A DISCRETE TIME MODEL FOR EPIDEMIC SPREAD: TRAVELING WAVES AND SPREADING SPEEDS

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ABSTRACT. In this paper, we aim to study spread of an epidemic in a spatially stratified population with non-overlapping generations. We consider mean field equation of an endemic chain-binomial process and allow individuals to disperse in the spatial habitat. To be able to model the spatial movement, we used an averaging kernel. The existence of traveling waves for traveling wave speeds greater than a certain minimum is proved. In addition, an explicit formula for the critical wave speed is given in terms of the moment generating function of the dispersal kernel and the basic reproductive ratio of the infectives.

1. INTRODUCTION

Mollison [14] modeled infectious disease spread by using a nonlocal contact model and this study triggered many other studies concerning the invasion of a new territory by intruder species. Mollison's model does not allow individuals to disperse. Medlock and Kot [13] compared the nonlocal contact model and distributed infectives model. Both of these approaches assume that the dynamics of the populations under consideration are governed by continuous time processes.

On the other hand, discrete time models have also been used to model dynamics of epidemic processes [1, 2]. One of the most popular processes is the chain-binomial models(see for example [7, 11].) Chain binomial processes assume that the population has non-overlapping generations. Recently extinction time of a generalized endemic model with no immunity against the disease is studied using its mean field dynamics [5]. The mean field equation of the model, not surprisingly, is a difference equation.

Ecological difference equations with dispersal have been studied by Kot [9] in terms of traveling waves. Traveling waves in ecological processes are used to study invasion of a new habitat by an intruder species. Similarly in continuous time epidemiological processes such solutions have been studied to determine the conditions

for invasion of a previously occupied habitat by infective individuals. For continuous time systems, the analysis of epidemic spread in terms of traveling waves and spreading speeds have been studied by many authors (see *e.g.* [17] and references therein.)

Here we consider the mean field equation of a generalized endemic chain-binomial process as given in [5] with spatial dispersal. This equation keep tracks of the frequency of infective individuals in discrete generations. We also allow individuals to disperse in the spatial habitat. The resulting equation is an integro-difference equation describing an epidemic process with no immunity against the disease.

Given a spatially inhomogeneous epidemic model it is natural to look for traveling wave solutions. We show that there exists a critical traveling wave speed such that if the traveling wave speed exceeds this value there exists a solution connecting the disease free equilibrium and the endemic equilibrium. We also show that the model is linearly determinate, and thus we are able to give an explicit formula to calculate the critical wave speed in terms of the basic reproductive ratio of the model and the moment generating function of the dispersal kernel.

The paper is organized as follows: In section 2, we briefly introduce our model and some of its properties. In section 3, we give our main results concerning the invasion of the spatial habitat by infective individuals. In section 4, we provide simulation results concerning the model and verify that our theoretical findings are in accordance with the simulation results. In addition, the effect of parameters, range of nonlocality and basic reproductive ratio, is investigated numerically. In section 5, we conclude our paper.

2. Model

Given a contact of a susceptible in period t, the probability that it is with an infective is $\iota_t = I_t/(N-1)$ where I_t denotes the number of infectives in a population of size N. More generally, the probability that no effective contact with susceptibles can be taken as a function of $1 - \iota_t$. To define the function we follow the method suggested in [7] *i.e.* we assume that this function is a probability generating function of a discrete distribution and has the following form:

$$f(x) = \sum_{k=0}^{\infty} p_k x^k$$

where p_k is the probability that a susceptible makes k contacts during a time interval.

Assume that all infected individuals I_t at time t will return to susceptible class at time $t + 1$. In this case, there is no removed state so that $S_t + I_t = N$ for any $N \in \mathbb{N}$ where S_t is the number of susceptible individuals in the population. In [5], the Markov chain modeling this process was taken by the following conditional

probability distribution:

$$Pr\left(I_{t+1} = x_{t+1} | I_t = x_t\right) = \binom{N - x_t}{x_{t+1}}(1 - f(1 - \imath_t))^{x_{t+1}}(f(1 - \imath_t))^{N - x_t - x_{t+1}}.$$

We consider the mean field equation of the above defined Markov process as following difference equation:

$$i_{t+1} = (1 - i_t)(1 - f(1 - i_t)) =: g(i). \tag{2.1}$$

As an easy consequence of [5, Theorem 5], one can easily verify that for any $\varepsilon > 0$ and $T \in \mathbb{N}$

$$\lim_{N \to \infty} Pr\left(\max_{1 \le n \le T} |\imath_n - i_n| > \varepsilon\right) = 0$$

where $\imath_n = I_n/(N - 1)$.

