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ANOMALOUS TRANSPORT OF INFECTIOUS DISEASES IN STRUCTURED POPULATIONS

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ABSTRACT

Structural properties of populations strongly influence dynamic transport and interaction such as the transmission of infectious diseases. In this paper a hierarchical block model is investigated, that schematically describes the configuration of social communities. It is shown that these models can produce super-diffusive spread and that the balance between intra- and inter-community contacts is a suitable factor for controlling the expression of this feature. The qualitative characteristics of epidemics simulated with this model are reproduced in numerical simulations of a basic fractional-in-space differential equation SIR model. Based on a stochastic formulation of the block structure, a preliminary connection between both models can be derived. The results of this paper confirm that certain structural characteristics of interacting populations can be simulated in aggregated models by employing fractional derivatives.

1 INTRODUCTION

Investigating the structural features of dynamical systems is important for understanding emerging patterns and effects. It is often the *topological* structure of a natural system that in turn leads to a particular choice of a mathematical formalism for modeling.

In the simulation of epidemic spread, a broad range of modeling approaches has been employed. Most commonly a population is segregated into compartments that represent different disease states (Kermack and McKendrick 1927) and the transitions between healthy and infected is formalized in terms of differential equations (classical SIR model). More elaborate approaches include the decomposition of a population according to social attributes like age and gender (Keyfitz and Keyfitz 1997), which can involve distinct disease parameters (e.g. infection likelihood). The ultimate refinement of this approach results in individual-based models, where each member of a population is simulated according to statistical and demographic data (Bicher et al. 2015). In a different or complementary approach, focus is put on the reconstruction of contact patterns that are responsible for disease transmission (Danon et al. 2012; Danon et al. 2011). Elaborate mixing terms, the formulation of pairwise contacts and data-driven network models are required in this context. As a third pillar, the mapping of social communities can increase the quality or insight into the dynamics of epidemic progression. This approach is often implemented in so-called meta-population models (Watts et al. 2005; Vazquez 2007; Logak and Passat 2017; Li and Zou 2009) where sub-populations are not aggregated according to individual attributes (compare basic compartment models) but in a way that mimics the social structure of populations.

Information on the demographic composition of a population, on the social structure, on the contact behavior of individuals and on the (virological) properties of infectious diseases are used to increase the quality and scope of such models. Elaborate data-driven models are for instance used to investigate herd immunity thresholds and occurrence of serotypic shift, which are important aspects in the development of vaccination strategies (Fraser et al. 2009). Occasionally, the required data is not available and many modeling decisions are hypotheses or assumptions. Hence, the results and insights of such models can only be of qualitative and technical nature. In this paper an abstracted structural model of interacting populations

is investigated. The models in this paper are not suitable for predicting the course of epidemic outbreaks or to simulate specific scenarios. Rather, the purpose of this investigation is to characterize certain phenomena (anomalous transport and diffusion) that result from particular hypothetical and simplified structural assumptions. In general, anomalous (and fractional) patterns are known to emerge in the course of epidemic outbreaks. For instance, in Colizza et al. (2006) and Hufnagel et al. (2004), long-range effects in the spread of infectious diseases were investigated in connection with global flight-data. In Lengyel et al. (2015), Watts et al. (2005), and Siudem and Hołyst (2019) hierarchically structured networks and geographic embedding are discussed. Further structural features of populations in connection with epidemic spread were investigated in Vazquez (2007) and Li and Zou (2009).

The aforementioned hypotheses correspond to the following conceptional model of an interacting population (Schneckenreither and Popper 2017): The social structure of populations is composed of latent and organizational *communities*. The former includes social relations that evolve as a network such as friendships, the latter represent distinct quantifiable units such as households and workplaces (Kobayashi et al. 2019). Membership in these communities or blocks is not a one-to-one relation, but individuals can be a member of different blocks (*overlapping blocks*). To further reproduce the structural (e.g. administrative and geographic) configuration of a population, communities can be combined into larger communities such as municipalities, countries, etc., which results in a *hierarchical* layout (Lengyel et al. 2015; Watts et al. 2005; Ravasz and Barabási 2003). A feature often associated with communities that are relevant in the transmission of infectious diseases is *locality*. Hence, the spatial embedding of hierarchical community structures is often encountered in models for the simulation of epidemic spread (Lengyel et al. 2015; Watts et al. 2005; Siudem and Hołyst 2019). We assume furthermore that contacts that happen within communities share distinct qualities (e.g. duration, periodicity, infection likelihood), which can also be recognized in statistical surveys (Mossong et al. 2008; Mastrandrea et al. 2015). In general, contacts among individuals that are not members of a common block, occur less frequently and are not periodic. This includes for instance contacts that occur during transportation. It turns out that these *random or weak contacts* can play a significant role in the spread of diseases (Colizza et al. 2006; Hufnagel et al. 2004; Gustafson et al. 2017; Schneckenreither and Popper 2017).

