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ORIGINAL ARTICLE

Theory of the Transmission of Infection in the Spread of Epidemics: Interacting Random Walkers with and Without Confinement

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Abstract A theory of the spread of epidemics is formulated on the basis of pairwise interactions in a dilute system of random walkers (infected and susceptible animals) moving in n dimensions. The motion of an animal pair is taken to obey a Smoluchowski equation in $2n$-dimensional space that combines diffusion with confinement of each animal to its particular home range. An additional (reaction) term that comes into play when the animals are in close proximity describes the process of infection. Analytic solutions are obtained, confirmed by numerical procedures, and shown to predict a surprising effect of confinement. The effect is that infection spread has a *non-monotonic* dependence on the diffusion constant and/or the extent of the attachment of the animals to the home ranges. Optimum values of these parameters exist for any given distance between the attractive centers. Any change from those values, involving faster/slower diffusion or shallower/steeper confinement, hinders the transmission of infection. A physical explanation is provided by the theory. Reduction to the simpler case of no home ranges is demonstrated. Effective infection rates are calculated, and it is shown how to use them in complex systems consisting of dense populations.

Keywords Smoluchowski · Interacting random walks · Diffusion · Infection transmission · Epidemics

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1 Introduction

The purpose of the following is to construct an analytic theory of the transmission of infection in the spread of epidemics on the basis of a simple but exactly soluble model of interacting random walkers representing animals moving about on the terrain and infecting one another on encounter. Seminal contributions (Anderson and May 1991; Okubo and Levin 2001; Hethcote 2000; Brauer and Castillo-Chávez 2001) involving concepts such as mass action, SIR, and the basic reproductive rate R_0 launched this field of research. The field derives its importance from human relevance as well as the theoretical difficulties of its intellectual challenge. Spatial considerations were introduced into the investigations independently by various authors (Dickmann et al. 2000; Okubo and Levin 2001; Abramson and Kenkre 2002; Aguirre et al. 2002; Cantrell and Cosner 2003; Kenkre 2003; McKane and Newman 2004; Kenkre et al. 2007; MacInnis et al. 2008) giving the studies a kinetic equation flavor. Missing from some of these studies were confinement features that arise in animal motion from home ranges and yet are clear and compelling in the light of field observations (Giuggioli et al. 2005, 2006; Abramson et al. 2006; MacInnis et al. 2008). These and other issues have made it essential to undertake a fundamental study of the transmission of infection in terms of interacting random walks specially under confinement.

2 Model and Method of Analysis

Our model starts with just two animals, one initially infected and the other initially uninfected (susceptible), respectively, denoted by 1 and 2, performing random walks around respective attractive centers at $\boldsymbol{R}_1$ and $\boldsymbol{R}_2$, with a diffusion constant D, there being the possibility of the uninfected individual getting infected at a rate proportional to $\mathcal{C}$ when the two occupy the same position. The central quantity that serves as the focus of our calculation is the joint probability density $P(\boldsymbol{r}_1, \boldsymbol{r}_2, t)$ that the infected animal is at $\boldsymbol{r}_1$ and the susceptible animal is at $\boldsymbol{r}_2$. Given this definition, $P(\boldsymbol{r}_1, \boldsymbol{r}_2, t)$ vanishes when the susceptible animal gets infected and the infection problem becomes formally similar to a Frenkel exciton annihilation problem analyzed a number of years ago in a system of molecular aggregates (Kenkre 1980). The present problem is considerably more complex, however, as a consequence of the tethering of the individuals to separate centers. Guided by the procedures set out in the exciton analysis (Kenkre 1980), we consider a capture problem in a space of twice the number of dimensions as the space in which each walker moves, introduce attractive quadratic potentials of steepness γ around the centers at $\boldsymbol{R}_1$ and $\boldsymbol{R}_2$, and write, applicable to s-dimensions in general,

$$\frac{\partial P}{\partial t} = \nabla_1 \cdot \left[\gamma\,(\boldsymbol{r}_1 - \boldsymbol{R}_1)\,P\right] + \nabla_2 \cdot \left[\gamma\,(\boldsymbol{r}_2 - \boldsymbol{R}_2)\,P\right] + D\left(\nabla_1^2 + \nabla_2^2\right) P - \delta(\boldsymbol{r}_1 - \boldsymbol{r}_2)\mathcal{C}P. \tag{1}$$

