Persistence and extinction of species in a disease-induced ecological system under environmental stochasticity

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Population extinction is a serious issue both from the theoretical and practical points of view. We explore here how environmental noise influences persistence and extinction of interacting species in presence of a pathogen even when the populations remain stable in its deterministic counterpart. Multiplicative white noise is introduced in a deterministic predator-prey-parasite system by randomly perturbing three biologically important parameters. It is revealed that the extinction criterion of species may be satisfied in multiple ways, indicating various routes to extinction, and disease eradication may be possible with the right environmental noise. Predator population cannot survive, even when its focal prey strongly persists if its growth rate is lower than some critical value, measured by half of the corresponding noise intensity. It is shown that the average extinction time of population decreases with increasing noise intensity and the probability distribution of the extinction time follows the log-normal density curve. A case study on red grouse (prey) and fox (predator) interaction in presence of the parasites *trichostrongylus tenuis* of grouse is presented to demonstrate that the model well fits the field data.

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I. INTRODUCTION

Predator-prey (PP) interactions in presence of infection are common in natural systems [1–6] and consequently a large number of mathematical models of predator-prey interactions have appeared in the recent past taking into account the effect of disease [7–16]. Study of such predator-prey models in presence of infection, popularly known as predator-prey-parasites (PPP) or eco-epidemiological models, is extremely important because it encapsulates both the ecological and epidemiological issues simultaneously. Mathematical models of PPP interactions extend, in most of the cases, the basic predator-prey model of either Rosenzweig-MacArthur (RM) type or Leslie-Gower (LG) type (also known as Holling-Tanner type). In the first case, only the prey has logistic (i.e., density-dependent) growth limited by a predetermined constant value, K, called the environmental carrying capacity, but not the predator. On the other hand, the logistic growth of both the populations is considered in the second type of PP models. In fact, the carrying capacity of the predator is not a constant here but rather depends on the prey density, known as the emerging carrying capacity [17]. Another distinguishing feature of these PP models lies in the fact that the predator in an LG model is a generalist one while in an RM model it is a specialist one. In an LG type PP model, the predator has a focal prey, which predators prefer to consume when in abundance, though it has other secondary food [18] on which the predator can survive in absence of its focal prey. The RM model, however, assumes a single prey for its predator [19].

All the PPP models mentioned earlier consider that the models are deterministic and therefore all model parameters (viz., birth rate, death rate, etc.) are constant. In a real ecosystem, however, these parameters are not constant due to various environmental noises and therefore fluctuate around some mean value [20,21]. Experimental evidence also supports the claim of such impact of environmental noise [22]. To make the models closer to reality, stochastic population models, therefore, have received significant attention from the researchers. There are few stochastic PPP models which either assume that the predator consumes infected prey only or the predator's functional response (prey attack rate) is type I [23–26]. These assumptions are simplifications of actual phenomena and usually done to make the analysis tractable. For example, Wei *et al.* [23] recently considered a predator-prey-parasite model with prey infection, where a predator feeds only on infected prey following the Beddington-DeAngelis response function and LG type growth of the predator. They have shown that the corresponding stochastic model has a unique positive global solution and established conditions for disease eradication and its persistence. Li and Wang [24] also studied a similar stochastic predator-prey model with disease in the predator, where the predator-prey relationship was modeled with RM type interaction. A stochastic predator-prey model with prey infection and type I response function was analyzed in [25]. It is shown that the deterministic stability results are preserved in the stochastic system. Ji and Jiang [26] considered an RM type PPP model, where the predator consumes only infected prey with type II response function. It is shown that both the deterministic system and its stochastic counterpart (with parameter perturbation in the disease transmission coefficient) have similar behavior if the noise intensity is low but the stability may be lost if the noise

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intensity is high. A four-dimensional deterministic predator-prey model with infection in both the prey and predator and type I response function was analyzed by Jang and Baglama [27]. They also simulated the corresponding continuous-time Markov chain model to study the population interaction under random effects but did not study it analytically. Stochastic PP models with different biological attributes, however, have been studied extensively [18,28–34]. Though these studies have made significant contributions in the theory and application of noise-induced population dynamics, none of these stochastic PP or PPP models has tried to fit the model with experimental data and therefore these models and the corresponding outcomes remained unverified.

