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Mass action law versus local contagion dynamics. A mean-field statistical approach with application to the theory of epidemics

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Abstract. A mean-field approach for epidemic processes with high migration is suggested by analogy with non-equilibrium statistical mechanics. For large systems a limit of the thermodynamic type is introduced for which both the total size of the system and the total number of individuals tend to infinity but the population density remains constant. In the thermodynamic limit the infection rate is proportional to the product of the proportion of individuals susceptible to infection and the average probability of infection. The limit form of the average probability of infection is insensitive to the detailed behaviour of the fluctuations of the number of infectious individuals and may belong to two universality classes: (1) if the fluctuation of the number of infectives is non-intermittent it increases with the increase of the partial density of infectives and approaches exponentially the asymptotic value one for large densities; (2) for intermittent fluctuations obeying a power-law scaling the average probability of infection also displays a saturation effect for large densities of infectives but the asymptotic value one is approached according to a power law rather than exponentially. For low densities of infectives both expressions for the average probability of infection are linear functions of the proportion of infectives and the infection rate is given by the mass-action law.

1. Introduction

The theory of epidemics is an independent branch of mathematical biology (Bailey 1975, Anderson 1982, Hadeler 1984, Becker 1989, Murray 1993). Although in the first stages of development of this field there had been almost no connections between the study of epidemics and the theoretical methods of statistical physics, the situation has changed radically during the last ten years. In this last period percolation theory (Bunde and Havlin 1991, Stauffer and Aharony 1992, Isichenko 1992) and the theory of cellular automata (Grassberger 1983, 1985, Boccara and Cheong 1992, Boccara *et al* 1994) have been applied to the study of propagation of space-dependent epidemics. In the cellular automata epidemic models, the main emphasis is on the influence of the motion of the individuals, an aspect which cannot be easily taken into account in classical treatments and is usually neglected. Only a few papers deal with the classical description of the motion in terms of partial

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differential equations (Murray *et al* 1986, Murray 1993) or of stochastic functional equations (Bartlett 1956, 1966).

The simplest space-independent description of an epidemic process can be given in terms of the Kermack–McKendrick model (1927, 1932, 1933, 1937, 1939). For a constant population size, with no births, deaths or migration, and constant probabilities of recovery and resensibilization the Kermack–McKendrick model is described in terms of the ordinary differential equations

$$\mathrm{d}S/\mathrm{d}t = -\phi(S, I, R) + \gamma R \tag{1}$$

$$\mathrm{d}I/\mathrm{d}t = \phi(S, I, R) - \alpha I \tag{2}$$

$$\mathrm{d}R/\mathrm{d}t = \alpha I - \gamma R \tag{3}$$

where S, I, R are the proportions of susceptible, infectious and recovered individuals within the population ($S + I + R = 1$), α is the rate of recovery of an infected individual, γ is the rate of resensibilization of an immune individual recovered from the sickness and the term $\phi(S, I, R)$ is the rate of infection. In the classical Kermack–McKendrick model the infection rate is assumed to have a bilinear form of the mass-action-law type

$$\phi(S, I, R) = \text{constant} \times IS. \tag{4}$$

The name mass-action law has been borrowed from chemical kinetics, a field in which rate laws of the type (4) have been used for the description of time evolution of certain chemical reactions for 130 years (Seinfeld *et al* 1989). In the biological literature it has been suggested that the validity range of the mass-action law (4) is limited to low population densities and that for large population densities the function $\phi(S, I, R)$ should display a saturation effect (Capasso and Serio 1978).

Recent studies of cellular automata epidemic models (Boccara and Cheong 1992, Boccara *et al* 1994, Schönfisch 1993, 1995) have shown that a description in terms of ordinary differential equations of the type (1)–(3) is also possible for space-dependent processes provided that the migration process is sufficiently fast. In the corresponding differential equations the infection rate $\phi(S, I, R)$ is generally strongly nonlinear and different from the mass-action-law form and displays a saturation effect similar to the one suggested by Capasso and Serio (1978). These studies show that the saturation effect of the infection rate $\phi(S, I, R)$ is due to the local nature of the interaction between healthy and infectious individuals which is limited to the immediate neighbourhoods of the susceptible individuals.

The purpose of the present paper is to point out some analogies between the statistical mechanics of irreversible processes (Kubo *et al* 1985, Grandy 1988) and the theory of space-dependent epidemics and to use them for the theoretical evaluation of the infection rate $\phi(S, I, R)$. Our approach stems from recent studies by one of the present authors (Schönfisch 1993, 1995) concerning the description of the cellular automata epidemic models by ordinary differential equations. Schönfisch (1993, 1995) has shown that for high migration the infection rate can be expressed as

$$\phi(S, I, R) = \nu S \psi(I) \tag{5}$$

where ν is a characteristic frequency and $\psi(I)$ is the probability that a susceptible individual is infected by the infective individuals from his or her immediate neighbourhood. In the following we aim to generalize this model for a continuous distribution of individuals in space. Rigorously speaking this is a very complicated many-body problem because the infection rate depends on the number and the space distribution of the infectives surrounding a healthy individual; on the other hand, this distribution is determined by the infection

rate. This feedback coupling leads to a complicated nonlinear evolution equation for a set of many-body probability densities describing the stochastic properties of the number and positions of the different individuals. Such an equation is similar to the nonlinear evolution equations used in plasma physics (Grandy 1988) or in astrophysics (Saslaw 1985). Work on the self-consistent treatment of such a many-body stochastic epidemic equation is in progress and the main results will be presented elsewhere (Vlad *et al* 1996b). The treatment presented in this paper is much simpler. Our aim is to investigate whether the above-mentioned feedback coupling is important or not in the limit of very large systems. The conclusion of our analysis is that for very large systems the expression of the infection rate is insensitive with respect to the details of the random distribution of the infectives. It is possible to identify two types of universal asymptotic behaviour which depend only on the total average number of infective individuals and not on the superior moments.

The outline of the paper is as follows. In sections 2 and 3 we give a general formulation of the problem and derive a relationship for the probability of infection $\psi(I)$ in terms of the characteristic function of the probability distribution of the number of infectives. Sections 4 and 5 deal with the two universal laws which emerge in the limit of large systems for non-intermittent and intermittent fluctuations, respectively. Finally in sections 6 and 7 some open problems and possibilities for generalizing our approach are pointed out.

