Lévy-flight Spreading of Epidemic Processes leading to Percolating Clusters

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We consider two stochastic processes, the Gribov process and the general epidemic process, that describe the spreading of an infectious disease. In contrast to the usually assumed case of short-range infections that lead, at the critical point, to directed and isotropic percolation respectively, we consider long-range infections with a probability distribution decaying in d dimensions with the distance as $1/R^{d+\sigma}$. By means of Wilson’s momentum shell renormalization-group recursion relations, the critical exponents characterizing the growing fractal clusters are calculated to first order in an $\varepsilon$-expansion. It is shown that the long-range critical behavior changes continuously to its short-range counterpart for a decay exponent of the infection $\sigma = \sigma_c > 2$.

I. INTRODUCTION

A. Epidemic Processes

The investigation of the formation and of the properties of random structures has been an exciting topic in statistical physics for many years. In the case that the formation of such structures obeys local rules, these processes can often be expressed in the language of population growth. It is well known that two special growth processes called (in the language of a disease) simple epidemic with recovery (Gribov process) [1-3], also known in elementary particle physics as Reggeon field theory [3-5], the stochastic version of Schlögl’s first reaction [6,7] and epidemic with removal (general epidemic process (GEP) [8-10]) lead to random structures with the properties of percolation clusters: directed percolation [11-13] in the first case and isotropic percolation (for a recent overview see [14]) in the last one [15-17]. These stochastic processes describe the essential features of a vast number of growth phenomena of populations of infected individuals near their extinction threshold and are relevant to a wide range of models in physics, chemistry, biology, and sociology. The transition between survival and extinction of such a growing population is a nonequilibrium continuous phase transition phenomenon and is characterized by universal scaling laws.

The Gribov process with short-range infection belongs to the universality class of local growth processes with absorbing states [18,19] such as the contact process [19-21] and certain cellular automata [22,23]. This universality class is characterized by the following four principles:

1. Infection of susceptible (“birth”) and spontaneous annihilation (“recovery”) of infected individuals.
2. Interaction (“saturation”) between the infected individuals.
3. Diffusion (“spreading”) of the disease in a d-dimensional space.
4. The state without infected individuals is absorbing.

To model these principles in a universal form, we use from the beginning a mesoscopic picture in which all microscopic length- and time-scales are considered as very short. Thus we take a continuum approach with the density of the infected individuals (the ills) at time $t$ as an order parameter. Note that the spontaneous annihilation of the ills makes it possible to avoid complications arising in the case of only diffusion controlled reactions which need creation and destruction operators as order parameters for a correct description.

The Langevin equation is constructed in accordance with the four principles as

$$\partial_t n = \lambda \nabla^2 n + R[n] n + \zeta,$$

where $\zeta(x,t)$ denotes a Gaussian Markovian noise with short range correlations which has to vanish if $n(x,t) = 0$ to model the absorbing state and the reaction rate $R[n]$ models birth, recovery and saturation. In a low density expansion we may set

$$R[n] = -\lambda \left( \tau + \frac{q}{2} n \right),$$

$$\langle \zeta(x,t) \zeta(x',t') \rangle = \lambda \gamma' n(x,t) \delta(x-x') \delta(t-t').$$

In contrast to the Gribov process (GP), the general epidemic process (GEP) introduces besides the susceptibles, $S$, who can catch the disease, and the infectives or ills, $I$, who have the disease and can transmit it, as a third class the removed, $R$, namely those who have had the disease and are now immune or death. Thus the first principle above is to be modified to

1. Infection of susceptible and spontaneous annihilation but without recovery of susceptible individuals.
Therefore the reaction rate now also depends on the number of the removed individuals, introducing a memory term into the process. Because this term is the leading one in the long-time and large-distance limit we now have 

$$R[n] = -\lambda (\tau + gm),$$ \hspace{1cm} (4)

$$m(x, t) = \lambda \int_{-\infty}^{t} dt' n(x, t').$$ \hspace{1cm} (5)

In a microscopic realization of the Gribov process a single species of (quasi)particles, the $I$’s, is introduced. The $I$’s represent the infected individuals (sites of a lattice). They perform simple random walks and undergo the following “chemical” reaction scheme built from reversible branching and irreversible spontaneous annihilation:

Gribov Process \[
\begin{aligned}
I &\rightarrow 2I \\
I &\rightarrow \emptyset
\end{aligned}
\]

Above some value of the branching rate of the reaction $I \rightarrow 2I$, the stationary state has a finite density of $I$’s. As the branching rate goes down to a threshold value, the stationary state density of $I$’s goes continuously to zero, which is an absorbing state below the threshold. This threshold value corresponds to the critical point which is an absorbing state below the threshold. This model the key parameter is the initial density of immune:

$$\text{General Epidemic Process} \begin{cases} 
S + I \rightarrow 2I \\
I \rightarrow R
\end{cases}$$

The history of this model goes back to 1927 when it was first introduced in the mathematical biology literature \[10\]. Here of course the stationary state is $I$-free. In this model the key parameter is the initial density of $S$’s, denoted by $\rho$: depending on the value $\rho$ with respect to a threshold value $\rho_c$, the infected individuals may either start to proliferate as a solitary wave before dying out in a finite system, which occurs for $\rho > \rho_c (\tau < \tau_c$ in Eq. \[4\]), or their number decreases from the outset, which takes place for $\rho < \rho_c (\tau > \tau_c)$.

