Braz J Phys (2012) 42:248–252
DOI 10.1007/s13538-012-0076-z

Coherence Resonance in an Epidemic Model with Noise

Yi Wang · Jianzhong Wang

Received: 11 November 2011 / Published online: 27 April 2012

Abstract Epidemic models frequently contain thresholds that determine the survivability of competing strains. In the model we study in this paper, if the basic reproduction number is less than one, the disease will disappear, otherwise the disease will persist. Interestingly, we find that additive noise of an appropriate intensity and temporal correlation can reverse this in a resonance-like manner. By using analogies from the theory of noise-driven dynamical system, we thus observe an evolutionary coherence resonance, similar to what has been reported previously in evolutionary games. Potential implications of our results for real-life field observations are also discussed.

Keywords Basic reproduction number · Noise · Coherence resonance

1 Introduction

Throughout history, infectious diseases, which are illnesses caused by disease agents that can be transmitted from organism to organism, have had a large impact on the human population. As a result, modeling the process of epidemics has been one of the most important works in mathematical biology [1–3]. Many scholars consider the dynamics of the disease as well as the life history of the disease-carrying agents by using a system of deterministic differential equations [4].

However, it is not suitable to explain some epidemics by using the application of deterministic differential equations, such as whooping cough [5]. In order to well understand the contradiction between the dynamics predicted by deterministic differential models and the data observed in the real world, some researchers relaxed the assumption of determinism and examined stochastic models [6]. These models were consistent with data in the England and Wales, in both the prevaccine and the vaccination eras. From then on, a number of authors assumed that the inconsistence appears to be the result of the interaction between seasonality, nonlinearity, and stochasticity [5, 7, 8]. As a result, noise terms have been introduced in epidemic models.

In the recent 10 years, noise-induced phenomena in nonlinear systems have been the topic of a number of physical and biological investigations [9, 10]. Noise can induce counterintuitive dynamical changes. In particular, noise has organizing rather than disruptive effects. Some of the more important examples are stochastic resonance [11] and noise-induced transitions [12, 13].

Coherence resonance is a phenomenon whereby addition of certain amount of noise makes its oscillatory responses most coherent, and a measure of stochastic oscillations attains an extremum at an optimal noise intensity [14–18]. The concept of coherence resonance in terms of dynamics and evolution has been found in prisoner's dilemma game [19–21], noise-guided evolution within cyclical interactions [22], and other models [23–26]. Now, it is natural to ask whether

Y. Wang (✉) · J. Wang
National Key Laboratory for Electronic Measurement Technology, North University of China, Taiyuan, 030051 Shanxi, People's Republic of China
e-mail: wangyi12345678@yahoo.com.cn

Y. Wang · J. Wang
Key Laboratory of Instrumentation Science and Dynamic Measurement, Ministry of Education, North University of China, Taiyuan, 030051 Shanxi, People's Republic of China

there is coherence resonance in epidemic models. In order to understand the mechanism, we investigate a susceptible–infected–removed (SIRS) model with noise.

We find a novel and interesting phenomenon in a SIRS model with noise, i.e., appropriate levels of noise can revert the extinction of disease in a resonant manner. The paper is organized as follows. In Section 2, we give the description of the SIRS model. Evidence for the coherence resonance is presented in Section 3. In the last section, we summarize and discuss the results.

2 Model

In general, the population, in which a pathogenic agent is active, consists of three subgroups: the healthy individuals who are susceptible (S) to infection, the already infected individuals (I) who can transmit the disease to the healthy ones, and the removed (R) who can not 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 placed in isolation, or they have died [27, 28]. The standard SIRS model is given by the following form:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\beta SI + \tau_R R, \tag{1a}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - \tau_I I, \tag{1b}$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \tau_I I - \tau_R R, \tag{1c}$$