2. Formulation of the problem

We consider a large population of individuals confined in a large region Σ of d_s-dimensional Euclidean space which is simply connected. As mentioned in the introduction, the total number M of individuals is constant and made up of the additive contributions M_S, M_I and M_R of susceptible, infectious and recovered individuals, respectively:

$$M = M_S + M_I + M_R. \tag{6}$$

The corresponding proportions are

$$S = M_S/M \qquad I = M_I/M \qquad R = M_R/M. \tag{7}$$

We assume that the different individuals may be placed in any position within the domain Σ and that all regions of Σ are accessible to migration. A strong migration process takes place within the system with a characteristic time scale which is at least one order of magnitude smaller than the characteristic time scales for infection, recovery and resensibilization. The analysis of the many-body stochastic description of the process (Vlad *et al* 1996b) shows that under these circumstances in the characteristic time scale of the epidemic process the individuals are uniformly and randomly distributed within the domain Σ. The probability density of the position vector of an individual

$$P(\boldsymbol{r})\,\mathrm{d}\boldsymbol{r} \qquad \text{with} \qquad \int_\Sigma P(\boldsymbol{r})\,\mathrm{d}\boldsymbol{r} = 1 \tag{8}$$

is simply given by the random uniform law

$$P(\boldsymbol{r})\,\mathrm{d}\boldsymbol{r} = \mathrm{d}\boldsymbol{r}/V_\Sigma \tag{9}$$

where

$$V_\Sigma = \int_\Sigma \mathrm{d}\boldsymbol{r} \tag{10}$$

is the size of the domain Σ (length, surface or volume). The emergence of the probability law (9) has a simple explanation. Even though the progress of the epidemic process leads to

correlations among the positions of the different individuals, the migration process, which is much faster, destroys these correlations. We can make an analogy with a chemical reaction occurring in a dense fluid (Seinfeld *et al* 1989). Although the chemical reaction eats up the more energetic molecules, destroying the equilibrium energy distribution, a local equilibrium energy distribution is restored and preserved by the non-reactive collisions, which in a dense fluid are usually more frequent than the reaction events themselves. For an epidemic process the migration of the individuals is similar to the non-reactive collisions; it plays the role of a thermostat restoring and maintaining the uniform random distribution (9) which is similar to the local equilibrium energy distribution.

Concerning the infection process, we assume that it has a local character. An infective placed at a relative position $\boldsymbol{r}$ from a healthy individual susceptible to receive the illness is characterized by a probability $p(\boldsymbol{r})$ of transmitting the infection. The infection process is limited to a relatively small neighbourhood D of a healthy individual. We have

$$p(\boldsymbol{r}) \begin{cases} \neq 0 & \text{for } \boldsymbol{r} \in D \\ = 0 & \text{for } \boldsymbol{r} \notin D. \end{cases} \tag{11}$$

The epidemic process is assumed to be translationally invariant and isotropic and thus the probability of infection $p(\boldsymbol{r})$ is a function of the absolute value $r = |\boldsymbol{r}|$ of the relative position vector $\boldsymbol{r}$ rather than of $\boldsymbol{r}$ itself:

$$p(\boldsymbol{r}) = p(r). \tag{12}$$

We denote by

$$B(M_I; t) \qquad \text{with } \sum_0^M B(M_I; t) = 1 \tag{13}$$

the probability of the number of the infectives enclosed in the large domain Σ at time t. The evaluation of this probability is a tough problem. For large systems Vlad *et al* (1996b) have reduced the determination of $B(M_I; t)$ to the solving of a Schrödinger equation in imaginary time, which by means of an eikonal (WKB-like) approximation can be reduced to a partial differential equation of the Hamilton–Jacobi type. In this paper we do not assume a concrete form for this probability. The only assumption made is that we have a summary knowledge of the nature of the fluctuations of the number of infectives for large systems. We define the characteristic function of the probability $B(M_I; t)$ as a discrete Fourier transform

$$G(b) = \sum \exp(\mathrm{i}bM_I)B(M_I; t) \tag{14}$$

where b is the Fourier variable conjugate to M_I. The cumulants $\langle\langle M_I^m \rangle\rangle$, $m = 1, 2, \ldots$ are given by a Taylor expansion of the characteristic function

$$\ln G(b) = \sum_{m=1}^{\infty} \frac{(\mathrm{i}b)^m}{m!} \langle\langle M_I^m \rangle\rangle \tag{15}$$

that is

$$\langle\langle M_I^m \rangle\rangle = (-\mathrm{i})^m \partial^m \ln G(0)/\partial b^m \qquad m = 1, 2, \ldots. \tag{16}$$

In terms of these cumulants we introduce the relative fluctuations of different orders

$$c_m = \langle\langle M_I^m \rangle\rangle / \langle\langle M_I \rangle\rangle^m \qquad m = 2, 3, \ldots. \tag{17}$$

If for large values of the average number of infectives $\langle\langle M_I \rangle\rangle = \langle M_I \rangle$, c_m tend to zero

$$c_m \to 0 \qquad \text{as } \langle M_I \rangle \to \infty \tag{18}$$

then the fluctuations have a non-intermittent behaviour. Otherwise, if as $\langle M_I \rangle \to \infty$ the relative fluctuations of different orders do not decrease to zero but tend towards constant values or diverge to infinity, then the fluctuations are intermittent. In this paper we consider both these two cases without making any particular assumptions concerning the shapes of $B(M_I; t)$, $G(b)$ or the values of the cumulants.

Since we are mainly interested in large systems made up of very large numbers of individuals, we consider a limit similar to the thermodynamic limit from statistical mechanics, i.e. that both the size V_Σ of the domain Σ and the total number of individuals M tend to infinity but the total population density $\rho = M/V$ remains constant

$$V_\Sigma, M \to \infty \qquad \text{with } \rho = M/V_\Sigma \text{ constant.} \tag{19}$$

In the following we shall be searching for possible asymptotic universal scaling laws for the probability of infection $\psi(I)$ in the thermodynamic limit (19) corresponding to non-intermittent and intermittent fluctuations, respectively.

3. Evaluation of the probability of infection

We introduce the instantaneous probability of infection $\xi(M_I; \boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I})$ of a healthy individual by a given number M_I of infectives placed at positions given by the relative displacement vectors $\boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I}$, respectively. We assume translational invariance and thus ξ is independent of the absolute position of the susceptible individual considered. We can derive the equation

$$\xi(M_I; \boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I}) = 1 - \prod_{l=1}^{M_I} [1 - p(r_l)] \tag{20}$$

which expresses the fact that at least one of the M_I infectives is enough for triggering the infection process. The M_I individuals have the probabilities $1 - p(r_l)$ of not spreading the infection and thus the probability that none of them trigger the infection is $\prod(1 - p(r_l))$; the complementary probability $1 - \prod(1 - p(r_l))$ equals $\xi(M_I; \boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I})$.

The probability of infection $\psi(I)$ can be expressed as an average of $\xi(M_I; \boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I})$ over the coordinates $\boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I}$ and over the total number of infectives M_I:

$$\psi(I) = \sum_{M_I=0}^{M} B(M_I) \int_\Sigma \cdots \int_\Sigma P(\boldsymbol{r}_1)\, \mathrm{d}\boldsymbol{r}_1 \ldots P(\boldsymbol{r}_{M_I})\, \mathrm{d}\boldsymbol{r}_{M_I} \xi(M_I; \boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I}). \tag{21}$$

