

ASYMPTOTIC SPEED OF SPREAD AND TRAVELING WAVES FOR A NONLOCAL EPIDEMIC MODEL

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(Communicated by Hal Smith)

ABSTRACT. By applying the theory of asymptotic speeds of spread and traveling waves to a nonlocal epidemic model, we established the existence of minimal wave speed for monotone traveling waves, and show that it coincides with the spreading speed for solutions with initial functions having compact supports. The numerical simulations are also presented.

1. Introduction. Consider the epidemic model proposed in [10, 8]

$$\begin{cases} \partial_t u_1(t, x) &= d\Delta u_1(t, x) - a_{11}u_1(t, x) + a_{12}u_2(t, x), \\ \partial_t u_2(t, x) &= -a_{22}u_2(t, x) + g(u_1(t, x)), \end{cases} \quad (1)$$

where d, a_{11}, a_{12} and a_{22} are positive constants, $u_1(t, x)$ and $u_2(t, x)$ denote the spatial densities of infectious agents and the infective human population at time $t \geq 0$, respectively. $1/a_{11}$ is the mean lifetime of the agents in the environment, $1/a_{22}$ is the mean infectious period of the infective human, a_{12} is the multiplicative factor of the infectious agents due to the human population, $g(u_1)$ is the force of infection on human population due to a concentration u_1 of the infectious agents. This model has some basic assumptions: (i) the total susceptible human population is large enough, with respect to the infective population, to be considered as constant; (ii) the infectious agents diffuse randomly in the habitat Ω due to a particular transmission mechanism; (iii) the infective population at $x \in \Omega$ only contributes to the infectious agents at the same spatial point.

Note that some infection agents u_1 (e.g., bacteria or viruses in the air), at a spatial point x , depend on the infective humans u_2 not only at the spatial point x , but also at spatial neighbor points of x , and even points in the whole region Ω . As mentioned in [1], to deal with indirect transmission diseases, typical of infectious diseases transmitted via the pollution of the environment due to the infective population (typhoid fever, schistosomiasis, malaria, etc.), a different approach should be used to model the mechanism of production of the pollutants. A possible model is the one proposed in [5]. Assume that the growth rate of bacteria or pollutants due to the infective population can be modelled by

$$\int_{\Omega} K(x, y)u_2(t, y)dy, \quad x \in \Omega, t \geq 0,$$

2000 *Mathematics Subject Classification.* Primary: 35K57, 37B40; Secondary: 92D30.

Key words and phrases. Spreading speed, traveling waves, nonlocal epidemic model.

Supported in part by the NSERC of Canada.