where S and I denote the density of the susceptible and infective population, respectively. The density of recovered individuals R is obtained by conservation of the entire population, i.e., $R(t) = 1 - S(t) - I(t)$. Here, β is the infection rate, τ_I is the period of the infective population, and τ_R is the time of the loss of resistance. The study of the rate equation is based on the analysis of epidemic spreading and outbreak. Particularly, the properties of the rate equations are extremely useful for assessing epidemic persistence by using estimated values for the classical expression for R_0-the basic reproduction number. R_0 is defined as the number of secondary infections caused by a single infected individual during its infectious period in an entirely susceptible population. For the model (1), the basic reproduction number is given by

$$R_0 = \frac{\beta}{\tau_I}. \tag{2}$$

If $R_0 > 1$ and the initial relative number of susceptible is greater than a critical value $S_c = 1/R_0$, the disease will spread. As the density of the infective population individuals increases, the number of susceptible S decreases, and thus the number of contacts of the infected individuals with susceptible ones decreases until $S = S_c$; then the epidemic reaches its maximum and subsequently decays. If $R_0 < 1$, the density of infective population would decrease to zero regardless of initial conditions.

Combined with noise term, the system of equations will have the following form:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\beta SI + \tau_R R, \tag{3a}$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - \tau_I I + \eta(\mathbf{r}, t). \tag{3b}$$

In (3), the stochastic factors are taken into account by the term $\eta(\mathbf{r}, t)$, which is obtained from microscopic interaction in the space [29, 30] where the typical white noise will emerge. Recently, colored noise and white noise have both been used in describing biological evolution [31]. White noise is the limited case of colored noise, so we consider the more general case–colored noise. In the present paper, we only consider the noise presented in Eq. (3b). The noise term $\eta(\mathbf{r}, t)$ is introduced additively in space and time, which defines the Ornstein–Uhlenbeck process that obeys the following stochastic partial differential equation [32–35]:

$$\frac{\partial \eta(r,t)}{\partial t} = -\frac{1}{\tau}\eta(r,t) + \frac{1}{\tau}\xi(r,t), \tag{4}$$

where $\xi(r, t)$ is a Gaussian white noise with zero mean and correlation,

$$\langle \xi(r,t) \rangle = 0, \tag{5a}$$

$$\langle \xi(r,t)\xi(r',t') \rangle = 2\sigma\delta(r-r')\delta(t-t'), \tag{5b}$$

where τ controls the temporal correlation, and σ measures the noise intensity.

3 Coherence Resonance

We will systematically analyze the effects of nonzero σ and τ on the stochastic SIRS model, with the aim of reporting noise-induced transitions in a resonance-like manner depending on σ and τ, thus evidencing coherence resonance in the epidemic system and revising the threshold principle.

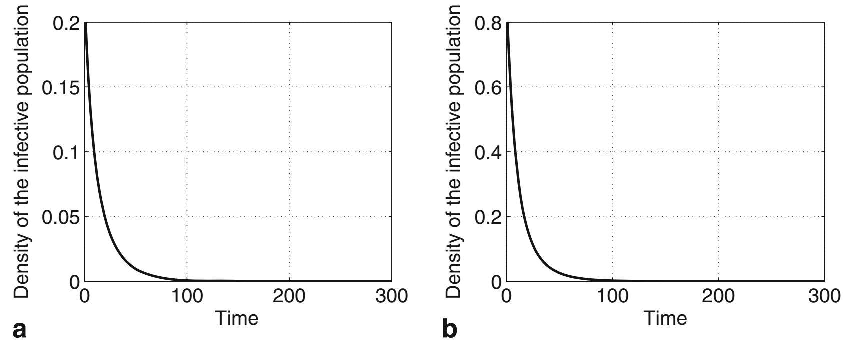


Fig. 1 Time series of the infective population. Parameters are used as follows: $\beta = 0.05$, $\tau_I = 0.1$, and $\tau_R = 0.1$. Initial conditions: **a** $S(0) = 0.1$ and $I(0) = 0.2$; **b** $S(0) = 0.01$ and $I(0) = 0.8$. Note that $R_0 = 0.5 < 1$, the density of the infective population tends to zero

In Fig. 1, we show the time series of the infective population without noise. As one can see in the figure, the density of the infective population always tends to zero. That is to say that if $R_0 < 1$, the infective population will disappear regardless of the initial conditions, which is called as threshold principle.