Various studies [35–38] show that the stochastic model improves the predictive features of the models analyzed and fits the data well by mimicking the random fluctuations. In this paper, we first theoretically analyze an LG-type stochastic PPP model, where the predator consumes both the susceptible and infected preys with type II response function, and then validate our model with empirical data of long time population interaction. We determine some restrictions on the system parameters and find limits on the noise intensities so that population persistence is possible almost surely. Sufficient conditions for eradication of infection are determined. A relation between the intrinsic growth rate of the predator with the corresponding noise intensity is established to show that predator persistence is possible even in the absence of its focal prey provided its intrinsic growth rate exceeds some threshold value, but extinct otherwise. We have estimated numerically the average extinction time distribution for susceptible prey, infected prey, and predator populations for different noise intensities and the corresponding probability distribution curve. Finally, a case study on red grouse (prey) and fox (predator) interaction in presence of the parasites *trichostrongylus tenuis* of grouse is presented to demonstrate that the model well fits the field data collected during 1995 to 2019 and further predicts the population densities beyond the study period. It is demonstrated that both the species can coexist for a long time if some parameters are not perturbed significantly; otherwise, there is a chance of fox extinction.

II. THE MODEL

A. Deterministic model

We consider an LG type predator-prey model with x and z as the prey and predator densities at time t, where the prey follows density-dependent growth and the predator follows the type II response function:

$$\begin{aligned}\frac{dx}{dt} &= ax - bx^2 - \frac{cxz}{m_1 + x},\\ \frac{dz}{dt} &= z\left[r - \frac{fz}{m_2 + x}\right],\end{aligned} \tag{1}$$

where a is the intrinsic growth rate of the prey, b is the intraspecies competition coefficient, c is the predator's attack rate, r is the intrinsic growth rate of the predator, f is the intraspecies competition of the predator, and m_2 is the half-saturation constant of the predator. A parasitic infection divides the prey population into a susceptible group and an infected group. The disease spreads horizontally, having disease transmissibility λ, and there is no vertical transmission. Infection may cause various modifications to its host, e.g., conspicuousness, castration, lower competitive ability, higher mortality, altered behavior, increased vulnerability, etc. [1–6]. Based on this empirical evidence, it is assumed that infected preys are unable to give birth and do not recover. The infected prey dies due to infection at a rate of γ and has intraspecies competition but no interspecies competition. Predators are not affected by the parasites and they consume that prey which is readily available. Both the susceptible and infected preys give the same reproductive gain to the predator population. These assumptions provide the following PPP model:

$$\begin{aligned}\frac{dx}{dt} &= ax - bx^2 - \lambda xy - \frac{cxz}{m_1 + x + y},\\ \frac{dy}{dt} &= \lambda xy - my^2 - \frac{eyz}{m_1 + x + y} - \gamma y,\\ \frac{dz}{dt} &= z\left[r - \frac{fz}{m_2 + x + y}\right],\end{aligned} \tag{2}$$

where y is the density of the infected prey at time t. The parameter m is the intraspecies competition coefficient of infected prey and e is the predation rate of infected prey. This model has similarity to the models [13,39]. In fact, Haque and Venturino [13] did not consider the inter- and intraspecies competition between and among the hosts, while it was considered in [39]. Inclusion of inter- and-intra-species competitions produces product terms like xy and x^2, y^2, respectively, which may be combined to deduce the model (2) from [39]. They mainly studied the stability and instability (through Hopf bifurcation) of different equilibrium points. Global stability of the interior equilibrium has been shown under nontrivial parametric restrictions. The basic reproduction number, secondary cases produced by an infected individual, for the deterministic system is shown to be $R_0^D = \frac{a\lambda}{b\gamma}$, and infection eradication is possible if $R_0^D < 1$ [39]. It was pointed out that the competition coefficient, b, contributes positively in the disease eradication process and makes a difference with the earlier study [13] which does not contain the intraspecific competition. Similar PPP systems, however, may show more complicated dynamics (including chaos) in presence of disease transmission delay [40]. A recent study [41], however, shows that chaos in a delay-induced PPP system may be suppressed through proper harvesting of prey species. It is mentionable that all these studies are described in a deterministic setting and have not been explored under environmental stochasticity.