Hence, trajectories of the stochastic model and its mean field equation stay close to each other for any finite time with probability 1. Define the basic reproductive number as follows:

$$\mu = f'(1).$$

This value plays an important role in the dynamics of mean field equation (2.1). In [5], conditions for the existence and stability of an endemic equilibrium are given using the basic reproductive number as follows:

Theorem 1. [5] *The following statements hold for any initial condition $i_0 \in (0, 1)$.*

(i) *If $\mu \le 1$ then solutions to (2.1) approach the disease-free equilibrium i.e. $\lim_{t \to \infty} i_t = 0$.*

(ii) *If $\mu > 1$ then solutions to (2.1) approach the unique endemic equilibrium $i_e \in (0, \frac{1}{2})$ i.e. $\lim_{t \to \infty} i_t = i_e$.*

Equation (2.1) does not allow spatial movements of individuals. To be able to consider such movements, we denote the frequency of infective individuals at spatial location x and time t by $i_t(x)$. Changes in these frequencies are modeled in two alternating steps. In the first step $i_t(x)$ is mapped into $g(i_t(x))$ The second stage is spatial shuffling. This stage is called dispersal stage and modeled by an integral operator. Hence the model is given as an integro-difference equation of the form:

$$i_{t+1}(x) = \int_\Omega k(x, y)g(i_t(y))\, dy.$$

Here the kernel $k(x, y)$ describes the dispersal of individuals from y. In particular, it is the probability that an infective individual in an interval of length dy about y disperses to an interval of the same length about x. Since $k(x, y)$ is a probability kernel, it must be nonnegative. In a biological point of view, It is reasonable to assume that the dispersal weight depends on the relative distance i.e. $k(x, y) = k(x - y)$.

Here we assume also that the domain is infinite and the redistribution or dispersal kernel has exponentially bounded tails. This is to say that the moment generating function of the probability kernel exists.

We state our standing assumptions on the probability generating function f and moment generating function of the dispersal kernel k as follows:

Assumption 1.

- *Random variables with probability generating function f and k have their first and second moments.*
- *There exists a $\phi_0 > 0$*

$$\int_{\mathbb{R}} e^{\phi y} k(y) \, dy < \infty$$

  for all $|\phi| \leq \phi_0$.
- *Dispersal kernel k is piecewise continuous.*

3. Traveling waves and spreading speeds

Consider the following integro-difference equation

$$i_{t+1}(x) = Q[i_t](x) := \int_{\mathbb{R}} k(x, y) g(i_t(y)) \, dy. \tag{3.1}$$

Simple traveling waves are the solutions satisfying

$$i_t(x) = I(x - ct) \tag{3.2}$$

for some constant c. This constant is called traveling wave speed. In this setting, each iterate yields an extension of the solution with no change in the shape of it. Plugging the equation (3.2) in (3.1) one obtains

$$I(x - c) = \int_{\mathbb{R}} k(x - y) g(I(y)) \, dy. \tag{3.3}$$

Here it is easy to see that the constant solutions to this equation are given by $I^* = 0$ and $I^* = i_e$. Here we consider the traveling wave equation (3.3) with the following boundary conditions:

$$I(-\infty) = i_e \text{ and } I(\infty) = 0. \tag{3.4}$$

For the existence of such solutions see Appendix A. To be able to compute the critical wave speed explicitly we need to show the operator is linear determinate *i.e.*

$$g(i) \leq \mu i.$$

To be able to show this recall Bernoulli's inequality:

$$(1 - i)^n \geq 1 - in$$

for $n \geq 1$. Using this, we have the following inequality:

$$1 - f(1-i) \quad \leq \quad 1 - p_0 - \sum_{k=1}^{\infty} p_k(1-ik) = \mu i.$$

Thus we have $g(i) \leq \mu i(1-i) < \mu i$ for $i \in (0,1)$.

