

Investigating The Travelling Wave Solution For an SIR Endemic Disease Model With No Disease Related Death (When The Spatial Spread Of The Susceptible Is Not Negligible).

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Abstract

This paper presents the travelling wave solution for an SIR endemic disease model with no disease related death when the spatial spread of the susceptible is not negligible. In this case the disease is driven by both the susceptible and the infective classes. The population is open since the disease is habitually prevalent in the population.

Keywords and phrases: Endemic model, spatial spread, travelling wave Solution.

1.0 Introduction

The prevalence of diseases in human population cannot be over emphasized. Spatial models of epidemics and epizootics have existed for at least forty years. A wide variety of methods have been used for the study of spatially structured epidemics.

One of the first applications of reaction-diffusion theory to spatial epidemiology was an effort by Noble (1974) to describe the spread of plague through Europe in the mid-fourteenth century. Recently Caraco et al (2002) have used a reaction-diffusion model to describe the spatial aspects of Lyme disease transmission. Perhaps the most well known and well studied spatial epizootic model is that of Kallen et al (1985), which described the spatial dynamics of rabies in fox populations. In its original form, this model is a simplification of Noble's plague model (1974). In Noble's plague model, he assumed that if S is the concentration of fox susceptible to rabies infection and I is the concentration of rabid fox (fox infested with rabies) and also assuming mass action kinetics and no latency period between infection and symptomatic behaviour then the rate at which susceptible fox becomes infested will be proportional to both the concentration of susceptible fox and the concentration of rabid fox. Customarily, a travelling wave is taken to be a wave which travels without change of shape and this will be our understanding here. So, if a solution $u(x, t)$ represents a travelling wave, the shape is a constant which we denote by c . If we look at this wave in a travelling frame moving at constant speed c , it will appear stationary. A Mathematical way of saying this is that if the solution u can be represented as

$$u(x, t) = u(x + ct) = u(z), \text{ where } z = x + ct \tag{1.1}$$

Then $u(x, t)$ is the travelling wave and it moves at constant speed c in the negative $x -$ direction. Clearly, if $x + ct$ is constant, so is u ; it also means that the coordinate system moves with speed c . A wave which moves in the positive $x -$ direction is of the form $u(x - ct)$ with positive c . the wave speed c generally has to be determined. The dependent variable z is sometimes called the wave variable. When we look for travelling wave solutions of an equation or system of equations in x and t in the form

$$u(x, t) = u(x + ct) = u(z), \text{ where } z = x + ct \tag{1.2}$$

We have

$$\frac{\partial u}{\partial t} = c \frac{du}{dz}, \quad \frac{\partial u}{\partial x} = \frac{du}{dz} \tag{1.3}$$

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So the partial differential equations in x and t become ordinary differential equations in z . To be physically realistic, $u(z)$ has to be bounded for all z and non-negative with the quantities with which we are concerned, such as chemicals, populations, bacteria and cells.

Let us first point out that without reaction there can be no travelling wave. To see this, consider a solution of the form

$$u(x, t) = u(x + ct) = u(z), z = x + ct \quad (1.4)$$

to the equation,

$$u_t = Du_{xx} \quad (1.5)$$

which is the diffusion equation, and then we have,

$$D \frac{d^2 u}{dz^2} - c \frac{du}{dz} = 0 \quad (1.6)$$

That is

$$D \frac{d^2 u}{dz^2} - c \frac{du}{dz} = 0 \text{ since } \frac{\partial u}{\partial t} = u_t = \frac{du}{dz} \quad (1.7)$$

This implies

$$u(x, t) = A + Be^{\frac{c}{D}z}$$

$$u(x, t) = A + Be^{\frac{c}{D}(x+ct)} \text{ since } z = x + ct \quad (1.8)$$

Where A and B are constants. Since u has to be bounded for all x and t , B must be zero since the exponential becomes unbounded as $x + ct \rightarrow -\infty$. $u(x, t) = A$, a constant, is not a wave solution. In marked contrast the parabolic reaction diffusion equation

$$u_t = Du_{xx} + f(u) \quad (1.9)$$

can exhibit travelling wave solutions, depending on the form of the reaction/interaction term $f(u)$. This solution behaviour was a major factor in starting the whole mathematical field of reaction diffusion theory.

