Proceedings of the 6th WSEAS Int. Conf. on NEURAL NETWORKS, Lisbon, Portugal, June 16-18, 2005 (pp126-133)

Dynamics and Travelling Waves in CNN Vector-Disease Model

Angela Slavova
Institute of Mathematics
Bulgarian Academy of Sciences
Sofia 1113, Bulgaria

Abstract - **In this paper a CNN vector-disease model is investigated from the point of view of its dynamics. Existence of the travelling waves for such model is proved.**

1 INTRODUCTION

In his pioneering work, Fisher [1] used a logistic-based reaction-diffusion model to investigate the spread of an advantageous gene in a spatially extended population. Kermack and McKendrik [4] proposed a simple deterministic model for a directly transmitted viral or bacterial agent in a clossed population consisting of susceptibles, infectives and recovereds.

Some vector-borne diseases as malaria, yellow fever, typhus, which arrive and spread in new areas are one of the main public health problems throughout the world. The investigations of the spatial spread of newly introduced diseases are interesting and challenging for both theory and applications.

We will consider a host in a bounded region $\mathcal{V} \in \mathbf{R}^N (N \leq 3)$, where a disease is carried by a vector. The host is divided into two classes - susceptible and infectious, and the vector population is divided into three classes- infectious, exposed and susceptible. Let us denote by $v(t,x)$ - normalized spatial density of infectious host at time t in x, and by $w(t,x)$ - normalized spatial density of susceptible host at time

t in x. Mention that normalization is done with respect to the spatial density of the total population, therefor we have $v(t,x)+w(t,x)=1$, $(t,x)\in \mathbf{R}_+\times\mathcal{V}$, $\mathbf{R}_+=[0,\infty)$. We define $IV(t,x)$ as normalized spatial density of infectious vector at time t in x, and $SV(t,x)$ as normalized spatial density of susceptible host at time t in x. Let β denotes the host-vector constant rate, then the density of new infections in host is given by $\beta w(t,x)IV(t,x)=\beta[1-v(t,x)]IV(t,x)$. We obtain the following equation

$$\frac{\partial v}{\partial t}(t,x)=D\Delta v(t,x)-bv(t,x)+ \qquad (1)$$
$$+\beta[1-v(t,x)]IV(t,x),$$

where $bv(t,x)$ is the rate at which the density of infectious vanishes, D is a diffusion constant, Δ is the Laplacian operator. In equation (1) we can substitute $IV(t,x)$ by

$$IV(t,x)=\int_0^\infty\int_{\mathcal{V}}\xi(t,s,x,y)SV(t-s,y).$$
$$.\eta(s)dy\,ds=$$
$$=\int_0^\infty\int_{\mathcal{V}}\xi(t,s,x,y)h\eta(s)v(t-s,y)dy\,ds,$$

where h is a positive constant, $\xi(t,s,x,y)$ is the proportion of vectors that arrive in x at time t, starting from y at time $t-s$, $\eta(s)$ is the proportion of vectors that are still infectious s units of time after they became exposed. After substituting $IV(t,x)$ into (1), changing the limits and denoting $d=\beta h$, $F(t,s,x,y)=\xi(t,s,x,y)\eta(s)$ we obtain the following diffusive integro-differential equation modelling the vector disease:

$$\frac{\partial v}{\partial t}(t,x)=D\Delta v(t,x)-bv(t,x)- \qquad (2)$$
$$-d[1-v(t,x)]\int_{-\infty}^t\int_{\mathcal{V}}F(t,s,x,y).$$
$$.v(s,y)dyds,(t,x)\in\mathbf{R}_+\times\mathcal{V}.$$

The initial and boundary conditions are:

$$v(\Theta,x)=\Phi(\Theta,x),(\Theta,x)\in(-\infty,0]\times\mathcal{V}, \qquad (3)$$

$$\frac{\partial v}{\partial n}(t,x) = 0, (t,x) \in \mathbf{R}_+ \times \mathcal{V},$$

where Φ is a continuous function for $(\Theta, x) \in (-\infty, 0] \times \mathcal{V}$, $\frac{\partial}{\partial n}$ is the outward normal derivative on $\partial\mathcal{V}$. The convolution kernel $F(t,s,x,y)$ is a positive continuous function in its variables $t \in \mathbf{R}$, $s \in \mathbf{R}_+$, $x, y \in \mathcal{V}$, and $\int_0^\infty \int_{\mathcal{V}} F(t,s,x,y)dy\,ds = 1$. We will suppose that the convolution kernel F has the following form $F(t,s,x,y) = \delta(x-y)\delta(t-s)$. Therefore we derive from (2) the following reaction- diffusion model:

$$\frac{\partial v}{\partial t} = D\Delta v(t,x) - bv(t,x) + \tag{4}$$
$$d[1 - v(t,x)]v(t,x), (t,x) \in \mathbf{R}_+ \times \mathcal{V}.$$

In Section 2 we shall introduce the appropriate CNN representation of the vector-disease model (4). The dynamics of this model will be studied by using the describing function technique. In Section 3 the existence of travelling waves of our CNN vector-disease model will be proved.