Expressing the probability $\xi(M_I, \boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I})$ by means of equation (20), using the normalization conditions of $B(M_I)$ and $P(\boldsymbol{r})$ and taking into account that the individual probability of infection $p(r)$ vanishes outside the neighbourhood D we can express the probability of infection $\psi(I)$ in terms of the characteristic function $G(b)$ given by equation (14)

$$\begin{aligned} \psi(I) &= 1 - \sum_{M_I=0}^{M} B(M_I) \left[1 - \int_D p(r) P(\boldsymbol{r})\, \mathrm{d}\boldsymbol{r} \right]^{M_I} \\ &= 1 - G\left(b = -\mathrm{i} \ln \left[1 - \int_D p(r) P(\boldsymbol{r})\, \mathrm{d}\boldsymbol{r} \right] \right) \end{aligned} \tag{22}$$

from which, by using the expression (9) for $P(\boldsymbol{r})$ we come to

$$\psi(I) = 1 - G[b = -\mathrm{i} \ln(1 - V^*/V_\Sigma)] \tag{23}$$

where

$$V^* = \int_D p(r)\,\mathrm{d}\boldsymbol{r} \leqslant \int_D \mathrm{d}\boldsymbol{r} = V_D \tag{24}$$

is an effective 'epidemic' volume of the neighbourhood D which is at most equal to the corresponding geometrical volume V_D. V^* expresses the local character of the infection process; in the definition (24) of V^* different contributions are ascribed to the different regions of the neighbourhood: the bigger the probability of contagion $p(r)$ the bigger the contribution to V^*. Note that the local character of the infection process is conserved even in the thermodynamic limit (19). Indeed, according to equation (24) the effective epidemic volume V^* depends only on the probability $p(r)$ and on the size and shape of the neighbourhood D but it is independent of the total volume V_Σ of the system.

Equation (23) is general; it is valid for any probability distribution $P(M_I)$ of the number of infected individuals and for any domain Σ, small or large. In the following two sections we shall apply it in the thermodynamic limit (19) for non-intermittent and intermittent fluctuations, respectively.

4. Universal behaviour for non-intermittent fluctuations

By combining equations (15) and (17) and taking into account that

$$\langle\langle M_I \rangle\rangle = \langle M_I \rangle = \langle I \rangle M = \langle I \rangle \rho V_\Sigma \tag{25}$$

where $\langle I \rangle$ is the average proportion of infectives we can express the characteristic function $G(b)$ in the following form,

$$G(b) = \exp\left\{ \sum_{m=1}^{\infty} \frac{(\mathrm{i}b)^m}{m!} c_m (\rho V_\Sigma \langle I \rangle)^m \right\} \tag{26}$$

and thus expression (22) for the probability of infection $\psi(I)$ becomes

$$\psi(I) = 1 - \exp\left\{ \sum_{m=1}^{\infty} \frac{c_m}{m!} \rho^m \langle I \rangle^m [V_\Sigma \ln(1 - V^*/V_\Sigma)]^m \right\} \tag{27}$$

where $c_1 = 1$ and c_m, $m = 2, 3, \ldots$, are given by equation (17). Here and in the following we deal only with the average value $\langle I \rangle$ of the proportion of the infectives, and thus, we use the simplified notation I for $\langle I \rangle$.

By assuming that the fluctuations of the number of infectives are non-intermittent in the thermodynamic limit (equation (18)) in equation (27) for $V_\Sigma \to \infty$ the contributions of all cumulants with the exception of the first one tend to zero and the expression of the probability of infection $\psi(I)$ becomes

$$\psi(I) \sim 1 - \exp(-I\rho V^*) \qquad \text{as } V_\Sigma \to \infty. \tag{28}$$

This is the sought-for universal scaling law for non-intermittent fluctuations. Although not stated explicitly, a similar scaling law is implicitly included in a system of difference equations analogous to the SIR model (1)–(3) derived by Boccara and Cheong (1992) in the case of an infinite range interaction epidemic cellular automaton. There are, however, two main differences between their equation and our universal scaling law (28): the Boccara and Cheong's equation is derived for a particular model whereas equation (28) holds in the thermodynamic limit for any system with high migration and non-intermittent fluctuations; besides, their proportionality coefficient in the exponent is different from ours.

5. Universal behaviour for intermittent fluctuations

We start out with the particular case of marginal intermittent fluctuations for which all relative fluctuations c_m tend towards finite values different from zero in the thermodynamic limit:

$$c_m \to \text{finite} \neq 0 \qquad \text{for } M_I \to \infty. \tag{29}$$

In this case equation (27) leads to

$$\psi(I) = 1 - \exp\left\{ \sum_{m=1}^{\infty} \frac{(-1)^m c_m}{m!} (\rho V^*)^m I^m \right\}. \tag{30}$$

This is, however, a non-generic situation which is hardly encountered in the real world. An expansion such as the series in the exponent of equation (30) is usually divergent and thus the relationship (30) is useless; such divergences can be, however, removed through resummation by applying a suitable renormalization procedure.

A possible choice is the use of a stochastic version of the Shlesinger–Hughes (1981) renormalization technique which has been recently suggested by one of the present authors (Vlad 1994) in the context of random point processes. We consider that a probability distribution $\tilde{B}(M_I)$ displaying an intermittent behaviour can be generated starting from a non-intermittent probability distribution $B(M_I)$ through a succession of random decimation processes. Following Vlad (1994), this succession of decimation processes is characterized by two constant probabilities: the probability λ that a decimation process takes place and the probability β that during a step an infectious individual is decimated. The decimation does not represent the actual removal of an individual from the system but merely a way of extracting the essential information about the population fluctuations.

For describing the decimation process we introduce the probability $B^{(q)}(M_I)$ of the number of infectives after q decimation steps and the probability

$$\chi_q = (1 - \lambda)\lambda^q \tag{31}$$

that there are q decimation steps. The final renormalized probability $\tilde{B}(M_I)$ resulting after the application of a random number of decimation steps described by the probability law (31) is

$$\tilde{B}(M_I) = \sum \chi_q B^{(q)}(M_I). \tag{32}$$

As the decimation processes are assumed to act in a random way and each infectious individual has the same probability β of being removed, the conditional probability $B^{(q)}(M_{Iq}|M_{I(q-1)})$ that at the qth step the number of remaining individuals is M_{Iq}, provided that at the $(q-1)$th step this number was $M_{I(q-1)}$, is given by a binomial

$$B^{(q)}(M_{Iq}|M_{I(q-1)}) = \frac{M_{I(q-1)}!}{M_{Iq}!(M_{I(q-1)} - M_{Iq})!} (\beta)^{M_{I(q-1)} - M_{Iq}} (1 - \beta)^{M_{Iq}}. \tag{33}$$

We have

$$B^{(q)}(M_{Iq}) = \sum_{M_{I(q-1)}} B^{(q)}(M_{Iq}|M_{I(q-1)}) B^{(q-1)}(M_{I(q-1)}) \tag{34}$$

with

$$B^{(0)}(M_{I0}) = B(M_{I0}) \tag{35}$$

where $B(M_I)$ is the initial probability distribution. No specific assumptions concerning $B(M_I)$ are made; the only assumption is that the relative fluctuations corresponding to $B(M_I)$ tend to zero in the thermodynamic limit.