**B. Lévy-flight Infections**

In the standard version of the epidemic models the susceptible individuals can become contaminated by already infected neighboring individuals. At the same time infected individuals are subject to spontaneous healing or immunization processes.

In realistic situations the infection can be also long ranged. This may be e.g. by a disease in an orchard where flying parasites contaminate the trees practically instantaneous in a widespread manner if the timescale of the flights of the parasites is much shorter as the mesoscopic timescale of the epidemic process itself. Thus following a suggestion of Mollison \[8\], Grassberger \[24\] introduced a variation of the epidemic processes with infection probability distributions $P(R)$ which decays with the distance $R$ as a power-law like

$$P(R) \propto \frac{1}{R^{d+\sigma}}, \quad \text{for} \quad R \rightarrow \infty.$$ \hspace{1cm} (6)

We will in general denote such long-range distributions as Lévy-flights although a true Lévy-flight is defined by its Fourier transform as $\tilde{P}(q) \propto \exp(-b|q|^\sigma)$ \[25\], and then only Lévy-exponents with $0 < \sigma \leq 2$ give rise to positive distributions \[26\]. The infection rate in the Langevin equation \[1\] is now given by

$$\frac{\partial n(x, t)}{\partial t} \bigg|_{\inf} = \int d^d x' P(|x - x'|) n(x', t)$$ \hspace{1cm} (7)

After Fourier transformation of this equation and after a small momentum expansion that is relevant in our mesoscopic consideration we get

$$\frac{\partial \tilde{n}(q, t)}{\partial t} \bigg|_{\inf} = (p_0 - p_2 q^2 + p_\sigma q^\sigma + o(q^2, q^\sigma)) \tilde{n}(q, t)$$ \hspace{1cm} (8)

where the analytical terms stem from the short-range part of $P(R)$ and the nonanalytical ones arise from the power-law decay \[1\]. The constant $p_0$ is included in the reaction rate as a negative (“birth”) contribution to $\tau$ whereas $p_2$ yields a diffusional term. Naively the parameter $p_\sigma$ is relevant or irrelevant in the long wave-length limit if $\sigma$ is smaller or bigger than 2 respectively, and this fact has mislead some authors to neglect this term from the outset if $\sigma > 2$. But this naive (“Gaussian”) argumentation may be wrong in an interacting theory because the critical behavior is in general determined by an non-trivial fixed point of a renormalization group transformation. To decide which one of the terms in Eq. \[8\] are relevant, one has to compare with the scaling behavior of the Fourier transformed susceptibility $\chi(q, \omega) \propto q^{2-\eta}$. If $\sigma < 2 - \eta$, the parameter $p_\sigma$ is a relevant perturbation and must be included in a renormalization group procedure. Prominent examples of systems with $\eta < 0$ are $\phi^3$-models as e.g. the Yang-Lee-singularity model \[27\]. In all these cases $p_\sigma$ is relevant also for $\sigma > 2$ and cannot be neglected.

In the following we define $\sigma = 2(1 - \alpha)$ and the diffusion term in Eq. \[8\] is now completed by a term proportional to $\propto q^{2(1-\alpha)} n(q, t)$. In real space we write the completed Langevin equation as

$$\partial n = \lambda \left[1 - \frac{v}{2\alpha} \left((-\nabla^2)^{-\alpha} + 1\right)\right] \nabla^2 n + R[n] n + \zeta,$$ \hspace{1cm} (9)
and the gradient-terms should be only considered (in Fourier space) up to a cutoff $\Lambda$ that we have set to 1. Then stability of this terms against inhomogeneous perturbation is guaranteed if $v \geq 0$.

Grassberger\textsuperscript{23} reported new critical exponents for $\alpha > 0$ from a 1-loop calculation that contain some numerical errors. These exponents are discontinuous in the limit $\alpha \to +0$ if one assumes irrelevance of the new terms for $\alpha < 0$. In this paper we will reconsider the problem of the anomalous susceptibility exponent of the epidemic models with long-range to short-range behavior. We will show that the critical exponents change continuously at the boundary $2\alpha = \tilde{\eta}_{SR}$ from long-range to short-range behavior.