Linear determinacy of this map implies that the critical wave speed can be characterized using the linearization of equation (3.1) near the equilibrium point 0 [19]. The critical wave speed c^* is the slowest value of the traveling wave speed c for which we can find a positive solution connecting two equilibria 0 and i_e.

The linearization (or the Frechet derivative) of the of (3.1) near 0 is given by

$$I(x-c) = \mathcal{M}[u](x) := \mu \int_{\mathbb{R}} k(x-y)I(y) \, dy. \tag{3.5}$$

For a moving wave in negative direction, one can consider the following ansatz

$$I(x) = A \exp(-\phi x) \tag{3.6}$$

for ϕ positive. Plugging (3.6) in (3.5), one obtains the characteristic equation:

$$\exp(\phi c) = \mu \int_{\mathbb{R}} k(s) \exp(\phi s) \, ds. \tag{3.7}$$

Since we require that $I(x)$ decussates the disease-free equilibrium 0, the appearance of such a solution coincides with the first appearance of a double root for (3.7). Define the following function

$$\Psi(\phi) = \phi c - \log(\mu) - \log \left(\int_{\mathbb{R}} k(s) \exp(\phi s) \, ds \right). \tag{3.8}$$

First observe that $\Psi''(0) < 0$ and hence we can conclude that $\Psi''(\phi) < 0$ for all $|\phi| \leq \phi_0$ for some $\phi_0 > 0$. Thus the double root of this equation can be calculated by taking $\Psi = 0$. Then, one can obtain a formula for the critical traveling wave speed which is given by

$$c^* = \min_{\phi > 0} \left\{ \frac{1}{\phi} \log \left[\mu \int_{\mathbb{R}} k(s) \exp(\phi s) \, ds \right] \right\} \tag{3.9}$$

We have the following two results regarding the long time behavior of the model (3.3).

Theorem 2. *If $c \geq c^*$, there exists solution of the equation (3.3) satisfying the boundary conditions (3.4).*

The result follows from [19, Theorem 6.6] and hypotheses are verified in Appendix A. This theorem implies that there exists a traveling wave solution connecting two equilibria 0 and i_e. Biological interpretation of the result is that infectives in the

population cannot spread in the population if traveling wave speed is larger than the critical wave speed c^*.

To be able to get the whole picture we give the following result related to the operator (3.1) that informs us about both of the cases $c < c^*$ and $c > c^*$.

Theorem 3. *Suppose that the initial function $0 \leq i_0(x) \leq 1$ is zero for sufficiently large x and $i_0(x) \geq \rho$ for some positive constant ρ and all sufficiently negative x. Then for any positive ε the solution $i_n(x)$ of the recursion (3.1) has the following properties:*

- $\lim_{n \to \infty} \sup_{x \geq n(c^*+\varepsilon)} i_n(x) = 0,$
- $\lim_{n \to \infty} \sup_{x \leq n(c^*-\varepsilon)} \left(i_e - i_n(x) \right) = 0.$

We used the theory developed in [10] to obtain above result and the conditions are verified in Appendix A. The last limit tells us that infectives can spread at a speed no higher than the critical wave speed c^*.

4. SIMULATION STUDIES

In this section, we turn to simulation studies on the effects of model parameters on epidemic spread. We are interested in effect of two parameters namely the basic reproductive number μ for the deterministic equation (2.1) and the range of nonlocality.

We concentrate on the normal distribution:

$$k(x - y) = \frac{1}{\sqrt{2\sigma^2 \pi}} \exp\left(- \frac{(x-y)^2}{2\sigma^2} \right) \tag{4.1}$$

for simulation studies. This distribution is centered around zero and parameter σ characterizes the range of nonlocality. Here we also need to specify the discrete distribution *i.e.* its mass function f. Here we use Poisson distribution for computation purposes. Hence we chose

$$f(x) = \exp\left(- \mu(1 - x) \right)$$

Thus our model without spatial dispersal can be read as

$$i_{t+1} = (1 - i_t)(1 - \exp(-\mu i_t)). \tag{4.2}$$

In our numerical simulations, we consider (3.3) corresponding to equation (4.2) with symmetric boundary conditions and use the discretization of the space variable $-50 \leq x \leq 50$ with a mesh interval of $\Delta x = 0.01$. The convolution terms is approximated by fast Fourier transform.

