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# Epidemic spreading on hierarchical geographical networks with mobile agents



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#### **ABSTRACT**

Hierarchical geographical traffic networks are critical for our understanding of scaling laws in human trajectories. Here, we investigate the susceptible-infected epidemic process evolving on hierarchical networks in which agents randomly walk along the edges and establish contacts in network nodes. We employ a metapopulation modeling framework that allows us to explore the contagion spread patterns in relation to multi-scale mobility behaviors. A series of computer simulations revealed that a shifted power-law-like negative relationship between the peak timing of epidemics  $\tau_0$  and population density, and a logarithmic positive relationship between  $\tau_0$  and the network size, can both be explained by the gradual enlargement of fluctuations in the spreading process. We employ a semianalytical method to better understand the nature of these relationships and the role of pertinent demographic factors. Additionally, we provide a quantitative discussion of the efficiency of a border screening procedure in delaying epidemic outbreaks on hierarchical networks, yielding a rather limited feasibility of this mitigation strategy but also its nontrivial dependence on population density, infector detectability, and the diversity of the susceptible region. Our results suggest that the interplay between the human spatial dynamics, network topology, and demographic factors can have important consequences for the global spreading and control of infectious diseases. These findings provide novel insights into the combined effects of human mobility and the organization of geographical networks on spreading processes, with important implications for both epidemiological research and health policy.

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# 1. Introduction

The complexity of human mobility and interaction patterns has attracted much recent attention in a variety of disciplines, including statistical physics and complex systems science  $[1-14]$ . Ever since the influential work of Brockmann et al. [\[4\]](#page--1-0) on the bank note dispersal, researchers have been intrigued by the revealed power-law distributions of human travel displacements [\[3,5\].](#page--1-0) Meanwhile, such empirical studies have increasingly benefited from a variety of advanced data collection technologies (e.g. mobile phone, GPS, social-network sites etc. [\[15\]](#page--1-0)), reporting the presence of many abnormal properties of

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human motion, including ultraslow diffusion [\[3,4\]](#page--1-0) and strikingly high predictability [\[6\].](#page--1-0) It is these abnormal properties that actually shape the difference between the natural human movements and the so-called Lévy flights [\[7,16\]](#page--1-0).

Several studies have been conducted so far to address the underlying mechanisms of the discovered abnormal properties of human spatial behavior. For the urban and intercity travels, Song et al. investigated the impact of exploration and preferential return on mobility patterns [\[8\]](#page--1-0). For the long-range travels, Han et al. [\[9\]](#page--1-0) demonstrated that hierarchical geographical organization of traffic systems plays a crucial role in the emergence of scaling in human trajectories.

An important, but less studied issue in this context, is the impact of the abnormal features of human motion on the dynamical evolution of spreading processes. For example, based on the mobile phone user data published in Ref. [\[3\],](#page--1-0) Wang et al. analyzed the spreading of a mobile-phone-virus outbreak [\[10\]](#page--1-0). Ni and Weng [\[11\]](#page--1-0) investigated the effects of heterogeneous spatial properties in metapopulation networks, and more recently, Belik et al. [\[12,13\]](#page--1-0) reported on the dynamics of a spreading process under bidirectional mobility, that was further investigated by Balcan and Vespignani  $[14]$ . All of these studies converge on the finding that spatially constrained mobility networks play a crucial role in shaping the key features of spreading processes.

While the focus of most previous studies has largely been on the local temporal development of diseases and epidemics  $[17,18]$ , their geographical, multi-scale circulation is less well understood  $[19,20]$ . In the present paper, we propose a metapopulation model [\[21\]](#page--1-0) that allows us to explore the contagion spread patterns in relation to multi-scale mobility behaviors and the organization of traffic systems embedded in realistic geography. Thus, we assume here that besides local, close-contact-driven and smaller outbreaks, it is the movement of individuals among the many different scales of a large geographical network that is actually essential to the spread of a global epidemic.

A previously reported model  $[9]$  demonstrated that a number of realistic mobility patterns can naturally be generated by random walks on a hierarchical geographical network, mimicking the properties of real-world traffic systems. Here, we directly implement a hierarchical geographical network with randomly-walking agents into the susceptible-infected (SI) epidemic model, combining thereby the study of epidemic dynamics with human-like mobility behaviors and the realworld-like organization of traffic systems (Section 2).

We first investigate the elementary properties of the generalized SI spreading process on hierarchical geographical networks and then compare the obtained epidemic patterns against the scenario generated with the Lévy flight model (Section [3](#page--1-0); details of the employed Lévy flight model are further presented in Appendix A). We then systematically investigate how population density and city size affect the epidemic curves in different layers of a hierarchically organized geographical network (Section [4\)](#page--1-0). A semi-analytical method is further introduced to better understand the importance of pertinent demographic factors (Appendix B). Finally, we study the effects of a border control measure on the timing and height of epidemic peak which is one of the main aspects of most mitigation strategies (Section [5](#page--1-0)).

### 2. The model

Models that explicitly address the hierarchical organization of geographical networks are capable of mimicking realworld traffic systems in which agents can randomly percolate along the network edges and thereby take part in long-range, inter-layer travels. In one such model [\[9\]](#page--1-0), agents can generate not only power-law-like travel displacement distributions, but are also able to display a scaling behavior in the probability density of having traveled a certain distance at a certain time, which is in agreement with recent empirical observations  $[4]$ . In addition, it has been shown that inter-event or waiting time distributions also display non-Poisson statistics. We therefore argue here that an implementation of a hierarchical network in our present study can help us understand highly relevant aspects of real epidemics that are otherwise difficult to capture with models that enable only local behavior, i.e. mixing at only one or two scales [\[22\].](#page--1-0)

With respect to the infectious disease dynamics, our model is based on the SI spreading process [\[23,24\],](#page--1-0) in which individuals can occupy one of the two possible states: 'susceptible', meaning they are not infected yet, and 'infected', meaning they already have the disease and can spread it to the susceptibles. In the present study, we examine the case of  $d = 2$ dimensional geographical hierarchical network with the following properties:

(i) Structure of the metapopulation network. We use the regular form of the hierarchical network reported in Ref. [\[9\]](#page--1-0). In our model, all units, or say subpopulations, are called cities, which can correspond to any kind of human settlements and not only to the real-world cities. These cities are organized in L layers of the network, where the first layer corresponds to the top network layer. A uniform 3-layer structure of the hierarchical geographical network is depicted in [Fig. 1](#page--1-0).

We first set the city located in the center of a square field as the top layer (i.e. the first layer) of the network, and we evenly divide this field into K regular sub-regions (with a 3  $\times$  3 arrangement for K  $=$  9). We then set all the centrally located cities of the respective sub-regions to be the 2nd-layer cities. We note here that the 2nd-layer city in the central sub-region is also regarded as the same node of the 1st-layer city due to the overlap of the positions. Here, the sub-regions are called ''2ndlayer sub-regions''. Similarly, each of the 2nd-layer sub-regions are further evenly divided into K 3rd-layer sub-regions with 3rd-layer central cities. This process is iterated until Lth-layer cities are generated. For  $2\leqslant n\leqslant L$ , there are  $K^{n-2}(K-1)$  nthlayer cities. The total number of cities in the network (the size of the network) is then defined as  $S = K^{L-1}$ .

In the  $(L - 1)$ th-layer sub-region, each Lth-layer city is connected to the central city of the sub-region. These Lth-layer cities belonging to the same  $(L-1)$ th-layer sub-region are also fully connected. Similarly, for  $1 < n < L$ , the *n*th-layer cities

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