radius and locations' density. When these quantities are negatively correlated, agents with larger radius belong to less dense (peripheral) locations, while when positively correlated, agents with larger radius belong to denser (central) locations. The endemic prevalence (in the SIS model) and the epidemic size (in the SIR model) are only marginally favored by the presence of many agents with large radius in the more central locations (positive correlation), while the diffusion of the epidemic is hindered if central locations are mostly assigned to agents with small radius of interaction (negative correlation).

Finally, we studied the effect of targeted vaccination strategies. We found that the vaccination of agents that belong to central locations is the most beneficial approach for the entire population, leading to the smallest epidemic prevalence. Our analysis corroborates our previous observation that central (and more dense) locations are crucial in the diffusion of disease processes. We emphasize that the proposed vaccination strategy can be implemented with information about the system at the mesoscopic level of locations, that is, without any information on the specific properties of single individuals (for instance, their radius of interaction). With information at the individual level, the proposed policy can be improved by combining knowledge about locations and radii of interaction prioritizing vaccination of central agents with large radius of interaction, which acts as "superspreaders."

A main limitation of our work resides in the assumption that each agent belongs to a unique location, while the remaining urban area is only seldom explored. A more realistic approach could consider agents that may be assigned to multiple locations. Our theoretical study of the one-dimensional case is able to provide insight into some aspects of our model. However, a general mathematical theory is missing. We believe that our preliminary results constitute a starting point for performing a more general theoretical analysis of the two-dimensional model. Furthermore, variations of the proposed model can be easily generated. For instance, the gravity law in our model could be replaced by the one recently proposed in [48].

Overall, our work determines that central urban areas are critical in the diffusion of epidemic diseases within a city, being the crossroad of most of the urban population, and thus should be carefully included into mathematical models of epidemic outbreaks. By vaccinating individuals in central urban areas, we can halt the overall contagion better than randomly distributing limited vaccination supplies. Our effort may help practitioners and epidemiologists to ease the development of proper containment strategies and to reach heard immunity [49] within urban environments.

Appendix A: Algorithm to generate a core-periphery structure

From a practical point of view, packing all convex regions L_ℓ (locations) in the square space $D \times D$ is a nondeterministic polynomial-time hardness (NP-hard) problem [50, 51], often requiring to find approximate methods [52, 53, 54]. In our study, we aim to reproduce the core-periphery structure present in real urban areas, as shown in Fig. 1 (a). To this end, we developed a heuristic algorithm that unfolds according to the following steps.

1 Place the center of the denser location, (x_1^c, y_1^c), in the center of the square space, $(x_1^c, y_1^c) = (D/2, D/2)$.

2 Initialize $\ell \leftarrow 1$, $\sigma_{\text{in}} \leftarrow 0$, and $\sigma_{\text{out}} \leftarrow \langle\sigma\rangle = \sum_{\ell=1}^{L} \sigma_\ell / L$.

3 Create a circular crown centered in $(D/2, D/2)$ with internal radius σ_{in} and external radius σ_{out}.

4 Randomly place the center of location $\ell+1$ in the crown and check for overlaps.

   i) If location $\ell+1$ does not overlap with other locations, then the location is placed. Increase the index ℓ by 1, that is, $\ell \leftarrow \ell + 1$. If $\ell = L$, then terminate the algorithm. Otherwise, resume it to step 4.

   ii) If an overlap occurs, then repeat the current assignment in 4. After a number of consecutive failed attempts (we set this limit to 100), stop the current iteration and move to step 5.

5 Set $\sigma_{\text{in}} \leftarrow \sigma_{\text{out}}$ and $\sigma_{\text{out}} \leftarrow \sigma_{\text{out}} + \langle\sigma\rangle$, and resume the algorithm to step 3.