B. Incorporating stochasticity

Note that the deterministic model (2) is based on the mean-field theory, where each quantity is averaged. This averaging of the quantity is valid if system population is large, but becomes invalid if the population is small [42], as small population behaves differently compared to its larger counterpart due to the loss of heterogeneity. There are three different types of stochasticity considered in ecological systems: demographic stochasticity, measurement stochasticity, and environmental stochasticity. The first type of stochasticity

is due to the endogenous causes and may appear through random variation in fecundity or survival due to genetic factor, disproportionate sex ratio, sexual selection, etc., and has a strong effect on small population [43]. The measurement stochasticity is caused by factors that randomly cause measurements of the variables up and down. The most important is the environmental stochasticity caused by exogenous factors. Stochasticity in the physical and biological environment may cause significant fluctuations even in a large population [44]. We are interested here in this environmental stochasticity. Introduction of such stochasticity in a population model may be done in various ways, e.g., following Markov process [45–47] and parameter perturbation technique [48–50].

The nature of the noise may be additive or multiplicative. The key difference between additive and multiplicative noises is that the noise is directly added to the system in the former case, and in the latter case, it is multiplied with the state variables [51]. The main disadvantage of additive noise is that, with an initial very low density of population, a negative fluctuation of noise could make the solution of the stochastic system negative, which is nonphysical because population density cannot be negative. In contrast, the multiplicative noise always ensures the non-negativity of the solution, even if there are initial negative fluctuations, because the noise appears in the exponential function. More specifically, for a linear multiplicative stochastic differential equation, the solution is purely exponential and the fluctuating term (i.e., the Wiener process) appears in the exponent, while for a nonlinear system a functional appears in the exponent [52]. Moreover, as the stochastic solution for multiplicative noise is exponential, the zero population density becomes a barrier for the solution, and for specific system parameters and noise strength level, strong negative fluctuation can bring the solution to zero density. Understanding the effectiveness of multiplicative noise in population dynamics, we use this noise to construct the stochastic system from its deterministic version (2).

The system to be analyzed may contain several parameters, but in which parameters perturbation will be introduced depending on biological and physical reasons. For example, rainfall, humidity, and temperature affect the food production as well as species growth [53,54]. Growth of many pathogens and their virulence is dependent on temperature [55,56]. Parameter perturbation technique considers such environmental stochasticity through direct perturbation of such parameters. We, therefore, considered random perturbations in the growth parameter of susceptible prey, virulence parameter, and growth parameter of the predator as

$$a \to a + \sigma_1 dW_1(t),\ -\gamma \to -\gamma + \sigma_2 dW_2(t),$$
$$r \to r + \sigma_3 dW_3(t),$$

where $W_i(t)$, $i = 1, 2, 3$ are mutually independent Wiener processes defined on a complete probability space (Ω, F, P) with a filtration $\{F_t\}_{t\in\Re^+}$ and σ_i^2 are the intensities of noises. The Wiener process $dW_i(t)$ satisfies the properties $\langle dW_i(t)\rangle = 0$ (gives the average value) and $\langle dW_i(t), dW_i(t')\rangle = \delta(t - t')$ (defines the correlation function), where δ is the Dirac delta function. Under these assumptions, the model (2) becomes

$$dx = \left[ax - bx^2 - \lambda xy - \frac{cxz}{m_1 + x + y}\right]dt + \sigma_1 x dW_1(t),$$
$$dy = \left[\lambda xy - my^2 - \frac{eyz}{m_1 + x + y} - \gamma y\right]dt + \sigma_2 y dW_2(t),$$
$$dz = \left[rz - \frac{fz^2}{m_2 + x + y}\right]dt + \sigma_3 z dW_3(t). \quad (3)$$

All parameters are non-negative. We analyze the stochastic model (3) with positive initial conditions $x(0) > 0$, $y(0) > 0$, $z(0) > 0$. There are, however, other techniques of considering stochasticity in a mathematical model. For example, one can consider stochastic perturbations as proportional to the distance of the state variable from the deterministic steady state [57,58]. This process is particularly useful to verify the robustness of various stability results of the corresponding deterministic system [57].

