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Lag-driven motion in front propagation

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HIGHLIGHTS

- An approximate front speed holds for systems presenting time-delayed dispersal.
- The approximate front speed explains the observed spread of several focal infections.
- Approximate spread rates for the Neolithic transition agree with observed data.
- Observed spread rates of tree colonizations are predicted by our approximate speed.
- The approximate speed is derived from the equations for the three systems above.

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ABSTRACT

Front propagation is a ubiquitous phenomenon. It arises in physical, biological and cross-disciplinary systems as diverse as flame propagation, superconductors, virus infections, cancer spread or transitions in human prehistory. Here we derive a single, approximate front speed from three rather different time-delayed reaction–diffusion models, suggesting a general law. According to our approximate speed, fronts are crucially driven by the lag times (periods during which individuals or particles do not move). Rather surprisingly, the approximate speed is able to explain the observed spread rates of completely different biophysical systems such as virus infections, the Neolithic transition in Europe, and postglacial tree recolonizations.

1. Introduction

Fronts are widely used in physical models of flame propagation [1], superconductors [2], virus infections [3], cancer spread [4], transitions in human prehistory [5], etc. In many systems, individuals or particles are at rest during some time intervals, and for this reason the corresponding fronts become time-delayed [5]. For single-species systems, the dynamics is governed by the hyperbolic reaction–diffusion (HRD) equation [6]:

$$\frac{\delta p}{\delta t}+\frac{T}{2}\frac{\delta^2 p}{\delta t^2}=D\left(\frac{\delta^2 p}{\delta x^2}+\frac{\delta^2 p}{\delta y^2}\right)+\left.\frac{\delta p}{\delta t}\right|_g+\left.\frac{T}{2}\frac{\delta^2 p}{\delta t^2}\right|_g, \tag{1}$$

where $p=p(x,y,t)$ is the population (or particle) number density at point (x,y) and time t, D is the diffusion coefficient, and the subindex $\cdots|_g$ indicates that the corresponding time derivatives take into account growth (i.e., net reproductive) but not diffusive processes [6]. In Eq. (1), terms proportional to T are second-order Taylor expansion terms [6] and account for the effects of the delay (or lag) time T which is elapsed between two successive motions of particles or individuals. If no delay time is considered (i.e., if $T=0$), Fisher's classical reaction–diffusion equation [7] is recovered.

A set of coupled equations is required when extending the scope to multiple-species systems. For example, focal infections provide a convenient experimental platform to study the replication (reaction) and spread (diffusion) of viruses

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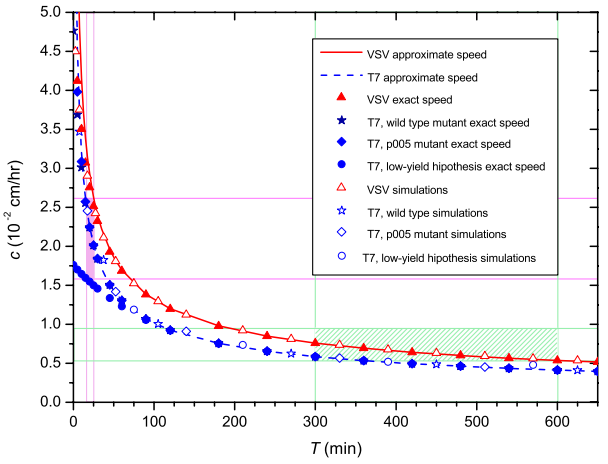

Fig. 1. Focal infections. Front speed versus lag time. The curves stand for the approximate solution $c = \sqrt{2D/T}$. The solid curve corresponds to VSV ($D = 1.44 \cdot 10^{-4}$ cm^2/h) and the dashed curve to the two T7 mutants ($D = 8.55 \cdot 10^{-5}$ cm^2/h). The full (empty) symbols correspond to the results of the exact theory (simulations). The triangles represent the results for VSV ($Y = 4983$ [11]). The stars stand for the wild T7 strain ($Y = 34.5$ [3]), and the rhombus for the p005 T7 mutant ($Y = 63.6$). The circles represent a hypothetical low-yield case $Y = 5$. The hatched (shaded) area corresponds to the observed ranges of T and c for VSV (T7) viruses. Details on the parameter values and the simulations appear in Appendix A.

in a cell monolayer [3,8,9]. The interactions between viruses (V), non-infected cells (C) and infected cells (I) give rise to the following evolution equations [3,10,11]:

$$\frac{\partial [C]}{\partial t} = -k_1 [C][V], \tag{2}$$

$$\frac{\partial [I]}{\partial t} = k_1 [V][C] - k_2 [I] \left(1 - \frac{[I]}{I_{\mathrm{MAX}}}\right), \tag{3}$$

where k_1 stands for the rate constant of adsorption of viruses V to non-infected cells C and k_2 is the death (or lysis) rate of infected cells I (each infected cell releases a new generation of Y viruses after a delay time T). If we replace p by the virus number density $[V](r, t)$ (where r is the radial coordinate centered at the inoculation point of the infection) in the above HRD equation (1), we obtain the evolution equation for the virus population. In agreement with Eqs. (2)–(3), the virus population growth reads

$$\left.\frac{\delta [V]}{\delta t}\right|_g = -k_1 [V][C] + k_2 Y [I] \left(1 - \frac{[I]}{I_{\mathrm{MAX}}}\right). \tag{4}$$

Hence, in order to determine the dynamics of focal infections, the set of differential equations (1)–(4) must be solved. Whereas the exact speed of front solutions to Eqs. (1)–(4) is very complicated (see Appendix A), recently we derived an approximate solution for the spread of virus infections which reads [11][1]

$$c = \sqrt{2D/T}. \tag{5}$$

Note that dimensional analysis could also suggest that c is proportional to $\sqrt{D/T}$, but other dependences are possible on this ground, e.g. $\sqrt{k_2 D}$ for Eqs. (1)–(4), $\sqrt{aD}$ (Fisher's speed [7]), $\sqrt{a^2 D/T}$ for Eq. (1), etc. Moreover, dimensional analysis cannot predict the factor $\sqrt{2}$, which was derived by marginal stability analysis in Ref. [11].

We would like to emphasize the following physical interpretation of Eq. (5). First, the parameter T is defined as the mean time a virus needs to reproduce inside an infected cell. If this lag T is substantially longer than the rest of the interval times involved in a virus life cycle (i.e., the death time of cells k_2^{-1}, the mean travel time in the extracellular medium, and the time viruses need to cross the cell membrane $k_1^{-1} C_0^{-1}$), then T becomes the only relevant time scale in our model. On the other hand, the parameter D is related to how easily the virus diffuses in the extracellular medium. It is thus reasonable that the parameters T and D determine the front speed under these assumptions. But is this framework valid in real situations? Before closing this introduction, we address this question by applying Eq. (5) to several virus infections.

Fig. 1 compares the results from the approximate speed (5), the exact theory, and simulations for the front speed of focal infections for Vesicular Stomatitis Virus (VSV) [11]. Moreover, for the first time Eq. (5) is applied to the T7 virus [3]: in Fig. 1

[1] In Ref. [11] we presented an approximate infection front speed in dimensionless variables which is equivalent to Eq. (5) in the present paper.