Now, we consider the effect of noise on the density of the infective population. We run the simulations until the density of the infective population reaches fixed values, or until it shows a behavior that does not seem to change its characteristics any longer. We choose 50 parameters set for numerical simulations. In Fig. 2, we give characteristic time course for different noise intensities. When $\sigma = 0.1$, 0.2, 0.3, and 0.4, the density of the infective population will be 0.082, 0.121, 0.074, and 0.017, respectively.

In order to reveal the effect of noise on the density of the infective population, we give the infective density with respect to noise intensity in Fig. 3. It shows that when $\sigma < 0.01$ or $\sigma > 0.479$, it has no effect on the density of the infective population. However, when $\sigma \in (0.01, 0.479)$, the density of the infective population is more than zero, which is induced by noise, and reaches the maximum value at $\sigma \approx 0.18$. That is to say, coherence resonance occurs in the SIRS model with noise.

The role of temporal correlation τ of the colored noise is also significant in controlling the density of the infective population. In Fig. 4, we give characteristic time course for different temporal correlation. When $\tau = 2, 4, 6$, and 8, the density of the infective population will be 0.03, 0.07, 0.062, and 0.028, respectively.

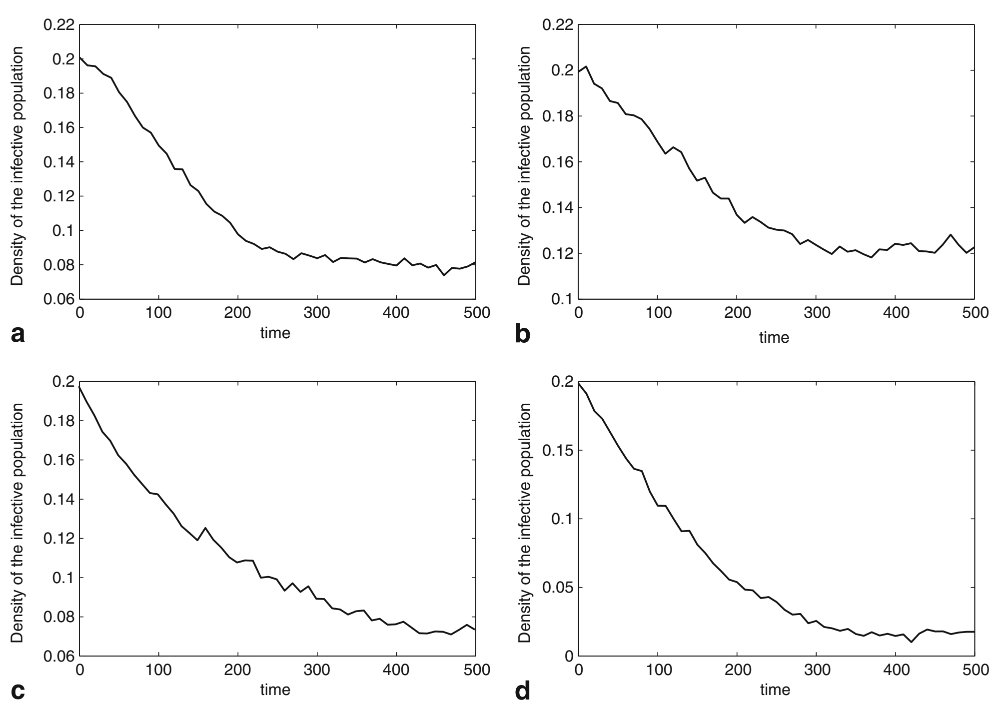


Fig. 2 Time series of the infective population for different noise intensities and $\tau = 1$. **a** $\sigma = 0.1$. **b** $\sigma = 0.2$. **c** $\sigma = 0.3$. **d** $\sigma = 0.4$