In terms of the propagator (Green function) for the homogeneous problem, $\Pi(\boldsymbol{r}_1, \boldsymbol{r}_1^0, \boldsymbol{r}_2, \boldsymbol{r}_2^0, t)$, the solution in the absence of the infection rate for any initial

placement of the two animals given by $P(\boldsymbol{r}_1^0, \boldsymbol{r}_2^0, 0)$ would be

$$\eta(\boldsymbol{r}_1, \boldsymbol{r}_2, t) = \int_{-\infty}^{\infty} \int_{-\infty}^{\infty} \mathrm{d}^s r_1^0 \mathrm{d}^s r_2^0 \; \Pi(\boldsymbol{r}_1, \boldsymbol{r}_1^0, \boldsymbol{r}_2, \boldsymbol{r}_2^0, t) P(\boldsymbol{r}_1^0, \boldsymbol{r}_2^0, 0). \tag{2}$$

When infection is present, we write, as a consequence of the linearity of the equations,

$$P(\boldsymbol{r}_1, \boldsymbol{r}_2, t) = \eta(\boldsymbol{r}_1, \boldsymbol{r}_2, t) - \mathcal{C} \int_0^t \mathrm{d}t' \int_{-\infty}^{\infty} \mathrm{d}^s r_1' \Pi(\boldsymbol{r}_1, \boldsymbol{r}_1', \boldsymbol{r}_2, \boldsymbol{r}_1', t - t') P(\boldsymbol{r}_1', \boldsymbol{r}_1', t'). \tag{3}$$

Defect technique procedures (Hemenger et al. 1974; Montroll and West 1979; Kenkre 1982a, b; Kenkre and Parris 1983; Szabo et al. 1984; Redner 2001; Redner and Ben-Avraham 1990; Spendier and Kenkre 2013; Spendier et al. 2013) along the lines originated in the analysis of the exciton annihilation problem mentioned above (Kenkre 1980) proceed by Laplace transforming Eq. (3), setting $\boldsymbol{r}_1 = \boldsymbol{r}_2$, and integrating over $\boldsymbol{r}_1$ in the appropriate space of s dimensions. An important result is

$$\int_{-\infty}^{\infty} \mathrm{d}^s r_1 \; \tilde{P}(\boldsymbol{r}_1, \boldsymbol{r}_1, \epsilon) = \int_{-\infty}^{\infty} \mathrm{d}^s r_1 \; \tilde{\eta}(\boldsymbol{r}_1, \boldsymbol{r}_1, \epsilon) - \mathcal{C} \int_{-\infty}^{\infty} \mathrm{d}^s r_1' \int_{-\infty}^{\infty} \mathrm{d}^s r_1 \; \tilde{\Pi}(\boldsymbol{r}_1, \boldsymbol{r}_1', \boldsymbol{r}_1, \boldsymbol{r}_1', \epsilon) \tilde{P}(\boldsymbol{r}_1', \boldsymbol{r}_1', \epsilon), \tag{4}$$

where ϵ is the Laplace variable and tildes denote Laplace transforms. Motivated by the so-called nu-function analysis introduced in capture problems (Kenkre 1982b; Kenkre and Parris 1983) [for a recent review and application, see Spendier and Kenkre (2013)] and assisted by the observation that the integral of $\tilde{\Pi}(\boldsymbol{r}_1, \boldsymbol{r}_1', \boldsymbol{r}_1, \boldsymbol{r}_1', \epsilon)$ over the entire domain of $\boldsymbol{r}_1$ (i.e., all space) appearing in Eq. (4) is independent of $\boldsymbol{r}_1'$, we introduce the symbol $\tilde{\nu}(\epsilon)$ to denote that integral,