The described hierarchical configuration results from a variety of social, demographic and geographic features. In a simplified and abstracted version of this complex structural system we can understand communities as local aggregations of the population that are characterized by elevated internal interaction. The same observations are valid in all layers of the hierarchy, that is, also higher level communities (e.g. schools as compared to school-classes) are characterized by locality and elevated internal interaction. However, we assume that with increasing level in the hierarchy, communities become 'less local' and 'less interacting'. In the following, the abstracted hierarchical block model is formalized in mathematical and stochastic terms (Section 2) and derived interaction networks are investigated with respect to non-locality of the (spatial) propagation of diseases (Section 3). Non-local effects can often be observed in anomalous (super-diffusive) processes, which are usually characterized by a nonlinear evolution of the mean squared deviation. Figure 1 visualizes the differentiation of diffusion processes according to the temporal evolution of the mean squared deviation. The microscopic behavior that is responsible for super-diffusive patterns, is the occurrence of spatial increments in all orders of magnitude (compare Lévy flights). A macroscopic model that can map anomalous diffusion is the fractional diffusion equation. In Section 4 the outline of a fractional-in-space reaction-diffusion equation is derived from a stochastic formulation of the dynamics of the block model. Simulations show that both models can produce the same qualitative and quantitative behavior. However, the exact parameterization of the fractional differential equation model is out of the scope of this paper.

Fractional differential equations are increasingly used for simulating the spread of infectious diseases (Gustafson et al. 2017; Hanert et al. 2011; Owolabi 2016; Stollenwerk et al. 2009). In space-fractional settings, often the (potential) underlying structural and topological features are not investigated. In this context, an additional structural explanation of anomalous effects and fractional dynamics can be useful.

However, also the super-diffusive and non-local characteristics of such models have proven to adequately reconstruct important phenomena observed in real epidemic outbreaks.

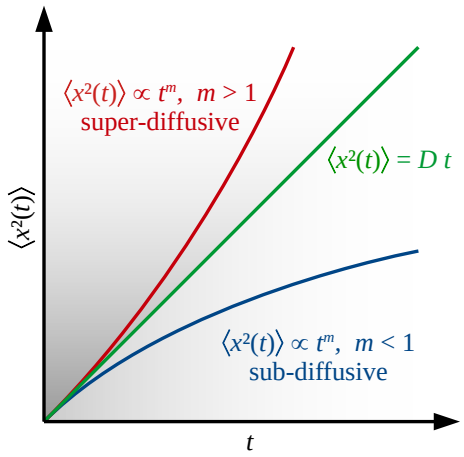

Figure 1: Different types of anomalous diffusion are distinguished by the behavior of the mean squared deviation.

2 HIERARCHICAL BLOCK MODEL AND TRANSPORT

To reproduce the spatial confinedness (locality) of communities in a mathematical model, blocks can be represented as probability densities with finite moments on the two-dimensional Euclidean space (spatial embedding). The prototypical and limiting case for probability distributions with finite moments is the normal distribution and the associated Gaussian densities. This is particularly useful to randomly generate block members in the vicinity of the block location or to retrospectively and stochastically assign locations to blocks. To display 'increase of locality' with lower levels, sub-blocks are assumed to have a certain fraction of the scale of their parent blocks. To obtain hierarchical encapsulation, the locations of sub-blocks are distributed according to the density function of their respective parent. We denote blocks in the same level of the hierarchy with a common *level parameter* l. Hence, if σ_l is the scale of a block in level l, then $\sigma_{l+1} = \sigma_l\, c$ is the scale of the parent block, where c is the *block scaling parameter*. Additionally, in order to define a hierarchical block structure, a maximum level L, the number of sub-blocks per block K and the number N of members (nodes or individuals) in each base level block is required. Starting from an arbitrary location of the single highest level block (level $L-1$), sub-blocks and their sub-blocks can be sampled in an iterative fashion. Finally for each base level block (level 0) a number of individuals can be generated. This approach for generating structured and heterogeneous populations reflects the spatial embedding of a *persistent* hierarchical block layout. In Figure 2 hierarchical block configurations are visualized for different parameterizations. The default measure for proximity or distance between two entities or individuals is the level number of the lowest level common parent block.

The stochastic formulation of blocks allows to describe the location of the sub-block $\mathbf{x}_l$ as a stochastic variation of the location of the parent $\mathbf{x}_{l+1}$ by

$$\mathbf{x}_l \overset{d}{=} \mathbf{x}_{l+1} + \mathbf{X}_{l+1}, \qquad \mathbf{X}_{l+1} \sim \mathrm{Norm}(\sigma_{l+1})$$