2 CNN MODEL AND ITS DYNAMICS

It is known [6] that some autonomous CNNs represent an excellent approximation to nonlinear partial differential equations (PDEs). In this section we will present the model (4) by a reaction-diffusion CNNs. The intrinsic space distributed topology makes the CNN able to produce real-time solutions of nonlinear PDEs. Consider the following well-known PDE, generally referred to us in the literature as a reaction-diffusion equation [1]:

$$\frac{\partial u}{\partial t} = f(u) + D\nabla^2 u,$$

where $u \in \mathbf{R}^N$, $f \in \mathbf{R}^N$, D is a matrix with the diffusion coefficients, and $\nabla^2 u$ is the Laplacian operator in $\mathbf{R}^2$. There are several ways to approximate the Laplacian operator in discrete space by a CNN synaptic law with an appropriate A-template. In our case we will take one-dimensional discretized Laplacian template:

$$A : (1, -2, 1).$$

Therefore the CNN representaion for our vector-disease model (4) will be the following:

$$\frac{dv_j}{dt} = (v_{j-1} - 2v_j + v_{j+1}) - \tag{5}$$
$$-bv_j + d(1 - v_j)v_j, 1 \leq j \leq N.$$

The above equation is actualy ordinary differential equation which is identified as the state euqation of an autonomous CNN made of N cells. For the output of our CNN model we will take the standard sigmoid function [2]. Let us denote by $N(v_j) = d(1 - v_j)v_j - bv_j$ the nonlinear part of this equation. We shall study the dynamics and the stability properties of (5) by using the describing function method [5]. Applying the double Fourier transform:

$$F(s, z) = \sum_{k=-\infty}^{k=\infty} z^{-k} \int_{-\infty}^{\infty} f_k(t) exp(-st) dt,$$

to the CNN equation (5) we obtain:

$$sV(s, z) = z^{-1}V(s, z) - 2V(s, z) + zV(s, z) + \tag{6}$$
$$+N(s, z).$$

According to describing function technique [5] the transfer function in this case is

$$H(s, z) = \frac{1}{s - z^{-1} + 2 + z}.$$

We are looking for possible periodic solutions of our CNN model (5) of the form:

$$v_j(t) = V_{m_0} sin(\omega_0 t + j\Omega_0) \tag{7}$$

If we take periodic boundary conditions [6] for our CNN model (5) we obtain the following values of the spatial frequency

$$\Omega_0 = \frac{2\pi k}{N}, 0 \leq k \leq N - 1. \tag{8}$$

After substituting $s = i\omega_0$, $z = exp(i\Omega_0)$ in the transfer function and deriving its real and imaginary part we obtain equations together with (8) for the unknowns ω_0, Ω_0 and V_{m_0}. According to the describing function technique [5] the following proposition then hold:

Proposition 1 *CNN equation (5) for the vector-disease model (4) with circular array of N cells has periodic solution $v_j(t)$ with a finite set of spatial frequences $\Omega_0 = \frac{2\pi k}{N}$, $0 \leq k \leq N$ and a period $T_0 = \frac{2\pi}{\omega_0}$.*

In order to complete our stability analysis we will define the equilibrium points of the CNN model (5). Let us rewrite (5) in the following way:

$$\frac{dv}{dt} = A * v + \mathcal{N}(v) = \mathcal{F}, \tag{9}$$

where v is N dimensional vector with elements $v_j(t)$, A is one dimensional discretized Laplacian template $(1-21)$, $*$ is convolution operator. According to the theory of dynamical systems [3] the equilbrium points of (9) are those satifying:

$$\mathcal{F}(v^e) = A * v^e + \mathcal{N}(v^e) = 0.$$

There are two steady states: $v_1^e = 0$ and $v_2^e = \frac{d-b}{d}$. The Jacobian matrix of the equilibrium points can be computed by the following formulae:

$$J_{ps} = \frac{\partial \mathcal{F}_p}{\partial v_s}|_{v=v^e}, 1 \leq p, s \leq N.$$

Then the following theorem hold for the stability of the steady states:

Theorem 1 *For our CNN model (5) with periodic boundary conditions:*

i). if $0 < d \leq b$ then the steady state $v_1^e = 0$ is asymptotically stable and $v_2^e = \frac{d-b}{d}$ is either stable or unstable for $0 \leq v \leq 1$;

ii). if $0 \leq b < d$ then the steady state $v_1^e = 0$ is either stable or unstable and $v_2^e = \frac{d-b}{d}$ is asymptotically stable in the interval $0 \leq v \leq 1$.

Proof: The Jacobian matrix J of the equilibrium points in our case is $J = A - (b-d)I_d - 2dv^e$. Then for the steady state $v_1^e = 0$ we have for the eigenvalues of J:

$$\sum_{q=1}^{N} \lambda_q = trace(A - (b-d)I_d) = (-2 - (b-d)),$$

which is negative for $d \leq b$ and either negative or positive for $b < d$. For the steady state $v_2^e = \frac{d-b}{d}$ the eigenvalues of the Jacobian J are:

$$\sum_{q=1}^{N} \lambda_q = trace(A + (b-d)I_d) = (-2 + (b-d)),$$

which will be either positive or negative for $d \leq b$, and negative for $b < d$. Theorem is proved.

