ORIGINAL ARTICLE

Gui-Quan Sun · Zhen Jin · Li-Peng Song
Amit Chakraborty · Bai-Lian Li

Phase transition in spatial epidemics using cellular automata with noise

Abstract One of the central issues in studying the complex population patterns observed in nature is the role of stochasticity. In this paper, the effects of additive spatiotemporal random variations—noise—are introduced to an epidemic model. The no-noise model exhibits a phase transition from a disease-free state to an endemic state. However, this phase transition can revert in a resonance-like manner depending on noise intensity when introducing nonzero random variations to the model. On the other hand, given a regime where disease can persist, noise can induce disappearance of the phase transition. The results obtained show that noise plays a tremendous role in the spread of the disease state, which has implications for how we try to prevent, and eventually eradicate, disease.

Keywords Cellular automata · Noise · Epidemiology · Coherence resonance · Extinction

Introduction

Infectious diseases, caused by a disease agent that can be transmitted from organism to organism, have had a large impact on human populations (Anderson and May 1992; Castillo-Chavez and Yakubu 2002; Kermack and McKendrick 1927; Roy and Pascual 2006; Shirley and Rushton 2005). In recent years, there has been increased awareness of the threat posed by newly emerging infectious diseases, for example SARS, the H5N1 strain of avian influenza, HIV, Ebola, whooping cough, Dengue fever, and so on. These diseases exhibit large-scale spatial contagions and long-term spatial patterns (He and Stone 2003; Nguyen and Rohani 2008; van Ballegooijen and Boerlijst 2004; Viboud et al. 2006).

Theoretical epidemiologists have used many different approaches to model the dynamics of spatially structured populations (Dieckmann et al. 2000; Hilker et al. 2006; MacArther 1958; Malchowa et al. 2004). The classical modeling approaches are reaction-diffusion models and patch-structured models (meta-populations, coupled-map lattices and deme-structured populations). The advantage of such models is that mathematical analysis is feasible. However, this advantage always comes at the expense of various simplistic assumptions that limit the applicability of these models, especially in evolutionary contexts. The advent of modern computing facilities has encouraged ecologists to turn to individual-based spatial simulations, such as cellular automata (CA) (Durrett and Levin 1994a, b). In CA, space is represented by a network of sites, and the state of the network is updated through probabilistic rules representing demographic events (Lion and van Baalen 2008).

However, there is some misunderstanding between the application of the above approaches and explaining epidemics such as, for example, whooping cough (Nguyen and Rohani 2008). To understand the stark contradiction between the dynamics predicted by deterministic models and those observed in data, some researchers have used models with noise to investigate the spread of epidemics (Bauch and Earn 2003b; Keeling et al. 2001a; Rohani et al. 1999, 2002). Since then, a number of authors have focused on this question, and the general consensus seems to be that pertussis epidemics result from the interaction between seasonality, nonlinearity and, especially, stochasticity (Alonso et al. 2007; Bauch and Earn 2003b; Perc 2006, 2007; Perc and Szolnoki 2008, 2010; Simões et al. 2007). Alonso et al. (2007) used household data on the incubation period to explain that noisy and somewhat irregular epidemics of

G.-Q. Sun · Z. Jin (✉) · L.-P. Song · B.-L. Li
Department of Mathematics,
North University of China,
Taiyuan 030051 Shanxi, People's Republic of China
E-mail: gquansun@yahoo.com.cn
E-mail: jinzhn@263.net
E-mail: slp880@gmail.com
E-mail: bai-lian.li@ucr.edu

G.-Q. Sun · A. Chakraborty · B.-L. Li
Ecological Complexity and Modeling Laboratory,
Department of Botany and Plant Sciences,
University of California, Riverside, CA 92521-0124, USA

whooping cough may arise due to stochasticity and its interaction with nonlinearity in transmission and seasonal variation in contact rates. Simões et al. (2007) presented a stochastic theory for the major dynamic transitions in epidemics from regular to irregular cycles. In addition, previous studies have revealed that noise can sustain damped cycles (Bartlett 1957; Perc and Szolnoki 2007; Perc et al. 2007), allow the system to remain in a transient regime (Bauch and Earn 2003a, b; Rand and Wilson 1991; Sidorowich 1992), and lead to irregular behavior (Earn et al. 2000; Keeling et al. 2001a).