Spatially structured epidemic models are useful tools in the study of geographic spread. In particular, spatial models can be used to estimate the speed of geographic spread. Estimates of rapidity of disease dissemination can in turn, be used to guide policy decisions. Research has shown that for many linear models there is a minimum speed c^* for travelling wave solution and that in many biologically realistic setting, solutions tend to approach advancing fronts that travel no faster than c^* .

However, a fundamental challenge in Mathematical epidemiology is determining how the structure of a population influences disease transmission. One important aspect is the spatial structure. For instance, the Severe Acute Respiratory Syndrome (SARS) epidemic spread through twelve countries within a few weeks and the recent swine flu has spread through all the continents (including some parts of Africa, e.g. Badagry in Lagos, Nigeria) within a few months of its outbreak. Projections of the spatial spread of an epidemic will facilitate the assessment of policy alternatives. Spatially-explicit models are necessary to evaluate the efficacy of movement controls [19]. Models that ignore spatial structure can lead to inaccuracy in the prediction of population dynamics [19].

2.0 Model Formulation And Notation

We consider spatial spread for an endemic disease - that is for a disease which is habitually prevalent in a population. In this kind of disease model, we are interested in long term behaviours of the disease. In this case, it will be unreasonable to lump together immune and death people into the same (removed) class, as their differences are now important. The removed class, R should now be considered as the immune class.

Since we are interested in long term behaviour, we cannot neglect birth and disease unrelated death. With births and deaths included, the population is no longer closed and the total population size N will only be a constant under

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additional assumptions on the birth and death rates. We consider a population with birth rate (not per capital birth rate) B and per capital disease-related and disease-unrelated death rates C and d respectively. For simplicity, we shall take C and d to be constants, but we shall make different assumptions about B . All births are assumed to enter the susceptible class (no vertical transmission). Vertical transmission is transmission from parent to foetus or newborn offspring.

The model described above without spatial spread

$$\begin{aligned}\frac{dS}{d\tau} &= bN - \beta IS - dS, \\ \frac{dI}{d\tau} &= \beta IS - \gamma I - CI - dI, \\ \frac{dR}{d\tau} &= \gamma I - dR\end{aligned}\tag{2.1}$$

Where βI is called the force of infection, β is the infection contact rate (that is the rate of infection per susceptible and per infective) and γ is the rate of recovery.

The population can approach an endemic steady state for no disease related death by letting $B = bN$, $b = d$ and $C = 0$ [4].

Equilibrium Analysis

To determine the disease-free steady state and the endemic steady state of the system (2.1), we set the right hand side of the system (2.1) to zero excluding the diffusion term because wherever in space the disease free and endemic steady state is achieved.

$$\begin{aligned}\eta(1 - \bar{u}) - R_0 \bar{u} \bar{v} &= 0 \\ (R_0 \bar{u} - 1) \bar{v} &= 0 \text{ i.e. } \bar{v} = 0 \text{ or } \bar{u} = \frac{1}{R_0} \\ \varepsilon \bar{v} - \eta \bar{w} &= 0\end{aligned}\tag{2.2}$$

Where u , v and w depends on x

when $\bar{v} = 0$, $\bar{u} = 1$ and $\bar{w} = 0$. Also, when

$$\bar{u} = \frac{1}{R_0}, \bar{v} = \eta \left(1 - \frac{1}{R_0}\right) \text{ and } \bar{w} = \varepsilon \left(1 - \frac{1}{R_0}\right)$$

The disease free steady state is at $(\bar{u}, \bar{v}, \bar{w}) = (u_0^*, 0, 0)$ where $u_0^* = 1$ and the endemic steady state is at

$$(\bar{u}, \bar{v}, \bar{w}) = (u_1^*, v_1^*, w_1^*) \text{ where } u_1^* = \frac{1}{R_0}, v_1^* = \eta \left(1 - \frac{1}{R_0}\right) \text{ and } w_1^* = \varepsilon \left(1 - \frac{1}{R_0}\right)\tag{2.3}$$

It is imperative to note that the endemic equilibrium (EE) exists only when $R_0 > 1$. If $R_0 = 1$, we obtain the disease free equilibrium (DFE).

3.0 Spatial Spread Model

We consider the spatial spread of the infective and susceptible. We shall introduce diffusion term D_s and D_I to represent diffusion coefficients of susceptible and infective respectively into the normal SIR endemic disease model.