We remark that the interest in reaction-diffusion problems involving particles that perform Lévy flights is not new. In the physics literature, they have most recently arisen as follows. Particles performing simple random walks are subject to the reactions arisen as follows. Particles performing simple random walks are subject to the reactions

$$G_0 (q, t) = \Theta (t) \exp \left\{ -\lambda \left[ \tau + q^2 + \frac{v}{2\alpha} (q^{-2\alpha} - 1) q^2 \right] t \right\}$$

as a function of momentum $q$ and time $t$. This propagator guarantees stability for all $\alpha$ as long as $\tau \geq 0$, $v \geq 0$, and $q = |q| \leq 1$. For simplicity we have set the momentum cut-off $\Lambda = 1$.

To study the critical behavior of this system near the critical point we use Wilson’s renormalization procedure. We introduce the usual coarse graining parameter $b > 1$ and split the fields $s$ and $\tilde{s}$ into components which are non zero on the momentum shell $\Omega_b = \{ q | 1/b \leq |q| \leq 1 \}$ and components defined on the complement of $\Omega_b$, the latter being denoted by $s^{\infty}$ and $\tilde{s}^{\infty}$. We integrate out the short scale degrees of freedom in the weight $\exp (-J)$, that is, those defined on $\Omega_b$, and rescale the fields according to

$$s(x, t) \to s^{\prime} (b^{-1} x, b^{-2\gamma} t) = b^{(d + \gamma)/2} s^{\infty} (x, t),$$

$$\tilde{s}(x, t) \to \tilde{s}^{\prime} (b^{-1} x, b^{-2\gamma} t) = b^{(d + \gamma)/2} \tilde{s}^{\infty} (x, t).$$

Renormalized parameters $\tau^\prime$, $\nu^\prime$, and $g^\prime$ are defined in such a way that the coarse grained functional looks like the old one. The one-loop calculation is standard and does not present any technical difficulties. For infinitesimal renormalization transformation with $b - 1 \ll 1$ we obtain

$$i\omega + \lambda \left\{ \tau^\prime + q^2 + \frac{\nu^\prime}{2\alpha} (q^{-2\alpha} - 1) q^2 \right\}$$

$$= i\omega b^{-\gamma} \left[ 1 - \frac{u}{4 (1 + \tau)^2} \ln b \right]$$

$$+ \lambda b^{2\gamma - \gamma} \left[ \tau^\prime + \frac{u}{2 (1 + \tau)} \ln b \right]$$

$$+ \lambda q^2 b^{\gamma - \gamma} \left[ 1 + \frac{v}{2\alpha} (b^{2\alpha} q^{-2\alpha} - 1) \right]$$

and

$$u^\prime = ub^{1 - d + 2\gamma - 3\gamma} \left[ 1 - \frac{2u}{(1 + \tau)^2} \ln b \right].$$

II. THE GRIBOV PROCESS WITH LÉVY-FLIGHTS

A. Renormalization Group Analysis

In order to develop the renormalization group analysis we recast the Langevin equation (1) as a dynamic functional\textsuperscript{31,33,34}

$$J [\tilde{s}, s] = \int d^d x d t \tilde{s} \left\{ \partial_t + \lambda \frac{g}{2} (s - \tilde{s}) + \lambda \left[ \tau - \nabla^2 + \frac{v}{2\alpha} \left( (-\nabla^2)^{1-\alpha} + \nabla^2 \right) \right] s \right\}$$

where $\tilde{s}$ is Martin-Siggia-Rose response field\textsuperscript{35}. We note that he dynamic functional can also be derived using the methods developed in\textsuperscript{33,34} from a microscopic master equation. By a suitable rescaling of the density $n \propto s$, the constant $g^\prime$ in Eq. (1) is made equal to $g$. The dynamic functional (10) is then symmetric in the absorbing phase under the exchange $s (x, t) \leftrightarrow -\tilde{s} (x, -t)$. All correlation and response functions can be calculated as functional integrals with weight $\exp (-J)$ in a perturbation expansion involving the propagator (the unperturbed response function)
We use \( l = \ln b \) as the flow parameter of the renormalization transformation that yields then from the other terms in Eqs. \([13, 14]\) the flow equations
\[
\frac{d\tau}{dl} = \left(2 - \gamma\right)\tau + \frac{u}{2(1+\tau)} + O\left(u^2\right),
\]
\[
\frac{du}{dl} = \left[4 - d - \gamma - 2\gamma - \frac{2u}{(1+\tau)^2}\right] + O\left(u^2\right),
\]
\[
\frac{dv}{dl} = (2\alpha - \gamma) v.
\]

The last equation is exact since the operator \( s \left(-\nabla^2\right)^{1-\alpha} \) in the dynamic functional \([10]\) is not renormalized. The reason is that the renormalization procedure can only generate contributions that are analytic in the moments.