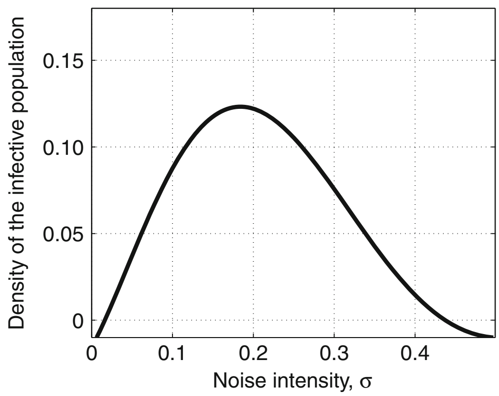


Fig. 3 An illustration of density of the infective population for different values of noise intensity. Parameters and initial conditions are the same as in Fig. 2

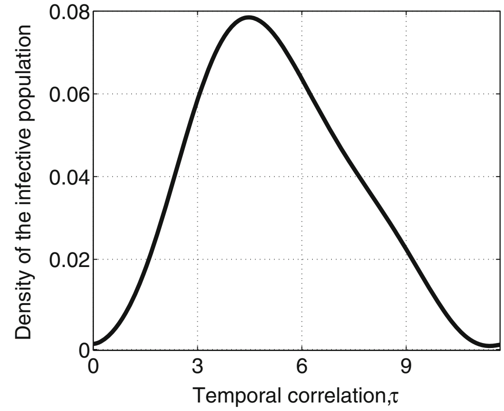


Fig. 5 Density of the infective population for different values of temporal correlation with $\sigma = 0.2$. Other parameters and initial conditions are the same as in Fig. 2

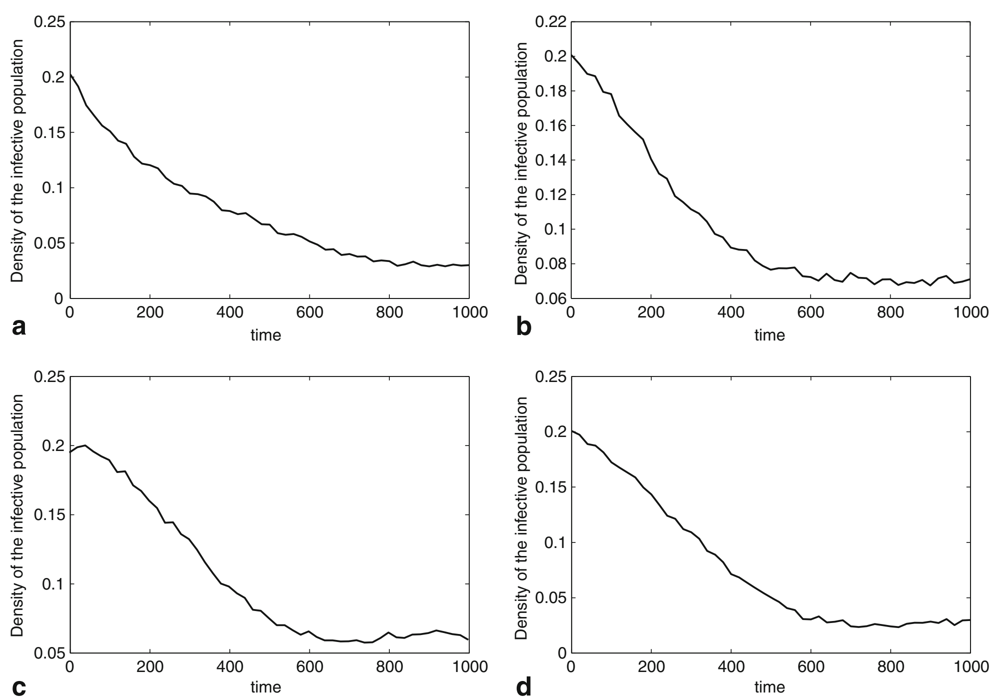
We also performed a series of simulations for fixed noise intensity σ, which is shown in Fig. 5. Results presented in the panels of Fig. 5 clearly show that the density of infective population will more than zero by nonzero values of τ in a resonant manner. More specifically, small τ is able to sustain only small density of the infective population. As τ being increased, the density of the infective population will reach the maximal value. However, for the larger τ, the density of infective population is small. In other words, the results presented in Fig. 3 indicate a typical coherence resonance scenario [23–26, 36, 37].