III. MATHEMATICAL RESULTS

We are interested in the equilibrium point $E^*(x^*, y^*, z^*)$, where all populations coexist. In the absence of noise, the existence of a unique equilibrium point of the system (3) and its stability can be deduced from [39]. A unique positive interior equilibrium point exists (see Appendix A) if $e > c$, $\lambda > \lambda^*$, $m < m^*$ and $x^* < \frac{ae+\gamma c}{be+\lambda c}$. The first condition says that the infected prey is predated at a higher rate compared to its healthy counterpart. This is a reasonable restriction because, by assumption, predators do not discriminate between infected and healthy preys, rather they consume that prey which is readily available. Since the infected prey has reduced mobility, the attack rate is expected to be higher on infected prey compared to healthy prey. Understandably, the infection cannot persist if the force of infection is too weak. Consequently, for the existence of an infected class, disease transmission rate should exceed some lower threshold value, λ^*. It is mentionable that the intraspecies competition arises when the same species compete for limited resources. Due to the lower fitness, the intraspecies competition among the infected prey is much smaller than that of the susceptible prey, i.e., $m < b$. The third restriction $m < m^*$ describes the threshold level of intraspecies coefficient of the infected preys. The last condition prescribes an upper bound in the equilibrium density of healthy prey for the existence of infected prey. Infected prey cannot exist if the last inequality is reversed (see Appendix A). In addition to these conditions, the parameters need to satisfy some nontrivial conditions for the local stability of this equilibrium (see Appendix A).

We are mainly concerned about the solutions of the stochastic system (3). For any population model, the first thing one needs to investigate is the non-negativity of the stochastic solution and its global existence. Introduction of multiplicative noise can induce population explosion [59,60]. It is, therefore, essential to show the boundedness of solutions, which means that the interacting species will not grow abruptly or exponentially for a long time. We have shown, for any positive initial value $(x(0), y(0), z(0)) \in \mathbb{R}^3_+$, that there exists a unique solution $(x(t), y(t), z(t)) \in \mathbb{R}^3_+$ of the system (3) (see Appendix B) which remains positive and bounded for all $t \geqslant 0$ with probability 1 (see Appendix C). An important aspect of population biology is the extinction and persistence of interacting species. It is important to know whether the

species of the system will die out in finite time or survive. From a mathematical point of view, the persistence of species may be weak or strong [61]. Suppose the function $g(t)$ represents a population at any time t and $g(t) > 0$ for all $t \geqslant 0$, then $\frac{1}{t}\int_0^t g(\theta)\,d\theta$ is the average value of population in the time span $[0, t]$. The population is said to be strongly persistent if the infimum of these limiting averages is always positive. Therefore, the eventual average population size will always remain away from zero, ensuring the existence of populations for all time. In the case of weak persistence, the supremum of these eventual averages is always positive; it, however, does not give any guarantee that the average population will always remain away from zero and therefore population may be arbitrarily closed to zero. Nonpersistent implies that the supremum of these eventual averages is zero. However, extinction is guaranteed if the supremum of these eventual averages becomes negative. The following results regarding persistence and nonpersistence of the species can be proved (see Appendices D and E).

Theorem III.1 (i) If $a < \frac{\sigma_1^2}{2}$ then $x(t)$ will go to extinction almost surely (a.s.).

(ii) If $a = \frac{\sigma_1^2}{2}$ then $x(t)$ is nonpersistent in the mean a.s.

(iii) If $a > \frac{\sigma_1^2}{2} + \lambda\bar{y} + \frac{c\bar{z}}{m_1}$ then $x(t)$ is strongly persistent in the mean a.s.

Theorem III.2 (a) For $a < \frac{\sigma_1^2}{2}$, $y(t)$ will go to extinction a.s.

(b) For $a > \frac{\sigma_1^2}{2}$,

(i) if $\lambda(a - \frac{\sigma_1^2}{2}) < b(\gamma + \frac{\sigma_2^2}{2})$ then $y(t)$ will go to extinction a.s.

(ii) if $\lambda(a - \frac{\sigma_1^2}{2}) = b(\gamma + \frac{\sigma_2^2}{2})$ then $y(t)$ is nonpersistent in the mean a.s.

(iii) if $\lambda(a - \frac{\sigma_1^2}{2}) > b(\gamma + \frac{\sigma_2^2}{2}) + \lambda^2\bar{y} + (\lambda c + be)\frac{\bar{z}}{m_1}$ then $y(t)$ is strongly persistent in the mean a.s.

Theorem III.3 (i) If $r < \frac{\sigma_3^2}{2}$ then $z(t)$ will go to extinction a.s.

(ii) If $r = \frac{\sigma_3^2}{2}$ then $z(t)$ is nonpersistent in the mean a.s.