In order to study the fixed point structure we find it useful to introduce \( \bar{\varepsilon} = 4(1-\alpha) - d = \varepsilon - 4\alpha \). Near the fixed points we linearize the flow equation \([13, 14]\) for the relevant variable \( \tau \) about \( \tau_c = \tau_\ast \) as \( d\tau/dl \approx \nu^{-1}(\tau - \tau_\ast) \). The flow equations \([13, 14]\) have, besides the trivial short-range Gaussian fixed point \((\tau^\ast, u^\ast, v^\ast) = (0, 0, 0)\), stable for \( \alpha < 0 \) and \( d > 4 \), and the trivial Lévy-Gaussian fixed point \((\tau^\ast, u^\ast, v^\ast) = (0, 0, 2\alpha)\) with \( \eta_{LG} = \gamma^\ast = 2\alpha \), \( z = 2 + \zeta^\ast = 2(1-\alpha) \), stable for \( d > 4(1-\alpha) \) and \( \alpha > 0 \), two non trivial fixed points. The first one is the already known short-range directed percolation fixed point \((\tau^\ast, u^\ast, v^\ast) = (-\varepsilon/3, 2\varepsilon/3, 0) + O\left(\varepsilon^2\right)\), with \( \eta_{DP} = \gamma^\ast = -\varepsilon/12 + O\left(\varepsilon^2\right), z_{DP} = 2 + \zeta^\ast = 2 - \varepsilon/12 + O\left(\varepsilon^2\right), \nu_{DP}^{-1} = 2 - \varepsilon/4 + O\left(\varepsilon^2\right) \) stable for \( \varepsilon > 0, \alpha < -\varepsilon/24 + O\left(\varepsilon^2\right) \).

The second one is the new Lévy-directed-percolation fixed point
\[
u^\ast = \frac{4}{\varepsilon} + O\left(\varepsilon^2\right),
\]
\[
\nu^\ast = \frac{28\alpha + \bar{\varepsilon}}{14 + c\bar{\varepsilon}} + O\left(\varepsilon^2\right),
\]
\[
\tau^\ast = \tau_c = -\frac{\bar{\varepsilon}}{7(1-\alpha)} + O\left(\varepsilon^2\right)
\]

which is stable for \(-\varepsilon/24 + O\left(\varepsilon^2\right) < \alpha < \varepsilon/4\). We obtain for this fixed point the critical exponents
\[
\eta_{LDP} = \gamma^\ast = -\bar{\varepsilon}/7 + O\left(\varepsilon^2\right),
\]
\[
\bar{\eta}_{LDP} = \gamma^\ast = 2\alpha,
\]
\[
z_{LDP} = 2 + \eta_{LDP} - \bar{\eta}_{LDP} = 2(1-\alpha) - \frac{\bar{\varepsilon}}{7} + O\left(\varepsilon^2\right),
\]
\[
\nu_{LDP}^{-1} = 2(1-\alpha) - \frac{2\bar{\varepsilon}}{7} + O\left(\varepsilon^2\right).
\]

FIG. 1. Stability regions of the Gribov process with long range spreading in the \((\alpha, d = 4 - \varepsilon)\) plane. G, LG, DP and LDP indicate the respective stability regions of the short range Gaussian, Lévy Gaussian, short range directed percolation and Lévy directed percolation fixed point

B. Scaling analysis

In the following we consider the scaling behavior of two key quantities: the time dependent order parameter (the density of infected individuals) \( \rho(t) = \langle s(x, t) \rangle_{\rho_0} \) for \( t > 0 \) if the initial state at time \( t = 0 \) is prepared with a homogeneous initial density \( \rho_0 \), and the response function \( \chi(x, t) = \langle s(x, t) s(0, 0) \rangle \) that yields the density of infected individuals after the epidemic is initialized by a pointlike source at \( t = 0 \) and \( x = 0 \). Here we are interested in the Lévy-flight case only, thus we will disregard the subscripts at all critical exponents. We set \( \tau - \tau_c \rightarrow \tau \) in the following.

\[\text{From the rescaling Eqs. \([12]\) we get at the Lévy fixed point (which is approached for } b \gg 1 \text{) the relationship}
\]
\[
\rho(t, \tau, \rho_0) = b^{-(d+n)/2} \rho \left(b^{-\tau / \nu_{DP}} b^{\nu_{DP} - \nu_{DP}} \right),
\]

where the critical exponents are displayed by Eqs. \([20, 23]\). The scaling of the initial density is easily found by noting that \( \rho_0 \) arises in the Langevin equation \([8]\) as an additive source term \( q(x, t) = \rho_0 \delta(t) \) that translates in the dynamic functional \( \mathcal{J} \), Eq. \([10]\), to a further additive contribution \( \int d^d x \rho_0 \tilde{s}(x, 0) \) from which one directly reads off the scaling behavior of \( \rho_0 \) if one knows that \( \tilde{s}(x, 0) \) scales as \( \tilde{s}(x, t) \) \([38]\). At criticality, for \( \tau = 0, \)
one obtains from Eq. (24) that the order parameter first increases in a universal initial time regime \(39,33\) as
\[
\rho(t, \rho_0) \propto \rho_0 t^\theta
\]
where the universal initial time exponent \(\theta\) is given by
\[
\theta = \frac{-\eta}{z} = \frac{\bar{\varepsilon}}{1\sigma} + O(\bar{\varepsilon}^2),
\]
and we have set \(\sigma = 2(1 - \alpha)\). Then, after some crossover time, the order parameter decreases as
\[
\rho(t) \propto t^{-(d+\eta)/2z},
\]
with \(d + \eta / 2z = 1 - 3\bar{\varepsilon} / 7\sigma + O(\bar{\varepsilon}^2)\). \(27\)

