

# Quasi-Neutral Theory of Epidemic Outbreaks

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## Abstract

Some epidemics have been empirically observed to exhibit outbreaks of all possible sizes, i.e., to be scale-free or scale-invariant. Different explanations for this finding have been put forward; among them there is a model for “accidental pathogens” which leads to power-law distributed outbreaks without apparent need of parameter fine tuning. This model has been claimed to be related to self-organized criticality, and its critical properties have been conjectured to be related to directed percolation. Instead, we show that this is a (quasi) *neutral model*, analogous to those used in Population Genetics and Ecology, with the same critical behavior as the voter-model, i.e. the theory of accidental pathogens is a (quasi)-neutral theory. This analogy allows us to explain all the system phenomenology, including generic scale invariance and the associated scaling exponents, in a parsimonious and simple way.

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## Introduction

Many natural phenomena such as earthquakes, solar flares, avalanches of vortices in type II superconductors, or rainfall, to name but a few, are characterized by outbursts of activity. These are typically distributed as power-laws of their size, without any apparent need for fine tuning – i.e. they are generically scale invariant – [1–6]. This is in contrast to what occurs in standard criticality, where a control parameter needs to be carefully tuned to observe scale invariance. The concept of *self-organized criticality*, which generated a lot of excitement and many applications in different fields, was proposed to account for generic scale invariance, i.e. to explain the “ability” of some systems to self-tune to the neighborhood of a critical point [1–3].

The spreading of some epidemics, such as meningitis in human populations, has been repeatedly reported to exhibit scale-invariant traits, including a wide variability of both durations and sizes of outbreaks. Moreover, the ratio of the variance to the mean of the distribution of meningitis and measles outbreak sizes have been empirically found to be very large and to grow rapidly with population-size [7]. This is the hallmark of anomalously large fluctuations such as those characteristic of the heavy tails of power-law distributions [8]. Actually, power-laws have been proposed to fit the statistics of some epidemics such as measles, pertussis or mumps in some specific locations as the Faroe islands or Reykjavik for which accurate long-term epidemiological data are available [9–12]. Remarkably, in some cases, more than four orders of magnitude of scaling have been found [7]. Other features of scale invariance have been reported for measles [7] and other infectious child diseases [13,14], rabies and bovine tuberculosis [15], or cholera [16].

At a theoretical level, as pointed out by Rhodes and Anderson[9,10], the lack of a characteristic scale in epidemic

outbreaks is reminiscent of earthquakes and their associated (Gutenberg-Richter) power-law distribution. Actually, the presence of scale-invariance in measles, pertussis and others has been justified in [12] by exploiting the analogies between simple models for such epidemics and well-known (self-organized) earthquake models [1–3]. In what follows, we focus now on meningitis, for which a related self-organized mechanism has been recently proposed [17–22].

The bacteria responsible for meningitis, *Neisseria meningitidis* or meningococcus, is a human commensal: it is typically harmless and it is present in up to one fourth of the human population [23]. Infection is transmitted through close contact with previously infected individuals. It is noteworthy that killing their hosts is a highly undesirable outcome for bacteria; therefore it makes sense that evolution selected for hardly harmful bacteria strains. Nevertheless, the meningococcus can *accidentally* mutate into a potentially dangerous strain, becoming highly damaging or even lethal for the host. This is an example of a more general type of “accidental pathogens” that innocuously cohabit with the host but that eventually –even if rarely– mutate causing symptomatic disease [23].

Aimed at modeling accidental pathogens and to shed some light on the reasons for the emergence of scale invariance in meningococcal epidemics, Stollenwerk, Jansen and coworkers proposed a simple and elegant mathematical model [17–22]. The **Stollenwerk-Jansen (SJ) model** is a variant of the basic susceptible-infected-recovered-susceptible (SIRS) model. In the SIRS, individuals can be in any of the following states: “susceptible” (S), “infected” (I), or “recovered” (R) (also called “immune”) [24–26]. Perfectly mixed populations are usually considered, i.e. every individual is neighbor of any other (which is a mean-field assumption in the language of Statistical Physics or a “panmictic” one in the Ecology jargon). The additional key

ingredient introduced in the SJ model is a second, potentially dangerous, strain of infected individuals labeled  $Y$ . Dangerous strains appear at a very small (mutation) rate at every contagion event. The dynamics of  $Y$  is almost identical to that of  $I$  except for the fact that at a certain rate  $\epsilon$  they can cause meningococcal disease of newly infected hosts and eventually kill them,  $Y \rightarrow X$  (see below) [17–22].

