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Research paper

Dynamics of a stochastic multi-strain SIS epidemic model driven by Lévy noise

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ABSTRACT

A stochastic multi-strain SIS epidemic model is formulated by introducing Lévy noise into the disease transmission rate of each strain. First, we prove that the stochastic model admits a unique global positive solution, and, by the comparison theorem, we show that the solution remains within a positively invariant set almost surely. Next we investigate stochastic stability of the disease-free equilibrium, including stability in probability and *p*th moment asymptotic stability. Then sufficient conditions for persistence in the mean of the disease are established. Finally, based on an Euler scheme for Lévy-driven stochastic differential equations, numerical simulations for a stochastic two-strain model are carried out to verify the theoretical results. Moreover, numerical comparison results of the stochastic two-strain model and the deterministic version are also given. Lévy noise can cause the two strains to become extinct almost surely, even though there is a dominant strain that persists in the deterministic model. It can be concluded that the introduction of Lévy noise reduces the disease extinction threshold, which indicates that Lévy noise may suppress the disease outbreak.

1. Introduction

One of the major challenges in the study and modeling of infectious diseases is investigating the evolution, persistence, and extinction of multiple pathogen strains. Many infectious diseases such as influenza, gonorrhea and HIV/AIDS have incredibly high incidences in the world, providing the necessary environment and opportunities for the evolution of new strains [1–4]. Biologists and mathematical modelers have long been concerned with appropriate epidemic models and disease management strategies. Recently, compartmental epidemiological models with multiple pathogen strains have received considerable attention because of their importance in the evolution of infectious diseases [5,6]. Furthermore, these types of compartmental epidemiological models have been applied to many infectious diseases such as dengue fever, malaria, influenza and HIV/AIDS [7–9]. Especially, the principle of competitive exclusion has been demonstrated in some deterministic multi-strain epidemic models, which implies that the strain with the largest basic reproduction number outcompetes all others [10–12].

In particular, Allen et al. formulated and analyzed a discrete-time multi-strain SIS epidemic model, where it is assumed that each strain is transmitted horizontally by direct contact between an infected individual and a susceptible one, and there is no coinfection or superinfection [12]. Under these assumptions, we present the multi-strain SIS epidemic model that takes

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the form of a system of differential equations and choose the standard incidence rate. Let $S(t)$ denote susceptible individuals and $I_k(t)$ represent individuals infected with strain k at time t, for $k = 1, 2, \ldots, n$. Then the model can be described as

$$\begin{cases} S'(t) = \sum_{k=1}^{n} \gamma_k I_k(t) - \sum_{k=1}^{n} \beta_k \frac{S(t)}{N} I_k(t), \\ I_k'(t) = \beta_k \frac{S(t)}{N} I_k(t) - \gamma_k I_k(t), \quad k = 1, 2, \ldots, n. \end{cases} \tag{1.1}$$

Note that the parameters in the model (1.1) are all deterministic irrespective of the effects of environmental variability and randomness, but, from an epidemiological perspective, it is more reasonable to consider how environmental noise affects the spread of infectious diseases with multiple pathogen strains. Many studies have indicated that environmental fluctuations have a huge impact on the transmission of an epidemic [13,14]. Usually, one of the frequently used methods is to consider environmental white noise, which arises from a nearly continuous series of small perturbations on the characteristic parameter such as the transmission rate [15–17]. In some stochastic models, environmental white noise has a counterintuitive effect on the spread of diseases, and dynamics of the stochastic systems can be markedly different from their associated deterministic versions. For example, Gray et al. obtained the basic reproduction number of a stochastic SIS epidemic model, which is less than the corresponding deterministic counterpart; the disease becomes extinct, even though it may persist in the deterministic case [18]. For the same kind of stochastic model, Vicenc et al. found that the external noise can induce initial epidemic growth, even if no invasion occurs in the deterministic model [13]. For more stochastic epidemic models perturbed by white noise, we refer the readers to [19–22]. Therefore, we assume that the transmission rate β_k of each strain k is not completely deterministic but fluctuates around a mean value due to continuous perturbations in the environment; i.e., $\beta_k \to \beta_k + \sigma_k \dot{B}_t^k$, $k = 1, 2, \ldots, n$, where $\dot{B}_t^k$ is white noise and σ_k is the noise intensity. As a result, model (1.1) becomes

$$\begin{cases} dS(t) = \Big(\sum_{k=1}^{n} \gamma_k I_k(t) - \sum_{k=1}^{n} \beta_k \frac{S(t)}{N} I_k(t) \Big) dt - \sum_{k=1}^{n} \sigma_k \frac{S(t)}{N} I_k(t) dB_t^k, \\ dI_k(t) = \Big(\beta_k \frac{S(t)}{N} I_k(t) - \gamma_k I_k(t) \Big) dt + \sigma_k \frac{S(t)}{N} I_k(t) dB_t^k, \quad k = 1, 2, \ldots, n. \end{cases} \tag{1.2}$$