Now we introduce the characteristic functions of the probabilities $B^{(q)}(M_I)$ attached to the different decimation steps

$$G^{(q)}(b) = \sum_{M_I} \exp(\mathrm{i}M_I b) B^{(q)}(M_I) \tag{36}$$

and the characteristic function of the renormalized probability $\tilde{B}(M_I)$:

$$\tilde{G}(b) = \sum_{M_I} \exp(\mathrm{i}M_I b) \tilde{B}(M_I) = \sum_{q=0}^{\infty} \chi_q G^{(q)}(b). \tag{37}$$

After some standard but lengthy computations from equations (31)–(37) we get the following expressions for $G^{(q)}(b)$ and $\tilde{G}(b)$,

$$G^{(q)}(b) = G[-\mathrm{i}\ln[1-(1-\exp(\mathrm{i}b))(1-\beta)^q]] \tag{38}$$

$$\tilde{G}(b) = (1-\lambda)\sum_{q=0}^{\infty} \lambda^q G[-\mathrm{i}\ln[1-(1-\exp(\mathrm{i}b))(1-\beta)^q]] \tag{39}$$

where the characteristic function of the initial non-intermittent probability $B(M_I)$ is given by equation (14). Equation (39) can be written in the self-similar form

$$\tilde{G}(b) = (1-\lambda)G(b) + \lambda\tilde{G}[-\mathrm{i}\ln[1-(1-\exp(\mathrm{i}b))(1-\beta)]]. \tag{40}$$

Equation (40) has a structure typical for a renormalization group (RG) equation; its form suggests the existence of a power-law scaling in $1-\exp(\mathrm{i}b)$ for $b \to 0$. Indeed, by using the Poisson summation technique (Titchmarsh 1948, Vlad 1994) the renormalized characteristic function $\tilde{G}(b)$ can be expressed as

$$\tilde{G}(b) = \tfrac{1}{2}(1-\lambda)G(b) + (1-\exp(\mathrm{i}b))^{-H}\,\Xi(\exp(\mathrm{i}b), \ln(1-\exp(\mathrm{i}b))) \tag{41}$$

where

$$\begin{aligned}\Xi(\alpha, c) = \frac{1-\lambda}{-\ln(1-\beta)}\Bigg\{ &\int_\alpha^1 (1-x)^{H-1} G(-\mathrm{i}\ln x)\,\mathrm{d}x + 2\sum_{l=1}^{\infty}\Bigg[\cos\left(\frac{2\pi l c}{-\ln(1-\beta)}\right) \\ &\times \int_\alpha^1 (1-x)^{H-1} G(-\mathrm{i}\ln x)\cos\left(\frac{2\pi l \ln(1-x)}{-\ln(1-\beta)}\right)\mathrm{d}x + \sin\left(\frac{2\pi l c}{-\ln(1-\beta)}\right) \\ &\times \int_\alpha^1 (1-x)^{H-1} G(-\mathrm{i}\ln x)\sin\left(\frac{2\pi l \ln(1-x)}{-\ln(1-\beta)}\right)\mathrm{d}x\Bigg]\Bigg\}\end{aligned} \tag{42}$$

where

$$H = \ln\lambda/\ln(1-\beta) \tag{43}$$

is a positive fractal exponent. In equation (42) and in the following we assume that the Fourier variable is imaginary

$$b = \mathrm{i}g \qquad \text{with } g = |b| \geqslant 0 \tag{44}$$

and thus the integrals over x are in fact real rather than complex. The scaling law (41) displays logarithmic oscillations in $\ln(1-\exp(\mathrm{i}b))$ which are due to the discrete nature of the decimation process. To avoid the complications generated by these logarithmic oscillations we consider the limit

$$\lambda \nearrow 1, \beta \searrow 0 \qquad \text{with } H = \ln\lambda/\ln(1-\beta) = \text{constant}. \tag{45}$$

This type of limit has been recently introduced in the context of stochastic renormalization (Vlad 1993); although it leads to the vanishing of the logarithmic oscillations, the scaling

behaviour characterized by the fractal exponent H is still present. In the limit (45) the RG equation (40) becomes an ordinary differential equation

$$-\mathrm{i}[1-\exp(\mathrm{i}b)]\exp(-\mathrm{i}b)\partial\tilde{G}(b)/\partial b = H[\tilde{G}(b)-G(b)] \qquad \text{with } \tilde{G}(0)=1 \tag{46}$$

where the initial condition results from the normalization condition of $B(M_I)$, $\sum B(M_I) = 1$. By integrating equation (46) we come to

$$\tilde{G}(b) = H[1-\exp(\mathrm{i}b)]^{-H}\int_{\exp(\mathrm{i}b)}^{1}(1-x)^{H-1}G(-\mathrm{i}\ln x)\,\mathrm{d}x. \tag{47}$$

We introduce the factorial moments

$$F_m = \sum_{M_I} M_I(M_I-1)\ldots(M_I-m+1)B(M_I) \tag{48}$$

$$\tilde{F}_m = \sum_{M_I} M_I(M_I-1)\ldots(M_I-m+1)\tilde{B}(M_I) \tag{49}$$

of the number of infectious individuals described by the initial and renormalized probabilities $B(M_I)$ and $\tilde{B}(M_I)$, respectively. From the definitions (14) and (37) of the characteristic functions $G(b)$ and $\tilde{G}(b)$ we obtain

$$F_m = \partial^m G(b=0)/\partial[\exp(\mathrm{i}b)]^m \tag{50a}$$

$$\tilde{F}_m = \partial^m \tilde{G}(b=0)/\partial[\exp(\mathrm{i}b)]^m. \tag{50b}$$

From equation (50*a*) it follows that the initial characteristic function $G(b)$ can be expressed in a Taylor series of the form

$$G(b) = 1+\sum_{m=1}^{\infty}\frac{F_m}{m!}[\exp(\mathrm{i}b)-1]^m. \tag{51}$$

By inserting equation (51) into equation (47), evaluating the integral over x and differentiating the resulting equations m times with respect to b we get the following relations between the renormalized and non-renormalized factorial moments:

$$\tilde{F}_m = F_m H/(H+m). \tag{52}$$

The hyperbolic dependence on m of the coefficient of F_m leads to intermittency. Indeed, by using the relations between the factorial moments and cumulants (Van Kampen 1992) we obtain

$$\langle\langle\tilde{M}_I\rangle\rangle = \frac{H}{H+1}\langle\langle M_I\rangle\rangle \tag{53}$$

$$\langle\langle\tilde{M}_I^2\rangle\rangle = \frac{c_2(H+1)^2+1}{H(H+2)}\langle\langle\tilde{M}_I\rangle\rangle^2 + \frac{\langle\langle\tilde{M}_I\rangle\rangle}{H+2} \qquad \text{etc.} \tag{54}$$

From equations (53) and (54) it follows that the renormalized relative fluctuation of order two

$$\tilde{c}_2 = \langle\langle\tilde{M}_I^2\rangle\rangle/\langle\langle\tilde{M}_I\rangle\rangle^2 = \frac{c_2(H+1)^2+1}{H(H+2)} + \frac{1}{\langle\langle\tilde{M}_I\rangle\rangle(H+2)} \tag{55}$$

does not decrease to zero in the thermodynamic limit but tends towards a positive value

$$\tilde{c}_2 \to [H(H+2)]^{-1} \qquad \text{as } \langle\langle\tilde{M}_I\rangle\rangle \to \infty, M\to\infty \tag{56}$$

which shows that the population fluctuations are intermittent.