By working out explicitly the analytical solution of this mean-field model as well as performing computer simulations, the authors above came to the counterintuitive conclusion that the smaller the value of  $\epsilon$  the larger the total amount of individuals killed on average in a given outbreak [17,18]. This apparent paradox is easily resolved by realizing that the total number of individuals infected with  $Y$  grows upon decreasing  $\epsilon$ . Actually, it has been shown that, whilst for high pathogenicity  $\epsilon$  the distribution of the number of observed  $Y$ -cases,  $s$ , is exponential, in the limit  $\epsilon \rightarrow 0$  it becomes power-law distributed, obeying

$$P(s) \sim s^{-(\tau=3/2)} F(s/s_c). \tag{1}$$

where  $\mathcal{F}$  is some scaling function and  $s_c$  a maximum characteristic scale controlled by  $1/\epsilon$  [17,18].

Observe that the exponent  $\tau=3/2$  matches that of a critical branching process, for which the average population of infected individuals does not either grow or decay in time but stays constant [27]. Also,  $\tau=3/2$  coincides with the exponent for the distribution of first return times of an unbiased random-walk [28], which describes generically the scaling of avalanches in mean-field models.

However, the value  $\tau=3/2$  predicted by the SJ model in its mean-field version, does not necessarily correspond to the best fit to empirical data [7,9,10]. To scrutinize the possible origin of such discrepancies, one would like to go beyond the mean-field/panmictic hypothesis by considering structured populations in which each individual has a finite local neighborhood. In what follows we shall study the SJ model on populations distributed on regular (Euclidean) lattices in dimensions  $d=2$  and  $d=1$ . The study of more complex networks (as small-world networks or networks with communities), aimed at describing more realistically the net of social contacts, is left for a future work.

As already pointed out [17,18], the SJ model can be straightforwardly made spatially explicit. Even if analytical or numerical calculations have not been performed, it was conjectured in [17–20] that in  $d$ -dimensions the SJ model should be in the *directed percolation* class, i.e. the broad and robust universality class characterizing phase transitions between an active and an absorbing state [29–34]. In the present case, the absorbing state would be the  $Y$ -free state obtained for sufficiently small  $Y$ -infection rates, while the active or endemic phase would correspond to a non-vanishing density of  $Y$ 's. In particular, if the critical behavior was indeed that of directed percolation, then  $\tau \approx 1.268$  for two-dimensional populations (and  $\tau \approx 1.108$  for epidemics propagating in one dimension) (see [35] and [36]).

A priori, the prediction of directed percolation scaling is somehow suspicious if the model is indeed self-organized; it has been shown that in general models of self-organized criticality, as sandpiles, ricepiles, earthquake models, etc. are *not* in the directed percolation class [37–41]. Furthermore, models of self-organized criticality lacking of any conservation law (as is the case of the SJ model) have been shown not to be strictly critical, i.e. they are just approximately close to critical points [42]; instead the exact solution by Stollenwerk and Jansen proves that their model is exactly critical [17,18]. These considerations cast some doubts on the conjecture of the SJ model being a model of self-organized

criticality [1,2,37–41]. Therefore, one is left with the following open questions: what is the SJ model true critical behavior?, what is the key ingredient why accidental pathogens – as described by the SJ model – originate scale invariant outbreaks?, does such an ingredient appear in other epidemics?

The purpose of the present paper is to answer these questions by analyzing the SJ model in spatially extended systems.

## Methods

The spatially explicit SJ model is defined as follows. Each site of a  $d$ -dimensional lattice is in one of the following states  $S, I, Y, R$ , or  $X$ . The dynamics proceeds at any spatial location  $x$  and any time  $t$  according to the following one-site processes:

- Spontaneous recovery of the benign strain:  $I \rightarrow R$ , at rate  $\gamma$ .
- Spontaneous recovery of the dangerous strain:  $Y \rightarrow R$ , at rate  $\gamma$ .
- Loss of immunity:  $R \rightarrow S$  at rate  $\alpha$ .
- Replacement or recovery of diseased:  $X \rightarrow S$ , at rate  $\varphi$ ,

and two-site processes:

- Infection with the benign strain:  $S + I \rightarrow I + I$ , at rate  $\beta - \mu$ .
- Infection with the dangerous strain:  $S + Y \rightarrow Y + Y$ , at rate  $\beta - \epsilon$ .
- Mutation:  $S + I \rightarrow Y + I$ , at rate  $\mu$ .
- Disease:  $S + Y \rightarrow X + Y$ , at rate  $\epsilon$ .