$$\tilde{\nu}(\epsilon) = \int_{-\infty}^{\infty} \mathrm{d}^s r_1 \; \tilde{\Pi}(\boldsymbol{r}_1, \boldsymbol{r}_1', \boldsymbol{r}_1, \boldsymbol{r}_1', \epsilon), \tag{5}$$

and succeed in obtaining, in the Laplace domain, an *explicit* solution for the joint probability (density) that the two animals occupy the same position,

$$\int_{-\infty}^{\infty} \mathrm{d}^s r_1' \; \tilde{P}(\boldsymbol{r}_1', \boldsymbol{r}_1', \epsilon) = \frac{\tilde{\mu}(\epsilon)}{1 + \mathcal{C}\tilde{\nu}(\epsilon)}. \tag{6}$$

The expression in Eq. (6) contains two quantities that are key to the analysis. The first of these, $\nu(t)$, whose Laplace transform is defined in Eq. (5), is the probability (density) that the locations of the two animals coincide (whatever that location) if at a time t earlier their locations also coincided. The second key quantity, $\mu(t)$, whose Laplace transform is

$$\tilde{\mu}(\epsilon) = \int_{-\infty}^{\infty} \mathrm{d}^s r_1' \ \tilde{\eta}(\boldsymbol{r}_1', \boldsymbol{r}_1', \epsilon)$$
$$= \int_{-\infty}^{\infty} \mathrm{d}^s r_1' \ \times \int_{-\infty}^{\infty}\int_{-\infty}^{\infty} \mathrm{d}^s r_1^0 \mathrm{d}^s r_2^0 \ \tilde{\Pi}(\boldsymbol{r}_1', \boldsymbol{r}_1^0, \boldsymbol{r}_1', \boldsymbol{r}_2^0, \epsilon) P(\boldsymbol{r}_1^0, \boldsymbol{r}_2^0, 0), \quad (7)$$

is the probability (density) that the two animals occupy the same location at the present time (whatever that location) if at a time t earlier they occupied locations as per the *given initial condition* of the problem. Both refer to the problem without infection ($\mathcal{C} = 0$). They are integrals (over the s-dimensional space) of the two-particle joint probability density and have the dimensions of reciprocal length raised to s. The rest of the calculation is straightforward. Knowledge of the propagators of the system generally in the presence of constraining potentials gives ν and, in combination with the given initial conditions, yields μ. The two together with Eq. (6) provide all that is necessary to obtain the infection probability and the nuances of its behavior.

3 Infection Curve and its Non-monotonic Dependence

When a definite infection event occurs, the joint probability density $P(\boldsymbol{r}_1, \boldsymbol{r}_2, t)$ drops to zero. The infection probability is, therefore,

$$\mathcal{I}(t) = 1 - \int_{-\infty}^{\infty}\int_{-\infty}^{\infty} \mathrm{d}^s r_1 \mathrm{d}^s r_2 \ P(\boldsymbol{r}_1, \boldsymbol{r}_2, t), \quad (8)$$

and, from Eq. (3), is obtained in the Laplace domain as

$$\tilde{\mathcal{I}}(\epsilon) = \frac{1}{\epsilon}\left[\frac{\tilde{\mu}(\epsilon)}{(1/\mathcal{C}) + \tilde{\nu}(\epsilon)}\right]. \quad (9)$$

Further insight requires the evaluation of the key quantities μ and ν, which follows from the form of the propagators appropriate to Eq. (1). These are well known to be Gaussian, to be multiplicative in Cartesian coordinates as one proceeds to higher dimensions, and to involve the saturating time $\mathcal{T}(t) = (1/2\gamma)(1-e^{-2\gamma t})$ that emerges from standard Ornstein–Uhlenbeck arguments (Reichl 2009; Risken 1989). The $2s$-dimensional propagator and the resulting ν and μ functions, the latter for arbitrary initial placement, are