Let $B = dN$, $b = d$ and $C = 0$ then the model (2.1) becomes,

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$$\begin{aligned}\frac{\partial S}{\partial \tau} &= bN - \beta IS - bS + \bar{\nabla}^2 S, \\ \frac{\partial I}{\partial \tau} &= \beta IS - \gamma I - bI + D_I \bar{\nabla}^2 I, \\ \frac{\partial R}{\partial \tau} &= \gamma I - bR\end{aligned}\tag{3.1}$$

Where $S = S(\xi, t)$, $I = I(\xi, t)$, $R = R(\xi, t)$,

$\bar{\nabla} S$ and $\bar{\nabla} I$ are Laplacian operators in one dimension representing the diffusion of the susceptible and infective densities respectively.

Next, we non-dimensionalised (3.1) and also rescale the time variable τ and space variable ξ , by using the following substitutions

$$\bar{u} = \frac{S}{N}, \quad \bar{v} = \frac{I}{N}, \quad \bar{w} = \frac{R}{N}\tag{3.2}$$

$$t = (\gamma + b)\tau \quad \text{and} \quad x = \xi \sqrt{\frac{\gamma + b}{D}}\tag{3.3}$$

The expected length of time an infective remains infectious is $\frac{1}{\gamma + b}$.

From (3.2) and (3.3), we obtain

$$\partial s = N\partial \bar{u}, \quad \partial I = N\partial \bar{v}, \quad \partial T = N\partial \bar{w}\tag{3.4}$$

$$\partial t = (\gamma + b)\partial \tau \quad \text{and} \quad \partial x = \partial \xi \sqrt{\frac{\gamma + b}{D}}\tag{3.5}$$

Substituting (3.2), (3.3), (3.4) and (3.5) into (3.1) and simplifying, we have

$$\begin{aligned}\frac{\partial \bar{u}}{\partial t} &= \eta(1 - \bar{u}) - R_0 \bar{u} \bar{v} + \frac{\partial^2 \bar{u}}{\partial x^2}, \\ \frac{\partial \bar{v}}{\partial t} &= (R_0 \bar{u} - 1)\bar{v} + \frac{\partial^2 \bar{v}}{\partial x^2}, \\ \frac{\partial \bar{w}}{\partial t} &= \varepsilon \bar{v} - \eta \bar{w}\end{aligned}\tag{3.6}$$

Where

$$R_0 = \frac{\beta N}{\gamma + b}, \quad \varepsilon = \frac{\gamma}{\gamma + b}, \quad \eta = \frac{b}{\gamma + b}$$

4.0 Travelling Wave Solution

We seek for a constant shape travelling wave solution of (3.1) by setting

$$\begin{aligned}\bar{u}(x, t) &= u(s) = u(x + ct), \quad \bar{v}(x, t) = v(s) = v(x + ct) \quad \text{and} \\ \bar{w}(x, t) &= w(s) = w(x + ct)\end{aligned}\tag{4.1}$$

where c is the wave speed which has to be determined. Substituting (4.1) into (3.1), we have

$$\begin{aligned}cu' &= \eta(1 - u) - R_0 uv + u'' \\ cv' &= (R_0 u - 1)v + v'' \quad \text{and} \\ cw' &= \varepsilon v - \eta w\end{aligned}\tag{4.2}$$

where the prime denotes differentiation with respect to s . The system (4.2) is to be analysed subject to the disease free steady state and the endemic steady state in (2.3).

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We model the spatial spread as a diffusive process where both the susceptible and infective classes (i.e. $S(x, t)$ and $I(x, t)$, which are function of spatial variable x as well as of time t) have diffusion coefficients D_s and D_I not equal to zero. We do not need to think that the individuals are actually diffusing; we can imagine them as fixed on a lattice with contacts to their nearest neighbours through which the disease propagates. The rates of transition from susceptible to infective and of removal from infective are the same as in the mean field model.

To write (4.2) as a system of first order ordinary differential equations, we set

$$m = u' \text{ and } m' = u'' \text{ Also } n = v' \text{ and } n' = v''$$

The system (4.2) becomes

$$\begin{aligned} u' &= m, \\ m' &= cm + R_0uv - \eta(1-u), \\ v' &= n \\ n' &= cn - (R_0u - 1)v \text{ and} \\ w' &= \frac{1}{c}(\varepsilon v - \eta w) \end{aligned} \tag{4.3}$$

In the (u, v, w, m, n) phase space, there is the disease free steady state $(1, 0, 0, 0, 0)$ and the endemic steady state $(u^*, v^*, w^*, m^*, n^*)$ where u^* , v^* and w^* have their usual meanings and $m^* = n^* = 0$.