For evaluating the behaviour of the probability of infection $\psi(I)$ in the thermodynamic limit we should express the renormalized characteristic function $\tilde{G}(b)$ in terms of the non-renormalized relative fluctuations c_m given by equation (17). By using the integration variable

$$z = (1 - x)/[1 - \exp(\mathrm{i}b)] \tag{57}$$

and making use of equations (15), (17) and (53), equation (47) becomes

$$\tilde{G}(b) = H \int_0^1 z^{H-1}\,\mathrm{d}z\; G[-\mathrm{i}\ln[1 - z(1 - \exp(\mathrm{i}b))]] \tag{58}$$

where

$$G(b) = \exp\left\{\sum_{m=1}^{\infty} \frac{(\mathrm{i}b)^m}{m!} c_m[\rho(1 + 1/H)V_\Sigma I]^m\right\}. \tag{59}$$

By inserting equations (58) and (59) into the general expression (23), where $G(b)$ is replaced by the corresponding renormalized function $\tilde{G}(b)$, we obtain the following relationship for the infection probability $\psi(I)$:

$$\psi(I) = 1 - H \int_0^1 z^{H-1} \exp\left\{\sum_{m=1}^{\infty} \frac{c_m}{m!}[\rho(1 + 1/H)]^m I^m [V_\Sigma \ln(1 - zV^*/V_\Sigma)]^m\right\} \mathrm{d}z. \tag{60}$$

In the thermodynamic limit all c_m-dependent terms with the exception of the first one vanish, resulting in the sought-for universal law for intermittent fluctuations

$$\begin{aligned}\psi(I) &= 1 - \int_0^1 Hz^{H-1} \exp\{-\rho(1 + 1/H)V^* I z\}\,\mathrm{d}z \\ &= 1 - H[\rho(1 + 1/H)V^* I]^{-H}\gamma[H, \rho(1 + 1/H)V^* I]\end{aligned} \tag{61}$$

where

$$\gamma(a, x) = \int_0^x t^{a-1}\exp(-t)\,\mathrm{d}t \qquad a > 0, x \geqslant 0 \tag{62}$$

is the incomplete gamma function.

6. Discussion

The relationship between the two universal laws is simple: the expression (28) for non-intermittent fluctuations is a particular case of equation (61) corresponding to the limit $H \to \infty$. Indeed, by using the properties of the incomplete gamma function it is easy to show that

$$\begin{aligned}\psi(I) = 1 - H[\rho(1 + 1/H)V^* I]^{-H}\gamma[H, \rho(1 + 1/H)V^* I] \to 1 - \exp(-I\rho V^*) \\ \text{as } H \to \infty.\end{aligned} \tag{63}$$

As the fractal exponent H becomes larger and larger, in the thermodynamic limit the intermittent character of fluctuations becomes smaller and smaller, and in the limit $H \to \infty$ it vanishes completely; in this limit the renormalized functions become identical to the non-renormalized ones.

For low total population densities, both functions $\psi(I)$ increase linearly with the fraction of infectives

$$\psi(I) \to kI \qquad \text{as } \rho \to 0 \tag{64}$$

where

$$k = \rho V^*. \tag{65}$$

In the other extreme of large total population densities, $\rho \to \infty$, both functions $\psi(I)$ tend to unity

$$\psi(I) \to 1 \qquad \text{as } \rho \to \infty \tag{66}$$

but the rate of approach to the limit value 1 is different. For non-intermittent fluctuations the difference between the asymptotic value 1 and the current value of the probability of infection $\psi(I)$ decreases exponentially with the total population density

$$1 - \psi(I) = \exp(-\rho V^* I) \tag{67}$$

whereas for intermittent fluctuations the decrease of the same difference is much slower, being given by an inverse power law

$$1 - \psi(I) \sim \Gamma(H+1)[\rho(1+1/H)V^* I]^{-H} \qquad \text{as } \rho \to \infty, \Gamma(H+1) = \gamma(H+1, \infty). \tag{68}$$

The conclusion of this analysis is that the intermittency of fluctuations decreases the efficiency of the saturation effect due to the local nature of the infection process. It is easy to check that to reach the same value of the infection probability $\psi(I)$ close to unity for intermittent fluctuations the total population density should be bigger than for non-intermittent fluctuations due to the fact that the exponential (67) decreases faster than the inverse power law (68). The intensity of this effect increases with the decrease of the value of the fractal exponent H. The reciprocal value of the exponent H

$$\mathcal{F} = 1/H \tag{69}$$

is a measure of the degree of intermittency of population fluctuations; for values of H close to zero the intermittency is very strong, whereas for $H \to \infty$ we recover the non-intermittent behaviour.

Concerning the validity range of the limit expressions (28) and (61) for the probability of infection $\psi(I)$, we expect that the scaling law (28) for non-intermittent fluctuations is valid in the thermodynamic limit for any values of the total population density ρ and of the proportion of infectives I. In contrast, the intermittent scaling law (61), being based on a renormalization approach is strictly valid only for those regions where a power-law scaling occurs, that is, for low and for large population densities (equations (64), (65) and (68)). The expression (61) for intermediate values of ρ and I depending on the incomplete gamma function, although mathematically properly defined, is only an extrapolation law between low and high population densities without a deep physical significance.

Our approach does not specify under what circumstances the population fluctuations are non-intermittent or intermittent. An answer to this question can be given only by using a many-body approach (Vlad *et al* 1996b). For illustration, in appendix A and appendix B we have checked the character of fluctuations for a number of probability distributions. The class of non-intermittent probability distributions includes the Poissonian and the positive binomial laws,

$$B(M_I) = (M_I!)^{-1}\langle M_I\rangle^{M_I} \exp(-\langle M_I\rangle) \tag{70}$$

$$B(M_I) = \frac{M!}{M_I!(M-M_I)!}\varepsilon^{M_I}(1-\varepsilon)^{M-M_I} \tag{71}$$

where ε is the probability of occurrence of an infective and the other symbols have the same significance as before.

In the many-body approach (Vlad *et al* 1996b) the probability $B(M_I)$ is evaluated by making use of the Kubo extensivity ansatz (Kubo *et al* 1973, Kitahara 1975, Vlad and Ross 1994)

$$B(M_I) \sim \text{constant} \times \exp(M\mathcal{J}(I)) \tag{72}$$

where

$$\mathcal{J}(I) \sim \mathrm{O}(M^0) \tag{73}$$

is an epidemic action similar to the action function from classical analytical mechanics depending on the proportion I of the infectives but which is independent of the total population size M. In appendix B we show that the fluctuations corresponding to equation (72) are also non-intermittent (see also Vlad *et al* 1994).