Remark 1 Recall that d represents the contact rate and b represents the recovery rate. The above stability results indicate that there is a threshold at $d = b$. If $d \leq b$, then the proportion v of infectious individuals tends to zero as t becomes large and disease dies out. If $d > b$, the proportion of infectious individuals tends to an endemic level $v_2 = \frac{d-b}{d}$ as t becomes large. There are periodic solutions in the region $0 \leq v \leq 1$.

3 TRAVELLING WAVES OF THE CNN MODEL

Let us consider our CNN equation (5). The travelling wave solutions will be presented in the following form:

$$v_j(t) = v(\eta), 1 \leq j \leq N, \tag{10}$$

where $\eta = t - jh$, $h > 0$ is a parameter. Note that η is the coordinate moving along the array with a velocity equal to $c = 1/h$. Substituting (10) in (5) we obtain

$$\dot{v} = v(\eta - h) - 2v(\eta) + v(\eta + h) + \mathcal{N}(v),$$

where the dot denotes differentiation with respect to η. The two difference terms $[v(\eta - h) - v(\eta)] - [v(\eta) - v(\eta + h)]$ can be replaced approximately by the first derivatives: $-\dot{v}/h$ and $+\dot{v}/h$, respectively. Hence, we obtain

$$\dot{v} = \frac{1}{1 + 2c}\mathcal{N}(v). \tag{11}$$

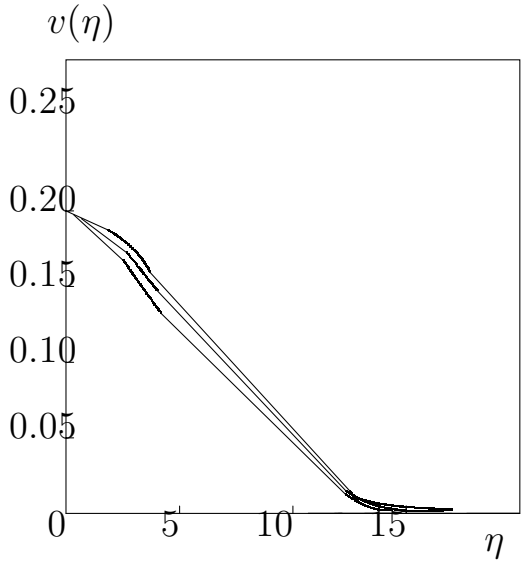
Clearly, $v \equiv 0$ and $v \equiv \frac{d-b}{d}$ are solutions of the stationary problem. So there are two equilibria $E_0 = (0, 0)$ and $E_1 = (\frac{d-b}{d}, 0)$. The following theorem for the travelling waves of vector-disease CNN model (5) hold:

Theorem 2 *For vector-disease CNN model (5), there exists $c > 0$ and $d > b$, such that there is a heteroclinic orbit connecting the equilbria E_0 and E_1 and the travelling wave $v(\eta)$ is strictly monotonically decreasing.*

Proof: We require a trajectory from $(0,0)$ to $(\frac{d-b}{d},0)$ in the phase plane remaining in the strip $0 \leq v \leq 1$. Any such wave front must be monotonic. The equilibria $(0,0)$ and $(\frac{d-b}{d},0)$ cannot be centers or foci, since the solutions close to such points must oscillate. Trajectories which pass from one equilibria to another are known as heteroclinic orbits [3]. It is easy to see that (11) has a heteroclinic solution $v^*(\eta)$ from E_0 to E_1 for the certain parameter values $d > b$. This solution corresponds to a travelling wave of the vector disease CNN equation (5) which satisfies:

$$lim_{\eta \to -\infty} v^*(\eta) = E_0, lim_{\eta \to +\infty} v^*(\eta) = E_1.$$

We obtain the following travelling front for the wave equation (11), here $b = 3.8$, $d = 4.8$ and $c = 2.0 - 2.4$:



References

[1] N.F.Britton, "Reaction-Diffusion Equations and Their Applications to Biology", Acad. Press, New York, 1986.

[2] L.O.Chua, L.Yang, "Cellular neural networks: Theory", IEEE Trans. Circuits Syst.,35:1257-1272,1988.

[3] Fenichel N., Geometric singular perturbation theory for ordinary differential equations, J.Diff.Eqns 31, pp. 53-98, 1979.

[4] J.Guckenheimer, Ph.Holmes, "Nonlinear Oscillations, Dynamical Systems, and Bifurcations of Vector Fields", Springer, 1983.

[5] W.O.Kermack, A.G.McKendrik, "A contribution to the mathematical theory of epidemics", Proc.R.Soc.Lond. A 115, pp.700-721, 1927.

[6] A.I.Mees, "Dynamics of Feedback Systems", Wiley, London, 1981.

[7] A.Slavova, "Applications of some mathematical methods in the analysis of celullar neural networks", J.Comp.Appl.Math. 114, pp. 387-404, 2000.