In the non absorbing stationary state, i.e. for \(\tau < 0\) and \(t \to \infty\), the order parameter behaves as
\[
\rho_{\text{stat}}(\tau) \propto |\tau|^{\beta},
\]
where the order parameter exponent \(\beta\) is found as
\[
\beta = \frac{\nu (d + \eta)}{2} = 1 - \frac{2\bar{\varepsilon}}{7\sigma} + O(\bar{\varepsilon}^2).
\]
Finally, at the critical dimension \(d = 2\sigma = 4(1 - \alpha)\), the scaling behavior is mean-field like with logarithmic corrections,
\[
\rho(t) \propto \rho_0 \ln^{1/7} t
\]
in the initial time regime at criticality,
\[
\rho(t) \propto \ln^{3/7} t
\]
in the long time regime at criticality,
\[
\rho_{\text{stat}}(\tau) \propto |\tau| \ln^{2/7} \left(\frac{1}{|\tau|}\right)
\]
in the stationary state, \(30\)

which we mention for completeness.

The scaling behavior of the response function is given by
\[
\chi(x, t) = b^{-(d + \eta)} \chi(b^{-1} x, b^{-z} t, b^{1/\nu} \tau).
\]
First we read off the correlation lengths for space and time and the corresponding exponents as
\[
\xi_\perp \propto |\tau|^{-\nu_\perp}
\]
with \(\nu_\perp = \frac{\nu}{\sigma} = \frac{1}{\sigma} + \frac{2\bar{\varepsilon}}{1\sigma^2} + O(\bar{\varepsilon}^2)\), \(32\)
\[
\xi_\parallel \propto |\tau|^{-\nu_\parallel}
\]
with \(\nu_\parallel = z\nu = 1 + \frac{\bar{\varepsilon}}{7\sigma} + O(\bar{\varepsilon}^2)\). \(33\)

At criticality, \(\tau = 0\), we have
\[
\chi(x, t) = t^{-(d + \eta)/z} \mathcal{F}\left(x/t^{1/z}\right)
\]
with a universal scaling function \(\mathcal{F}(x)\). This relation shows that the density of infected individuals as the result from a pointlike seed dies out with an exponent \((d + \eta)/z = 2\beta/\nu_\sigma\). Comparing with the general decay law \(24\), we find that the probability to find after a time \(t\) an infected individual if there was a pointlike seed at time \(t = 0\) decays with an exponent \(\beta/\nu_\sigma\). The Fourier transformed susceptibility at the critical point scales as
\[
\chi(q, \omega) = q^{-2 + \nu} \tilde{\mathcal{F}}(\omega/q^z) \propto q^{-\sigma}.
\]
As the last scaling exponent that can be deduced in the usual way from the given critical exponents we present the fractal dimension of the clusters of the infected individuals:
\[
d_f = \frac{d - \beta}{\nu} = \frac{d - \eta}{2} \]
\[
= \sigma - \frac{3\bar{\varepsilon}}{7} + O(\bar{\varepsilon}^2).
\]
We note that the values of the exponents \(\nu_\parallel\) \(33\), \(\nu_\perp\) \(33\), \(\beta\) \(29\), and \(d_f\) \(30\) that we have found are different from those given by Grassberger \(24\). The values of all the exponents changes continuously at the stability boundary \(\sigma = 2(1 - \alpha) = 2 - \bar{\eta}_{DP} = 2 + \varepsilon/12 + O(\bar{\varepsilon}^2)\) to their short-range directed percolation values.

C. Comparison with existing simulations

In a recent letter Albano \(40\) has presented a numerical study of one-dimensional branching and annihilating random walks (BARW) in which the individuals perform Lévy flights. For Brownian particles in \(d < 2\) dimensions the BARW (which is defined by the equations \(A \rightarrow (m + 1)A\) and \(A + A \rightarrow \emptyset\) is known to belong for \(m\) odd to the universality class of directed percolation \(41,42\). For \(d > 2\) the systems shows a phase transition at zero branching rate which can be described by mean field exponents. If the random walk is replaced by Lévy flights, noise becomes irrelevant above \(d_\sigma = \sigma\).