Some comments on these reaction rules are in order. Accordingly to [21] it is assumed that being infected with one strain protects against co-infection with a second one. This assumption, which is supported by some empirical observations [21], prevents transitions as  $I + Y \rightarrow Y + Y$  from appearing.  $X$  (diseased/dead) individuals are immediately replaced by new susceptible ones, so that the total population size is kept fixed; i.e.  $\varphi \rightarrow \infty$ . A “back mutation” ( $S + Y \rightarrow Y + I$ ) reaction could be introduced, but for most purposes its rate is so small that it can be neglected. The dynamics is easily implemented in computer simulations by using the Gillespie’s algorithm [43] or a variation of it in a rather standard way. In the well-mixed case writing the densities of the different species as  $S, I, Y, R$ , and  $X$ , one readily obtains the following mean-field or rate equations:

$$\begin{aligned} \dot{S} &= \alpha R + \phi X - \beta S(I + Y) \\ \dot{I} &= (\beta - \mu)SI - \gamma I \\ \dot{Y} &= (\beta - \epsilon)SY - \gamma Y + \mu SI \\ \dot{R} &= \gamma(I + Y) - \alpha R \\ \dot{X} &= \epsilon SY - \phi X \end{aligned} \tag{2}$$

satisfying the constraint  $S + I + Y + R + X = 1$ . Stollenwerk and Jansen [17,18] worked out the exact solution of this set of equations, concluding that it is critical in the limit  $\epsilon \rightarrow 0$ , and obtaining the explicit form of Eq.(1).

Instead, in spatially explicit models this mean-field approach breaks down: (i) the densities need to be replaced by spatio-temporal fields, as  $S(x,t), I(x,t), Y(x,t)$ , etc; (ii) new (Laplacian) terms describing the map of nearest-neighbor contacts appear; (iii) fluctuations become relevant and noise terms need to be added to account for demographic fluctuations. A full set of such stochastic Langevin equations can be derived from the microscopic dynamics

by using standard procedures [28,44,45] and numerically investigated [46] (results not shown here).

### Results

We now report on extensive numerical simulations for the SJ model. We have chosen the following parameter values:  $\alpha=0.3$ ,  $\beta=0.2$  and  $\gamma=0.1$ , and have tried other different sets to confirm the robustness of the results. We consider the mutation rate  $\mu$  to be sufficiently small, such that strains generated by consecutive mutations do not overlap; i.e. outbreaks finish well before a new mutation appears. For this reason we take as initial condition for any outbreak a state with a single mutant of the potentially dangerous strain  $Y$  and effectively fix  $\mu=0$  during outbreaks.

### Two dimensions

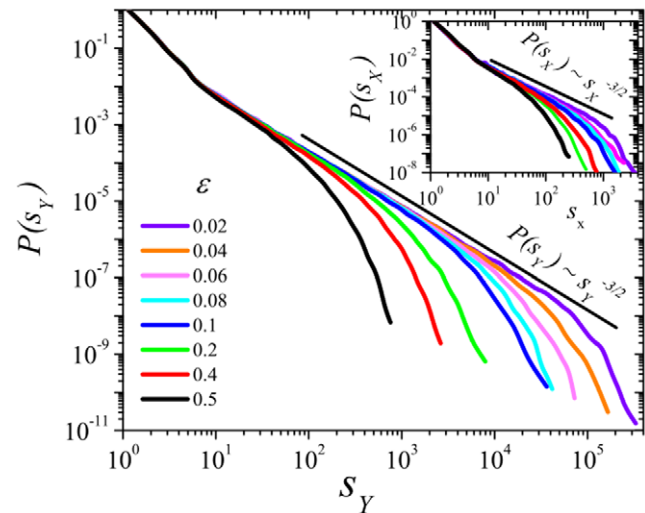
We consider square lattices of linear size  $L=32,64,128,256,512$ , and  $L=1024$ , take a random initial condition with only  $S$  and  $I$  individuals, and run the dynamics with periodic boundary conditions, keeping  $\mu=0$ , until a steady state is reached. For instance, for  $L=128$ , the steady state is characterized by  $\langle I \rangle \approx 0.38$ ,  $\langle R \rangle \approx 0.12$  and  $\langle S \rangle \approx 0.50$  for the set of parameters above.

Once the system sets into its steady state, we place a  $Y$  individual at the geometrical center of the lattice and study its spreading [31–34]. To avoid finite size effects, spreading experiments are stopped once the  $Y$ -strain touches the system boundary, and the described procedure is iterated. Depending on system size we ran up to  $10^7$  independent realizations. As customarily done, we monitor: (i) the epidemics size distribution, analogous to Eq.(1), for both  $Y$  and  $X$ ; (ii) the average total number of  $Y$  as a function of time  $N(t)$ ; (iii) the surviving probability  $P_s(t)$  that the  $Y$  strain is still present in the system at time  $t$ , and (iv) the average square radius from the origin of  $Y$ -infected individuals,  $R^2(t)$ . At criticality, these quantities are expected to scale algebraically as  $N(t) \sim t^\eta$ ,  $P_s(t) \sim t^{-\delta}$ , and  $R^2(t) \sim t^z$ , while they should show exponential cut-offs in the sub-critical (or absorbing) phase.