(iii) If $r > \frac{\sigma_3^2}{2}$ then $z(t)$ is strongly persistent in the mean a.s.

These results provide a quantitative measure on the system parameters and/or prescribe some limits on the environmental noises for which both the prey and predator populations can persist together or in isolation. They show that species extinction may occur through many routes. For example, if the intrinsic growth rate of a healthy prey is less than half of the corresponding noise intensity, then a healthy prey cannot survive but can survive provided its growth rate exceeds this critical value. It is obvious that an infected prey also cannot survive in absence of a susceptible prey (see Theorem III.2 a). Theorem III.2 b says that infected prey $y(t)$ will go extinct while sound prey $x(t)$ may persist if the basic reproduction number $R_0^S < 1$, where $R_0^S = \frac{\lambda(a-\frac{\sigma_1^2}{2})}{b(\gamma+\frac{\sigma_2^2}{2})}$. This is an extremely important measure from the infection management point of view. It describes that disease control may be possible by tuning some system parameters as well as the noise parameters. The parameters b and γ, measuring the intraspecies competition and removal rate of the infected prey, have a negative correlation with the basic reproduction number, and R_0^S can be reduced to below unity by increasing these rate parameters. In contrast, the infection rate parameter λ is positively correlated with R_0^S. Interestingly, both the noise parameters are negatively correlated with R_0^S. Thus, proper adjustment of environmental noise can potentially change the disease state of a system. The last theorem prescribes a relationship between the predator's intrinsic growth rate and noise intensity that allows its survival.

It is to be mentioned that most of the stochastic systems have no exact equilibrium; instead, they may have a time-independent probability distribution [62]. The following theorem (see Appendix F for proof) shows the existence of such stationary distribution for the populations of system (3).

Theorem III.4 Let $(x(t), y(t), z(t)) \in \mathbb{R}^3$ be a solution of the stochastic system (3) with initial value $(x(0), y(0), z(0)) \in \mathbb{R}^3$. If the conditions (i) $\frac{fz^*}{rm_2^2} > \max\{\frac{Ac}{m_1+\bar{x}+\bar{y}}, \frac{Ae}{m_1+\bar{x}+\bar{y}}\}$, (ii) $Ab > \frac{cz^*}{m_1} + \frac{fz^*}{2rm_2^2} + \frac{(c+e)z^*}{2m_1}$, (iii) $m + \frac{Ae}{m_1+\bar{x}+\bar{y}} > \frac{ez^*}{m_1} + \frac{fz^*}{2rm_2^2} + \frac{(c+e)z^*}{2m_1}$, and (iv) $\frac{f}{r(m_2+\bar{x}+\bar{y})} + \frac{A(e+c)}{2(m_1+\bar{x}+\bar{y}} > \frac{fz^*}{rm_2^2}$ are satisfied, then

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t [(x(s)-x^*)^2 + (y(s)-y^*)^2 + (z(s)-z^*)^2]ds \leqslant G\Theta \text{ a.s.},$$

where $A = m_1 + x^* + y^*$, $\Theta = \frac{A\sigma_1^2 x^*}{2} + \frac{A\sigma_2^2 y^*}{2} + \frac{\sigma_3^2 z^*}{2r}$, $G = \frac{1}{\min\{K,Q,T\}}$, $K = Ab - \frac{cz^*}{m_1} - \frac{fz^*}{2rm_2^2} - \frac{(c+e)z^*}{2m_1}$, $Q = m - \frac{ez^*}{m_1} - \frac{fz^*}{2rm_2^2} - \frac{(c+e)z^*}{2m_1} + \frac{Ae}{2(m_1+\bar{x}+\bar{y})}$, and $T = \frac{f}{r(m_2+\bar{x}+\bar{y})} + \frac{A(e+c)}{2(m_1+\bar{x}+\bar{y}} - \frac{fz^*}{rm_2^2}$; $\bar{x}$, $\bar{y}$, $\bar{z}$ are the stochastic bounds of $x(t)$, $y(t)$, $z(t)$, respectively; and (x^*, y^*, z^*) is the interior equilibrium point of the deterministic system (2).

This shows that $\Theta \to 0$ if the noise intensities σ_1, σ_2, σ_3 tend to zero, and we then have

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t [(x(s)-x^*)^2 + (y(s)-y^*)^2 + (z(s)-z^*)^2]ds \to 0,$$

yielding $\lim_{t\to\infty}(x(t), y(t), z(t)) = (x^*, y^*, z^*)$. Therefore, the stochastic solution will remain close and eventually approach the time-independent equilibrium solution of the deterministic system when noise intensities are negligible.