An examination of the literature reveals that the effects of noise on epidemic models using CA, especially on the persistence and extinction of the disease, have generally been overlooked despite their potential ecological reality and intrinsic theoretical interest. These structures may in fact correspond to the real world. For this reason, we have proposed an epidemic model using cellular automation with noise and investigated the effect of noise on disease spread. In the present paper, we present an interesting noise-induced phenomenon in an epidemic model. In particular, we study the effects of temporally and spatially white additive Gaussian noise introduced into the parameters of our spatial epidemic model. The results obtained show that appropriate levels of noise can cause the extinction of disease in a resonant manner. Since no additional deterministic inputs are introduced into the system, the phenomenon reported is thus conceptually identical to coherence resonance (Pikovsky and Kurths 1997). In addition, we reveal the dual role of noise, i.e., to induce both the appearance and disappearance of the disease.

The paper is organized as follows. In Methods, we give a description of the spatial epidemic model using CA. Evidence for coherence resonance is presented in the Results. The extinction induced by noise is shown, and extinction time with respect to the standard deviation of noise and system size is given. Finally, we summarize and discuss the results in the Discussion and conclusion.

Methods

A spatial susceptible-infected-recovered-susceptible (SIRS) model will be illustrated. Generally speaking, the population in which a pathogenic agent is active comprises three subgroups: healthy individuals who are susceptible (S) to infection; already infected individuals (I) who can transmit the disease to healthy subjects; and the "removed" (R), who cannot get the disease or transmit it: either they have a natural immunity, or they have recovered from the disease and are immune from getting it again, or they have been isolated, or they have died (Anderson and May 1992; Keeling et al. 2003, 2001b; Murray 1993; Ostfeld et al. 2005; Smith et al. 2002; Sun et al. 2008). Within a subpopulation, the dynamics of these local populations obey a basic reac-

tion scheme, which has been studied both in physics and mathematical epidemiology, that conserves the population number, namely the infection dynamics process identified by the following set of reactions (van Ballegooijen and Boerlijst 2004):

$$S + I \xrightarrow{\beta} I + I, \tag{1a}$$

$$I \xrightarrow{\tau_I} R, \tag{1b}$$

$$R \xrightarrow{\tau_R} S. \tag{1c}$$
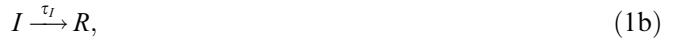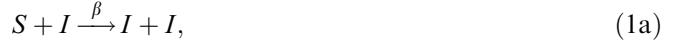
In the cellular automation, S, I and R mean the state in one discrete lattice. The first reaction (Eq. 1a) reflects the fact that an infectious host (I) can infect susceptible (S) neighbors with infection rate β; the second reaction (Eq. 1b) indicates the acquisition of resistance, i.e., that hosts are infectious for a fixed period τ_I, after which they become recovered (R); and the third reaction (Eq. 1c) indicates the loss of resistance, i.e., that after a fixed period τ_R, recovered hosts once again become susceptible.

Van Ballegooijen and Boerlijst (2004) showed that there were three different types of spatial patterns in model (1): (1) localized disease outbreaks that are self-limiting in size, (2) turbulent waves, and (3) stable spiral waves. Furthermore, they predicted that there was a trade-off between the parameters of infection period and infection rate, which emerges from the evolutionary dynamics of the system on the stable spiral waves, and referred to this relationship as emergent trade-off.

However, in the present paper, we want to demonstrate the effects of noise on the CA model. Therefore, a noise term is added and modeled by introducing Gaussian noise additively to all parameters so that the modified parameters satisfy $P = p + \xi$, where $p \in (\beta, \tau_I, \tau_R)$ and ξ is a random variable (dynamic noise) being normally distributed with zero mean and σ standard deviation. The Gaussian noise satisfies the correlation function

$$\langle \xi_i(h)\xi_j(l) \rangle = \sigma^2 \delta_{ij}\delta_{hl}, \tag{2}$$

where indexes i and j mark the central location of two individual pairs on the lattice, while h and l denote two consecutive pair interactions. In a word, Eq. 2 means that the noise is not correlated in space (δ_{ij}) or time (δ_{hl}).