4 Conclusion and Discussion

It has been observed in the literature that the effect of noise in epidemic models, especially on the persistence of the disease, has been generally overlooked despite their potential ecological reality and intrinsic theoretical interest. These structures may, in fact, correspond to the real world. For such reason, we pose a SIRS model with noise and investigated the noise effect on the spread of the disease.

We show that the color of noise introduced in the system of an epidemic model can revise the thresh-

Fig. 4 Time series of the infective population for different temporal correlation and $\sigma = 0.2$. **a** $\tau = 2$. **b** $\tau = 4$. **c** $\tau = 6$. **d** $\tau = 8$



old principle in a resonant manner depending on the intensity of noise and temporal correlation. The reported phenomenon is called coherence resonance. In practice, noise effects should not be neglected when considering the process of the spread of disease, which may have effect on the dynamics of behavior, particularly revising some rules. Furthermore, we note that the phenomenon may be found in the predator–prey models [38], which means that it may be widely applicable in various fields of research ranging from epidemiology to ecology.

Acknowledgements We are grateful for the constructive suggestions of the anonymous referees on our original manuscript.

References

1. W.O. Kermack, A.G. McKendrick, A contribution to the mathematical theory of epidemics. Proc. R. Soc. Lond. A **115**, 700–721 (1927)
2. R.M. Anderson, R.M. May, *Infectious Disease of Humans: Dynamics and Control* (Oxford University Press, Oxford, 1992)
3. W.H. Hetcote, The mathematics of infectious diseases. SIAM Rev. **42**, 599–653 (2000)
4. J.M. Hyman, J. Li, Behavior changes in SIS STD models with selective mixing. SIAM J. Appl. Math. **57**, 1082–1094 (1997)
5. H.T.H. Nguyen, P. Rohani, Noise, nonlinearity and seasonality: the epidemics of whooping cough revisited. J. R. Soc. Interface **5**, 403–413 (2008)
6. P. Rohani, D.J. Earn, B.T. Grenfell, Opposite patterns of synchrony in sympatric disease metapopulations. Science **286**, 968–971 (1999)
7. P. Rohani, M.J. Keeling, B.T. Grenfell, The interplay between noise and determinism in childhood diseases. Am. Nat. **159**, 469–481 (2002)
8. C.T. Bauch, D.J.D. Earn, Transients and attractors in epidemics. Proc. R. Soc. Lond B **270**, 1573–1578 (2003)
9. J. García-Ojalvo, J.M. Sancho, *Noise in Spatially Extended Systems* (Springer-Verlag, New York, 1999)
10. M. Sieber, H. Malchow, L. Schimansky-Geier, Constructive effects of environmental noise in an excitable prey–predator plankton system with infected prey. Ecol. Complex. **4**, 223–233 (2007)
11. L. Gammaitoni, P. Hanggi, P. Jung, F. Marchesoni, Stochastic resonance. Rev. Mod. Phys. **70**, 223–287 (1998)
12. W. Horsthemke, R. Lefever, *Noise-Induced Transitions* (Springer, Berlin, 1984)
13. F. Lesmes, D. Hochberg, F. Morán, J. Pérez-Mercader, Noise-controlled self-replicating patterns. Phys. Rev. Lett. **91**, 238301 (2003)
14. B. Lindner, L. Schimansky-Geier, Analytical approach to the stochastic Fitzhugh–Nagumo system and coherence resonance. Phys. Rev. E. **60**, 7270–7276 (1999)
15. S. Lee, A. Neiman, S. Kim, Coherence resonance in a Hodgkin–Huxley neuron. Phys. Rev. E **57**, 3292–3297 (1998)
