Epidemic models with an infected-infectious period

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(Received 14 October 1997)

The introduction of an infective-infectious period on the geographic spread of epidemics is considered in two different models. The classical evolution equations arising in the literature are generalized and the existence of epidemic wave fronts is revised. The asymptotic speed is obtained and improves previous results for the Black Death plague. [S1063-651X(98)01403-2]

PACS number(s): 05.70.−a, 05.40.+j

I. INTRODUCTION

Geographic spread of epidemics was studied by a pioneering work of Noble [1], but it is less understood and less well studied than its temporal evolution. However, recent works such as [2] and [3], for instance, propose new approaches taking into account the role of cross-diffusion or a variable population size. Here we consider a simple SI (susceptible-infectious) model that leads to a very good velocity of spread of epidemics in accordance with the experimental results obtained for the Black Death catastrophic plague pandemic. This model is compared with a model of three species.

Our main assumption is to consider a characteristic time τ of delay in the appearance of the infectious members, which measures the period between the infected-infectious transition. When a susceptible population is infected, there is a time $\tau>0$ during which the infectious agents develop within the susceptible individual organisms and it is only after that time that the infected population becomes itself infectious (or infective). The corresponding model mechanisms for the development and spatial spread of the disease are phenomenologically derived. The traveling wave analysis of the model is carried out and the asymptotic velocity for an infectious solitary wave is found and it is compared with the older results of Noble.

II. THE FIRST MODEL

The SI model consists of only two populations, infectious $I(x,t)$ and susceptible $S(x,t)$, which interact. We model the spatial dispersal of the density of infectious individuals I and the density of susceptible individuals S by simple diffusion and consider the infectious and susceptible populations to be described by the same diffusion coefficient D . We consider the transition rate from susceptible to infected to be proportional to rSI, where r is a constant parameter. This means that rS is the number of susceptible individuals who catch the disease from each infectious unit. The susceptible members who catch the disease become infected members, an intermediate stage between susceptible and infectious. After a period τ, infected members become infectious and may transmit the disease. The parameter r measures the transmission efficiency of the disease from infectious to susceptible individuals. We assume that the infectious members have a disease-induced mortality rate αI, where $1/\alpha$ is the life expectancy. The evolution equations for the susceptible and infectious populations take the form

$$\frac{\partial \mathbf{U}}{\partial t}=D\frac{\partial^2 \mathbf{U}}{\partial x^2}+\mathbf{f},$$

where $\mathbf{U}=(S,I)^T$, and $\mathbf{f}=(f_s,f_I)$ is given by

$$f_s=-rS(x,t)I(x,t),$$
$$f_I=rS(x,t-\tau)I(x,t-\tau)-\alpha I(x,t).$$

Introducing the dimensionless variables

$$I^*=I/S_0,\quad S^*=S/S_0,\quad t^*=rS_0t,\quad \text{and } x^*=\sqrt{\frac{rS_0}{D}}x, \tag{1}$$

where S_0 is a representative population, the evolution equation system is

$$\frac{\partial S}{\partial t}=-I(x,t)S(x,t)+\frac{\partial^2 S}{\partial x^2},$$

$$\frac{\partial I}{\partial t}=\frac{\partial^2 I}{\partial x^2}+S(x,t-a)I(x,t-a)-\lambda I, \tag{2}$$

where $a=\tau rS_0$ and we have omitted the asterisks for notational simplicity. The dimensionless parameter λ is given by

$$\lambda\equiv\frac{\alpha}{rS_0}.$$

We look for traveling wave solutions, in the usual way by setting $z=x-ct$ in Eq. (2) where c is the wave speed, which must be determined. This will represent a wave of constant shape traveling in the positive x direction. Substituting this into Eq. (2) yields the ordinary differential system for $I(z)$ and $S(z)$,

$$S''+cS'-IS=0, \tag{3}$$

$$I''+cI'-\lambda I+\left(\sum_{n=0}^{\infty}\frac{(ca)^n}{n!}\frac{d^nI}{dz^n}\right)\left(\sum_{n=0}^{\infty}\frac{(ca)^n}{n!}\frac{d^nS}{dz^n}\right)=0,$$

where we have expanded in Taylor series the terms $S(x,t-a)$ and $I(x,t-a)$ by assuming S and I infinitely derivable. The only homogeneous steady state is $(0,\hat{S})$ where $\hat{S}$ may be any positive real value. The problem consists of finding the range of values of λ such that a solution exists with positive wave speed c and non-negative I and S such that [1]

$$I(-\infty)=I(\infty)=0 \quad \text{and} \quad 0\leqslant S(-\infty)<S(\infty)=1.$$