Fig. 1 shows the epidemic outbreak size distribution for different values of  $\epsilon$  and system size  $128 \times 128$  (size of  $Y$ -infected outbreaks,  $s_Y$ , in the main plot, and size of  $X$ -infected outbreaks,  $s_X$ , in the inset). The probability distribution of avalanches sizes  $s_X$  for  $X$ -infected sites is observed to inherit the statistics of  $Y$ -infected ones. Both distributions can be well fitted by Eq.(1) where  $F$  is a cut-off (exponential) function and  $s_c \sim \epsilon^{-1/\sigma}$  determines the maximum size. The best fit gives  $\tau \approx 1.48(5)$  (fully compatible with  $3/2$  as shown in Fig. 1) and  $1/\sigma \approx 2$  (not shown) both for  $P(s_Y)$  and  $P(s_X)$ . From these plots we conclude that the system becomes critical in the limit of vanishing  $\epsilon$ , as occurs in mean-field. Observe that for small pathogeneicities, as  $\epsilon=0.02$ , the system, even if sub-critical, exhibits scaling along more than three orders of magnitude.

Both the mean and the variance of the above distributions are too observed to diverge as power-laws in the limit  $\epsilon \rightarrow 0^+$ . Actually, as shown in Fig. 2 the ratio of the variance  $\sigma_{s_Y}$  (resp.  $\sigma_{s_X}$ ) to the mean  $\langle s_Y \rangle$  (resp.  $\langle s_X \rangle$ ) diverges when  $\epsilon \rightarrow 0^+$  as  $\sigma_{s_Y} / \langle s_Y \rangle \sim \sigma_{s_X} / \langle s_X \rangle \sim \epsilon^{-2.2(3)}$  in the infinitely large system-size limit (otherwise, for finite  $L$  a size-induced cut-off appear as illustrated in Fig. 2 by comparing simulations for two different sizes).

Analogously, fixing  $\epsilon=0$  we measure the mean and variance as a function of system size; the ratio of the variance to the mean diverges very fast as  $L$  grows,  $\sigma_{s_Y} / \langle s_Y \rangle \sim L^{3.8(3)}$  (results not shown). Observe that, trivially, in this case  $P(s_X) = \delta(s_X)$ , i.e. no death is produced.



**Figure 1. Main: Avalanche size distribution,  $P(s)$  for various values of  $\epsilon$  in a two dimensional lattice of size  $128 \times 128$ , and associated distribution for avalanche sizes of  $Y$ -infected individuals,  $s_Y$  (main plot), and  $s_X$  of  $X$ -infected ones (inset). doi:10.1371/journal.pone.0021946.g001**

Fig. 3 shows results obtained for spreading quantities for various sizes and  $\epsilon=0$ , i.e. at criticality. The best fits we obtain for the asymptotic behavior of these three magnitudes are

$$N(t) \sim 1, \quad P_s(t) \sim \frac{\log(t)}{t}, \quad R^2(t) \sim t \quad (3)$$

An effective power-law, with exponent slightly smaller than unity can be fit to our numerical results for  $P_s(t)$  (see Fig. 3); however, such an effective value of the exponent can be seen to grow upon extending the maximum time, making the case for a logarithmic correction as described by Eq.(3) and illustrated in Fig. 4. Observe also that the reported values,  $\eta=0$ ,  $\delta=1$ ,  $z=1$  satisfy the hyper-scaling relation  $\delta + \eta = dz/2$  using  $d=2$  [35].

Contrarily to a priori expectations and to a previous conjecture, these asymptotic laws have nothing to do with directed percolation values (which predicts pure power law behavior for the three spreading quantities [31–34] with  $\eta \approx 0.229$ ,  $\delta \approx 0.450$ ,  $z \approx 1.132$ ,  $\tau \approx 1.268$ ; see [35] for a set of spreading and avalanche exponent numerical values in different universality classes).