Appendix B: Analysis of the temporal network of interactions

Here, we compute the quantities $|\mathcal{C}_i|$ and $\langle\sigma^2\rangle_i$, in the limit of large systems $N \to \infty$. We start with $|\mathcal{C}_i|$. The probability density function $G(\sigma)$ of the power law distribution of the radii of interaction, $G(\sigma) \sim \sigma^{-\omega}$ with cutoffs, so that $\sigma \in [\sigma_{\min}, \sigma_{\max}]$, as

$$G(\sigma) = \begin{cases} \dfrac{\omega - 1}{\sigma_{\min}^{1-\omega} - \sigma_{\max}^{1-\omega}} \sigma^{-\omega} & \text{if } \sigma \in [\sigma_{\min}, \sigma_{\max}]\,, \\ 0 & \text{otherwise}\,. \end{cases} \tag{17}$$

From the expression of $G(\sigma)$, we compute $|C_i|$ using a central limit theorem argument [55], which ensures that, for large N,

$$|\mathcal{C}_i| \approx (N-1) \int_{\sigma_i}^{\sigma_{\max}} G(\sigma) d\sigma = (N-1) \frac{\sigma_i^{1-\omega} - \sigma_{\max}^{1-\omega}}{\sigma_{\min}^{1-\omega} - \sigma_{\max}^{1-\omega}}\,. \tag{18}$$

We define the conditional probability density function

$$G_i(\sigma) := G(\sigma \,|\, \sigma \geq \sigma_i) = \frac{G(\sigma)}{P[\sigma > \sigma_i]} = \frac{G(\sigma)}{\int_{\sigma_i}^{\sigma_{\max}} G(\sigma) d\sigma} = \frac{\omega - 1}{\sigma_i^{1-\omega} - \sigma_{\max}^{1-\omega}} \sigma^{-\omega} \,, \quad (19)$$

where the first equality holds due to scale invariance of the power law distribution, and then explicit computation is performed using the expression of $G(\sigma)$. Using again the central limit theorem [55] and Eq. (19), we compute $\langle \sigma^2 \rangle_i$ as

$$\langle \sigma^2 \rangle_i \approx \int_{\sigma_i}^{\sigma_{\max}} \sigma^2 G_i(\sigma) d\sigma = \frac{(\omega - 1)(\sigma_{\max}^{3-\omega} - \sigma_i^{3-\omega})}{(3 - \omega)(\sigma_i^{1-\omega} - \sigma_{\max}^{1-\omega})} \,. \quad (20)$$

Next we calculate the probability for a node to be in its base location when $\alpha = 0$, which corresponds to the case in which agents move deterministically outside of their base location. We consider the generic agent i that belongs to location $\Omega_{\beta(i)}$. Since $A_{\beta(i)} \ll D^2$, we use the approximation $D \to \infty$. We introduce the following partition of the planar space,

$$C_h^{(i)} := \left\{ (x, y) \in [0, D]^2 : (h-1)v < \min_{(\xi,\eta) \in \Omega_{\beta(i)}} \sqrt{(x - \xi)^2 + (y - \eta)^2} \leq hv \right\} , \quad (21)$$

for any $h \in \mathbb{Z}_{\geq 0}$. Note that $C_h^{(i)}$ is the region of the plane from which agent i reaches its base location $\beta(i)$ in exactly h time-steps. Consequently, when $h = 0$ agents are inside their base location, that is, $C_0^{(i)} = \Omega_{\beta(i)}$. Any point (x, y) of the $D \times D$ planar space can be mapped onto this partition through the projection $z^{(i)} : [0, D] \times [0, D] \longrightarrow \mathbb{Z}_{\geq 0}$, defined as

$$z^{(i)}(x, y) = h \iff (x, y) \in C_h^{(i)} \,. \quad (22)$$

Using the mapping $z^{(i)}$, for each agent $i \in \mathcal{V}$, we define the stochastic process $z_i(t) : \mathbb{Z}_{\geq 0} \longrightarrow \mathbb{Z}_{\geq 0}$ as $z_i(t) := z^{(i)}(x_i(t), y_i(t))$. Since $\alpha = 0$, when an agent is outside its base location, then its law of motion is purely deterministic and it moves in the direction of the location. Therefore, if $z_i(t) = h \neq 0$, then, $z_i(t+1) = h - 1$. If $z_i(t) = 0$, the agent is inside its base location, from which it exits only through a jump, which is statistically characterized by Eq. (6). Hence, with probability $1 - p$ the process $z_i(t)$ remains in state 0 at the following time-step. Else, if a jump occurs, the process z_i evolves to state h with probability equal to

$$q_h = \int_{(h-1)v}^{hv} P_{\text{jump}}(x) \, dx = \int_{(h-1)v}^{hv} c e^{-cx} \, dx = e^{-cv(h-1)} - e^{-cvh} \,. \quad (23)$$