Albano has investigated the behavior of the critical exponents for \(m = 1\) as a function of \(\sigma\), for \(0.25 < \sigma < 1\). His results are summarized in the following table:

| \(\sigma\) | \(z\) | \(\eta\) | \(z - \sigma - \eta\) |
|---|---|---|---|
| 2 | 1.590 | -0.482 | 0.072 |
| 1.5 | 1.585 | -0.483 | 0.568 |
| 1 | 1.583 | -0.489 | 1.073 |
| 0.75 | 1.581 | -0.512 | 1.343 |
| 0.5 | 1.575 | -0.559 | 1.628 |
| 0.25 | 1.569 | -0.774 | 1.893 |

The critical dimension \(d_\sigma\) is lower than 1 for \(\sigma \in \{0.25, 0.5, 0.75\}\). (For \(\sigma = 0.25\) even the critical dimension of the Lévy-flight directed percolation \(d_\sigma = 4(1 - \alpha) = 2\sigma\) is lower than 1). Therefore the phase
transition should occur at zero branching rate, and critical exponents should be the mean field ones \((\beta_{\text{MF}} = 1, z_{\text{MF}} = \sigma \text{ and } \eta_{\text{MF}} = 0)\); this is clearly not the case for the exponents given in table \([32]\). The hyperscaling relation \(z = \sigma + \eta\) Eqs. \([21, 22]\), which we have shown to hold in any dimension, is violated as \(\sigma\) is decreased. These facts cast doubt on the reliability of the simulations performed in \([40]\). A possible explanation is to be found in the Lévy flight generation procedure. Indeed the author uses a distribution Eq. \([16]\) truncated at some distance cut-off, the effect of which is to produce an effectively short range motion. This interpretation is confirmed by the slow variation of the exponents as a function of \(\sigma\), their values remaining close to that of directed percolation with simple random walk displacement.

III. THE GENERAL EPIDEMIC PROCESS WITH LÉVY-FLIGHTS

A. Renormalization Group Analysis

The renormalization group analysis of the GEP is performed analogously to the corresponding analysis of the Gribov process presented in section 2. The Langevin equation for the GEP \([41]\), where now the reaction rate is given by Eq. \([43]\), is recast in the dynamic functional \([44]\):

\[
\mathcal{J} [s, \tilde{s}] = \int dx dt \left\{ \partial_t + \lambda g S - \frac{\lambda}{2} \tilde{s}^2 \right\} s + \lambda \left[ \tau - \nabla^2 + \frac{v}{2\alpha} \left( (-\nabla^2)^{\alpha} + \nabla^2 \right) \right] \tilde{s}.
\]

(38)

The field \(S(x, t) = \lambda \int_{t=0}^{t} dt' s(x, t')\), a rescaled form of Eq. \([42]\), introduces a memory term in the dynamics. In analogy to the Gribov process we have rescaled the fields so that \(g' = g\). The dynamic functional \([43]\) is then symmetric under the exchange \(S(x, t) \leftrightarrow \tilde{s}(x, t)\), or \(s(x, t) \rightarrow \partial_t \tilde{s}(x, t)\) \([44]\). From this symmetry follows that we only need to consider one coupling coefficient \(g\) for the two interaction terms in \(\mathcal{J}\). The perturbation expansion involves also the propagator displayed in Eq. \([41]\), and \(v \geq 0\) is needed for stability. We integrate out the short scale degrees of freedom in the weight \(\exp (-\mathcal{J})\), and rescale now the fields according to

\[
\begin{align*}
\tilde{s}(x, t) &\rightarrow \tilde{s}'(b^{-1} x, b^{-2} \tau t) = b^{(d+2\gamma)/2} \tilde{s} \\left( x, t \right), \\
\tilde{s}(x, t) &\rightarrow \tilde{s}'(b^{-1} x, b^{-2} \tau t) = b^{(d+2\gamma)/2} \tilde{s} \\left( x, t \right).
\end{align*}
\]

(39)

Note that from the exchange symmetry follows exactly

\[
\zeta = \frac{\gamma - \tilde{\gamma}}{2}.
\]

(40)

The renormalized parameters \(\tau', v', \) and \(g'\) are now defined by the coarse graining equations calculated to one-loop order with \(b - 1 \ll 1\)

\[
\begin{align*}
\bar{s} &\rightarrow \frac{1}{\xi} + \mathcal{O}(\xi^2), \\
v &\rightarrow \frac{g_{\text{MF}} + 2\xi}{4\xi} + \mathcal{O}(\xi^2), \\
\tau &\rightarrow \frac{\xi}{16(1 - \alpha)} + \mathcal{O}(\xi^2).
\end{align*}
\]