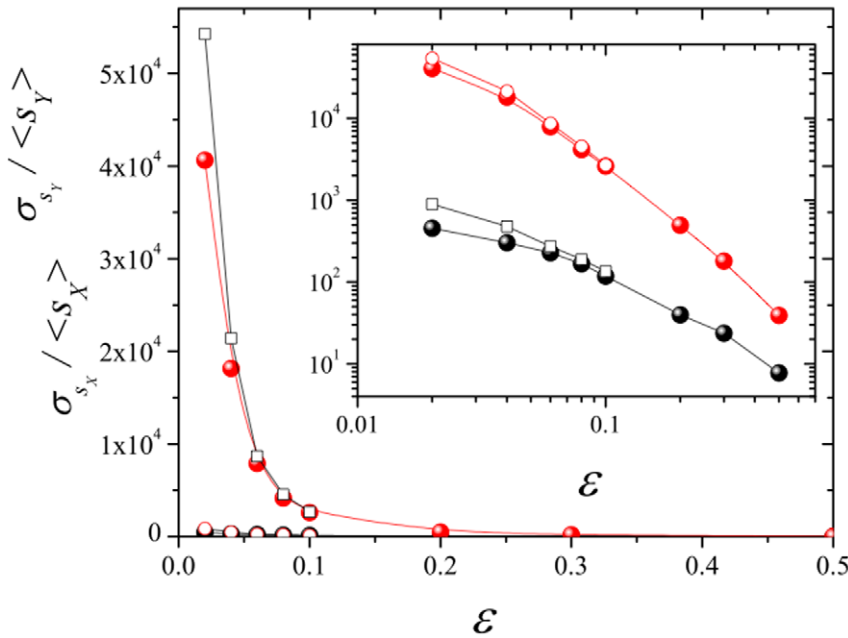
Instead, our results for spreading are in excellent agreement with the expectations for the two-dimensional voter-model [47–49] universality class (also called “compact directed percolation class” [31–33,50] [34,35,51] (see below). Also, our results for the size distribution are in excellent agreement with the two-dimensional voter class expectations,  $\tau=3/2$  and  $1/\sigma=2$ . In particular, using these theoretical values, the variance to mean ratio should scale as  $L^4$  in good agreement with the numerical finding above.

### One dimension

To further confirm the conclusion above, we have also performed studies of one-dimensional lattices, for which the expected behavior in the voter-model class is:

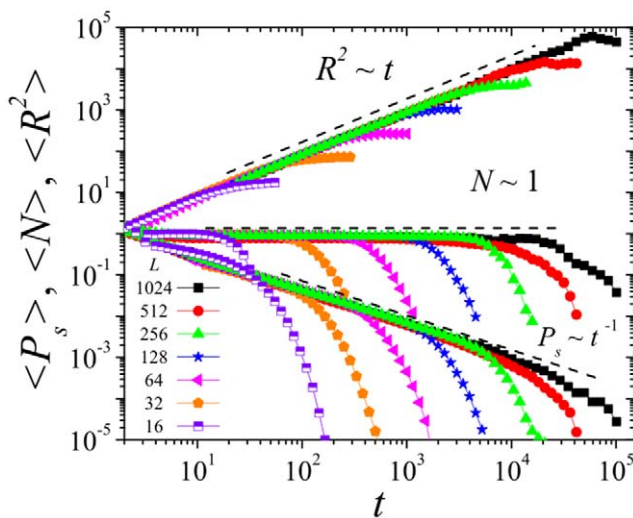
$$N(t) \sim t^0, \quad P_s(t) \sim t^{-1/2}, \quad R^2(t) \sim t, \quad (4)$$

while the avalanche size exponent is  $\tau=4/3$  [35].



**Figure 2.** Ratio of the variance ( $\sigma_s$ ) to the mean size ( $s$ ) for  $Y$  (red) and for  $X$  (black), for different values of  $\epsilon$  and system sizes  $128 \times 128$  (full symbols) and  $256 \times 256$  (empty symbols). The ratio diverges in the double limit  $\epsilon \rightarrow 0, L \rightarrow \infty$ . Inset: as the main plot, but in double logarithmic scale: as system-size increases the curves converge to straight lines, i.e. power-laws, but finite size effects are significant. doi:10.1371/journal.pone.0021946.g002