The transition probabilities of $z_i(t)$ depend only on the state h in which the process is and on the model parameters. The process $z_i(t)$ is a Markov chain, whose structure is illustrated in Fig. 9 and whose transition matrix is

$$M = \begin{bmatrix} 1-p & pq_1 & pq_2 & pq_3 & \dots \\ 1 & 0 & 0 & 0 & \dots \\ 0 & 1 & 0 & 0 & \dots \\ 0 & 0 & 1 & 0 & \dots \\ \vdots & \vdots & \vdots & \ddots & \ddots \end{bmatrix} . \tag{24}$$

We observe that, if $p > 0$, then the Markov chain is ergodic and it converges to its unique stationary distribution π, which can be computed as the left eigenvector of M associated with the eigenvalue 1 [56]. When the system has reached its steady state, the probability for each agent to be inside its base location, $q_{\text{in}} = \pi_0$, that is derived from the left eigenvalue equation for M in Eq. (24) (with unitary eigenvalue), that is,

$$\begin{cases} \pi_0 = (1-p)\pi_0 + \pi_1, \\ \pi_h = \pi_{h+1} + pq_h\pi_0 \,, \qquad \forall\, h \in \mathbb{Z}_{>0} \,. \end{cases} \tag{25}$$

From Eq. (25), the expression of q_h in Eq. (23), and using that $\sum_{h=0}^{\infty} \pi_h = 1$, we derive

$$q_{\text{in}} = \pi_0 = \frac{e^{cv} - 1}{(1+p)e^{cv} - 1} \,, \tag{26}$$

while the probability that the agent is outside its base location is

$$q_{\text{out}} = 1 - q_{\text{in}} = 1 - \frac{e^{cv} - 1}{(1+p)e^{cv} - 1} = \frac{pe^{cv}}{(1+p)e^{cv} - 1} \,. \tag{27}$$

Appendix C: Analytical study of SIS and SIR in a one-dimensional lattice

Here, we propose a simplified version of the agent-based model which provides some analytical intuitions on the results of the numerical simulations in Figs. 5 and 6. Our simplified model considers that agents move in a discrete, infinitely long, one-dimensional lattice with periodic boundary conditions (that is, a ring). The L locations occupy consecutive positions on the lattice and a fixed number of $n = N/L$ agents belong to each one, as their base location.

To generate a contact, agents should occupy the same position along the lattice. Agents belong to a unique base location in the lattice, which they may leave with probability p. We use a geometric distribution [55] to describe the agents' law of motion, that is, the probability of jumping at a distance d from the base location is equal to

$$P_{\text{jump}}(d) = (1-c)^{d-1}\, c \,, \tag{28}$$

where $c > 0$ is a constant parameter that governs the decay rate, similar to Eq. (6). Once outside their base location, agents move toward their base location by making one step toward it, similar to the two-dimensional model with $\alpha = 0$. A schematic representation of the one-dimensional model is provided in Fig. 10.

We remark that this one-dimensional agent-based model maintains some key features of the model studied in the main text, that is: (i) the presence of closely-spaced base locations, (ii) a stochastic mechanism that governs the probability of jumping outside the base location, and (iii) a gravity law that biases the agents to jump close to the base location according to an exponential distribution.