Furthermore, the population may suffer sudden environmental shocks and catastrophes such as abrupt climate changes and unpredictable disasters, which can cause jumps in epidemic dynamics [23,24]. Here, a jump indicates a sudden change in the number of individuals, and the mathematical explanation is that the sample paths are not continuous almost surely. Nevertheless, the classical stochastic model (1.2) is a pure diffusion-type stochastic process with continuous sample paths, which cannot explain large, occasional environmental fluctuations. Instead, the non-Gaussian Lévy noise, which extends standard Brownian noise to many types of impulsive jump-noise processes, should be suitable for describing the abrupt phenomena [25–28]. Additionally, the jump-diffusion model has intuitive appeal because it allows the number of individuals to change continuously most of the time, but it may also cause large jumps to occur occasionally [29,30]. Therefore, we aim to introduce Lévy noise into the multi-strain SIS epidemic model (1.1) and investigate the effects of Lévy noise on the spread of infectious diseases with multiple pathogen strains. Introducing Lévy noise into system (1.1) and imposing the effects of Lévy noise on the transmission rate β_k of each strain k, we have

$$\beta_k \to \beta_k + \dot{L_t^k}, \quad k = 1, 2, \ldots, n.$$

Here, L_t^k is real-valued independent Lévy process for $k = 1, 2, \ldots, n$. According to the Lévy–Itô decomposition and the interlacing technique [25], the Lévy process with jumps bounded by 1 can be interpreted as

$$L_t^k = \mu^k t + \sigma_k B_t^k + \int_0^t \int_{|u^k|<1} u^k \tilde{N}^k(ds, du^k), \quad k = 1, 2, \ldots, n,$$

where μ^k determines the velocity of the deterministic drift process $\mu^k t$, while B_t^k is real-valued independent standard Brownian motion. $N^k(dt, du^k)$ is an independent Poisson random measure on $R^+ \times (R \setminus \{0\})$ with compensated (mean-subtracted) Poisson process $\tilde{N}^k(dt, du^k) = N^k(dt, du^k) - \nu^k(du^k)dt$ and the Lévy measure ν^k satisfies $\int_{R\setminus\{0\}} (1 \wedge (u^k)^2) \nu^k(du^k) < \infty$. Assume that $\mu^k = 0$. Hence we obtain the following stochastic multi-strain SIS epidemic model driven by Lévy noise:

$$\begin{cases} dS(t) = \Big(\sum_{k=1}^{n} \gamma_k I_k(t) - \sum_{k=1}^{n} \beta_k \frac{S(t)}{N} I_k(t) \Big) dt - \sum_{k=1}^{n} \frac{S(t-)}{N} I_k(t-) dL_t^k, \\ dI_k(t) = \Big(\beta_k \frac{S(t)}{N} I_k(t) - \gamma_k I_k(t) \Big) dt + \frac{S(t-)}{N} I_k(t-) dL_t^k, \quad k = 1, 2, \ldots, n, \end{cases} \tag{1.3}$$

where $S(t-)$ and $I_k(t-)$ indicate the left limit of $S(t)$ and $I_k(t)$. Note that $N'(t) = S'(t) + I_1'(t) + \ldots + I_n'(t) = 0$, then the total population $N(t)$ is a constant. Here, we choose $N = 1$, so $S(t) = 1 - \sum_{m=1}^{n} I_m(t)$ represents the proportion of susceptible individuals and $I_k(t)$ denotes the proportion of individuals infected with strain k at time t, for $k = 1, 2, \ldots, n$. Denote $I(t) = (I_1(t), \ldots, I_n(t))^T$, $b_k(I(t)) = I_k(t)(\beta_k(1 - \sum_{m=1}^{n} I_m(t)) - \gamma_k)$, $c_k(I(t)) = I_k(t)(1 - \sum_{m=1}^{n} I_m(t))$. Then, the model (1.3) can be