$$\Pi(\boldsymbol{r}_1, \boldsymbol{r}_1^0, \boldsymbol{r}_2, \boldsymbol{r}_2^0, t) = \left(\frac{1}{4\pi D\mathcal{T}(t)}\right)^s$$
$$\times \prod_{\beta=1}^{s} e^{-\frac{\left(x_1^\beta - h_1^\beta - (x_1^{0\beta} - h_1^\beta)e^{-\gamma t}\right)^2 + \left(x_2^\beta - h_2^\beta - (x_2^{0\beta} - h_2^\beta)e^{-\gamma t}\right)^2}{4D\mathcal{T}(t)}},$$

$$\nu(t) = \left(\frac{1}{\sqrt{8\pi D\mathcal{T}(t)}}\right)^s \prod_{\beta=1}^{s} e^{-\frac{\left(1-e^{-\gamma t}\right)^2\left(h_1^\beta - h_2^\beta\right)^2}{8D\mathcal{T}(t)}},$$

$$\mu(t) = \left(\frac{1}{\sqrt{8\pi\, D\mathcal{T}(t)}}\right)^s \prod_{\beta=1}^{s} e^{-\frac{\left(h_1^\beta - h_2^\beta + \left(\left(x_1^{0\beta} - h_1^\beta\right) - \left(x_2^{0\beta} - h_2^\beta\right)\right)e^{-\gamma t}\right)^2}{8D\mathcal{T}(t)}}, \quad (10)$$

where the label β runs from 1 to s, and the initial position and home range center of the susceptible animal have the respective x-components $x_2^{0\beta}$ and h_2^β. The rest of the notation is obvious.

For the motion of two 1-dimensional walkers ($s = 1$), we do not need the index β, and if we make the natural assumption that the animals are located initially at their own respective centers, the quantities $\nu(t)$, $\mu(t)$, which are closely related to Smoluchowski propagators connecting the two home range centers, are given by

$$\nu(t) = \frac{e^{-\frac{H^2}{8D\mathcal{T}(t)}(1-e^{-\gamma t})^2}}{\sqrt{8\pi\, D\mathcal{T}(t)}}; \quad \mu(t) = \frac{e^{-\frac{H^2}{8D\mathcal{T}(t)}}}{\sqrt{8\pi\, D\mathcal{T}(t)}}. \quad (11)$$

They equal each other for large times but begin quite differently at the initial time: $\mu(0)$ vanishes while $\nu(0)$ is infinite. Here, $H = h_1 - h_2$ is the distance between the two home range centers.

The infection curve $\mathcal{I}(t)$ is now obtained by calculating (see e.g., Roberts and Kaufman 1966) the Laplace transforms of Eq. (11), substituting them in Eq. (9), and inverting the transform. We perform the inversion numerically, with the help of a simple code implemented in MATLAB, and verify the results by direct numerical solution of the partial differential Eq. (1). The reader might find it useful to consult a related analysis given in an appendix (Spendier et al. 2013) where a similar procedure is explained in detail. The validation procedure results in agreement that is excellent except for confining potentials that are so steep that the direct numerical procedure used for verification breaks down. Our calculated $\mathcal{I}(t)$ for initial location of the animals at their home range centers, and for an assumed contact rate parameter $\mathcal{C}_1$ equal to 0.3 in units of $2D/H$, is displayed in Fig. 1 as a function of t scaled to τ_H, for various steepness values of the confining potential. Here, $\tau_H = H^2/2D$ is the time required for either animal to traverse diffusively the distance between the two home centers. We attach the suffix 1 to $\mathcal{C}$ to emphasize that this result is $1d$. Striking behavior is apparent in Fig. 1.

Recall that $\sigma = \sqrt{2D/\gamma}$ is the width of the steady-state distribution of the Smoluchowski walker in $1d$. We keep D and the inter-center distance H constant, and increment γ, thereby changing σ. The case of no confining potential corresponds to the thick solid curve ($H/\sigma = 0$). We gradually increase the steepness of the confinement potential , giving the latter parameter the respective values 0.6 (thin solid line), 1.0 (dotted), 1.64 (dot-dashed) and 2.12 (dashed). Generally, as time proceeds, $\mathcal{I}(t)$ rises from 0 and saturates to 1. Infection may be said to occur faster as the confining potential becomes steeper *but only for relatively small values* of γ. Further increases make the infection proceed more slowly. Vertical arrows between curves show this march graphically. Reversal in their direction marks the interesting phenomenon.