We start our analysis by computing the probability that agents are in their base location, denoted by ψ_{in}, or in a position at a distance d from it, ψ_d, when the system is at steady state. For $p > 0$, the system is ergodic and we can compute ψ_{in} and ψ_d at steady state [56]. Similar to Appendix B, from the steady-state equation we derive the following recursive system of equations:

$$\begin{cases} \psi_{\text{in}} = (1-p)\psi_{\text{in}} + 2\psi_1 \,, \\ \psi_d = \psi_{d+1} + \dfrac{p}{2} P_{\text{jump}}(d)\psi_{\text{in}} \,, \qquad \forall\, d \in \mathbb{Z}_{>0} \,, \end{cases} \tag{29}$$

where the factor 2 is because there are two positions at a distance d from any location $\ell \in \mathcal{L}$, as in Fig. 10.

From Eq. (29), the expression of $P_{\text{jump}}(d)$ in Eq. (28), and using that $\psi_{\text{in}} + 2\sum_{d=1}^{\infty} \psi_d = 1$, we derive

$$\psi_{\text{in}} = \frac{c}{c+p} \,, \tag{30}$$

and

$$\psi_d = \frac{cp\,(1-c)^{d-1}}{2(c+p)} \,. \tag{31}$$

Given that each agent is randomly assigned to one of the L locations, the probability that a generic agent $i \in \mathcal{V}$ is inside location ℓ is equal to

$$q_\ell = \frac{1}{L}\psi_{\text{in}} + \frac{1}{L}\sum_{x=1}^{L-\ell} \psi_x + \frac{1}{L}\sum_{x=1}^{\ell-1} \psi_x \,, \tag{32}$$

where the first term refers to the probability that the agent is in its base location and its base location is ℓ, while the second and third terms correspond to the probability that the agent belongs to another base location and it occupies location ℓ. Similarly, we compute the probability that agents are in a position not occupied by any location and at a distance d from the closest location as

$$q_{\text{out},d} = \frac{1}{L}\sum_{x=d}^{L+d-1} \psi_x \,, \tag{33}$$

where we assume that the closest location is $\ell = 1$. By a simple change of variables, we can write an equivalent expression when the closest location is $\ell = L$.

In the SIR and SIS processes, the disease propagates from infected agents to susceptible ones occupying the same position of the one-dimensional lattice. We define as $s(t)$, $i(t)$, and (for the SIR model only) $r(t)$ the fractions of susceptible, infected, and recovered agents at time t, respectively. For large-scale systems, we can compute the fraction of susceptible, infected, and recovered agents along the lattice by using the central limit theorem [55]. The fraction of susceptible, infected, and recovered agents inside location ℓ is $s_\ell(t) = q_\ell s(t)$, $i_\ell(t) = q_\ell i(t)$, and $r_\ell(t) = q_\ell r(t)$, respectively. Similarly, the fraction of susceptible, infected, and recovered agents at a distance d from the closest location is $s_{\text{out},d}(t) = q_{\text{out},d} s(t)$, $i_{\text{out},d}(t) = q_{\text{out},d} i(t)$, and $r_{\text{out},d}(t) = q_{\text{out},d} r(t)$, respectively.

We now compute the probability that an agent becomes infected at time t. We first consider the probability of not being infected. In location ℓ, such a probability is equal to $1-\lambda i_\ell(t)$ for each contact. On average, an agent contacts $q_\ell N$ other agents, the probability of not being infected in location ℓ is equal to $\overline{\Lambda}_\ell(t) = (1-\lambda i_\ell(t))^{q_\ell N}$. Similarly, the probability of not being infected at a distance d from the closest location is equal to $\overline{\Lambda}_{\text{out},d} = (1-\lambda i_{\text{out},d}(t))^{q_{\text{out},d}N}$. Thus, the contagion probability of an agent inside its base location ℓ is the complement of $\overline{\Lambda}_\ell(t)$, that is,

$$\Lambda_\ell(t) = 1 - \big(1-\lambda i_\ell(t)\big)^{q_\ell N} = 1 - \big(1-\lambda q_\ell i(t)\big)^{q_\ell N}\,, \tag{34}$$

and the contagion probability when the agent is at a distance d from the base location is the complement of $\overline{\Lambda}_{\text{out},d}(t)$

$$\Lambda_{\text{out},d}(t) = 1 - \big(1-\lambda i_{\text{out},d}(t)\big)^{q_{\text{out},d}N} = 1 - \big(1-\lambda q_{\text{out},d} i(t)\big)^{q_{\text{out},d}N}\,, \tag{35}$$