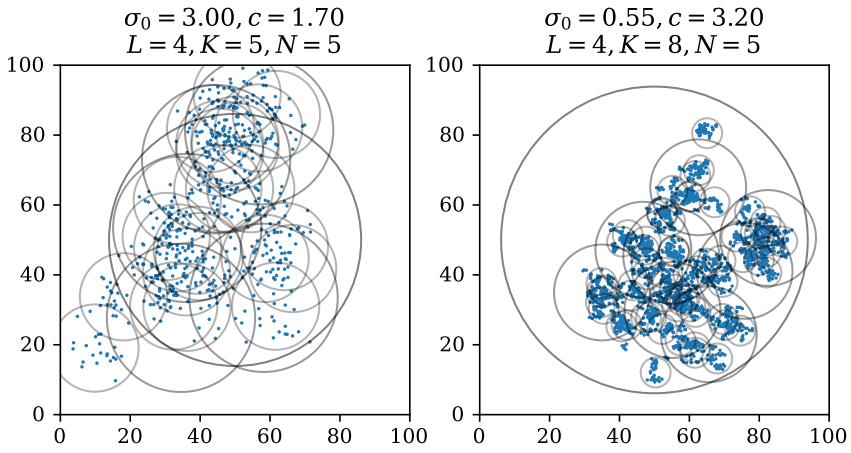

Figure 2: Different hierarchical block configurations, for given parameters σ_0, c, K, N and a maximum level L. Blocks are represented as the 95% confidence ellipse of the corresponding density functions. The resulting nodes (blue) represent a population that is hierarchically structured.

or vice versa. Let σ_0 be the scale of 0-level blocks (*base block scale*), then a block in level l has the scale $\sigma_l = \sigma_0 c^l$. From the additivity of normal random variables it follows that

$$\mathbf{x}_0 \overset{d}{=} \mathbf{x}_l + \sum_{\lambda=1}^{l} \mathbf{X}_\lambda, \qquad \sum_{\lambda=1}^{l} \mathbf{X}_\lambda \sim \text{Norm}\left(\sqrt{\sum_{\lambda=1}^{l} \sigma_\lambda^2}\right)$$

if $\mathbf{x}_l$ is the location of the l-level parent block. And if $\mathbf{x}_{0,1}$ and $\mathbf{x}_{0,2}$ are the locations of two 0-level blocks with their common lowest level parent in level l, then

$$\mathbf{x}_{0,1} - \mathbf{x}_{0,2} \sim \text{Norm}\left(\sqrt{2\sum_{\lambda=1}^{l} \sigma_\lambda^2}\right). \tag{1}$$

The scale parameters in above formulations are square roots of truncated geometric series such that a closed-form expression for the scale of the *stochastic distance* between two entities (with lowest common level block in level l) can be found according to (1)

$$\sigma(l) := \sqrt{2\sum_{\lambda=0}^{l} \sigma_\lambda^2} = \sqrt{2\sum_{\lambda=0}^{l} \sigma_0^2 c^{2\lambda}} = \sqrt{2\sigma_0^2 \frac{c^{2(l+1)}}{c^2 - 1}}.$$

Hence, for any two individuals with their lowest common parent level l, the stochastic distance is given by $\mathbf{X}\sigma(l)$ where $\mathbf{X}$ is a bivariate standard normal random variable.

The topological and stochastic proximity measures introduced above can be used to simulate spatial transport or interaction. However, to this end a (stochastic) model for the selection of an interaction level is required. This model should reflect the conceptual observation that intra-community interaction is significantly different from inter-community interaction and that blocks in higher levels of the hierarchy are 'less interacting' than blocks in lower levels. The most parsimonious stochastic model to differentiate between intra- and inter-community interaction is by a *intra-block interaction likelihood* p such that the probability for intra-block interaction as compared to extra-block interaction is Bernoulli distributed. As a consequence, the random selection of an interaction level l can be formulated as a geometrically distributed

random variable $\Lambda \sim \text{Geom}(p)$. If the sampled value of Λ is 0, then interaction shall happen within a base block, for a value $l > 0$ interaction happens with an individual that is a member of the same l-level parent block. The geometric distribution, as the discrete analogous to the exponential distribution (finite mean), can be regarded as a prototypical model.

Hence, in the stochastic model for any two individuals the distance is given by $\mathbf{X}\,\sigma(\Lambda)$. In (Schneckenreither 2020) it was shown how this stochastic formulation can be used as spatial increments in random walks (Lévy-flights), which in turn reproduce to the diffusive behavior described by the fractional diffusion equation in the Riesz-Feller sense. Because the density of the product distribution $\mathbf{X}\,\sigma(\Lambda)$ has power-law tails

$$\sim |\mathbf{x}|^{\frac{\ln(1-p)}{\ln(c)}-2}, \tag{2}$$

in the limit (generalized central limit theorem (Uchaikin and Zolotarev 1999; Nolan 2018)) α- or Lévy-stable increments are obtained. As a consequence, the diffusion behavior of the fractional diffusion equation in the Riesz-Feller sense is approximated (Gorenflo and Mainardi 2003), (Metzler and Klafter 2000). The order of the corresponding spatial fractional derivative α is linked to the parameterization of the hierarchical block model by $\alpha = -\ln(1-p)/\ln(c)$. The detailed connection is out of the scope of this paper, it is only necessary to recognize – what is also intuitive – that primarily the relation between the scaling parameter c and the parameter of the geometric distribution p determines the amount of non-locality and *anomaly* that is produced by the topological and stochastic block model.