This non-monotonic behavior is noteworthy, one of the primary results of our analysis, and is also observed if the diffusion constant of the animals is varied keeping the

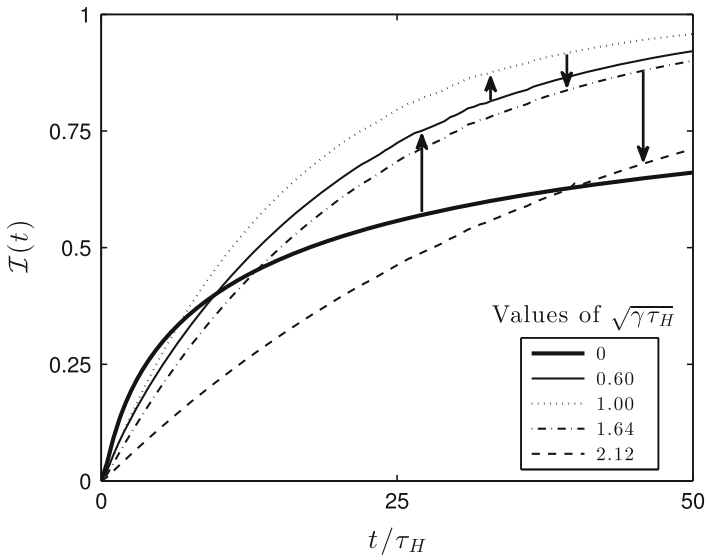

Fig. 1 Non-monotonic variation of the infection curve $\mathcal{I}(t)$ with change in γ, the steepness of the potential confining the animals to their home ranges. Time is scaled to τ_H; $\mathcal{C}_1$ scaled to $2D/H$ equals 0.3. Starting with the unconfined case ($\gamma = 0$), increase in γ makes infection more effective for small values of γ but less effective for larger values. The value shown for each *line* in the legend is of $\sqrt{\gamma\tau_H}$. This quantity equals H/σ, the ratio of the inter-center distance to the steady-state Smoluchowski width

potential steepness constant. It arises from the interplay of three quantities, the diffusion constant D, the steepness γ and the inter-center distance H which here is also the distance between the initial locations of the animals. For a given value of H, changes in D or γ exhibit the phenomenon. Varying H does not: maximum transmission occurs when $H = 0$, i.e., when the animals do not have to move to find each other for the infection to be propagated. The key parameter is $\gamma\tau_H = H^2\gamma/2D$ which is nothing other than $(H/\sigma)^2$: For a given H, optimum transmission of infection occurs when the parameter equals 1, particularly in the contact-limited case. More generally, the critical value is different from 1. By the term contact-limited, we mean the case that $\mathcal{C}$ is much smaller than the corresponding motion parameter. In such a case, the contact process of infection when the animals meet, rather than the motion, determines the overall infection event (see Eq. 9).

4 Reduction to the Case of No Confinement

Given that many of the previous quantitative theories do not explicitly incorporate home range confinement, it is important to ask what our model calculation predicts for the simpler case of such free diffusion. In that case, a full analytic solution is possible. With $\gamma \to 0$, $\nu(t)$ and $\mu(t)$ in $1d$ are simple propagators of the diffusion equation,

$$\nu(t) = \frac{1}{\sqrt{8\pi\, Dt}}; \quad \mu(t) = \frac{1}{\sqrt{8\pi\, Dt}} e^{-\frac{H^2}{8Dt}}. \tag{12}$$

Their Laplace transforms are known. With the introduction of a time $\theta = 8D/(\pi\mathcal{C}_1^2)$ that incorporates the diffusion constant and the contact parameter $\mathcal{C}_1$, we have for the infection probability in the Laplace domain,

$$\tilde{I}(\epsilon) = \frac{1}{\epsilon}\left(\frac{e^{-\sqrt{\epsilon\tau_H}}}{1+\sqrt{\epsilon\theta}}\right). \tag{13}$$