The evolution of the fraction of infected agents at time $t+1$ is determined by the following equation:

$$\begin{aligned} i(t+1) &= i(t) - \mu i(t) + \sum_{\ell=1}^{L} s_\ell(t)\Lambda_\ell(t) + 2\sum_{d=1}^{\infty} s_{\text{out},d}(t)\Lambda_{\text{out},d}(t) \\ &= i(t) - \mu i(t) + \sum_{\ell=1}^{L} q_\ell s(t)\Lambda_\ell(t) + 2\sum_{d=1}^{\infty} q_{\text{out},d} s(t)\Lambda_{\text{out},d}(t)\,. \end{aligned} \tag{36}$$

The evolution of the fraction of infected agents depends on four terms: (i) the fraction of infected at time t, (ii) the fraction of newly recovered, (iii) the fraction of newly infected in any location, and (iv) the fraction of newly infected outside all the locations.

The evolution of the SIS model is fully determined by Eq. (36), since $s(t) = 1-i(t)$. For the SIR model, instead, Eq. (36) should be coupled with the following equation, which describes the evolution of the fraction of recovered agents,

$$r(t+1) = r(t) + \mu i(t)\,, \tag{37}$$

and with the constraint that $s(t) = 1 - r(t) - i(t)$. The evolution of the fraction of recovered agents only depends on the fraction of recovered at time t and the fraction of newly recovered.

We compute the epidemic threshold of both SIR and SIS disease processes by studying the stability of the disease-free equilibrium in Eq. (36). We linearize

Eq. (36) and expand the expressions for the contagion probabilities in Eqs. (34) and (35) about the disease-free equilibrium $i^* = 0$, obtaining

$$i(t+1) = i(t) - \mu i(t) + \sum_{\ell=1}^{L} \lambda q_\ell^3 N i(t) + 2\sum_{d=1}^{\infty} \lambda q_{\mathrm{out},d}^3 N i(t)\,. \tag{38}$$

The epidemic threshold is computed by imposing $i(t+1) = i(t)$ in Eq. (38), obtaining

$$\frac{\lambda}{\mu} = \frac{1}{N\left(\displaystyle\sum_{\ell=1}^{L} q_\ell^3 + 2\sum_{d=1}^{\infty} q_{\mathrm{out},d}^3\right)}\,. \tag{39}$$

In the case of one location, $L = 1$, the threshold in Eq. (39) reduces to

$$\frac{\lambda}{\mu} = \frac{1}{N\left(q_1^3 + 2\displaystyle\sum_{d=1}^{\infty} q_{\mathrm{out},d}^3\right)} = \frac{4\,(c+p)^3\left(3 - 2c + c^2\right)}{Nc^2\left(2c\,(3 - 2c + c^2) + p^3\right)}\,, \tag{40}$$

where the last equality is obtained by substituting the explicit terms for q_1 and $q_{\mathrm{out},d}$ from Eqs. (32) and (33), respectively, and computing the sum of the obtained series. From inspection of Eq. (40), we observe that increasing the probability of jumping outside the location, p, contributes to increasing the epidemic threshold and thus lowers the endemic prevalence and epidemic size, similar to Fig. 5 (b).

When many locations are present, that is, $L \to \infty$, the contribution of the second term at the denominator yields a marginal contribution to the epidemic threshold in Eq. (40), so that,

$$\frac{\lambda}{\mu} \approx \frac{1}{N\displaystyle\sum_{\ell=1}^{\infty} q_\ell^3}\,. \tag{41}$$

We observe that the epidemic threshold is now independent from any choice of the probability of jumping outside the location, p, as suggested by the numerical studies of Fig. 6 (b).

Abbreviations

SIR: susceptible–infected–removed; SIS: susceptible–infected–susceptible

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Availability of data and materials

A sample of the algorithms generated is available at [57]. The entire set of algorithms is available upon request.