16. C. Zhou, J. Kurths, B. Hu, Array-enhanced coherence resonance: nontrivial effects of heterogeneity and spatial independence of noise. Phys. Rev. Lett. **87**, 098101 (2001)
17. A. Neiman, P. Saparin, L. Stone, Coherence resonance at noisy precursors of bifurcations in nonlinear dynamical systems. Phys. Rev. E **56**, 270–273 (1997)
18. G.-Q. Sun, Z. Jin, L.-P. Song, A. Chakraborty, B.-L. Li, Phase transition in spatial epidemics using cellular automata with noise. Ecol. Res. **26**, 333–340 (2011)
19. M. Perc, M. Marhl, Evolutionary and dynamical coherence resonances in the pair approximated prisoner's dilemma game. New J. Phys. **8**, 142 (2006)
20. M. Perc, Transition from Gaussian to Levy distributions of stochastic payoff variations in the spatial prisoner's dilemma game. Phys. Rev. E **75**, 022101 (2007)
21. M. Perc, Double resonance in cooperation induced by noise and network variation for an evolutionary prisoner's dilemma. New J. Phys. **8**, 183 (2006)
22. M. Perc, A. Szolnoki, Noise-guided evolution within cyclical interactions. New J. Phys. **9**, 267 (2007)
23. M. Perc, Spatial coherence resonance in excitable media. Phys. Rev. E **72**, 016207 (2005)
24. M. Perc, Spatial decoherence induced by small-world connectivity in excitable media. New J. Phys. **7**, 252 (2005)
25. M. Perc, Coherence resonance in a spatial prisoner's dilemma game. New J. Phys. **8**, 22 (2008)
26. A.S. Pikovsky, J. Kurths, Coherence resonance in a noise-driven excitable system. Phys. Rev. Lett. **78**, 775–778 (1997)
27. D.J.D. Earn, P. Rohani, B.M. Bolker, B.T. Grenfell, A simple model for complex dynamical transitions in epidemics. Science **287**, 667–670 (2000)
28. O. Diekmann, M. Kretzschmar, Patterns in the effects of infectious diseases on population growth. J. Math. Biol. **29**, 539–570 (1991)
29. T. Reichenbach, M. Mobilia, E. Frey, Noise and correlations in a spatial population model with cyclic competition. Phys. Rev. Lett. **99**, 238105 (2007)
30. T. Reichenbach, M. Mobilia, E. Frey, Mobility promotes and jeopardizes biodiversity in rock–paper–scissors games. Nature **448**, 1046–1049 (2007)
31. B. Blasius, A. Huppert, L. Stone, Complex dynamics and phase synchronization in spatially extended ecological systems. Nature **399**, 354–359 (1999)
32. D.J. Higham, An algorithmic introduction to numerical simulation of stochastic differential equations. SIAM Rev. **43**, 525–546 (2001)
33. G.-Q. Sun, Z. Jin, Q.-X. Liu, L. Li, The role of noise in a predator–prey model with Allee effect. J. Biol. Phys. **35**, 185–196 (2009)
34. G.-Q. Sun, L. Li, Z. Jin, B.-L. Li, Effect of noise on the pattern formation in an epidemic model. Numer. Methods Partial Differ. Equ. **26**, 1168–1179 (2010)
35. G.-Q. Sun, Z. Jin, Q.-X. Liu, B.-L. Li, Rich dynamics in a predator–prey model with both noise and periodic force. BioSystems **100**, 14–22 (2010)
36. D. Alonso, A.J. McKane, M. Pascual, Stochastic amplification in epidemics. J. R. Soc. Interface **4**, 575–582 (2007)
37. A.J. Black, A.J. McKane, Stochastic amplification in an epidemic model with seasonal forcing. J. Theor. Biol. **267**, 85–94 (2010)
38. A.J. McKane, T.J. Newman, Predator–prey cycles from resonant amplification of demographic stochasticity. Phys. Rev. Lett. **94**, 218102 (2005)