Inverse transformation gives the analytic time domain result

$$I(t) = \text{erfc}\left(\sqrt{\frac{\tau_H}{4t}}\right) - e^{\left(\sqrt{\frac{\tau_H}{4t}}+\frac{t}{\theta}\right)}\text{erfc}\left(\sqrt{\frac{\tau_H}{4t}}+\sqrt{\frac{t}{\theta}}\right). \tag{14}$$

We have not encountered this result in the epidemic literature earlier. However, curiously, we have found that the expression has been reported independently by several authors in totally unrelated reaction diffusion contexts (Carslaw and Jaeger 1959; Abramson and Wio 1995; Redner 2001; Spendier and Kenkre 2013). The further simplification of an infinite contact rate (motion limit), leading to a vanishing θ, yields the simple diffusion result that the infection curve is given by a complementary error function of argument $\sqrt{\tau_H/4t}$. The time dependence of Eq. (14) is depicted as the thick solid line $\gamma = 0$ in Fig. 1.

5 Effective Rates of Infection and Extension to Dense Systems

The foregoing analysis, while exact for dilute systems, is not immediately applicable to dense systems because they contain numerous (rather than one) interacting pairs. The dynamics, and even identity, of the pairs, evolve in time. We have developed, and plan to report in a forthcoming publication, an approximate kinetic equation theory applicable to such situations, along the lines of earlier analysis (Kenkre 2003; Kenkre et al. 2007). For use in that theory, we extract from the above single-pair analysis an effective infection rate. The spirit of this extraction is the same as in the calculation of a Fermi golden rule rate for describing transitions in a complex quantum system. The detailed procedure is, however, different. It is explained next.

First, we notice that Eq. (9) for the infection probability in the Laplace domain can be cast in the form

$$\tilde{\mathcal{I}}(\epsilon) = \frac{\tilde{\alpha}(\epsilon)}{\epsilon\,(\epsilon+\tilde{\alpha}(\epsilon))},$$

where we will term the quantity $\tilde{\alpha}(\epsilon)$ an infection *memory*. It is given in the Laplace domain by

$$\tilde{\alpha}(\epsilon) = \frac{\epsilon\tilde{\mu}(\epsilon)}{(1/\mathcal{C})+\tilde{\nu}(\epsilon)-\tilde{\mu}(\epsilon)}. \tag{15}$$

If the Markoffian approximation were to be made on this infection memory, i.e., if $\tilde{\alpha}(\epsilon)$ were to be replaced by an ϵ-independent constant α, the infection probability in the time domain, $\mathcal{I}(t)$, would be simply an exponentially rising function $1-e^{-\alpha t}$. It is clear that this function has, in essence, the typical shape seen in Fig. 1. Obviously, the extraction of a single infection rate from the full dynamics of the infection probability