We use in the following the expressions Υ_b, Υ_s and $\Upsilon_{\mathbf{x}}$ to denote the diffusive transport or interaction behavior of the hierarchical block model, the stochastic block model and the fractional diffusion equation.

3 EPIDEMIC SPREAD IN HIERARCHICAL BLOCK MODELS

In the persistent hierarchical block model, a basic SIR-type disease can be simulated by sampling a certain number of temporary links (let E be the number of outgoing links per node) among the individuals of the population and allowing the transmission of the infectious state with a certain probability. Hence, the number of new infections introduced by a single infected individual in a fully susceptible population (during one unit of time) can be approximated with a binomial distribution. Given a large enough number of nodes, with the parameterization $\text{Binom}(p_I/E, E)$ the expected number of new infections is p_I. In each simulation step, a recovery likelihood p_R is used to remove the infectiousness of individual nodes. In Figure 3 two scenarios with identical parameterization but different intra-block interaction likelihoods are compared. It is shown that by a small change of parameter, the super-diffusive behavior can be obtained. In this case the speed of spread is increased by orders of magnitude, since non-local interaction allows the disease to quickly spread to distant fully susceptible regions.

If $\Upsilon_b(t,b)$ describes the interaction of (remote and local) infected individuals with the individuals in base block b at time t, then $p_I\Upsilon_b(t,b)$ is the local force of infection and $\Upsilon_b(t,b)S(t,b)$ is called the mixing term. The following iterative scheme is a mathematical (meta-population) description of the dynamics of the hierarchical block model,

$$\begin{aligned}
S(t+1,b) &= S(t,b) - p_I\Upsilon_b(t,b)\,S(t,b)\\
I(t+1,b) &= I(t,b) + p_I\Upsilon_b(t,b)\,S(t,b) - p_R I(t,b)\\
R(t+1,b) &= R(t,b) + p_R I(t,b),
\end{aligned}$$

where $b \in B$. According to the parameterization of the model, the interaction term $\Upsilon_b(t,b)$ is the relative number of infectious contacts with a node in block b. The interaction term can be approximated by

$$\Upsilon_b(t,b) \approx \sum_{b' \in B} I(t,b')\,\kappa(b,b'),$$

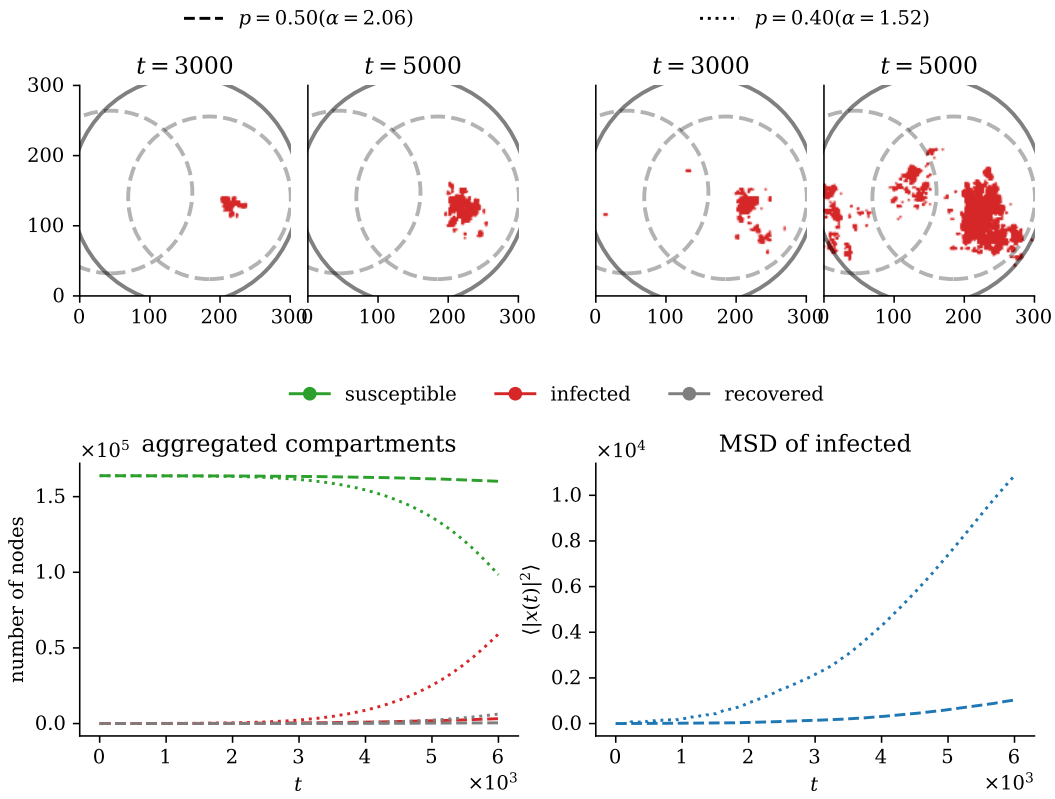

Figure 3: Effect of different intra-block interaction likelihoods on the dynamics of epidemic spread. The parameters in both scenarios are as follows: $L = 16, K = 2, N = 5, E = 2, \sigma_0 = 0.42, c = 1.4$. In the top the two highest level blocks are indicated by gray circles; infected nodes are shown in red. In the bottom left, the drastic difference in the number of infected is visible. In the bottom right, expressed super-linear mean squared deviation (MSD) for the anomalous case ($\alpha = 1.52$) and linear mean squared deviation for the normal case ($\alpha \approx 2$) can be distinguished.