IV. SIMULATION RESULTS

A simulation study has been performed in two steps. First, we illustrate the theoretical results presented in the previous section, and in the second step, we consider an experimental data set to demonstrate how our stochastic model fits these data.

A. Effect of environmental noise on the persistence and extinction of species

It is to be recalled that a deterministic model always gives a unique solution corresponding to a unique initial point when system parameters remain fixed. However, a stochastic solution of the same system gives different solutions for each simulation due to its inherent stochasticity even when the

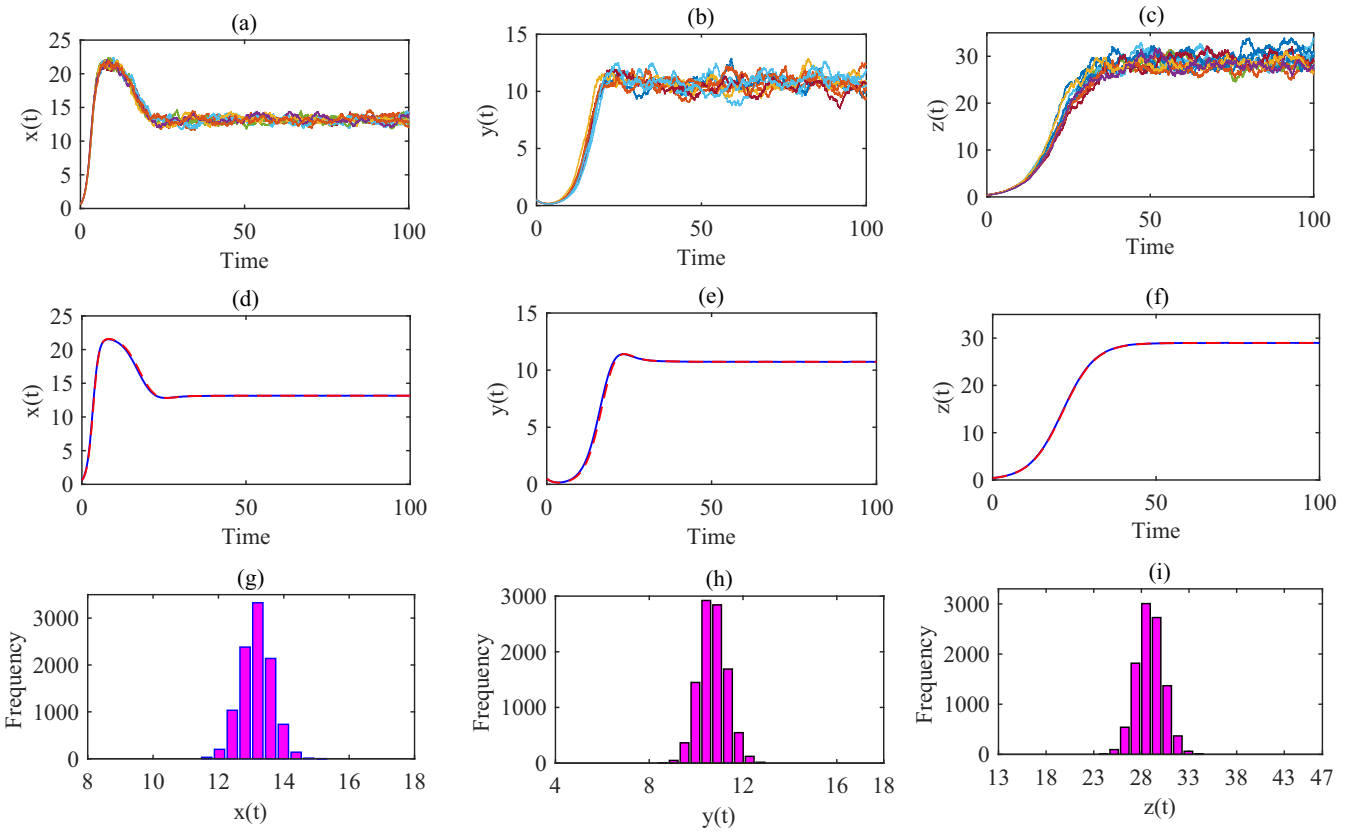

FIG. 1. Upper panel: Time series solutions of 15 simulations of the stochastic system (3) with noise intensity $\sigma_1 = 0.03$, $\sigma_2 = 0.03$, $\sigma_3 = 0.03$. Middle panel: Average value of 1000 time series solutions with the same noise intensity. It shows that the stochastic solution (solid blue curve) and the deterministic solution (red broken line) are qualitatively and quantitatively similar. Lower panel: Frequency distribution of the populations at $t = 100$ for 10 000 simulations of system (3). It shows small fluctuations in the population densities around the deterministic steady state value $E^*(x^*, y^*, z^*) = (13.16, 10.73, 28.98)$. Parameters are $a = 1.1$, $b = 0.05$, $\lambda = 0.04$, $c = 0.1$, $e = 0.12$, $f = 1.2$, $m_1 = 200$, $m = 0.001$, $\gamma = 0.5$, $r = 0.2$, $m_2 = 150$ and the initial value is (0.6,0.5,0.4).

initial value and system parameters remain the same (see Fig. 1, first row). It will be, therefore, prudent to plot the mean value of such solutions corresponding to a fixed value of the noises to represent the overall behavior of the system's solutions. We first demonstrate how different noise intensities can alter persistency of interacting species while the other system parameters remain unchanged. Choosing weak noise intensities like $\sigma_1 = 0.03$, $\sigma_2 = 0.03$, $\sigma_3 = 0.03$ so that the conditions of stochastic persistence [see Theorems III.1(iii), III.2 b(iii), and III.3(iii)] and stationary distribution (Theorem III.4) are satisfied, one can observe that the stochastic and deterministic solutions (Fig. 1, middle row) show similar behavior and