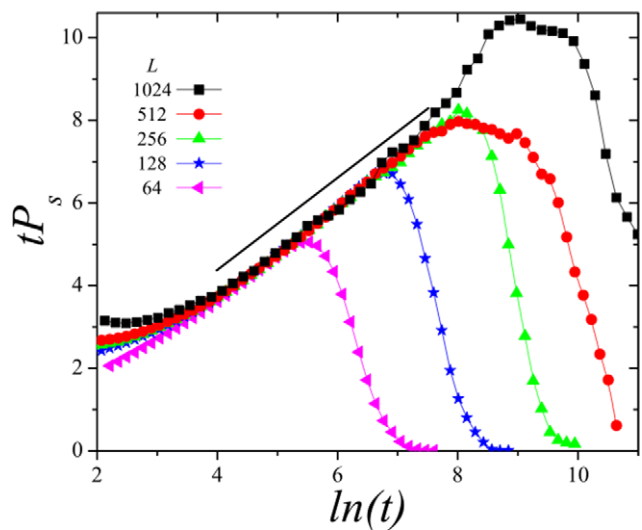
Fig. 5 shows the avalanche size distribution for various one dimensional sizes (512, 1024, 2048, 4096, and 8192) and  $\epsilon = 0$ . The measured  $\tau$  exponent,  $\tau = 1.33(3)$  is in excellent agreement with the voter-model class expectation. Fig. 6 shows the result of spreading experiments (for  $L = 1024, 2048, 4096$ , and 8192). The best fits to the slopes,  $\eta \approx 0$ ,  $\delta = 0.50(2)$ , and  $z = 1.01(1)$  are in excellent agreement with the theoretical prediction (see dashed lines in Fig. 6). These results confirm that the SJ model is in the voter-class also in one spatial dimension (and exclude one-dimensional directed percolation values  $\eta \approx 0.313$ ,  $\delta \approx 0.159$ ,  $z \approx 1.265$ ,  $\tau \approx 1.108$ ).



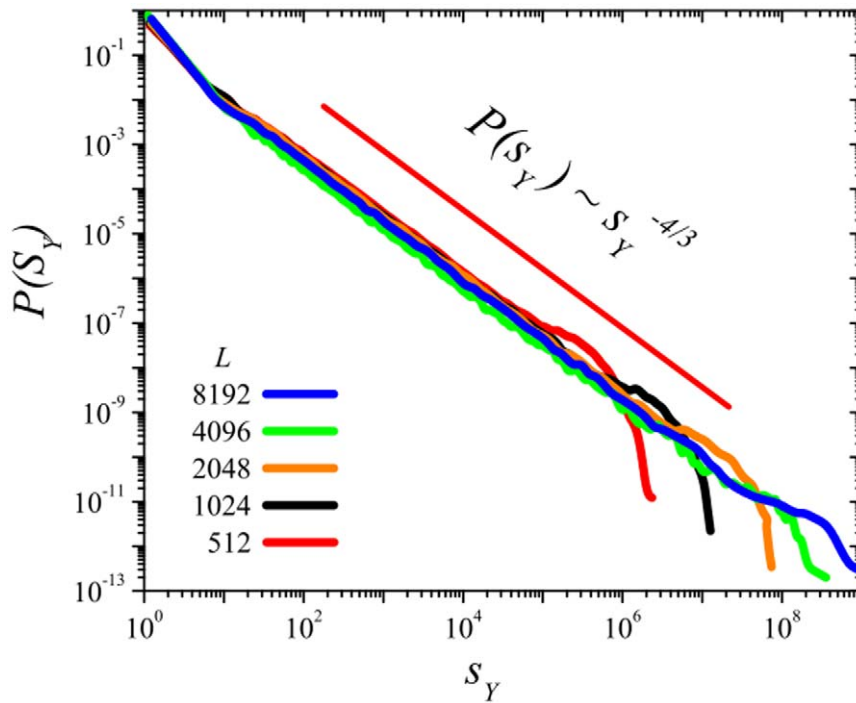
**Figure 3.** Log log plot of  $N(t)$ ,  $P_s(t)$ , and  $R^2(t)$ , for spreading experiments in different two-dimensional systems (linear sizes  $L = 32, 64, 128, 256, 512$ , and 1024) as a function of time  $t$ . Dashed lines are a guide to the eye. doi:10.1371/journal.pone.0021946.g003

### Discussion

Neutral theories date back to the sixties when Kimura introduced them in the context of population genetics [52]. Kimura assumed, as a null or neutral model, that each allele of a given gene (in haploid populations) is equally likely to enter the next generation, i.e. allele-type does not affect the prospects for survival or reproduction. Inspired in this, Hubbell proposed an analogous neutral theory of (forest) bio-diversity, in which the prospects of death and reproduction do not depend on the tree



**Figure 4.** Plot of  $tP_s(t)$  as a function of  $\ln(t)$  for different system sizes (from  $L = 64$  to  $L = 1024$ ), illustrating the presence of linear logarithmic corrections for the surviving probability in the two-dimensional SJ model. doi:10.1371/journal.pone.0021946.g004



**Figure 5. Avalanche size distribution,  $P(S_Y)$  for  $\epsilon=0$  and various one-dimensional lattices, of size  $L=512,1024,2048,4096,$  and  $8192,$  respectively.** The straight line corresponds to the theoretical prediction for the one-dimensional voter-model class. doi:10.1371/journal.pone.0021946.g005

species [53]. Both theories lead to correct predictions and also to interpretation controversies (see [54] for a recent review).