Author's contributions

M.N. and L.Z. designed the research, performed the analytical derivations, and wrote a first draft of the manuscript. M.N. performed the numerical studies. A.R. and M.P. supervised the research, secured the funding, and consolidated the manuscript in its present submission. All the authors reviewed and approved the submission.

Competing interests

The authors declare that they have no competing interests.

Author details

[1]Department of Mechanical and Aerospace Engineering, New York University Tandon School of Engineering, 11201 Brooklyn, USA. [2]Faculty of Science and Engineering, University of Groningen, 9747 AG Groningen, The Netherlands. [3]Dipartimento di Elettronica e Telecomunicazioni, Politecnico di Torino, 10129 Torino, Italy. [4]Office of Innovation, New York University Tandon School of Engineering, 11201 New York, USA. [5]Department of Biomedical Engineering, New York University Tandon School of Engineering, 11201 Brooklyn, USA.

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Figures

Figure 1 Qualitative comparison between real datasets from an experimental study [28], in (a), and the output of our algorithm, in (b). (a) Experimental results about human digital activity density in the cities of Milan and Rome, Italy. The highest density is registered in central areas, while a lower densities are observed in peripheral ones. (b) Using our algorithm, we generate $L = 1,000$ circular locations distributed in rings of decreasing densities. The first few rings contain the denser locations (dark central regions) and may parallel the city center of a urban environment, while the outer rings are less dense and represent peripheral areas (light gray regions). Source of (a): [28]. Parameters used to generate (b): $D = 1,000$, $\Sigma_{\text{min}} = 3$, $\Sigma_{\text{max}} = 30$, and $\gamma = 2.1$. Details of the generative algorithm used are available in Appendix A.

Figure 2 Schematic representation of two snapshots of our model with $N = 4$ agents and $L = 2$ locations. The entire space is delimited with solid black lines. In (a) and (c), we represent only the agents' motion and the border of the locations is represented with dashed black lines. Agents 1 and 2 are assigned to location $A := \beta(1) = \beta(2)$, while agents 3 and 4 are assigned to location $B := \beta(3) = \beta(4)$. Direction and modulus of the agents' velocity is drawn with solid red arrows. The position where agent 1 will jump is indicated with a dotted red arrow. The four arrows around a agent indicate that it will move in a random position inside its own location. In (b) and (d), we show the temporal network formation mechanism. Agents' radii of interaction are represented with solid circles, and undirected links are represented with solid blue lines.

Figure 3 Relationship between the agents' average degree and radius of interaction. (a) Comparison between numerical results and analytical predictions from Eq. (13), for the case without base locations. Simulation results are generated with the following parameter set: $L = 0$, $D = 100,000$, $\sigma_{\mathsf{min}} = 5$, and $\sigma_{\mathsf{max}} = 500$. (b) Comparison between numerical results and analytical predictions from Eq. (16), in the case of multiple base locations, uniformly distributed in the domain. In the simulations, we use the following parameters: $L = 10$, $D = 10^9$, $\Sigma_{\mathsf{min}} = 1,000$, $\Sigma_{\mathsf{max}} = 10,000$, $\sigma_{\mathsf{min}} = 10$, $\sigma_{\mathsf{max}} = 100$, $p = 0.3$, and $\alpha = 0$. Agents are initially inside their base location and interactions are recorded after 100 steps to allow the agents to reach a steady-state configuration. Other parameter values are $N = 10,000$, $v = 500$, $c = 4 \cdot 10^{-4}$, $\omega = 2.4$, and $\gamma = 2.1$, and $T = 5,000$.

Figure 4 Relationship between the agents' average degree and radius of interaction in an urban-like environment. Darker circles represent agents assigned to more peripheral locations, while brighter ones indicates agents belonging to more central locations. We set: (a) $p = 0.1$ and $\alpha = 0$, (b) $p = 0.4$ and $\alpha = 0.2$, and (c) $p = 0.8$ and $\alpha = 0.4$. Agents are initially inside their base location and contacts are recorded after 100 steps to allow the agents to reach a steady-state configuration. Other parameter values are $L = 100$, $N = 10,000$, $D = 10^9$, $\Sigma_{\mathsf{min}} = 100$, $\Sigma_{\mathsf{max}} = 10,000$, $\sigma_{\mathsf{min}} = 1$, $\sigma_{\mathsf{max}} = 1,000$, $v = 500$, $c = 4 \cdot 10^{-4}$, $\omega = 2.4$, $\gamma = 2.1$, and $T = 5,000$.