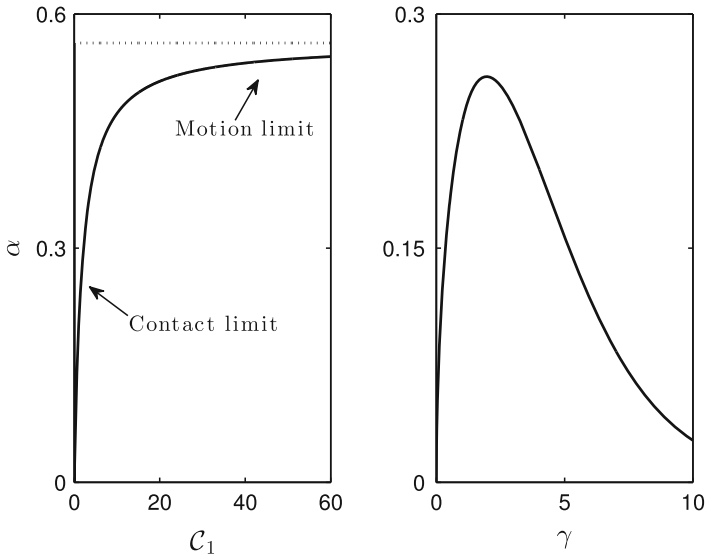

Fig. 2 Dependence of the effective infection rate α from Eq. (16) on the contact rate $\mathcal{C}_1$ (scaled to $2D/H$) in the *left panel* and on the confining potential steepness γ in the *right panel*. Both α and γ are scaled to $1/\tau_H$. The *left panel* shows that α is linear in the contact rate for small values of the latter but saturates to the motion-limited value (0.56 in this example) for large values. The *right panel* shows the non-monotonicity effect on infection: As confinement steepness γ increases, α rises to a peak and decreases for larger γ. For the *right panel*, $\mathcal{C}_1$ in units of $2D/H$ is 15

is provided by taking the Markoffian approximation of the infection memory $\tilde{\alpha}(\epsilon)$ through the limit $\epsilon \to 0$.

$$\alpha \equiv \lim_{\epsilon \to 0} \tilde{\alpha}(\epsilon) = \frac{\mu(\infty)}{(1/\mathcal{C}) + (1/\mathcal{M})}. \tag{16}$$

Here, $\mu(\infty)$ is the limit as $t \to \infty$ of $\mu(t)$, and we have introduced a *motion parameter* $\mathcal{M}$ as the reciprocal of $\int_0^\infty dt\,[\nu(t) - \mu(t)]$. An Abelian theorem has been used in the second equality in Eq. (16) to express α in terms of quantities in the time domain. The effective rate now appears as the product of the probability in the steady state that the two walkers occupy the same position, independently of the initial condition (essentially the numerator), and a combined rate involving the contact parameter and a motion parameter (essentially the reciprocal of the denominator). Thus, α equals simply $\mathcal{C}\mu(\infty)$ in the contact-limited case, i.e., when $\mathcal{C} << \mathcal{M}$. In the opposite limit $\mathcal{M} << \mathcal{C}$, infection is governed by the motion and α is $\mathcal{M}\mu(\infty)$.

This limiting behavior for extreme relative values of the contact and motion parameters is clear in the left panel of Fig. 2. The motion parameter $\mathcal{M}$ describes an accumulated integral of the difference between the two probability densities explained above of the two walkers coinciding in location. The non-monotonicity effect is displayed in the right panel of Fig. 2 where the infection rate α rises, peaks, and drops as the potential steepness is varied.

Equation (10) allows the evaluation of $\mu(\infty)$ in Eq. (16) for arbitrary dimensions s as being $\left[(1/\sigma\sqrt{2\pi})e^{-H^2/2\sigma^2}\right]^s$ where $\sigma = \sqrt{2D/\gamma}$ is the width of steady-state distribution in $1d$. Calculating $\mathcal{M}$ involves the evaluation of an improper integral

which is convergent in 1-d (Sugaya 2014) but presents the standard difficulties that arise in reaction diffusion problems in dimensions higher than 1 if reaction is taken to occur at points (regions of vanishing dimension) as we have done here. Generalizing the treatment to include reaction in finite regions solves this problem. It is also of interest to include the consequences of the introduction of an additional decay into the system. Such a decay may arise from radiative lifetimes as explained for excitons in molecular crystals earlier (Kenkre 1982a), from finite lifetimes τ of the infected animals as they may die from natural death or from predator attack, or from finite lifetime of the infection itself. The latter may be caused by the animals recovering from being infective. In such cases, one takes the limit $\epsilon \to 1/\tau$ rather than $\epsilon \to 0$, and Eq. (16) is replaced by

$$\alpha\tau = \frac{\int_0^\infty \mathrm{d}t e^{-t/\tau}\mu(t)}{(1/\mathcal{C}) + \int_0^\infty \mathrm{d}t e^{-t/\tau}\left[\nu(t) - \mu(t)\right]}. \tag{17}$$

In case a finite lifetime is absent in the given problem, it is perfectly natural to introduce it as a probe time associated with measurement.