where $\kappa(b, b')$ aggregates the structural features of the block model. Hence, $\kappa(b, b')$ depends on the parameterization of the block model and is a function of the level of the lowest common parent block of b and b' (i.e. the lowest level block that contains both blocks).

Using the alternative stochastic concept of proximity, a spatial version of the interaction term can be formulated

$$\Upsilon_s I(t, \mathbf{x}) = \int_{\mathbb{R}^2} \iota(\mathbf{y}) I(t, \mathbf{x} + \mathbf{y}) \, \mathrm{d}\mathbf{y}, \tag{3}$$

where $\iota(\mathbf{x})$ is the density of the random variable $\mathbf{X}\sigma(\Lambda)$ with a certain algebraic decay (2). This spatial formulation corresponds to a distributed non-discrete population and can be regarded as a continuous approximation of the persistent block structure model (Schneckenreither 2020). Accordingly several indirections between both models that can influence the quantitative behavior of transport have to be taken into account:

(OB) In the stochastic model, the block structure remains to exist only in a latent or volatile sense. Hence, the stochastic model implies retrospective stochastic assignment of nodes or locations to blocks, which corresponds to the concept of overlapping blocks in the persistent block model.

(UD) Either the continuous spatial distribution (density) of the population in the stochastic model must reproduce the spatial distribution of individuals in the persistent block model, or the block model must be configured to have a uniform distribution of individuals on a certain domain.

(BC) The hierarchical block model is not subject to boundary conditions because interaction does only depend on topological alignment but not spatial embedding.

In Schneckenreither et al. (2008), $\iota(\mathbf{x})$ with finite variance were investigated and linked to spatial reaction-diffusion equations. In essence, a Taylor series expansion of the function $I(t,\mathbf{y})$ in the integral (3) leads to an expression containing the Laplace operator. Here, our aim is to link the topological mixing behavior Υ_b and the corresponding non-local interaction term $\Upsilon_s(t,\mathbf{x})$ with the fractional Laplacian $-(-\Delta_\mathbf{x})^{\alpha/2}I(t,\mathbf{x})$. However, instead of applying a fractional Taylor expansion, a more informal approach is presented in the following section.

4 FRACTIONAL DIFFERENTIAL EQUATION MODEL

We note that the fractional Laplacian corresponds to the Riesz-Feller fractional derivative and permits the integral representation (Dyda et al. 2015; Samko 1998)

$$(-\Delta_\mathbf{x})^{\alpha/2} f(\mathbf{x}) = \frac{1}{|c(\alpha)|} \lim_{\varepsilon\to 0} \int_{\mathbb{R}^2\setminus B_\varepsilon(0)} \frac{f(\mathbf{x}) - f(\mathbf{x}+\mathbf{y})}{|\mathbf{y}|^{2+\alpha}}\,\mathrm{d}\mathbf{y}, \tag{4}$$

where $c(\alpha)$ is a certain normalizing constant. Despite this expression is very similar to (3), the mathematical connection to the spatial increments of Lévy-flights is usually derived in the Fourier picture.

By separating the shape of the density $\iota(\mathbf{y})$ around the origin from the asymptotic behavior $\frac{D^{\alpha/2}}{|c(\alpha)|}|\mathbf{y}|^{-(2+\alpha)}$ for large arguments (compare (2)), we can write

$$\Upsilon_s(t,\mathbf{x}) = \int_{\mathbb{R}^2} \iota(\mathbf{y}) I(t,\mathbf{x}+\mathbf{y})\,\mathrm{d}\mathbf{y} \approx \int_{B_\varepsilon(0)} \iota(\mathbf{y}) I(t,\mathbf{x}+\mathbf{y})\,\mathrm{d}\mathbf{y} + \int_{\mathbb{R}^2\setminus B_\varepsilon(0)} \frac{D^{\alpha/2}}{|c(\alpha)|}|\mathbf{y}|^{-(2+\alpha)} I(t,\mathbf{x}+\mathbf{y})\,\mathrm{d}\mathbf{y}.$$