The described spatial epidemic model can be iterated forward in time using either a synchronous or a random Monte Carlo update scheme (Perc 2008). Since the synchronous update scheme tends to converge more quickly to the equilibrium than the random iteration, we thus applied the latter, letting all individuals interact pairwise with all their nearest neighbors and then simultaneously update their strategies according to Eq. 1 (Perc 2008).

The key quality describing the infection is the basic reproduction number—R_0, which is the most fundamental parameter used by epidemiologists. For microparasites, it is defined as the average number of secondary cases caused by an infectious individual in a
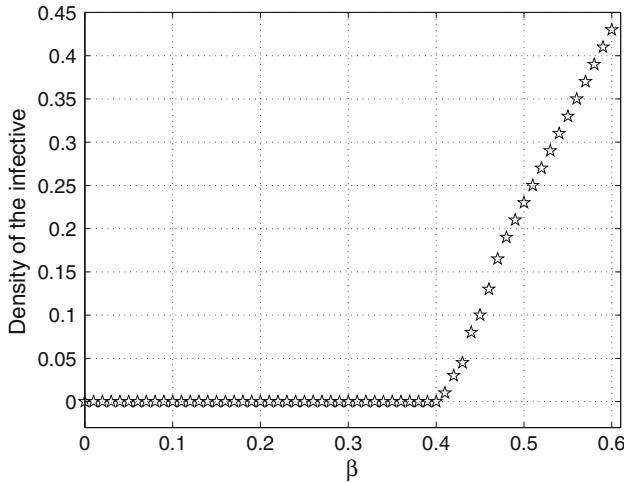
Fig. 1 An illustration of infective fraction for fixed τ_I without noise ($\sigma = 0$). Phase transition, from the disease-free state to an endemic state, occurs at $\beta_c = 0.4$. Here, $\tau_I = 0.4$ and $\tau_R = 1$

completely susceptible population (Anderson and May 1992; Murray 1993). Epidemiologically (and typically), if $R_0 > 1$, the number of infectious individuals grows, while if $R_0 < 1$ the number of infectious individuals would decrease to zero regardless of initial conditions (Sun et al. 2008). For model (1) with small value of β, as seen in the Appendix, it can be found that:

$$R_0 = \frac{\beta}{\tau_I}. \tag{3}$$

Note that, when β is larger than β^* ($\beta^* \approx 2.5$), the expression of R_0 should be changed because of the emergence of cluster extinction (Liu et al. 2009). Here, we focus our attention on the small β and assume $\tau_R = 1$ without loss of generality. Results presented in the Fig. 1 show the density of infectious individuals for various values of β obtained using the deterministic CA model ($\sigma = 0$). It is evident that the disease will die out when β is small enough. However, if the infection rate β becomes larger than the threshold $\beta_c = 0.4$, the disease will spread.

Results

Coherence resonance

In this section, we will systematically analyze the effects of nonzero σ on the epidemic model for $\beta < \beta_c$, with the aim of reporting noise-induced transitions to the mixed state in a resonance-like manner depending on σ, thus evidencing coherence resonance. The grid size for all panels is 300×300, and Moore neighborhood is used.

Although the noise may sometimes cause the density of the populations (S, I and R) to be less than zero, it will lead the system to a cutoff effect at low densities when species extinction is taken into account explicitly.

According to the spatial model, at each position in space, whenever the population densities fall below a certain value, ε, they are set to zero or a sufficiently small positive constant. In this paper, we set them equal to 0.000001 when the variables change to negative (Malchow et al. 2008; Petrovskii et al. 2004). Note that we are not overly concerned here with the exact value of ε, for the reason that an attempt to estimate the exact value would hardly make any ecological sense.

In order to see the effects of noise, we investigate the epidemic model by setting $\beta < \beta_c$. As evidenced in Fig. 1, when there is no noise ($\sigma = 0$), the model states that $I = 0$ for $\beta < \beta_c$. On the other hand, results presented in the panels of Fig. 2 clearly show that the density of I is more than zero by adding nonzero values of σ in a resonant manner. More specifically, small σ are able to sustain only small densities of I. The density of I will increase proportionately with the increase of σ when $\sigma < 3$, and reach a maximal value at $\sigma \approx 3$. After that, it has inverse relation with σ. In other words, the results presented in Fig. 2 indicate a typical coherence resonance[1] scenario[2] (Perc 2005a, b, c, d, 2008; Pikovsky and Kurths 1997).