6 Conclusions

The calculation we have presented is precise for the limited model considered and is valid for movement both with and without spatial constraints imposed on the moving animals. The spatial constraint would represent the existence of home ranges. In the presence of spatial constraints, the analysis has uncovered a remarkable phenomenon: Infection efficiency is non-monotonic when the steepness of the confining potential, or the animal diffusion constant, is varied. A similar simpler phenomenon occurs in reaction diffusion scenarios for *trapping* considerations under a confining potential, as we have recently shown (Spendier et al. 2013).

In our present context, each of the two quantities, the steepness of the confining potential and the animal diffusion constant, has a critical value on both sides of which infection becomes inefficient. An understanding of the curious effect we observe can be achieved at various levels. The effect involves three quantities, the distance H between the centers of the home ranges, the diffusion constant D, and the potential steepness γ. Combined into a single parameter $\sqrt{H^2\gamma/2D}$, which equals H/σ, the quantities signal inefficient transmission of infection when variations in D or γ make the parameter differ from its optimum value. In the contact-limited case, the optimum value is 1 and corresponds to the static statement that the width of the steady-state distribution of the Smoluchowski equation equals the distance between the home centers or to the dynamic statement that the time taken by the walker to traverse the inter-home range distance H diffusively equals the time $1/\gamma$ characteristic of free motion of the walker to the center under the action of the potential. Away from the contact limit, the optimum value changes from 1 because of contributions from what has been explained as the motion parameter $\mathcal{M}$ (see earlier text). Thus, in the right panel of Fig. 2, for the particular variable values we have assumed, it happens to equal 1.97.

Since our analysis has employed Laplace transforms and their numerical inversion, a validation procedure by comparison to direct numerical solution of the differential equation used is highly desirable. Such validation has been done for all cases considered. It has not been described in the present paper, except for a passing statement following Eq. (11). We have published a closely related validation in detail recently on a subject dealing, not with epidemics, but with trapping phenomena. It is given in its entirety in an appendix in Spendier et al. (2013). The results of the validation, in our present epidemics context, is excellent.

The (quadratic) confinement potential we have considered in Eq. (1), and consequently throughout the analysis, has been selected for two reasons. The first is the simplicity of a linear restoring force it represents. The second is the analytic tractability it provides. Explicit expressions for the propagators can also be obtained for linear (rather than quadratic) and box potentials (Chase et al. 2014). They too result in the behavior we have discussed. Generally, the analysis we have given carries over, in its qualitative conclusions, for any confinement potential.

The basic assumption of the present analysis, that a simple random walk, consequently a diffusion equation for the evolution of the probability density, is appropriate for the description of animal motion, is in keeping with most field observations. As an example, we cite the investigations of the movement of *Peromyscus maniculatus* rodents carried out in Abramson et al. (2006) in the context of the spread of the Hantavirus epidemic—distributions in mark-recapture experiments were found to be essentially Gaussian and the description of the simple random walk to be quite appropriate. In any particular case, when this is not true, one would modify Eq. (1) to incorporate such features as the Lévy nature of the walks (Plank and Codling 2009; Petrovskii et al. 2011), for instance, by introducing general memory functions to describe appropriate anomalous motion (Giuggioli and Kenkre 2014).

Our analysis is applicable for arbitrary initial conditions. In addition to being exact for the simplified model considered, it provides a sound basis for obtaining expressions for infection rates that can be used in approximate, but practical, theories of the spread of infection. Such extended theories are appropriate in realistic scenarios involving dense animal populations, will be reported elsewhere, and consist of a kinetic equations setup as in our earlier treatments (Abramson and Kenkre 2002; Kenkre 2003; Kenkre et al. 2007). The infection (aggression) rates in these theories are computed from the present analysis rather than being simply postulated. The formalism is directly useful for the study of the spread of zoonotic diseases such as the Hantavirus (Yates et al. 2002) in which infection spreads as the result of the movement of rodents on a terrain. It should also find use in other contexts as in the study of West Nile Virus (Nasci et al. 2001; Strausbaugh et al. 2001; Kenkre et al. 2005) within the field of epidemics and also in general studies of reaction diffusion and interacting random walks.

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