The factor $D^{\alpha/2}$ corresponds to a *generalized diffusion coefficient* (Metzler and Klafter 2000). Using (4), the second term can be rewritten such that

$$\Upsilon_s(t,\mathbf{x}) \approx -D^{\alpha/2}(-\Delta_\mathbf{x})^{\alpha/2} I(t,\mathbf{x}) + I(t,\mathbf{x}) \int_{B_\varepsilon(0)} \iota(\mathbf{y})\,\mathrm{d}\mathbf{y} + I(t,\mathbf{x}) \int_{\mathbb{R}^2\setminus B_\varepsilon(0)} \frac{D^{\alpha/2}}{|c(\alpha)|}|\mathbf{y}|^{-(2+\alpha)}\,\mathrm{d}\mathbf{y}.$$

Accordingly, $\Upsilon_x(t,\mathbf{x}) := -C_1 D^{\alpha/2}(-\Delta_\mathbf{x})^{\alpha/2} I(t,\mathbf{x}) + C_2 I(t,\mathbf{x})$, where $C_{1,2}$ are positive constants, is a possible choice for the interaction term in a fractional-in-space differential equation SIR model,

$$\begin{aligned}
\partial_t S(t,\mathbf{x}) &= -p_I \Upsilon_x(t,\mathbf{x})\, S(t,\mathbf{x}) \\
\partial_t I(t,\mathbf{x}) &= p_I \Upsilon_x(t,\mathbf{x})\, S(t,\mathbf{x}) - p_R I(t,\mathbf{x}) \\
\partial_t R(t,\mathbf{x}) &= p_R I(t,\mathbf{x}).
\end{aligned}$$

A more formal route to the (same) fractional interaction term is via the fractional Taylor series expansion of $I(t,\mathbf{x})$, which could indicate the correct formalization of the coefficients $C_{1,2}$.

In this work, for numerical simulation a simple Euler scheme ($dt = 1$) was used. The fractional Laplacian was approximated using the Fourier spectral method described in Bueno-Orovio et al. (2014). The space discretization aligns with the ε in the construction of $\Upsilon_x(t,\mathbf{x})$. Similar and further numerical methods for fractional differential equations are for instance discussed in Hanert and Piret (2014), Yang et al. (2011), and Bonito et al. (2018). The solution method used here is not very accurate or stable but suffices to reproduce the qualitative statements of the paper. Furthermore, the coefficients in $\Upsilon_x(t,\mathbf{x})$ were heuristically set to $C_1 = 1.0/\Gamma(2\alpha+1)$ and $C_2 = 1 - C_1$.