An intermittent probability law investigated in appendix A is the negative binomial

$$B(M_I) = \frac{\Gamma(H + M_I)}{\Gamma(H)M_I!} \frac{\langle M_I \rangle^{M_I} H^H}{(H + \langle M_I \rangle)^{M_I + H}} \tag{74}$$

where

$$\Gamma(x) = \int_0^\infty t^{x-1} \exp(-t)\, \mathrm{d}t \qquad x > 0 \tag{75}$$

is the complete gamma function and H is a positive fractal exponent similar to the one entering equation (61). The negative binomial law (74) has a long and distinguished history in the empirical description of the statistics of infectious diseases as well as of other contamination phenomena for which the probability of a further event increases whenever an event has occurred, for instance, for fire and sickness insurance (Bliss and Fisher 1953, Boswell and Patil 1970 and references therein, Beard *et al* 1977). Recently the negative binomial has been applied for describing a similar kind of contagion phenomenon in particle and nuclear physics for the study of multiplicity distributions in hadronic interactions and nuclear fragmentation cascades (Giovannini and Van Hove 1986, Van Hove 1987, Cugnon 1987, Carruthers and Shih 1987, Suzuki and Biyajima 1988, Chaudhuri 1992). A further recent application is the study of galaxy clustering in the large scale structure of the universe (Carruthers 1991).

For a negative binomial in the thermodynamic limit the relative fluctuations c_m tend towards the positive values

$$c_m \to H^{-(m-1)}(m-1)!\$_m^{(m)} \neq 0 \qquad \text{for finite } H, \langle M_I \rangle \to \infty \tag{76}$$

where

$$\$_m^{(l)} = \sum_{k=0}^{l} \frac{(-1)^{l-k} k^m}{k!(l-k)!} \tag{77}$$

are the Stirling numbers of the second kind. From equation (76) we note that the intermittency is present for any finite value of the fractal exponent H. The non-intermittent behaviour emerges for $H \to \infty$, a situation in which the negative binomial (74) tends towards the Poissonian law (70).

In appendix C we show that for a negative binomial distribution of the number of infectives the probability of infection $\psi(I)$ is given by

$$\psi(I) = 1 - [H/(H + \rho V^* I)]^H. \tag{78}$$

Equation (78) is valid for any values of the total volume V_Σ of the system, whether small or large. As expected, for small or large values of the total population density ρ the probability

of infection $\psi(I)$ given by equation (78) has the same scaling behaviour as the universal scaling law (61) for intermittent fluctuations:

$$\psi(I) \to \rho V^* I \qquad \text{as } \rho \to 0 \tag{79}$$

$$\psi(I) \to 1 - H^H/(\rho V^* I)^H \qquad \text{as } \rho \to \infty \tag{80}$$

(compare with equations (64) and (68)). The behaviour for intermediate population densities is different for the two equations (61) and (78). This fact is consistent with the above-mentioned observation that the intermittent scaling law (61) is universal only as $\rho \to 0$ or $\rho \to \infty$.

The results presented in this paper may have some further biological as well as physical implications. We note first that the present analysis is consistent with the many-body approach (Vlad *et al* 1996b) only for non-intermittent fluctuations. In the present stage of development of the many-body theory the consistency with the intermittent scaling behaviour for $\psi(I)$ found here (equation (61)) cannot be tested because the many-body approach is based on the Kubo extensivity ansatz (72) which is valid only for non-intermittent fluctuations. The statistical description of the empirical epidemic data in terms of the negative binomial (74) suggests, however, that in some cases the intermittent behaviour may occur in the spreading of an infectious disease. For a detailed description of the intermittent behaviour in terms of the many-body theory a renormalized approach of the stochastic evolution equations for the grand canonical number-position probability densities should be developed. At the present stage of research the finalization of such a project is rather uncertain.

The mean field approach suggested here can be easily extended to include the correlated behaviour for the population of infectives. The main steps of such an approach for the non-intermittent case are presented in appendix D. For translationally invariant systems the expression for the infection probability $\psi(I)$ has the following form,

$$\psi(I) = 1 - \exp\{-\rho V^* I + \mathcal{M}[g]\} \tag{81}$$

where $\mathcal{M}[g]$ is a linear functional depending on the correlation functions $g = (g_m(\boldsymbol{r}_1, \ldots, \boldsymbol{r}_m))$ of different orders $m = 2, 3, \ldots$ of the positions of infectives in clusters of different sizes $m = 2, 3, \ldots, \mathcal{M}[g]$ is given by a series expansion similar to the virial expansion in equilibrium statistical mechanics. For intermittent fluctuations the functional $\mathcal{M}[g]$ diverges. Further research should lead to the development of a suitable renormalization technique for the resummation of $\mathcal{M}[g]$.

Concerning the possible physical applications of our approach, we mention only two. There is a formal analogy between the mechanism of contamination suggested in this paper and the Klafter–Shlesinger (1986) generalization of the Förster model of non-exponential relaxation (Förster 1949, Klafter and Shlesinger 1986, Vlad and Mackey 1995a). The main idea is to search for different types of universal scaling behaviour for non-exponential relaxation in the thermodynamic limit by using the method presented in this paper. Such an approach will be presented elsewhere (Vlad *et al* 1996c, Vlad *et al* 1996a). A second possible application is related to the study of the cage effect in liquid state chemical kinetics (Seinfeld *et al* 1989) based on an analogy between the neighbourhood D of a susceptible individual and the cage of solvent.

7. Conclusions

In this paper a mean field approach in continuous space and time has been suggested for the description of the saturation effect generated by the short-range, local character of the

infection processes in the spreading of an epidemic in a population with high migration. The method is based on the use of a continuous analogue of the neighbourhood of a susceptible in cellular automata epidemic models. In the thermodynamic limit two universal scaling laws have been identified, corresponding to non-intermittent and intermittent fluctuations, respectively. For low population densities both expressions lead to the mass-action law. For high densities the infection probabilities tend towards unity with different rates in the two cases: exponentially for non-intermittent fluctuations and according to an inverse power law for intermittent fluctuations.

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Appendix A

For a Poissonian distribution of the number of infectives all cumulants are equal to the mean value (Van Kampen 1992). This can be checked by computing the characteristic function

$$G(b) = \exp[\langle M_I\rangle(\exp(\mathrm{i}b) - 1)] \tag{A.1}$$

from which, by applying equation (6) we come to

$$\langle\langle M_I^m\rangle\rangle = \langle M_I\rangle \qquad m = 1, 2.\ldots. \tag{A.2}$$

The relative fluctuations c_m corresponding to equation (A.2) tend to zero as $\langle M_I\rangle \to \infty$,

$$c_m = \langle M_I\rangle^{-(m-1)} \to 0 \qquad \text{as } \langle M_I\rangle \to \infty \tag{A.3}$$

and thus for a Poissonian distribution the fluctuations are non-intermittent.