Figure 5 Influence of the agents' randomness, α, and the probability of jumping outside the location, p, on the endemic prevalence of the SIS model, (a)–(b), and the epidemic size of the SIR model, (c)–(d). Curves represent the median of 100 independent simulations; 95% confidence bands are displayed in gray. Agents are initially inside their base location and the infection starts after 100 steps to allow the agents to reach a steady-state configuration. The fraction of randomly chosen initial infected is 0.01. Other parameter values are $L = 1$, $N = 10,000$, $D = 10^9$, $\Sigma_{\mathsf{min}} = 100$, $\Sigma_{\mathsf{max}} = 10,000$, $\sigma_{\mathsf{min}} = 1$, $\sigma_{\mathsf{max}} = 1,000$, $v = 500$, $c = 4 \cdot 10^{-4}$, $\omega = 2.4$, $\gamma = 2.1$, $\lambda = 0.15$, and $\mu = 0.1$.

Figure 6 Influence of the agents' randomness, α, and the probability of jumping outside the location, p, on the endemic prevalence (SIS model), (a)–(b), and the epidemic size (SIR model), (c)–(d). Curves represent the median of 100 independent simulations; 95% confidence bands are displayed in gray. Agents are initially inside their base location and the infection starts after 100 steps to allow the agents to reach a steady-state configuration. The fraction of randomly chosen initial infected is 0.01. Other parameter values are $L = 100$, $N = 10,000$, $D = 10^9$, $\Sigma_{\mathsf{min}} = 100$, $\Sigma_{\mathsf{max}} = 10,000$, $\sigma_{\mathsf{min}} = 1$, $\sigma_{\mathsf{max}} = 1,000$, $v = 500$, $c = 4 \cdot 10^{-4}$, $\omega = 2.4$, $\gamma = 2.1$, $\lambda = 0.15$, and $\mu = 0.1$.

Figure 7 Impact of different ways of assigning agents to their locations on the endemic prevalence (SIS model), (a), and the epidemic size (SIR model), (b). The "Uncorrelated" case represents a random assignment. In the "Pos. Correlated" case, agents with larger radii are assigned to the denser (central) locations, while, in the "Neg. Correlated" case, agents with larger radii belong to the less dense (peripheral) locations. Curves represent the median of 100 independent simulations; 95% confidence bands are displayed in gray. Agents are initially inside their base location and the the infection starts after 100 steps to allow the agents to reach a steady-state configuration. The fraction of randomly chosen initial infected is 0.01. Other parameter values are $L = 100$, $N = 10,000$, $D = 10^9$, $\Sigma_{\mathsf{min}} = 100$, $\Sigma_{\mathsf{max}} = 10,000$, $\sigma_{\mathsf{min}} = 1$, $\sigma_{\mathsf{max}} = 1,000$, $v = 500$, $c = 4 \cdot 10^{-4}$, $\omega = 2.4$, $\gamma = 2.1$, $\lambda = 0.15$, and $\mu = 0.1$.