(41)
which is stable for $-\varepsilon/42 + O(\varepsilon^2) < \alpha < \varepsilon/6$. We obtain for this fixed point the critical exponents

\begin{align}
\eta_{\text{GEP}} &= \gamma^* = -2\alpha - \frac{3\varepsilon}{8} + O(\varepsilon^2), \\
\eta_{\text{LGEP}} &= \bar{\gamma}^* = 2\alpha, \\
z_{\text{LGEP}} &= 2 + \zeta^* = 2(1 - \alpha) - \frac{3\varepsilon}{16} + O(\varepsilon^2), \\
\nu_{\text{LGEP}}^{-1} &= 2(1 - \alpha) - \frac{\varepsilon}{4} + O(\varepsilon^2).
\end{align}

Note again that at all fixed points $u^*$ and $v^*$ are non-negative as they should for stability of the theory. We have depicted in Fig. 2 the stability regions for each of the above fixed points in the $(\alpha, d)$ plane. Now a glance at the exact flow equation (47) of the parameter $v$ shows that the boundary between the domains of attraction of the short-range-GEP fixed point and the Lévy-GEP fixed point is given exactly by $\eta_{\text{GEP}} = \eta_{\text{LGEP}} = 2\alpha$. At this boundary all exponents are again continuous functions of the parameter $\alpha$.

![Fig. 2. Stability regions of the general epidemic process with long range spreading in the $(\alpha, d = 6 - \varepsilon)$ plane. G, LG, GEP and LGEP represent the stability regions of the short range Gaussian, Lévy Gaussian, short range percolation and Lévy percolation fixed point, respectively.](image)

**B. Scaling analysis**

At first we consider the scaling behavior of the time dependent density of infected individuals $\rho(t) = \langle S(x,t)\rangle_{\rho_0}$ for $t > 0$ if the initial state at time $t = 0$ is prepared with a homogeneous initial density $\rho_0$. We are interested in the Lévy-flight case only, thus we will disregard the subscripts at all critical exponents. Again we set $\tau - \tau_c \rightarrow \tau$.

Here we find from the rescaling Eqs. (39) at the Lévy fixed point (which is approached for $b \gg 1$) the relationship

$$
\rho(t, \tau, \rho_0) = \rho_{\text{stat}}(\tau > 0, \rho_0) \propto \rho_0^{\frac{1}{\delta}}
$$

with

$$
\delta = \frac{d + 2 - \bar{\eta}}{d - 2 + \bar{\eta}} = \frac{d + \sigma}{d - \sigma}.
$$

Below threshold, that is $\tau > 0$, the order parameter $\rho_{\text{stat}}(\tau > 0, \rho_0)$ plays here the role of an ordering field. In the infinite time limit at criticality, when $\tau = 0$, the order parameter $\rho_{\text{stat}} = \rho(t \rightarrow \infty)$ goes to zero with $\rho_0$ as

$$
\rho_{\text{stat}}(\tau = 0, \rho_0) \propto \rho_0^{\frac{1}{\gamma}},
$$

with

$$
\bar{\gamma} = (2 - \bar{\eta}) \nu = \sigma \nu = 1 + \frac{\varepsilon}{4\sigma} + O(\varepsilon^2).
$$

Where the critical exponents are displayed by Eqs. (49-52). The scaling of the initial density is also found by adding a source term $q(x,t) = \rho_0\delta(x)$ to the Langevin equation (3) that translates in the dynamic functional $\mathcal{F}$. Eq. (52) to the additive contribution $\int d^d x \rho_0 \delta(x,0)$ from which one reads off the scaling behavior of $\rho_0$ since $\bar{s}(x,0)$ scales as $\tilde{s}(x,t)$. At criticality, for $\tau = 0$, it follows from Eq. (24) that the infection density first increases in the universal initial time regime as

$$
\rho(t, \rho_0) \propto \rho_0^\theta
$$

where the universal initial time exponent $\theta$ is given by

$$
\theta = -\eta + \bar{\eta} = \frac{\sigma}{z} - 1 = \frac{3\varepsilon}{16\sigma} + O(\varepsilon^2).
$$

We have set $\sigma = 2(1 - \alpha)$.

As an order parameter we consider the density of the removed (immune) individuals $\tilde{s}$ namely

$$
\tilde{s}(t, \tau, \rho_0) \propto \langle S(x,t)\rangle_{\rho_0} = \lambda \int_0^t dt' \langle s(x,t)\rangle_{\rho_0}.
$$

The scaling properties of this order parameter are determined from Eq. (53) by

$$
\tilde{s}(t, \tau, \rho_0) = \tilde{s}(d-2+\bar{\eta})/\rho_0 \left(b^{-\beta} t^{b^\nu/\bar{\gamma} \tau}, b^{(d-2-\bar{\eta})/\rho_0}\right).
$$