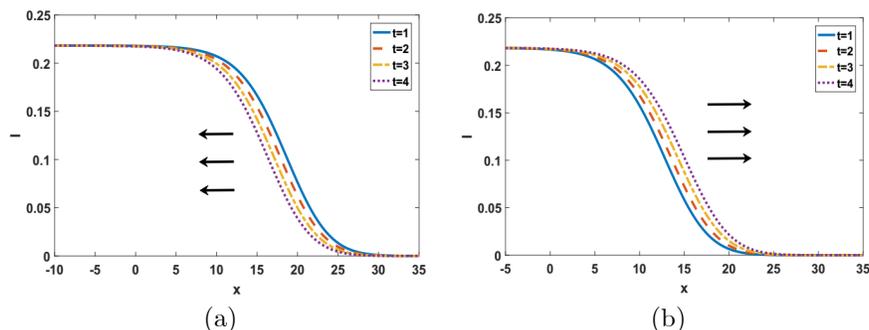(a)   (b)

FIGURE 1. Panels (a) and (b) display the existence and non existence of traveling waves. The model parameters chosen here are $\mu = 1.5$ and $\sigma = 0.5$. We set $c = 2$ for Panel (a) and $c = 0.2$ for panel (b).

For demonstration, we set $\mu = 1.5$ and $\sigma = 0.5$. For these values, the critical wave speed can be calculated as $c^* = 0.495$ using the formula (3.9). Theorem 2 implies that traveling waves exist only for traveling wave speed c larger than this critical value. Panel (a) in Figure 1 shows the formation of a traveling wave whose front towards the negative direction as time evolves, and panel (b) suggests that no traveling waves along the negative direction are formed and the solution converges to constant i_e on the domain. Hence, infective individuals spread in sustains in panel (b) in Figure 1 and they diminish for large traveling wave speeds as shown in panel (a) of the same figure.
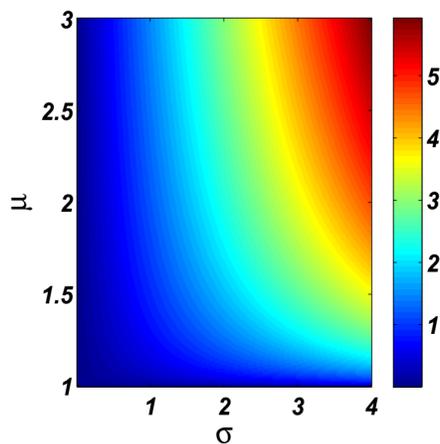

FIGURE 2. Effect of the parameters μ and σ on the critical wave speed c^*.

Here it is clear that the critical wave speed c^* measure the likelihood that infectives vanish in a population *i.e.* infection diminishes when the critical wave speed

is small. For our models (3.3) or (3.1), the critical speed is a function of two parameters, namely the basic reproductive ratio μ and the range of nonlocality σ. To be able to further investigate and illustrate the effect of these parameters we calculated the critical wave speed for a set of parameters. Here it is clear that the critical wave speed c^* measures the likelihood that infectives vanish in a population $i.e.$ infection diminishes when the critical wave speed is small using the kernel function (4.1) with moment generating function $\exp(\sigma^2 x^2/2)$.

As seen in Figure 2, the critical waves speed increases with the parameters μ and σ. As expected, this implies that the higher rate of infection favors the spread of infectives. On the other hand, as the range of nonlocality gets larger, the spread of infectives is favored again.

5. Conclusion

In this paper, we considered spatial extension of an epidemic process derived as mean field equation of a stochastic process. The resulting equation is an integro-difference equation modeling nonlocal dispersal of infective individuals.

We analyze the deterministic model on an unbounded region. We focus on studying traveling wave speed and/or the asymptotic speed of propagation. It has been shown that the model is *linearly determinate* which allows us to calculate the critical wave speed explicitly. Our main results can be summarized as follows:

(1) We investigate the critical traveling wave speed (or the spreading speed) as a function of extend of nonlocality of the dispersal kernel and the rate of infection. As a result, we found general conditions on the spread of infective individuals in terms of the critical traveling wave speed.

