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On a Cross-Diffusive SIS Epidemic Model with Singular Sensitivity

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Abstract. This work introduces a repulsive chemotaxis susceptible-infected-susceptible (SIS) epidemic model with logarithmic sensitivity and with mass-action transmission mechanism, in which the logarithmic sensitivity assumes that the chemotactic migration of susceptible populations is suppressed by large density of infected individuals while the biased movement is strongly sensitive to a density variation of small infected population. Under suitable regular assumption on the initial data, we firstly assert the global existence and boundedness of smooth solutions to the corresponding no-flux initial boundary value problem in the spatially one-dimensional setting. Second, we investigate the effect of strong chemotaxis sensitivity on the dynamics of solutions through extensive numerical simulations. Our numerical study suggests that although this chemotaxis model includes an unbounded force of infection, the blow-up of solutions cannot occur in two dimensions, which remains to be analytically verified. Moreover, the numerical studies on the asymptotic profiles of the endemic equilibrium indicate that the susceptible populations move to low-risk domains whereas infected individuals become spatially homogeneous when the repulsive-taxis coefficient is large. Furthermore, simulations performed in the one- and two-dimensional cases find that rich patterns, like periodic peaks, structured holes, dots and round circles, may arise at intermediate times, but eventually are smoothed out, and that clusters of infection can emerge in a heterogeneous environment. Additionally, our numerical simulations suggest that the susceptible population with larger chemosensitivity, tends to respond better to the infected population, revealing the effect of strong chemotaxis sensitivity coefficient on the dynamics of the disease.

AMS subject classifications: Primary: 35B40; Secondary: 35A01, 35K57, 92C17, 92D25

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1 Introduction

Transmission mechanisms play a key role in the dynamical behaviors of many infectious diseases. The traditional mass action assumption on incidence rate dates back to the pioneering Kermack-McKendrick model [16] in which the number of new infected hosts per unit area, per unit of time is βSI, where S and I are respectively the densities of susceptible and infective individuals, and β is the transmission coefficient. This classic mechanism is based on the homogeneous-mixing assumption: transmission occurs via direct contact between susceptible and infective hosts that mix completely with each other and move randomly in a fixed domain. As an alternative to mass action, frequency-dependent transmission term $\beta SI/(S+I)$ was proposed by de Jong [8]. This incidence rate arises from the random-mixing assumption: The probability that each susceptible individual S contacts the infection depends on the proportion $I/(S+I)$ of encounters involving infected individuals. However, there are some biological processes which lead to non-linearities in the transmission rates of parasites; for example, the transmission of parasites by intermediate hosts, such as biting arthropods, indicates some complex nonlinear infection mechanisms. On the other hand, there might exist physiological heterogeneity in susceptibility. Indeed, several theoretical studies have generalized the incidence rate βSI to a more general form $\beta S^p I^q$ with $p>0$ and $q>0$ (cf. [12, 18, 22, 30], for instance). In general, an approach to proposing appropriate forms of transmission function is to compare the fit of host-pathogen models to empirical data of observed diseases dynamics [22].

Environmental heterogeneity is another factor that has important impacts on the persistence and extinction of infectious diseases (see, e.g. [7, 11, 19, 20]). Since infection is almost always patchy in space, some patch models describing the spreading of infectious diseases have naturally been proposed (cf. [1, 4, 27, 29, 33, 34], for example).

Extensive studies on susceptible-infected-susceptible (SIS) epidemic models in the literature have demonstrated that the motilities of both susceptible and infected individuals closely link to outbreak and collapse of infectious diseases, and qualitative analysis in [1, 2, 5, 24] suggests that restricting the movement of susceptible individuals may eliminate the disease. Nevertheless, the authors in [36] pointed out that when the total size of the population is large, limiting the diffusion rate of infected individuals might be a better strategy for controlling the disease in a diffusive SIS model with mass-action-type transmission. Moreover, it has been revealed that the asymptotical profiles of the endemic steady state not only relate to the diffusion rates of susceptible and infected individuals but also depend on the disease transmission mechanisms: see [2, 24] for frequency-dependent incidence rate; cf. [5, 6, 9, 36] for mass-action transmission mechanism; and refer to [25] for a general nonlinear incidence function.