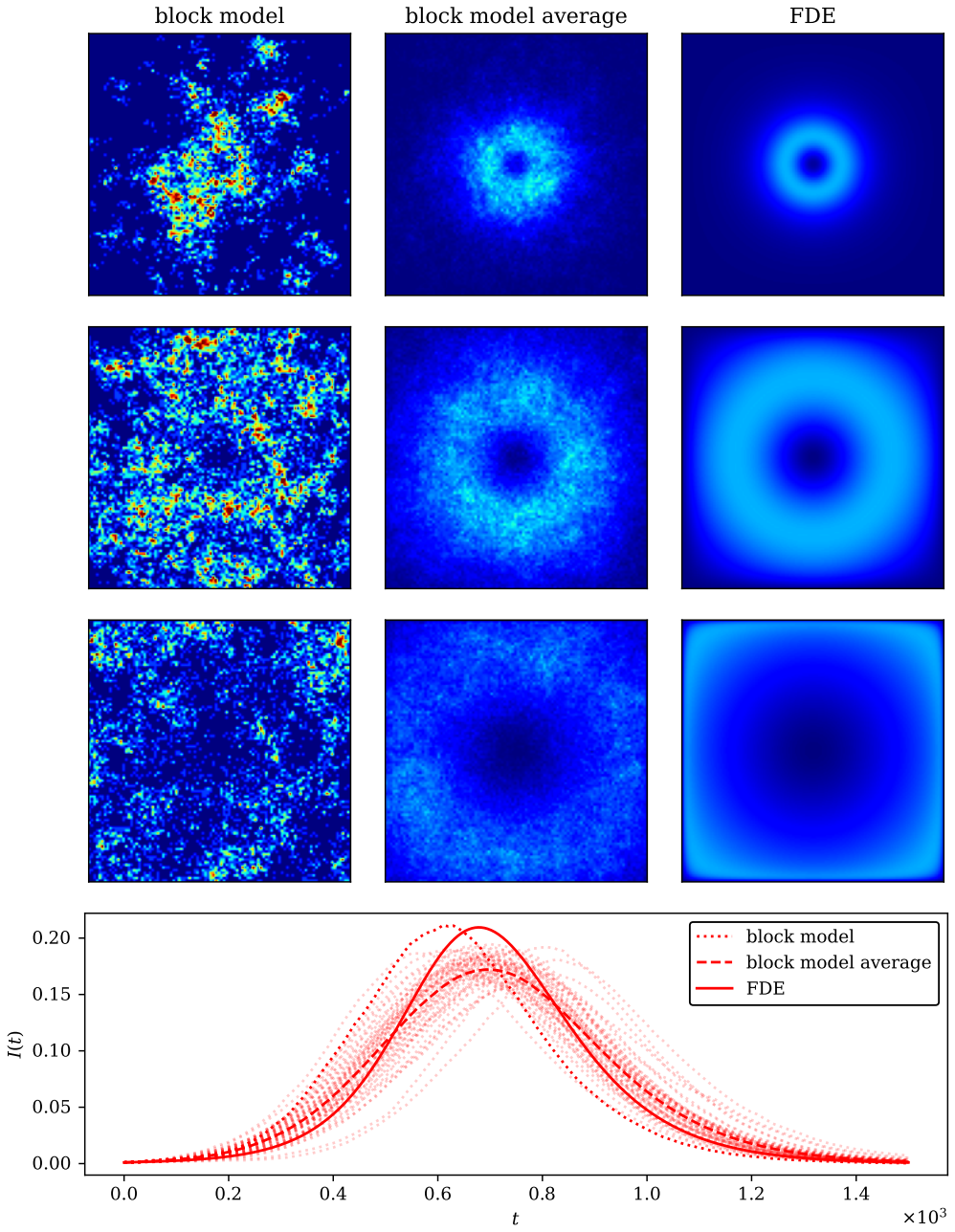

Figure 4: Comparison of the hierarchical block model and the fractional differential equations model. The stability parameter was $\alpha = 1.5$ and the diffusion coefficient was set to $D = 3.0$. Further parameterization of the block model was $L = 6, K = 4, N = 5, p = 0.7, E = 6$, which led to the block scaling parameters $\sigma_0 = 0.66$ and $c = 2.23$ with 10^5 nodes. The infection and recovery rates in both models were set to $p_I = 0.03$ and $p_R = 0.01$. Located in the center of the domain (100 units of length), a fraction of 0.001 of the total population was initially infected in both models. Solution of the differential equation was calculated on a 100×100 cell grid. The rows show the spatial configuration of infected $I(t)$ at $t = 400, 600, 800$. The center column shows the average of 50 simulation runs with the block model.