(2) We numerically studied the model and showed that the critical traveling wave speed increases as the range of nonlocality of the dispersal kernel and/or the rate of infection grows. Larger model parameters favors the spread of infectives.

One can extend these results to the multidimensional epidemic models using the theories developed in [10, 12]. These models include the multinomial AIDS models as presented in [18, pp. 89-92]. Another interesting question involves the study of asymptotic behavior of spatial generalization of SEIR type endemic processes with fractional time derivative [6, 15] .

Appendix A. Proof of Theorem 3

Here we sketch the proof of Theorem 3 by using the theory developed by Li et. all. [10] and Weinberger [19] Existence and uniqueness of solutions are clear. This also implies that map Q has semigroup property.

First of all note that for any constant β, $Q[\beta] = f(\beta)$. In addition it is easy to show that f maps $[0, 1]$ to itself. It was shown in [5, Theorem 1], $f(\beta) > \beta$ for $\beta \in (0, i_e)$ and $f(\beta) < \beta$ for $\beta \in (i_e, 1)$. Now we would like to note that the map Q satisfies the hypotheses presented in [10, 19].

    i. Suppose that $u \leq v \leq i_e$ $a.e.$ for some $u, v \in \mathcal{B}$. Then we have

$$Q[u] - Q[v] \quad = \quad \int_{\mathbb{R}} k(x - y)\bigg(g\big(u(y)\big) - g\big(v(y)\big)\bigg)\, dy.$$
(A.1)

    Recall that the endemic equilibrium i_e s globally stable and hence the map g is increasing for any function $u \leq i_e$. Then one can easily conclude that $g(u) - g(v) \leq 0$ for any $u \leq v \leq i_e$. Since the above given integrand is non-positive, we have the order preserving property of the recursion as desired.

    ii. It is clear that for our model there are only two spatially homogeneous fixed points 0 and i_e. It is easy to see that i_e is an asymptotically stable fixed point of Q_t for any constant initial condition by Theorem 1.

    iii. If $i_t(x)$ is a solution to equation (3.1) so does $v_t(x) = i_t(x - y)$. Hence map Q is translation invariant.

    iv. Continuity can be obtained using [16, Theorem 5.3].

    v. Existence of a subsequence v_{n_k} such that $Q[v_{n_k}]$ converges uniformly on a bounded set follows from Arzela-Ascoli theorem.

Consider the map Q and choose a continuous function with the following properties:

- $\phi(x)$ is non-increasing in x;
- $\phi(x) = 0$ for all $x \geq 0$;
- $0 \ll \phi(-\infty) \ll i_e$

To be able to define the spreading speed, let $a_0(c; s) = \phi(s)$ and define the sequence $a_n(c; s)$ by the recursion;

$$a_{n+1}(c; s) = \max\{\phi(s), Q[a_n(c; s)](s + c)\}.$$
(A.2)

    The above defined operator is also order preserving. By definition, it is easy to see that $a_1 \geq a_0 = \phi(s)$. It can be shown that $a_n \leq a_{n+1} \leq i_e$ by induction. Moreover, sequence $a_n(c; s)$ is not increasing in c an s. Thus the sequence a_n converges to a limit function $a(c; s)$ non-increasing in c and s and bounded by i_e. It follows from Lui's argument [12] that $a(c; \pm\infty)$ are equilibrium of the map Q. In particular $a(c; \pm\infty) = i_e$ as noted in [10].

    Now consider another initial condition $\hat{\phi}$ satisfying the above given properties. Hence there is another sequence $\hat{a}_n(c; s)$ whose limit function can be denoted by $\hat{a}(c; \infty)$. As discussed in [10], one can show that $a_N(c; x - \tau) \geq \hat{\phi}(x)$ for some integer N and a translation τ. Hence, by comparison principle, it can be shown that $\hat{a}(c; \infty) = a(c; \infty)$. Hence we conclude that vector $a(c; \infty)$ is independent of the choice of the initial function ϕ. We then define the slowest spreading speed by

$$c_+^* = \sup\{c : a(c; \infty) = 1\}.$$
(A.3)

Hence the result follows from [10, Theorem 2.2] for map Q. It is also possible to show that c_+^* is equal to critical traveling wave speed c^* given in (3.9) for the recursion (3.3) follows from [20, Theorem 3.4].

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