To quantify the ability of each particular σ to make the phase transition from a disease-free state to an endemic state more precisely, we calculate the fraction of I with various σ for $\beta < \beta_c$. We assume that the fraction of I uniquely determines the constructive effects of noise on the system, and thus has the same meaning as the signal-to-noise ratio (Gammaitoni et al. 1998) in classical stochastic and coherence resonance phenomena observed in dynamic systems. The results presented in Fig. 3 clearly show that an optimal level of additive spatiotemporal noise for which the fraction of the infective is maximal always exists, thus indicating the existence of coherence resonance in the studied spatial epidemic model using CA.

Extinction induced by noise

When the parameter values are such that disease persists ($\beta > \beta_{tr}$), a time series of I for fixed β, γ, and σ can be illustrated (Fig. 4). We find that, for a long time, the density of I will be zero, i.e., the disease will die out. Also, the average time to extinction[3] with respect to different values of σ is shown in Fig. 5 (in total, more

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[1]The term coherence resonance refers to a phenomenon whereby addition of certain amount of noise makes its oscillatory responses most coherent. As a result, a coherence measure of stochastic oscillations attains an extremum at optimal noise intensity (Lindner and Schimansky-Geier 1999; Lee et al. 1998; Zhou et al. 2001; Neiman et al. 1997; Neiman et al. 1998; Pikovsky and Kurths 1997).

[2]This phenomenon is associated with two-states: disease-free and epidemic state, and noise induces jumps between them at a suitable noise intensity.

[3]We perform numerical simulations by using different initial conditions (in total, more than ten different initial conditions are examined) and obtain the mean time to extinction, which is called "average time to extinction".