In its simplest spatially explicit version the neutral theory is as follows. Consider, for argument’s sake, the neutral theory of biodiversity with only two tree species. It can be formulated by considering the spatially explicit Moran process [55] or voter model [34,47–49]. This is defined by two symmetrical species (up/down, right/left, black/white, etc.) fully occupying a  $d$ -dimensional

lattice (or, more in general, an arbitrary network). At some rate a randomly chosen individual is removed and replaced by any of its nearest neighbors with homogeneous probability. This leads to a local tendency to create clusters of any of the two symmetrical species. Clusters of each of the two species will occupy different positions until eventually one of the two species will take over the whole (finite) space, leading to fluctuation induced monodominance in any finite system-size.

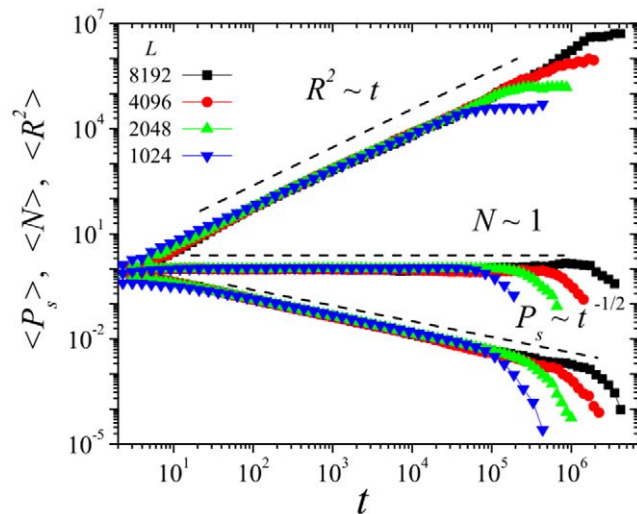
Such a type of coarsening process has been studied in depth; the asymptotic scaling of the spreading quantities,  $N(t)$ ,  $P_s(t)$ , and  $R^2(t)$  is analytically predicted to be given by Eq.(3). In particular, the logarithmic corrections for the surviving probability correspond to the fact  $d=2$  is the upper critical dimension of the voter universality class and are closely related to the marginality of the return time of two-dimensional random walkers [36,47–49]. The key ingredients of this universality class turn out to be the symmetry between the two existing absorbing (mono-dominated) states [51,56,57] and the absence of surface tension [51].

It is important to underline that, given the symmetry (neutrality) between the two species in the voter model, a given population of any of the species can either grow or decline with the same probability; i.e. there is no deterministic bias. Using the field theoretical jargon, the “mass” or “gap” term vanishes; all this is tantamount to the model being critical [58].

Indeed, for the voter model in mean-field, calling  $\phi$  the density in one of the two states (and  $1-\phi$  the complementary density for the second species), then

$$\dot{\phi} = -\phi(1-\phi) + (1-\phi)\phi = 0 \tag{5}$$

where the first term represents the loss of an individual in the first state by contact with a neighboring in the second one, and the second represents the reverse process. Therefore, the average



**Figure 6. Evolution of  $N(t)$ ,  $P_s(t)$ , and  $R^2(t)$  as a function of time  $t$  in log-log scale for spreading experiments performed on one-dimensional lattices of linear sizes  $1024,2048,4096,$  and  $8192.$**  Parameter values are  $\alpha=0.3, \gamma=0.1,$  and  $\beta=1.8.$  Results are averaged over 90000 independent realizations. doi:10.1371/journal.pone.0021946.g006

density of  $\phi$  is constant all along the system evolution. Actually, in the voter-model there is no control parameter to be tuned; the model lies, by definition at a critical point: criticality is imposed by the neutral symmetry. Instead, introducing a bias or preference towards one of the species,

$$\dot{\phi} = -(1 - \alpha)\phi(1 - \phi) + (1 + \alpha)(1 - \phi)\phi = \alpha(1 - \phi)\phi \quad (6)$$

where the constant  $\alpha > 0$  quantifies the degree of asymmetry, a non-vanishing linear or “mass” term is generated, bringing about an overall tendency for  $\phi$  to grow or to decrease depending on the sign of  $\alpha$ , i.e. deviating the system from criticality.

Introducing spatial dependence and stochasticity, Eq.(5) transforms into the Langevin equation for the voter class [51]

$$\dot{\phi}(x,t) = \nabla^2 \phi(x,t) + D\sqrt{\phi(x,t)(1 - \phi(x,t))}\eta(x,t) \quad (7)$$

where  $\eta(x,t)$  is a Gaussian white noise (observe that the equation is symmetrical under the change  $\phi \leftrightarrow (1 - \phi)$  and that there are two absorbing states at  $\phi = 0$  and  $\phi = 1$ , respectively).