To analyse the system (4.3), we determine the Eigen values by first considering the Jacobian of the system (4.3).

$$J = \begin{pmatrix} 0 & 1 & 0 & 0 & 0 \\ R_0v + \eta & c & R_0u & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ -R_0v & 0 & -(R_0u - 1) & c & 0 \\ 0 & 0 & \frac{\varepsilon}{c} & 0 & -\frac{\eta}{c} \end{pmatrix} \tag{4.4}$$

At the disease-free steady state $(1, 0, 0, 0, 0)$, we have

$$J^* = \begin{pmatrix} 0 & 1 & 0 & 0 & 0 \\ \eta & c & R_0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & (1 - R_0) & c & 0 \\ 0 & 0 & \frac{\varepsilon}{c} & 0 & -\frac{\eta}{c} \end{pmatrix} \tag{4.5}$$

The Eigen values are given by the roots of

$$\begin{vmatrix} -\lambda & 1 & 0 & 0 & 0 \\ \eta & c - \lambda & R_0 & 0 & 0 \\ 0 & 0 & -\lambda & 1 & 0 \\ 0 & 0 & (1 - R_0) & c - \lambda & 0 \\ 0 & 0 & \frac{\varepsilon}{c} & 0 & -\frac{\eta}{c} - \lambda \end{vmatrix} = 0 \tag{4.6}$$

The roots of the characteristic equation of (4.6) are given by:

$$\lambda_1 = -\frac{\eta}{c}, \quad \lambda_2, \lambda_3 = \frac{c \pm \sqrt{c^2 + 4(1 - R_0)}}{2},$$

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$$\lambda_4, \lambda_5 = \frac{c \pm \sqrt{c^2 + 4\eta}}{2} \quad (4.7)$$

Thus, there is an unstable manifold defined by the Eigen vectors associated with the Eigen values λ_2 and λ_3 which are positive for all $c > 0$. Furthermore $(1, 0, 0, 0, 0)$ is unstable in an oscillatory manner if $c^2 < 4(R_0 - 1)$. So the only possibility for a travelling wave front solution to exist with non-negative u, v and w is if

$$c \geq 2\sqrt{R_0 - 1}, \quad R_0 > 1 \quad (4.8)$$

With c satisfying this condition, a realistic solution with a lower bound on the wave speed may exist which tend to $u = 1, v = 0$ and $w = 0$ as $s \rightarrow -\infty$.

Next, we consider the travelling wave front as (u, v, w, m, n) approaches the endemic steady state $(u^*, v^*, w^*, 0, 0)$. The Eigen values is the roots of

$$\begin{vmatrix} -\lambda & 1 & 0 & 0 & 0 \\ R_0 v^* + \eta & c - \lambda & R_0 u^* & 0 & 0 \\ 0 & 0 & -\lambda & 1 & 0 \\ -R_0 v^* & 0 & -(R_0 u^* - 1) & c - \lambda & 0 \\ 0 & 0 & \frac{\varepsilon}{c} & 0 & -\frac{\eta}{c} - \lambda \end{vmatrix} = 0 \quad (4.9)$$

This gives

$$\begin{aligned} \lambda &= -\frac{\eta}{c}, \quad \lambda^4 - 2c\lambda^3 + (R_0 u^* - R_0 v^* - 1 + c^2 - \eta + c)\lambda^2 \\ &- c(R_0 u^* - R_0 v^* - 1)\lambda - (R_0 u^* \eta - R_0 v^* + \eta) = 0 \end{aligned} \quad (4.10)$$

After simplification (4.10) becomes

$$\lambda^4 - 2c\lambda^3 + (c^2 - \eta R_0 + c)\lambda^2 + c\eta(R_0 - 1)\lambda - \eta(R_0 - 1) = 0 \quad (4.11)$$

Applying Descartes' Rule of signs on (4.11)

Case 1: when $R_0 > 1$ and $\eta R_0 > 1$

There are three variations in sign implying that the polynomial has three or one positive real zeros.

Case 2: when $R_0 < 1$ and $\eta R_0 < 1$

Equation (4.11) becomes

$$\lambda^4 - 2c\lambda^3 + (c^2 - \eta R_0 + c)\lambda^2 - c\eta(R_0 - 1)\lambda + \eta(R_0 - 1) = 0 \quad (4.12)$$

There are four variations in sign implying that the polynomial has four or two or no positive real zeros.