Fig. 2a–h (Color online) Spatial pattern of the susceptible (*white*), infective (*red*) and recovered (*black*) at different noise intensities. All panels are depicted on a 300×300 spatial grid. Parameter values used are: $\beta = 0.3$, $\tau_I = 0.4$, $\tau_R = 1$. **a** $\sigma = 0.06$, **b** $\sigma = 0.1395$, **c** $\sigma = 0.15$, **d** $\sigma = 0.22$, **e** $\sigma = 0.3$, **f** $\sigma = 0.5$, **g** $\sigma = 0.55$, **h** $\sigma = 0.75$
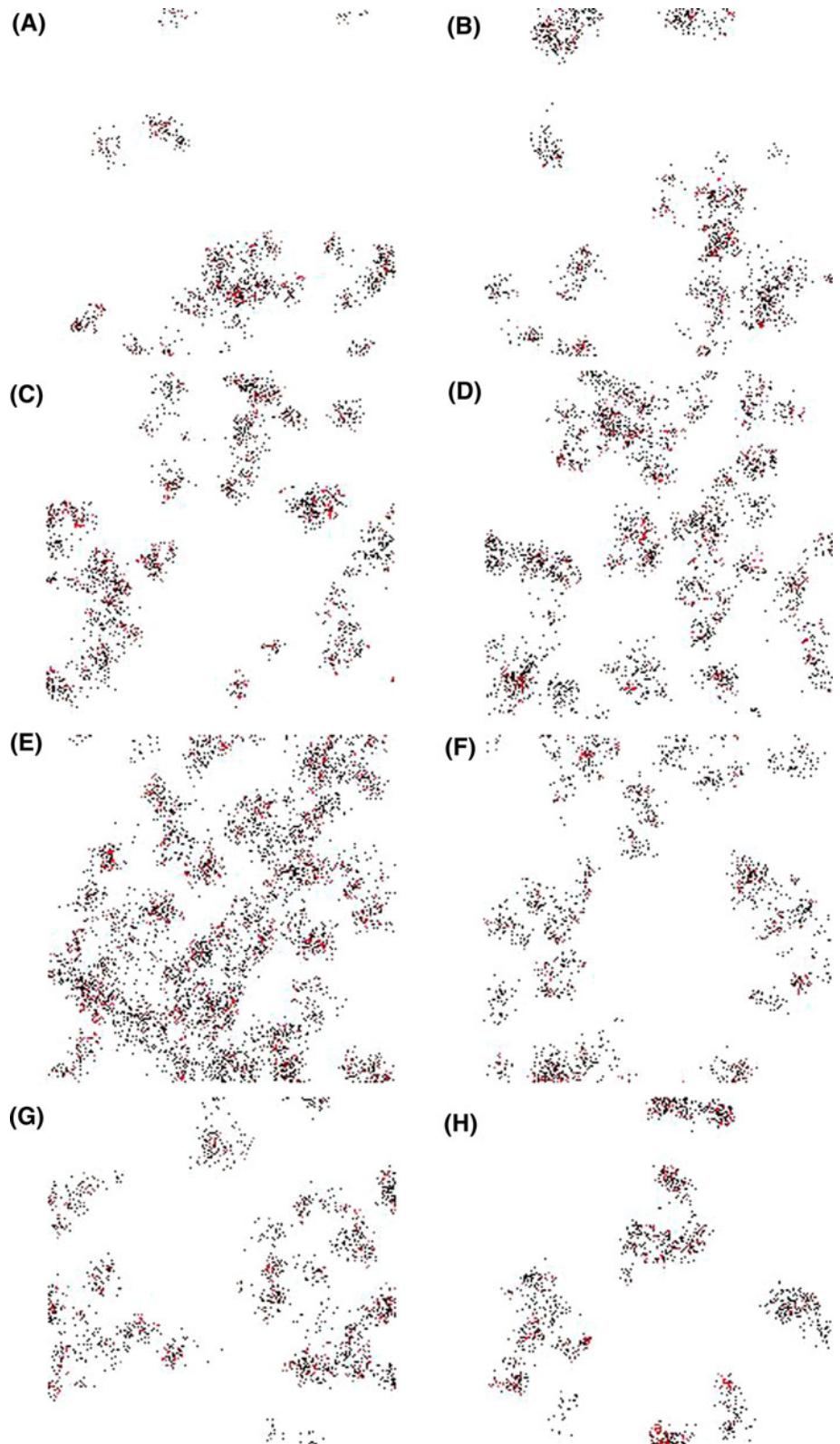
than 40 parameter sets were examined). One can see from this figure that, as σ increases, the extinction time decreases. However, when σ is increased further, the extinction time is an increasing function of σ.

In an early study (Bartlett 1957, 1960), Bartlett found that measles in large cities had recurring outbreaks, while it became extinct in small communities until reintroduced from external sources. In addition, Keeling
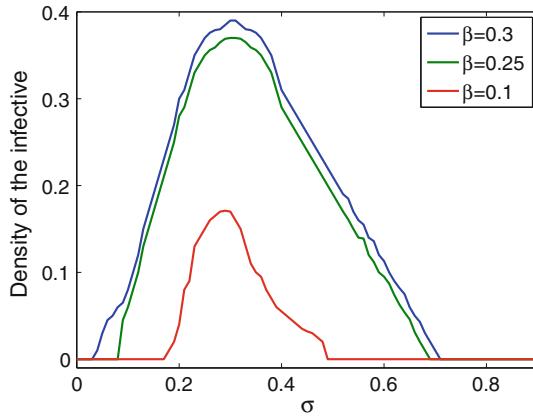
Fig. 3 An illustration of infective fraction for fixed τ_I and different values of σ. Note that, for the no-noise model, the parameter values ensure the disease-free state. Coherence resonance occurs when combined with noise. Here, $\tau_I = 0.4$ and $\tau_R = 1$
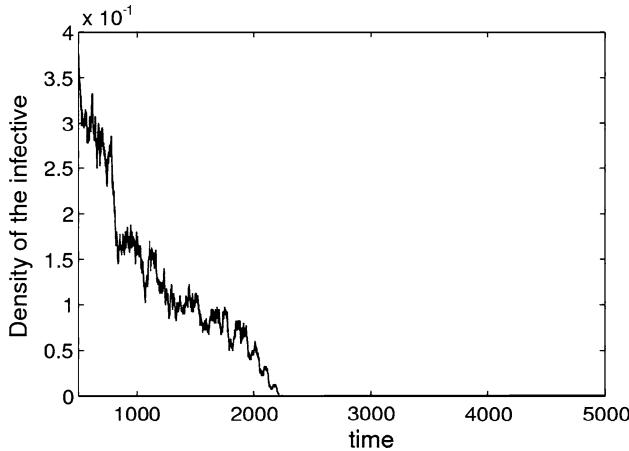