Similarly for the binomial law (71) the characteristic function $G(b)$ is

$$\begin{aligned} G(b) &= [\varepsilon\exp(\mathrm{i}b) + 1 - \varepsilon]^M = \exp\{M\ln[\varepsilon(\exp(\mathrm{i}b) - 1) + 1]\} \\ &= \exp\left\{M\sum_{l=1}^{\infty}\frac{(-1)^{l+1}}{l}(\exp(\mathrm{i}b) - 1)^l\varepsilon^l\right\}. \end{aligned} \tag{A.4}$$

By expressing the terms $(\exp(\mathrm{i}b) - 1)^l$ by means of the binomial formula and expanding the exponentials $\exp(\mathrm{i}bq)$, $q = 1, 2, \ldots, l$, in the resulting expressions in Taylor series after some algebraic manipulations we obtain

$$G(b) = \exp\left\{M\sum_{m=1}^{\infty}\frac{(\mathrm{i}b)^m}{m!}\sum_{l=1}^{m}(-1)^{l+1}\varepsilon^l(l-1)!\$_m^{(l)}\right\} \tag{A.5}$$

where $\$_m^{(l)}$ are the Stirling numbers of second kind defined by equation (77). The cumulants corresponding to the expansion (A.5) are

$$\langle\langle M_I^m\rangle\rangle = M\sum_{l=1}^{m}(-1)^{l+1}\varepsilon^l(l-1)!\$_m^{(l)}. \tag{A.6}$$

In particular, for $m = 1$ we obtain

$$\langle M_I\rangle = \langle\langle M_I\rangle\rangle = M\varepsilon. \tag{A.7}$$

By eliminating the total number of individuals M from equations (A.6) and (A.7) and computing the relative fluctuations c_m we obtain

$$c_m = \langle M_I \rangle^{-(m-1)} \left(\sum_{l=1}^{m} (-1)^{l+1} \varepsilon^{l-1} (l-1)! \$_m^{(l)} \right) \to 0 \qquad \text{as } \langle M_I \rangle \to \infty \tag{A.8}$$

and thus for the positive binomial (71) the fluctuations are also non-intermittent.

For the negative binomial (74) the characteristic function $G(b)$ can be computed by inserting equation (74) into the definition (14) and by summing the resulting binomial series

$$\begin{aligned} G(b) &= \exp\{-H \ln[1 + (1 - \exp(\mathrm{i}b))\langle M_I \rangle / H]\} \\ &= \exp\left\{ H \sum_{l=1}^{\infty} (\langle M_I \rangle / H)^l (\exp(\mathrm{i}b) - 1)^l / l \right\}. \end{aligned} \tag{A.9}$$

Now we can apply the same steps as for the case of the positive binomial, that is, to use the Newton formula for $(\exp(\mathrm{i}b) - 1)^l$ and to expand the exponentials $\exp(\mathrm{i}bq)$, $q = 1, \ldots, l$, in the resulting equations in Taylor series. In the end we obtain the following expressions for the cumulants $\langle\langle M_I^m \rangle\rangle$ and for the relative fluctuations c_m,

$$\langle\langle M_I^m \rangle\rangle = H \sum_{l=1}^{m} (l-1)! (\langle M_I \rangle / H)^l \$_m^{(l)} \tag{A.10}$$

$$c_m = \sum_{l=1}^{m} (l-1)! H^{-(l-1)} \langle M_I \rangle^{-(m-l)} \$_m^{(l)} \to H^{-(m-1)} (m-1)! \$_m^{(m)} \qquad \text{as } \langle M_I \rangle \to \infty \tag{A.11}$$

and thus for finite H the fluctuations described by the negative binomial law (74) are intermittent.

Appendix B

By using the Kubo extensivity ansatz (72) and (73), the expression (14) for the characteristic function $G(b)$ becomes

$$G(b) \sim \text{constant} \sum_{M_I} \exp\{M(\mathrm{i}bI + \mathcal{J}(I))\} \qquad \text{as } M \to \infty. \tag{B.1}$$

As $M \to \infty$ we evaluate the sum in equation (B.1) by applying the method of steepest descent. We come to

$$G(b) \sim \exp\{M(\varphi(\mathrm{i}b) + \mathrm{O}(M^{-1}))\} \qquad M \to \infty \tag{B.2}$$

where

$$\varphi(\mathrm{i}b) = \mathrm{i}b\eta(\mathrm{i}b) + \mathcal{J}(\eta(\mathrm{i}b)) \tag{B.3}$$

and

$$\eta(x) = [-\partial \mathcal{J} / \partial I]^{(-1)} \tag{B.4}$$

with

$$-\partial \mathcal{J}(\eta(x)) / \partial \eta(x) = x \qquad x = \mathrm{i}b \tag{B.5}$$

is the inverse function of $-\partial\mathcal{J}(I)/\partial I$. We assume that $\mathcal{J}(I)$ has only one maximum for $I = I^*$, which corresponds to the deterministic proportion of infectives and that its derivative is a decreasing function for any positive value of I,

$$\partial\mathcal{J}(I^*)/\partial I = 0 \tag{B.6}$$

$$\partial^2\mathcal{J}(I)/\partial I^2 < 0. \tag{B.7}$$

Due to the condition (B.7) according to the theorem of existence of implicit functions $\eta(x)$ exists and is unique.

Without loss of generality we assume that in equations (72) and (B.1) the normalization constant is chosen so that

$$\mathcal{J}(I^*) = 0. \tag{B.8}$$

As $\partial\mathcal{J}(I^*)/\partial I = 0$ (equation (B.6)), it follows that $\eta(0) = I^*$ and thus from equations (B.3) and (B.8) we have

$$\varphi(0) = 0. \tag{B.9}$$

By expanding in equations (B.2) and (B.3) the function $\varphi(\mathrm{i}b)$ in a Taylor series, using equation (B.9) and comparing the result with equation (15), we obtain

$$\langle\langle M_I^m \rangle\rangle = M\partial^m\varphi(0)/\partial x^m \qquad \text{as } M \to \infty \tag{B.10}$$

and the corresponding relative fluctuations c_m are given by

$$c_m \simeq \langle M_I\rangle^{-(m-1)}[(\partial^m\varphi(0)/\partial x^m)/(\partial\varphi(0)/\partial x)^m] \to 0 \qquad \text{as } \langle M_I\rangle \to \infty \tag{B.11}$$

and thus the population fluctuations described by the Kubo extensivity ansatz (72) and (73) are non-intermittent.

Appendix C

For computing the infection probability $\psi(I)$ in the case of a negative binomial distribution of the number of infectives, we write equation (A.9) for the characteristic function $G(b)$ in the form

$$G(b) = \left(\frac{H}{H + \langle M_I\rangle(1 - \exp(\mathrm{i}b))}\right)^H. \tag{C.1}$$

By expressing in equation (C.1) the average value $\langle M_I\rangle$ of the number of infectives in terms of the average proportion of infectives $\langle I\rangle = I$ and of the total population density ρ (see equation (25)) and using the general expression (23) for the infection probability $\psi(I)$ we come to equation (78). Equation (78) does not depend on the total volume V_Σ of the domain Σ and thus it is valid both for small and large systems.