the population densities of system (3) remain very close to the equilibrium solution $E^*(13.16, 10.73, 28.98)$ of the deterministic system (2). In the lower panel of Fig. 1, we presented the frequency distribution, where the widths of rectangles represent various classes, and their heights indicate the frequency of the class. It is to be mentioned that the coefficient of variation of the time series solution of the stochastic system (3) is very low after $t = 100$ and, therefore, the system was run for 100 to show the asymptotic behavior. The behavior will remain the same for higher runs. Distribution of the rectangles indicates how much the stochastic solutions will oscillate around the deterministic steady state for the considered noise intensity. It can be observed that x, y, and z populations are distributed in the range (11.6,15.2), (8.6,12.8), and (24,34.1), respectively, and the highest frequency is observed at 13.2, 10.4, 28.5, which is very close to the deterministic steady state value $E^*(x^*, y^*, z^*) = (13.16, 10.73, 28.98)$. If we increase the strength of noises then the fluctuation increases. For higher values of $\sigma_1 = 0.1, \sigma_2 = 0.1, \sigma_3 = 0.1$, the population densities (Fig. 2) fluctuate more around the deterministic steady state $E^*(x^*, y^*, z^*)$. In this case, the frequency distribution of $x(t)$, $y(t)$, and $z(t)$ populations is over a larger range (8.6,18), (5.1,16.8), (13.4,45.8) around the deterministic steady state values $x^* = 13.16$, $y^* = 10.73$, and $z^* = 28.98$. The "probabilistic smoke cloud" of the system (3) for the above two sets of forcing intensities is shown in Fig. 3. For the lower value of noises, populations are distributed in a smaller region around the deterministic equilibrium value in comparison to the higher value of noises.

Further increase of noise intensity may cause stochastic extinction of system populations, for example, an increase in the noise intensity of the infected prey, say $\sigma_2 = 0.95$, so that the conditions of Theorems III.1(iii), III.2 b(i), and III.3(iii) are satisfied and then the infected population is extinct (Fig. 4, upper row). Noticeably, when infected species die out then the susceptible population density lies above its deterministic steady state value. Thus, environmental noise can make a