Fig. 4 Time-series of infectious individuals (I) when noise is turned on ($\sigma = 0.75$). Parameters values: $\beta = 0.6$, $\tau_I = 0.4$, and $\tau_R = 1$. Note that, without noise, the density of I is more than zero. In other words, extinction of the disease is induced by noise. Here, the grid size for all panels is 300×300
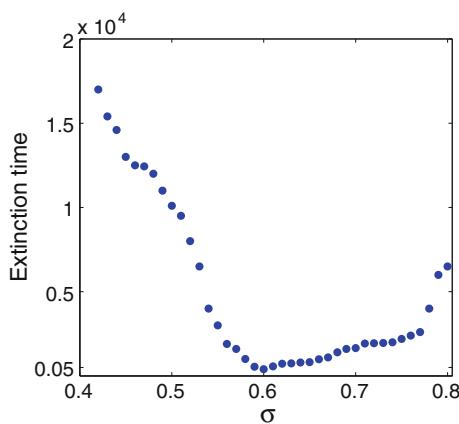

Fig. 5 Average time to extinction versus different values of σ for $\beta = 0.6$, $\tau_I = 0.4$, and $\tau_R = 1$. The grid size for all panels is 300×300
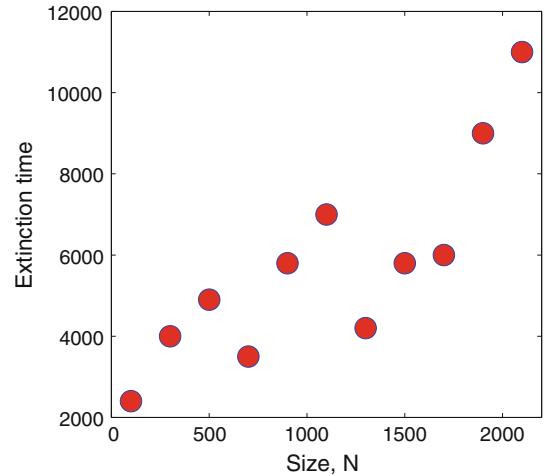

Fig. 6 Average time to extinction versus system size N for $\beta = 0.6$, $\tau_I = 0.4$, $\tau_R = 1$, and $\sigma = 0.54$

and Grenfell (1997) found that measles does not persist if the population size is below a critical value. That is to say, the extinction time is an increasing function of community size. Our present system (1) is a simple individual-based SIRS model with diffusion processes (Boland et al. 2008; Reichenbach et al. 2008; Simões et al. 2007). It is natural for us to ask whether the conclusion holds when noise is added. Here, the aim of the analysis is to derive information about the quasi-stationary distribution and the time to extinction. And an approximation of the discrete state Markov chain (1) is introduced, which leads to a truncated normal distribution as an approximation of the marginal distribution of infectious individuals in a quasi-stationary state (Nasell 1999, 2001).

Figure 6 presents a graph of the average extinction time as a function of system size, similar to the study by Petrovskii et al. (2004). Due to the existence of noise, extinction time does not always increase as community size increases. Instead, oscillatory behavior emerges. The relationship between system size, standard deviation of noise, and extinction time is shown in Fig. 7; when the noise intensity is small, e.g., $\sigma < 0.44$, extinction time increases as the community size becomes larger. However, extinction time becomes very much smaller when the noise intensity is large, see $\sigma > 0.72$ for example. However, in other regions, the noise can induce unpredicted extinction times at different system sizes.