Lastly for $\tau < 0$, the order parameter is independent of $\rho_0$ in the limit $\rho_0 \rightarrow 0$, and goes to zero with $\tau$ as

$$
\rho_{\text{stat}}(\tau < 0, \rho_0) \propto |\tau|^{\bar{\beta}},
$$

where the order parameter exponent is given by

$$
\bar{\beta} = \nu \frac{(d-2+\bar{\eta})}{2} = \nu \frac{(d-\sigma)}{2} = 1 - \frac{\varepsilon}{4\sigma} + O(\varepsilon^2).
$$
At the critical dimension $d = 3\sigma$, the scaling behavior of $\bar{\rho}_{\text{stat}}$

$$\bar{\rho}_{\text{stat}}(\tau = 0, \rho_0) \propto \rho_0^{1/2}$$
$$\bar{\rho}_{\text{stat}}(\tau > 0, \rho_0) \propto \rho_0 \frac{\ln^{1/4} \tau}{\tau}$$
$$\bar{\rho}_{\text{stat}}(\tau < 0) \propto |\tau| \ln^{1/4} \left( \frac{1}{|\tau|} \right). \quad (62)$$

is mean-field like with logarithmic corrections. At criticality we find from (57) the scaling behavior

$$\bar{\rho}(t, \rho_0) = \rho_0^{(d-\sigma)/(d+\sigma)} \mathcal{F} \left( t\rho_0^{2z/(d+\sigma)} \right) \quad (63)$$

with a universal scaling function

$$\mathcal{F}(x) \propto \begin{cases} 
\frac{x^{2/\nu}}{z} & \text{for } x \ll 1 \\
1 & \text{(exponentially) for } x \to \infty.
\end{cases} \quad (64)$$

Note that the “static” exponents $\bar{\beta}, \bar{\gamma}, \bar{\delta}, \bar{\eta}$ (and $\nu$) correspond to well known undirected percolation exponents but for long-range connectivity. They were already given in [24,44].

To study the spread of the infection by computer simulations one may investigate the response function $\bar{\chi}(x, t) = \langle S(x, t) s(0, 0) \rangle$ which describes the density of the immune percolating individuals at the time $t$ caused by an infection at $t = 0, x = 0$. Its scaling behavior is given by

$$\bar{\chi}(x, t, \tau) = b^{-(d-2+\bar{\eta})} \left( b^{-1} x, b^{-\bar{\gamma}} t, b^{1/\nu} \tau \right). \quad (65)$$

We read off the correlation lengths for space and time and the corresponding exponents as

$$\xi \propto |\tau|^{-\nu}$$

with

$$\nu = \frac{1}{\sigma} + \frac{\bar{\varepsilon}}{4\sigma^2} + O \left( \bar{\varepsilon}^2 \right), \quad (66)$$

$$\xi_t \propto |\tau|^{-\nu_t}$$

with

$$\nu_t = z\nu = 1 + \frac{\bar{\varepsilon}}{16\sigma} + O \left( \bar{\varepsilon}^2 \right). \quad (67)$$

At criticality, $\tau = 0$, we have

$$\bar{\chi}(x, t) = |x|^{-(d-2+\bar{\eta})} \mathcal{F}_\chi \left( |x|/t^{1/z} \right) \quad (68)$$

with an universal scaling function $\mathcal{F}(x)$. The Fourier transformed susceptibility at the critical point scales as

$$\chi(q, \omega) = q^{-2+\bar{\eta}-z} \mathcal{F}(\omega/q^2) \propto q^{-\sigma-z}. \quad (69)$$

As the last scaling exponent that can be deduced in the usual way from the given critical exponents we present the fractal dimension of the percolation clusters of the removed individuals:

$$d_f = d - \frac{\bar{\beta}}{\nu} = \frac{d + \sigma}{2}. \quad (70)$$

We note that the value of the exponent $\nu_t$ [24] that we have found is different from that given by Grassberger [24]. The values of all the exponents change continuously at the stability boundary $\sigma = 2(1 - \alpha) = 2 - \bar{\eta}_{\text{GEP}} = 2 + \varepsilon/21 + O \left( \varepsilon^2 \right)$ to their short-range undirected percolation values.

### IV. CONCLUSIONS

Epidemic processes are growth models for phenomena arising abundantly in nature. We have shown by imposing a Lévy flight type of infection spreading that new long-range determined universality classes come into play. We were able to characterize the universality classes by determining the critical exponents to first order in an $\varepsilon$-expansion around their upper critical dimension. There exist exact relationships between the exponents, and some critical exponents are given exactly as functions of spatial dimension and the exponent characterizing the long-range tail of the Lévy flight infection. Besides, we have been able to build a renormalization group flow that possesses a fixed point structure that allows to describe short-range and long-range infection in the same and to pass continuously from one behavior to another by varying the Lévy flight exponent. Because the anomalous susceptibility exponent, the analog of the Fisher exponent in critical equilibrium phenomena, is negative here, the continuous crossover between long- and short-range behavior arises at a Lévy exponent greater than 2. We hope that this work triggers more simulation work with a Lévy-like infection.

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