By linearizing Eq. (3) about the steady state and setting $\hat{S}=S(\infty)=1$ we obtain

$$\begin{aligned} v''+cv'-u&=0,\\ u''+cu'-\lambda u+\sum_{n=0}^{\infty}\frac{(ca)^n}{n!}\frac{d^nI}{dz^n}&=0, \end{aligned} \tag{4}$$

where $u\equiv I$ and $v\equiv S-\hat{S}$. The second equation for Eq. (4) is uncoupled from v and may be analyzed separately. Its characteristic equation is

$$\mu^2+c\mu-\lambda+e^{\mu ca}=0. \tag{5}$$

Since we require $I(z)\to 0$ with $I(z)>0$, $I(z)$ cannot oscillate about $I=0$, otherwise $I(z)<0$ for some z and therefore we must have real values for μ. In order to have two real solutions for Eq. (5) it is necessary that the restriction

$$e^{-c^2a/2}<\lambda+\frac{c^2}{4} \tag{6}$$

be fulfilled.

Application to the Black Death plague

In dimensional terms, the speed of the traveling waves, V say, is given by

$$V_{\min}=2\sqrt{rS_0D}c. \tag{7}$$

In order to apply our model to the experimental results, we must know the value of τ. This value could be related to the incubation period of the disease but we have not yet established a direct correspondence.

In order to analyze our results we take the same approximate values for the parameters used by Noble. The susceptible population density is assumed to be $S_0\approx 50/\text{miles}^2$, the diffusion coefficient is $D\approx 10^4$ miles^2/yr, the transmission coefficient is $r\approx 0.4$ miles^2/yr, and the life expectancy is about 3.5 weeks, so $\alpha\approx 15$/yr. With these parameters we obtain that the speed for the classical case ($a=0$) is 447.2 miles/yr, somewhat greater than the experimental results of 200–400 miles/yr quoted by Langer [4]. If we take the infected-infectious period of two weeks ($a=0.822$), which seems to be reasonable, the asymptotic speed is $V_{\min}$, where c fulfills the equality in Eq. (6) as may be shown by using the steepest descent method of Kolmogorov. This yields, after numerical calculation, 281.7 miles/yr, which lies entirely within the experimental range.

III. THE SECOND MODEL

A second model which takes into account the infected-infectious period may be developed by including a third species. Let $S(x,t)$ be the number density of susceptible members, $\hat{I}(x,t)$ the number density of infected members, and $I(x,t)$ the number density of infectious members. We assume in this section that the infected members have an infectious transition rate $\hat{I}/\tau$ where τ is the characteristic time of transition from infected to infectious or the infected-infectious period and assume that all the susceptible members who catch the disease become infected members. By assuming Ficks's law for the diffusive spread of members, we get the following set of equations:

$$\begin{aligned} \frac{\partial S}{\partial t}&=D\frac{\partial^2S}{\partial x^2}-rSI,\\ \frac{\partial \hat{I}}{\partial t}&=D\frac{\partial^2\hat{I}}{\partial x^2}+rSI-\frac{1}{\tau}\hat{I},\\ \frac{\partial I}{\partial t}&=D\frac{\partial^2I}{\partial x^2}-\alpha I+\frac{1}{\tau}\hat{I}. \end{aligned} \tag{8}$$