Appendix D

For a correlated spatial distribution of individuals the stochastic properties of the number M_I and of the positions $\boldsymbol{r}_1, \ldots \boldsymbol{r}_{M_I}$ of the infectives can be described in terms of a random point process (Van Kampen 1992). We introduce the Janossy probability densities

$$Q_0,\, Q_{M_I}(\boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I})\,\mathrm{d}\boldsymbol{r}_1, \ldots, \mathrm{d}\boldsymbol{r}_{M_I} \tag{D.1}$$

with the normalization condition

$$Q_0 + \sum_{M_I \geqslant 1} \frac{1}{M_I!}\int_\Sigma \cdots \int_\Sigma Q_{M_I}(\boldsymbol{r}_1, \ldots, \boldsymbol{r}_{M_I})\,\mathrm{d}\boldsymbol{r}_1 \ldots \mathrm{d}\boldsymbol{r}_{M_I} = 1. \tag{D.2}$$

$Q_{M_I}(\boldsymbol{r}_1,\ldots,\boldsymbol{r}_{M_I})\,\mathrm{d}\boldsymbol{r}_1\ldots\mathrm{d}\boldsymbol{r}_{M_I}$ is the probability that there are M_I infectives and that their positions are between $\boldsymbol{r}_1$ and $\boldsymbol{r}_1+\mathrm{d}\boldsymbol{r}_1$, ..., and $\boldsymbol{r}_{M_I}$ and $\boldsymbol{r}_{M_I}+\mathrm{d}\boldsymbol{r}_{M_I}$. In terms of the Janossy densities we introduce the product densities (Van Kampen 1992):

$$\eta_m(\boldsymbol{r}_1,\ldots,\boldsymbol{r}_m)=\sum_s\frac{1}{s!}\int_\Sigma\cdots\int_\Sigma Q_{M_I+S}(\boldsymbol{r}_1,\ldots,\boldsymbol{r}_m,\boldsymbol{r}_{m+1},\ldots,\boldsymbol{r}_{m+S})\,\mathrm{d}\boldsymbol{r}_{m+1}\ldots\mathrm{d}\boldsymbol{r}_{m+S} \tag{D.3}$$

and the generating functionals

$$\Lambda[W(\boldsymbol{r}')]=Q_0+\sum_{M_I}\frac{1}{M_I!}\int_\Sigma\cdots\int_\Sigma Q_{M_I}(\boldsymbol{r}'_1,\ldots,\boldsymbol{r}'_{M_I})W(\boldsymbol{r}'_1)\ldots W(\boldsymbol{r}'_{M_I})\,\mathrm{d}\boldsymbol{r}'_1\ldots\mathrm{d}\boldsymbol{r}'_{M_I} \tag{D.4}$$

$$\Xi[W(\boldsymbol{r}')]=1+\sum_{m\geqslant 1}\frac{1}{m!}\int_\Sigma\cdots\int_\Sigma \eta_m(\boldsymbol{r}'_1,\ldots,\boldsymbol{r}'_m)W(\boldsymbol{r}'_1)\ldots W(\boldsymbol{r}'_m)\,\mathrm{d}\boldsymbol{r}'_1\ldots\mathrm{d}\boldsymbol{r}'_M \tag{D.5}$$

where $W(\boldsymbol{r}')$ is a suitable test function. It can be shown that (Van Kampen 1992, Vlad and Mackey 1995b)

$$\Lambda[W(\boldsymbol{r}')]=\Xi[W(\boldsymbol{r}')-1]. \tag{D.6}$$

A cumulant expansion of the generating functional $\Xi[W(\boldsymbol{r}')]$ of the product densities

$$\Xi[W(\boldsymbol{r}')]=\exp\left\{\sum_{m\geqslant 1}\frac{1}{m!}\int_\Sigma\cdots\int_\Sigma g_m(\boldsymbol{r}'_1,\ldots,\boldsymbol{r}'_m)W(\boldsymbol{r}'_1)\ldots W(\boldsymbol{r}'_m)\,\mathrm{d}\boldsymbol{r}'_1\ldots\mathrm{d}\boldsymbol{r}'_m\right\} \tag{D.7}$$

provides a definition for the correlation functions $g_m(\boldsymbol{r}_1,\ldots,\boldsymbol{r}_m)$ of the positions of infectives from a cluster of size m:

$$g_m(\boldsymbol{r}_1,\ldots,\boldsymbol{r}_m)=\left.\frac{\delta^m\ln\Xi[W(\boldsymbol{r}')]}{\delta W(\boldsymbol{r}_1)\ldots\delta W(\boldsymbol{r}_m)}\right|_{W(\boldsymbol{r}')=0}. \tag{D.8}$$

For a correlated behaviour of the individuals the infection probability $\psi(I)$ can be expressed as an average of the instantaneous probability $\xi(M_I;\boldsymbol{r}_1,\ldots,\boldsymbol{r}_{M_I})$ of infection of a healthy individual by a given number of infectives with given positions

$$\psi(I)=\langle\xi(M_1;\boldsymbol{r}_1,\ldots,\boldsymbol{r}_{M_I})\rangle \tag{D.9}$$

where the average is computed in terms of the Janossy densities $Q_{M_I}(\boldsymbol{r}_1,\ldots,\boldsymbol{r}_{M_I})$. By using the expression (20) for $\xi(M_I;\boldsymbol{r}_1,\ldots,\boldsymbol{r}_{M_I})$, the normalization condition (D.2) of the Janossy densities and equations (D.4)–(D.6), we obtain

$$\begin{aligned}\psi(I)&=1-\sum\frac{1}{M_I!}\int_\Sigma\cdots\int_\Sigma Q_{M_I}(\boldsymbol{r}'_1,\ldots,\boldsymbol{r}'_{M_I})\prod_{l=1}^{M_I}[1-p(\boldsymbol{r}'_l)]\,\mathrm{d}\boldsymbol{r}'_1\ldots\mathrm{d}\boldsymbol{r}'_{M_I}\\&=1-\Lambda[W(\boldsymbol{r}')=1-p(\boldsymbol{r}')]=1-\Xi[W(\boldsymbol{r}')=-p(\boldsymbol{r}')].\end{aligned} \tag{D.10}$$

Now we use the expansion (D.7) of the generating functional $\Xi[W(\boldsymbol{r}')]$ and take into account the local character of the infection events (equation (11)); we come to

$$\psi(I)=1-\exp\left\{\sum_{m\geqslant 1}\frac{(-1)^m}{m!}\int_D\cdots\int_D g_m(\boldsymbol{r}_1,\ldots,\boldsymbol{r}_m)p(\boldsymbol{r}_1)\ldots p(\boldsymbol{r}_m)\,\mathrm{d}\boldsymbol{r}_1\ldots\mathrm{d}\boldsymbol{r}_m\right\}. \tag{D.11}$$

For a translationally invariant process we have

$$g_1=\langle M_I\rangle/V_\Sigma=\rho\langle I\rangle=\rho I\qquad\text{independent of }\boldsymbol{r} \tag{D.12}$$

$$g_m(\boldsymbol{r}_1,\ldots,\boldsymbol{r}_m)=g_m(\boldsymbol{r}_1-\boldsymbol{r}^*,\ldots,\boldsymbol{r}_m-\boldsymbol{r}^*) \tag{D.13}$$

where $\boldsymbol{r}^*$ is an arbitrary reference vector. By using equations (D.12) and (D.13), the expression (D.11) for the infection probability $\psi(I)$ can be written in the form (81), where the factor

$$\mathcal{M}[g] = \sum_{m\geqslant 2} \frac{(-1)^m}{m!} \int_D \cdots \int_D g_m(\boldsymbol{r}_1, \ldots, \boldsymbol{r}_m)\, p(\boldsymbol{r}_1) \ldots p(\boldsymbol{r}_m)\, \mathrm{d}\boldsymbol{r}_1 \ldots \mathrm{d}\boldsymbol{r}_m \tag{D.14}$$

is given by an expression similar to the virial expansion from equilibrium statistical mechanics.

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