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ASYMPTOTIC DYNAMICS OF DETERMINISTIC AND STOCHASTIC EPIDEMIC MODELS WITH MULTIPLE PATHOGENS

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Abstract. Emerging diseases in animals and plants have led to much research on questions of evolution and persistence of pathogens. In particular, there have been numerous investigations on the evolution of virulence and the dynamics of epidemic models with multiple pathogens. Multiple pathogens are involved in the spread of many human diseases including influenza, HIV-AIDS, malaria, dengue fever, and hantavirus pulmonary syndrome [9, 15, 16, 23, 24, 27]. Understanding the impact of these various pathogens on a population is particularly important for their prevention and control. We summarize some of the results that have appeared in the literature on multiple pathogen models. Then we study the dynamics of a deterministic and a stochastic susceptible-infected epidemic model with two pathogens, where the population is subdivided into susceptible individuals and individuals infected with pathogen j for $j = 1, 2$. The deterministic model is a system of ordinary differential equations, whereas the stochastic model is a system of stochastic differential equations. The models assume total cross immunity and vertical transmission. The conditions on the parameters for coexistence of two pathogens are summarized for the deterministic model. Then we compare the coexistence dynamics of the two models through numerical simulations. We show that the deterministic and stochastic epidemic models differ considerably in predicting coexistence of the two pathogens. The probability of coexistence in the stochastic epidemic model is very small. Stochastic variability results in extinction of at least one of the strains. Our results demonstrate the importance of understanding the dynamics of both the deterministic and stochastic epidemic models.

Key Words. Epidemic model, multiple pathogens, stochastic differential equation, vertical transmission, cross immunity

1. Introduction

The spread and persistence of an infectious disease depend on a multitude of factors. For example, two important factors are pathogen virulence and transmissibility. Pathogens that are too virulent, that kill their host too quickly before transmission to another host, reduce their chance of survival and ultimately, the

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persistence of the disease. This view has led to what has been called "conventional wisdom." Conventional wisdom asserts that pathogens evolve toward reduced virulence [6]. Some well-known natural experiments and theoretical results support this view. More recent evidence suggests that this view is too simplistic. The evolution of virulence has received much attention in the literature (see, e.g., [18, 19, 21, 48] and references therein). Epidemic models with multiple pathogens have provided an important theoretical tool to investigate how complex interactions between host and pathogen affect pathogen evolution and disease persistence [1, 2, 8, 9, 11, 13, 15, 16, 17, 20, 23, 24, 31, 37, 40, 41, 42, 43, 44, 47].

1.1. Evolution of Virulence. A well-known natural experiment that supports conventional wisdom involves the rabbit population in Australia. The European rabbit was introduced into Australia about 1859 and within 20 years, the rabbit population had increased to such high densities that they were a serious problem [25, 26, 37]. Various methods of control were tried but none were very successful until 1950 when the myxoma virus was introduced. Initially, the mortality rate due to myxoma virus was estimated at 99.8% [25, 37]. But over time, the virulence changed. Virulence was graded on a scale from I to V, grade I being the most virulent. It was found in studies by Fenner and Ratcliffe [26] that from 1950 to 1965, the average virulence grade in the rabbit population changed from the most virulent, grade I, to an intermediate level of virulence, grade III (see [6, 25, 37]).

Some theoretical results that also support conventional wisdom come from the study of epidemic models. The basic reproduction number is a well-known threshold parameter in the mathematical epidemiology literature [7, 29, 50]. The basic reproduction number is the number of secondary infections caused by an infected individual in an entirely susceptible population. For a simple epidemic model, this threshold parameter can be defined as the transmission rate (number of adequate contacts per time that result in infection) multiplied by the length of the infectious period. Therefore, the basic reproduction number is

$$\mathcal{R}_0 = \frac{\beta}{d+\alpha}. \tag{1}$$

The parameter β is the transmission rate and the fraction $1/(d+\alpha)$ is the length of the infectious period, where d is the natural death rate and α is the disease-related death rate. The parameter α is often used as a measure of the virulence of the disease. Generally, an epidemic occurs when $\mathcal{R}_0 > 1$. When $\mathcal{R}_0 < 1$ the disease does not persist. It can be seen from the definition of $\mathcal{R}_0$ that if the pathogen is highly virulent, as measured by α, then $\mathcal{R}_0 < 1$. In this case, the disease and the pathogen do not persist. However, if the virulence is decreased, that is, α is decreased, so that $\mathcal{R}_0 > 1$, then the disease persists in the population. Unfortunately, the relationship between reduced virulence and disease persistence is not quite as simple as this discussion appears (see e.g., [6, 18, 19, 38, 44, 47]). The parameters in the definition of $\mathcal{R}_0$ are generally not independent. In fact, transmission may be correlated with virulence [6]. The evolution of virulence becomes even more complicated when the model includes multiple pathogen strains with differing levels of virulence and transmissibility. The edited volume [21] by Dieckmann, Metz, Sabelis, and Sigmund is a good reference for the current knowledge of evolutionary ecology of infectious diseases and management goals for virulent pathogens.