Figure 8 Effect of different vaccination strategies on the endemic prevalence (SIS model), (a)-(b)-(c), and epidemic size (SIR model), (d)-(e)-(f). The vaccination coverage represents the fraction of immune agents prior to the disease onset. In "Random", we select the fraction of agents to vaccinate at random; in "Center", we vaccinate first the agents that are assigned to central base locations, while in "Peripheral", we prioritize vaccination for agents that belongs to the peripheral agents. We set: (a)-(d) $p = 0.1$ and $\alpha = 0.0$, (b)-(e) $p = 0.4$ and $\alpha = 0.2$, and (c)-(f) $p = 0.8$ and $\alpha = 0.4$. Curves represent the median of 100 independent simulations; 95% confidence bands are displayed in gray. Agents are initially inside their base location and the infection starts after 100 steps to allow the agents to reach a steady-state configuration. The fraction of randomly chosen initial infected is 0.01. Other parameter values are $L = 100$, $N = 10,000$, $D = 10^9$, $\Sigma_{\mathsf{min}} = 100$, $\Sigma_{\mathsf{max}} = 10,000$, $\sigma_{\mathsf{min}} = 1$, $\sigma_{\mathsf{max}} = 1,000$, $v = 500$, $c = 4 \cdot 10^{-4}$, $\omega = 2.4$, $\gamma = 2.1$, $\lambda = 0.15$, and $\mu = 0.1$.

Figure 9 Transition graph of the Markov chain $z_i(t)$.

Figure 10 Schematic of the one-dimensional simplified version of the agent-based model. (a) Scenario where only one base location is present (in black). (b) Scenario where multiple base locations are present.

Tables

Symbol	Description
$\mathcal{V}$	Set of all agents
$\mathcal{L}$	Set of all locations
N	Number of agents in the system
L	Number of locations in the system
n	Number of agents in each location
D	Side of the square planar space
T	Total number of discrete time-steps in the observation window
t	Index for discrete time instants
ℓ	Index for the locations in the system
i, j	Indices for the agents in the system
ρ_ℓ	Density of location ℓ in the system
$\beta(i)$	Function that maps agent i to its base location
Ω_ℓ	Region of space occupied by location ℓ
$x_i(t), y_i(t)$	Position occupied by agent i in the planar space at time t
$\theta_i(t)$	Direction of the motion of agent i at time t
$\Phi_i(t)$	Angle of the direction of the shortest path from $x_i(t), y_i(t)$ to $\Omega_{\beta(i)}$
$\Delta\theta_{it}$	Angle drawn uniformly at random in $[0, 2\pi)$ for agent i at time t
v	Velocity of agents outside their base location
α	Randomness in the agents' motion toward their base location
p	Probability of jumping outside the base location
$P_{\mathsf{jump}}(d)$	Probability of jumping at a distance d from the base location
q_{in}	Probability of being inside the base location
q_{out}	Probability of being outside the base location
q_ℓ	Probability of being inside Ω_ℓ
$q_{\mathsf{out},d}$	Probability of being at a distance d from the closest location
$\Psi_{\mathsf{in}}, \Psi_{\mathsf{out}}, \Psi_d$	Probability that agents are inside, outside, and at a distance d from their base location
$P(\Sigma)$	Probability density distribution of locations' radii
γ	Exponents of the power law distribution of locations' radii
$\Sigma_{\mathsf{min}}, \Sigma_{\mathsf{max}}$	Lower and higher cut-off of the distribution of locations' radii
$G(\sigma)$	Probability density distribution of agents' radii of interaction
ω	Exponents of the power law distribution of radii of interaction
$\sigma_{\mathsf{min}}, \sigma_{\mathsf{max}}$	Lower and higher cut-off of the distribution of radii of interaction
k_i	Degree of agent i
λ	Infection probability per contact
μ	Recovery probability per unit time
$s(t), i(t), r(t)$	Fraction of susceptible, infected, and recovered agents in the system
$s_\ell(t), i_\ell(t), r_\ell(t)$	Fraction of susceptible, infected, and recovered agents in Ω_ℓ
$s_{\mathsf{out},d}(t), i_{\mathsf{out},d}(t), r_{\mathsf{out},d}(t)$	Fraction of susceptible, infected, and recovered agents at distance d from the closest location
$\Lambda_\ell(t)$	Contagion probability in Ω_ℓ
$\Lambda_{\mathsf{out},d}(t)$	Contagion probability at a distance d from the closest location
$\langle\cdot\rangle$	Statistical average of the quantity "$\cdot$"
$\mathsf{E}[\cdot]$	Expected value of the quantity "$\cdot$"
$P[\cdot]$	Probability of an event "$\cdot$"

Table 1 Nomenclature of the variables and notation used in the paper.