It implies that the endemic equilibrium is not stable for both cases. It is difficult to characterize the travelling wave speed.

Discussions And Conclusions

The travelling wave solution for when the spatial spread of the susceptible is not negligible for the disease free steady state is obtainable that is, it exist. but it is difficult to characterize the travelling wave speed for the endemic equilibrium for the SIR model when the disease is driven by both the susceptible and infective class.

We have found that for the SIR endemic disease model, when the spatial spread of the susceptible is not negligible, the travelling wave front solution exist with non-negative u, v and w if $c^2 \geq 4(R_0 - 1)$, $R_0 > 1$ for the disease free equilibrium. Also that the wave with minimal speed $C = 2\sqrt{R_0 - 1}$ is the only one which can be stable as a

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solution of the original systems of partial differential equations and in dimensional variable C depending on the initial population since $R_0 = \frac{\beta N}{\gamma + b}$. Also, vaccination can be effected for the disease free steady state if $N \ll \frac{\gamma + b}{\beta}$. The travelling wave front for the endemic steady state (u^*, v^*, m^*, w^*) is difficult to characterize since all the zeros of (4.11) are non-negative.

References

- [1] Abramson G., Mathematical modelling of the spread of infectious diseases, A series of lectures given at PANDA, UNM (2001) unpublished.
- [2] Abramson G. and Kenkre V. M., Travelling waves of infection in the Hantavirus Epidemic, *Bulletin of Mathematical Biology* (2003), 65, pp. 519-534.
- [3] Abual-Rub M. S., Vaccination in a model of an epidemic, *international Journal of Mathematics and Mathematical sciences*, volume 23, No. 6 (2000), pp.425-429.
- [4] Britton N. F., *Essential Mathematical Biology*, Springer-Varlag London limited (2003), pp. 83-115; 147-172.
- [5] Burie J. B., Calonnec. A and Ducrot A., Singular perturbation Analysis of Travelling waves for a model in Phytopathology, *Mathematical Modelling of Natural Phenomena*, Vol. 1 No. 1 (2006): Population dynamics pp. 49-63.
- [6] Byrne H., Tutorial Notes on Model Building, LNS/EPSRC short course in Mathematical Biology, University of Manchester, UK (2002) – Unpublished.
- [7] Dickmann O and Heesterbeek J. A. P., *Mathematical Epidemiology of Infectious Diseases*, John Wiley & Sons Limited, Chichester, UK (2000).
- [8] Francesco M. D., *Mathematical Models in Life Sciences*, Lecture notes based on the book by J. D. Murray (2009), pp. 1-126.
- [9] Fowler A. C., *Techniques of Mathematical Modelling*, Mathematical Institute, Oxford University, United Kingdom (2004)- Unpublished.
- [10] Guardiola J. and Vecchio A., 1st di Genetica e Biofisica A. Buzzati Traverso-CNR, Napoli, Italy (2003).
- [11] Hethcote H. W., The Mathematics of infectious diseases, *SIAM Review*, 42 599-653 (2000).
- [12] Jing L. and Zou X., Modelling spatial spread of infectious diseases with a fixed latent period in a spatially continuous domain, *Bulletin of Mathematical Biology* (2009).
- [13] Jordan D. W. and Smith P., *Non linear ordinary differential equation*, Oxford University press Inc., New York (1999).
- [14] Lewis M., Renclawowicz J. and Driessche V., Travelling waves and spread rates for a west Nile model, *Bulletin of Mathematical Biology* (2006) 68; 3-23.
- [15] Li, M. Y and Muldowney J. S. Global Stability for the SEIR model in *Epidemiology, Math. Biosci.*, 125-155-164 (1995).
- [16] Murray, J. D., *Mathematical Biology*, Springer, Berlin (1983).
- [17] Real L. A. and Biek R., Spatial dynamics and genetic of infectious diseases on heterogeneous Landscape, *Journal of the Royal society interface* (2007) 4, 935-948.
- [18] Reluga T., A two-phase Epidemic driven by Diffusion Department of Applied Mathematics, University of Washington, Seattle, USA (2004), pp. 1-37.
- [19] Reluga T. C. Medlock J. and Galvani A. P., A model of spatial Epidemic spread when individuals move within overlapping Home ranges, Department of Epidemiology and Public Health, Yale University School of Medicine, New Haven (2005) – Unpublished.

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