Despite of the coincidence in the asymptotic scaling, let us underline that the SJ model is *not* a voter model. In the SJ model there are 5 different species and not just 2. In the case  $\epsilon = 0$ , however,  $I$  and  $Y$  are perfectly symmetrical; but as processes as  $Y + I \rightarrow Y + Y$  or  $Y + I \rightarrow I + I$  do not exist, replacement of one species by the other occurs only if mediated by  $S$  particles. It is, therefore, only at sufficiently coarse grained scales that the dynamics behaves as the voter-model; microscopically the two dynamics differ significantly.

Even if the SJ is not a voter model, the underlying reason for it to exhibit scale-invariance is that, in the limit  $\epsilon = 0$  and  $\mu = 0$  the model is “neutral” (i.e. strains  $I$  and  $Y$  are perfectly symmetrical) and, as a direct consequence, it is critical. More explicitly, calling  $Z$  the total density of infected sites which includes both  $I$  and  $Y$  then, at a mean field level, keeping  $\mu = 0$  and  $\epsilon = 0$

$$\begin{aligned} \dot{S} &= \alpha R - \beta SZ \\ \dot{Z} &= \beta SZ - \gamma Z \\ \dot{R} &= \gamma Z - \alpha R, \end{aligned} \quad (8)$$

and the steady state is  $Z_{st} = (\alpha\beta - \alpha\gamma)/(\alpha\beta + \beta\gamma)$ ,  $S_{st} = \gamma/\beta$ , and  $R_{st} = Z_{st}\gamma/\alpha$ . Let us suppose, for argument sake, that  $Z_{st} > 0$ ; i.e. that there is a non-vanishing stationary density of infected sites (otherwise epidemics would just extinguish in finite time) and that at time  $t = 0$  is  $Y(t = 0) = Y_0$ . The evolution of  $Y$  is controlled by

$$\dot{Y} = \beta S_{st} Y - \gamma Y = 0, \quad (9)$$

implying  $Y(t) = Y_0$ , i.e. there is no bias for  $Y$  to grow or decay, or

in other words, under these conditions the model is critical in what respects the  $Y$  variable. For example, in spreading experiments we fix  $Y_0 = 1/N$  (and  $I_0 = Z_{st} - 1/N$ ), implying that the total number of  $Y$ -sites is 1 on average, in agreement with the numerical findings in Fig. 3 and Fig. 6. Switching on a non-vanishing  $\epsilon$  is equivalent in the voter model to introduce a bias, inducing deviations from criticality, i.e. exponential cut-offs for the size distribution and spreading quantities, as indeed observed in our numerical simulations for  $\epsilon \neq 0$  (see, for instance, Fig. 3 and Fig. 6).

It is noteworthy that in the perfectly symmetrical (neutral) case,  $\epsilon = 0$ ,  $\mu = 0$ , the model is somehow dull: two benign strains compete in a critical way, but there is no observable consequence of this for the population under study. The interesting behavior of the SJ model comes from slight deviations from criticality, this is,  $\mu \rightarrow 0$  and  $\epsilon \rightarrow 0$  with  $\epsilon \gg \mu$ ; it is in this double limit that outbreaks leave behind an observable scale-invariant distribution of sick/dead individuals.

In summary, the Stollenwerk-Janssen model provides us with the most parsimonious explanation for the appearance of scale-invariant epidemic outbreaks caused by accidental pathogens such as the meningococcus. Our main finding is that the system is critical in the limit of vanishing pathogenicity in all dimensions, and the reason for this is that the SJ is a *neutral model*, which turns out to be critical for the very same reasons as other neutral theories in Population Genetics and Ecology are critical. As a consequence of this, the critical behavior of the model is *not* described by directed percolation as previously conjectured, but instead by the voter-model universality class, representative of neutral theories. Understanding the theory of accidental pathogens as a neutral theory gives us new insight into the origin of generic scale invariance in epidemics in particular and in propagation phenomena in general.

Similar ideas might apply to related problems as the spreading of computer viruses in the Internet or in the web of e-mail contacts: the largest overall damage is expected to occur for any type of spreading agents if their probability to cause damage is as small as possible. Being close to neutrality warrants success on the long term.

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### Author Contributions

Conceived and designed the experiments: MAM OAP. Performed the experiments: OAP MAM. Analyzed the data: OAP MAM. Contributed reagents/materials/analysis tools: OAP MAM. Wrote the paper: OAP MAM.

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