Using now the dimensionless variables (1) we obtain (we omit asterisks for notational simplicity)

$$\begin{aligned} \frac{\partial S}{\partial t}&=\frac{\partial^2S}{\partial x^2}-SI,\\ \frac{\partial \hat{I}}{\partial t}&=\frac{\partial^2\hat{I}}{\partial x^2}+rSI-\frac{1}{a}\hat{I},\\ \frac{\partial I}{\partial t}&=\frac{\partial^2I}{\partial x^2}-\lambda I+\frac{1}{a}\hat{I}. \end{aligned} \tag{9}$$

If we fix the reference frame onto the moving front by using the transformation $z=x-ct$, we obtain

$$S''+cS'-IS=0, \tag{10}$$

$$\begin{aligned} \hat{I}''+c\hat{I}'-\frac{1}{a}\hat{I}+S\hat{I}&=0,\\ I''+cI'-\lambda I+\frac{1}{a}\hat{I}&=0. \end{aligned} \tag{11}$$

Analogously to the previous model the homogeneous steady state is $(S_s,\hat{I}_s,I_s)=(1,0,0)$. Defining the new variables $v\equiv S-1$, $w\equiv\hat{I}$, and $u\equiv I$ and linearizing about the steady state, one obtains

$$\begin{aligned} v''+cv'-u&=0,\\ w''+cw'-\frac{1}{a}w+u&=0, \end{aligned} \tag{12}$$

$$u''+cu'-\lambda u+\frac{1}{a}w=0.$$

We define now the vector $\mathbf{U}=(v,w,u)^T$ so that Eq. (12) can be rewritten in the form

$$\mathbf{U}''+c\mathbb{I}\cdot\mathbf{U}'+\mathbb{A}\cdot\mathbf{U}=0, \tag{13}$$

where $\mathbb{I}$ is the unity matrix and

$$\mathbb{A}=\begin{pmatrix} 0 & 0 & -1 \\ 0 & -\frac{1}{a} & 1 \\ 0 & \frac{1}{a} & -\lambda \end{pmatrix}.$$

By linearizing $\mathbb{A}$ we obtain the following characteristic polynomial

$$\mu\left[\mu^2+\mu\left(\lambda+\frac{1}{a}\right)+\frac{1}{a}(\lambda-1)\right]=0. \tag{14}$$

In order to have real values in Eq. (13) it is necessary that

$$c>\sqrt{2}\left[\sqrt{\left(\lambda-\frac{1}{a}\right)^2+\frac{4}{a}}-\lambda-\frac{1}{a}\right]^{1/2},$$

with $\lambda<1$. Note that the constraint $\lambda<1$ is recovered both from Noble's work and from the first model.

Application to the Black Death plague

In dimensional units the asymptotic velocity has the form

$$V=\sqrt{2rS_0D}\left[\sqrt{\left(\frac{\alpha}{rS_0}-\frac{1}{\tau rS_0}\right)^2+\frac{4}{\tau rS_0}}-\frac{\alpha}{rS_0}-\frac{1}{\tau rS_0}\right]^{1/2}.$$

Taking the same characteristic values of Noble and assuming $\tau=2$ weeks, we get $V=339.5$ miles/yr which lies entirely in the experimental range 200–400 miles/yr.

We have shown with these two models that the introduction of an infected-infectious period τ, which is reasonable from the practical point of view, of two weeks, leads us to a speed of the disease propagation which lies entirely in the experimental range. In both models the speed of the disease is lower than in the classical model ($\tau=0$) due to the infected-infectious period. Murray [5] excuses the bad theoretical result by arguing that the classical model ($a=0$) is extremely simple and does not take into account the nonuniformity in population density, the stochastic elements, and so on. The fact is that, with a simple extension of the classical model, we are able to obtain two better results taking into account the infective-infectious period, which is also invoked in a recent work [6].

ACKNOWLEDGMENTS

I thank Professor José Casas-Vázquez. I also wish to express my gratitude to J. Camacho, A. Compte, and Professor J. E. Llebot and Professor D. Jou for useful discussions and for their interest in this work. I acknowledge the support of the program Formación de Personal Investigador under Grant No. CLI95-1867. Financial support from the DGICYT of the Spanish Ministry of Education under Grant No. PB94-0718 is acknowledged as well.

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