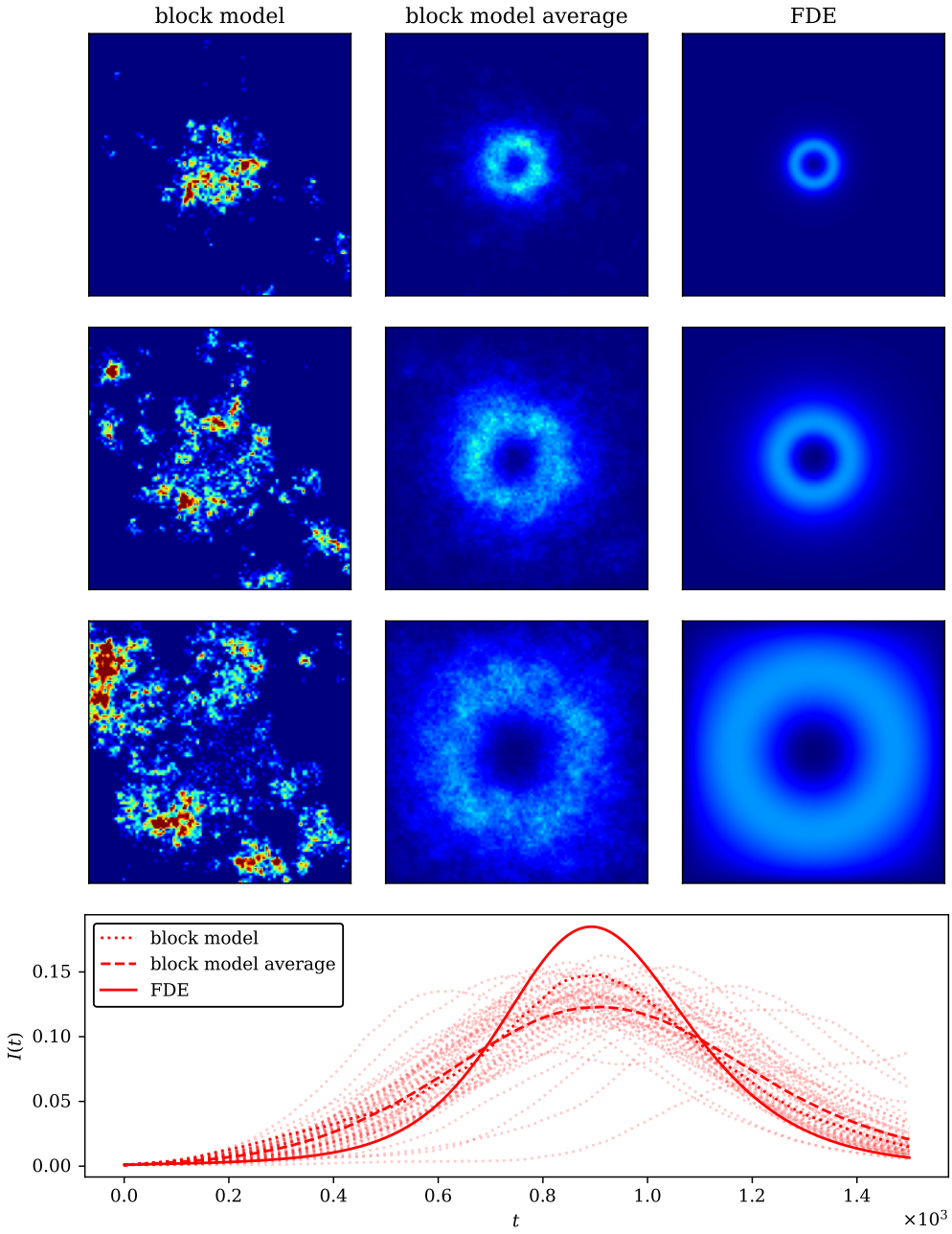

Figure 5: Comparison of the hierarchical block model and the fractional differential equations model. The parameterization is identical to the simulations in Figure 4 except for the stability parameter, which was set to $\alpha = 1.8$. The rows show the spatial configuration of infected $I(t)$ at $t = 400, 600, 800$.

In Figure 4 and Figure 5 simulations with the hierarchical block model are compared to the numerical solution of the fractional differential equations model. Due to the crude and heuristic parameterization of the fractional interaction term (coefficients $C_{1,2}$), the presented comparison is of qualitative nature at best. However, to accommodate for additional indirections between the hierarchical block model, its stochastic

approximation and also the fractional reaction-diffusion equation, several modifications have been applied to the hierarchical block model:

(OB) The stochastic model implies a configuration with overlapping blocks. Hence, during simulation nodes are stochastically assigned to base blocks before a topological neighbor is sampled.

(UD) To generate a uniform population density, multiple highest level blocks (e.g. 20 in the results presented above) are sampled with their center locations distributed uniformly on a bounded domain.

(BC) Nodes sampled from the resulting base blocks that lie outside of the domain are removed in order to obtain a boundary condition that aligns with the stochastic block model and the differential equations model. Because missing nodes introduce reduced interaction in the boundary regions, Dirichlet boundary conditions can be used for the fractional Laplacian in the continuous model.

The spatial discretization applied in the numerical solution of the fractional differential equation and the temporal scheme introduce additional errors. In general, the boundary conditions and initial conditions in a fractional setting can have a stronger influence on the solution than in the usual case (Kutner and Masoliver 2017). Therefore, careful adjustment of both conditions is necessary. Also the configuration of the block model involves a delicate balance between computational performance and accuracy (Schneckenreither 2020). For instance, a small number of layers introduces a cutoff in the spatial increment distribution, which in turn leads to a decreased diffusivity.

5 LIMITATIONS AND OUTLOOK

In this paper a highly simplified and abstracted model of structured interacting populations and the spread of infectious diseases was investigated. The presented model is capable of reproducing certain anomalous patterns observed in the progression of epidemic outbreaks. From a stochastic and spatial formalization of the topological structures and jumps in the hierarchical block model, the basic structure of a corresponding fractional-in-space reaction-diffusion differential equation model was derived. It is noteworthy that the reconstruction of anomalous diffusion and the fractional diffusion equation by simulating random walks on geometric fractals is not completely new.

The dynamic spread of infectious diseases in structured populations is a superposition of many different effects and mechanisms. For instance, when the epidemic reaches a fully susceptible block, all members can become infected in a relatively short period of time, which corresponds to a small local burst in the number of infected. In the global infection number and in aggregated models, local bursts can not be recognized directly. Hence, complex structural features and dynamic patterns of individual-based models can be included in aggregated models by introducing fractional spatial derivatives.

In Figure 3 it is evident that the clean linear and super-linear behavior of the mean squared deviation is only maintained for a rather small period of time. Especially if the spatial expansion of the epidemic is super-diffusive, the infection reaches all base blocks quickly. When this state is reached, no further significant spatial expansion can occur. In order to allow further spatial expansion it would be necessary to add additional blocks and block levels, which can drastically increase the requirements for computer memory and simulation time. However, this exhaustion does not necessarily correspond to the point in time when the peak number of infected is reached. The local saturation of the population is obtained once the infection is brought to a base block and intra-block transmission led to the infection of all individuals in the block. Depending on the configuration of the disease, this can happen with a certain delay when compared to the maximum spatial expansion. Because certain effects cannot be captured by the mean squared deviation, additional methods and measures for quantifying the anomaly of the spatial expansion of simulated epidemics should be applied.

In Figure 4 and Figure 5 simulation results from the hierarchical block model and from the fractional differential equation SIR model were compared. Despite the underlying mathematical connection between both models is very informal and preliminary, a certain qualitative and quantitative correspondence can

be recognized. Besides the inaccurate parameterization of the fractional interaction term, additional errors that emerge in the numerical scheme for the fractional differential equation and in the hierarchical block model were discussed. The presented approach and structural formulation of the interaction term could serve as a starting point for finding an exact and analytical connection between both models.

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