Discussion and conclusion

The main objective of our study was to show that white additive Gaussian noise spatially and temporally introduced into the parameters of an epidemic model can influence the spread of disease. The dynamics exhibit a noise-controlled transition from no disease to an endemic state (density of infectious individuals > 0), in a
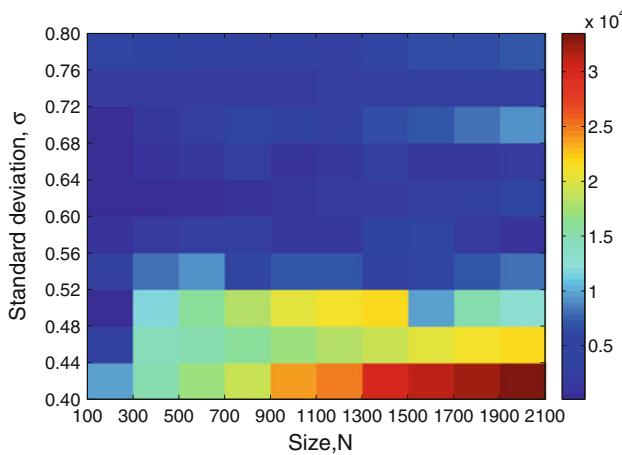
Fig. 7 Average time to extinction for different system size N and standard deviation σ with $\beta = 0.6$, $\tau_I = 0.4$, $\tau_R = 1$

resonant manner depending on the level of random variations. This reported phenomenon is known as coherence resonance. In other words, noise can enhance disease spread. In addition, the results also show that noise can lead to the disease becoming extinct. This finding may well explain inconsistencies between the dynamics predicted by deterministic models and those observed in data (Rohani et al. 2002).

Some scholars have found that noise can induce only a higher extinction risk of a population (Foley 1994; Johst and Wissel 1997; Mode and Jacobsen 1987; Roughgarden 1975; Wichmann et al. 2003a, b). At the same time, some studies have shown that noise leads only to populations having a lower extinction rate (Ripa and Heino 1999). That is to say that, for a specific model, attention was focused only on one aspect of the noise effects. Our work here reveals the dual effects of noise on epidemic spread, which extends the findings of the effects of noise on disease. Consequently, when attempting to prevent disease spread between different populations, we should take into account the dual effects of noise.

The noise used in this paper is Gaussian noise. However, colored noise, arising from either environmental variability or internal effects, has also been used to describe interactions between populations (Blasius et al. 1999; Sun et al. 2010a, b). The effects of color noise on disease spread remain unclear and will be the subject of future research.

Because of the uncertainty effects of noise, in practice we should reduce some noise terms in the process of epidemic spread. For example, spatio-temporal dynamic analyses of disease require that data on disease incidence be highly precise, both in space and time. So we should try our best to reduce errors (Grenfell et al. 2001). Moreover, collaborative efforts among ecologists, epidemiologists and health care professionals are required to evaluate feasibility and efficacy (Ostfeld et al. 2005). Thus, we should provide plenty opportunities for such successful collaborations. Maybe, in such a way, disease can be well predicted and thus controlled.

Appendix: Mean-field equations of reactions (1)

The mean-field equations describe the temporal evolution of the stochastic lattice system, defined by the reactions (1). They may be seen as a deterministic description of systems without spatial structure. The study of the rate equation forms the basis of the analysis of epidemic spread and outbreak. For the standard SIRS, mean-field approximations, also referred to in reaction (1), are as follows:

$$\frac{dS}{dt} = -\beta SI + \tau_R R, \tag{4a}$$

$$\frac{dI}{dt} = \beta SI - \tau_I I, \tag{4b}$$

$$\frac{dR}{dt} = \tau_I I - \tau_R R. \tag{4c}$$

By direct calculation, we first find the steady states as follows:

(1) $E_0 = (1, 0, 0)$, which corresponds to the disease-free state;
(2) Interior equilibrium point $E^* = (S^*, I^*, R^*)$, where

$$S^* = \frac{\tau_I}{\beta}, \tag{5a}$$

$$I^* = \frac{\tau_R(\beta - \tau_I)}{\beta(\tau_I + \tau_R)}, \tag{5b}$$

$$R^* = \frac{\tau_I(\beta - \tau_I)}{\beta(\tau_I + \tau_R)}, \tag{5c}$$

which corresponds to the endemic equilibrium. It is easy to show that E^* is positive under the condition $\beta > \tau_I$.

By using standard linear stability analysis, it can be readily seen that E_0 is a stable node if $\beta < \tau_I$; otherwise, it is a unstable node. Moreover, E^* is a stable node if it exists.

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