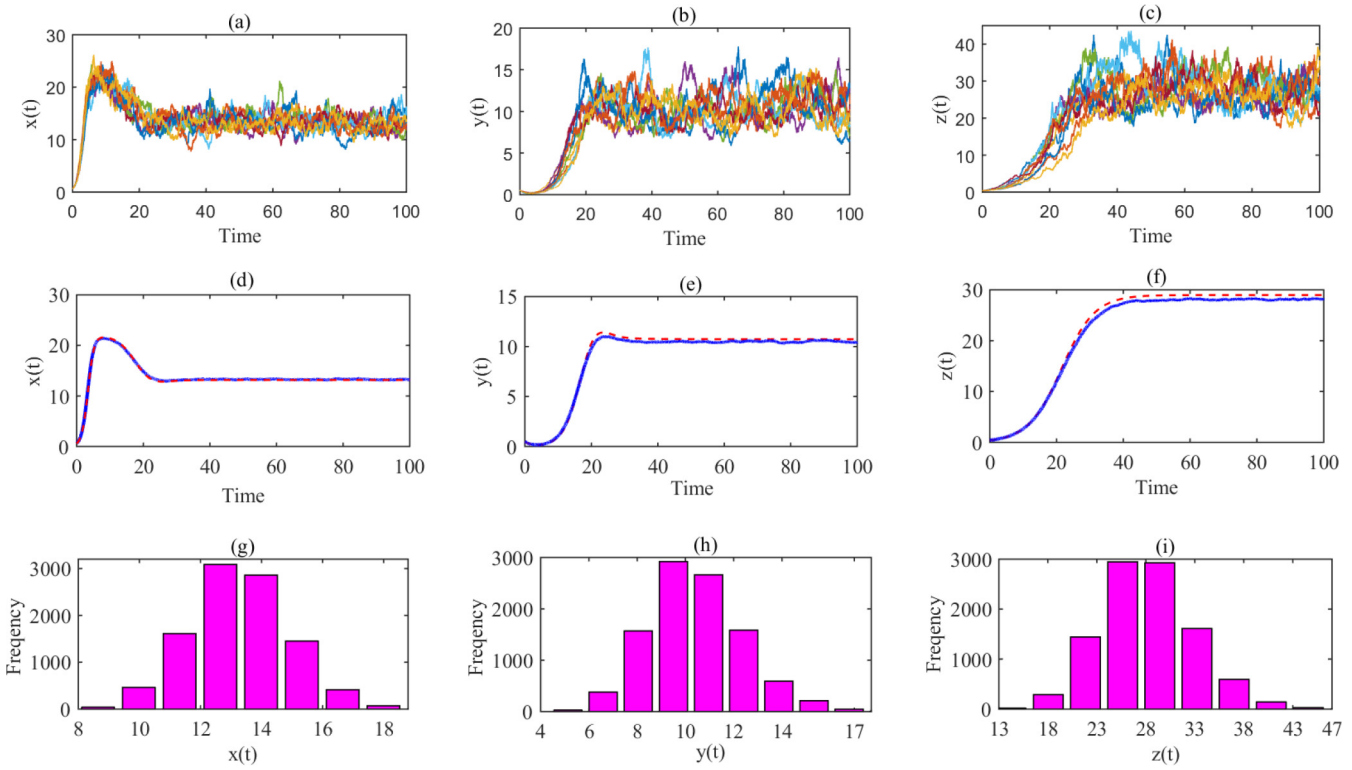

FIG. 2. Upper panel: Time series solutions of 15 simulations of the stochastic system (3) with noise intensity $\sigma_1 = 0.1, \sigma_2 = 0.1, \sigma_3 = 0.1$. Middle panel: Average value of 1000 solutions of system (3) with the same noise intensity (solid blue line) and the solution of the deterministic system (2) (red broken line). Lower panel: Frequency distribution of the respective populations obtained at $t = 100$ for 10 000 simulations of system (3). Parameters are as in Fig. 1.

system infection free provided the noise intensity has a higher impact on the infected prey; however, the infection persists in an unvarying environment. If we increase the noise intensity in the growth rate of the susceptible population to $\sigma_1 = 1.49$, keeping all other noise intensities and parameters as in Fig. 1, to satisfy the condition of Theorem III.1(i) [see also Theorem III.2(a)] then both the susceptible and infected populations go to extinction but the predator survives with a lower density (Fig. 4, second row) by consuming its alternative food. It is to be recalled that the considered prey is the primary food of the predator and the predator can survive in absence of its focal prey at a lower density by consuming its secondary prey. If the noise intensities are such that $\sigma_1 = 0.01 = \sigma_2$ and $\sigma_3 = 0.74$ so that the conditions of Theorems III.1(iii), III.2 b(iii), and III.3(i) are fulfilled then both the prey populations coexist but the extinction of predator population occurs

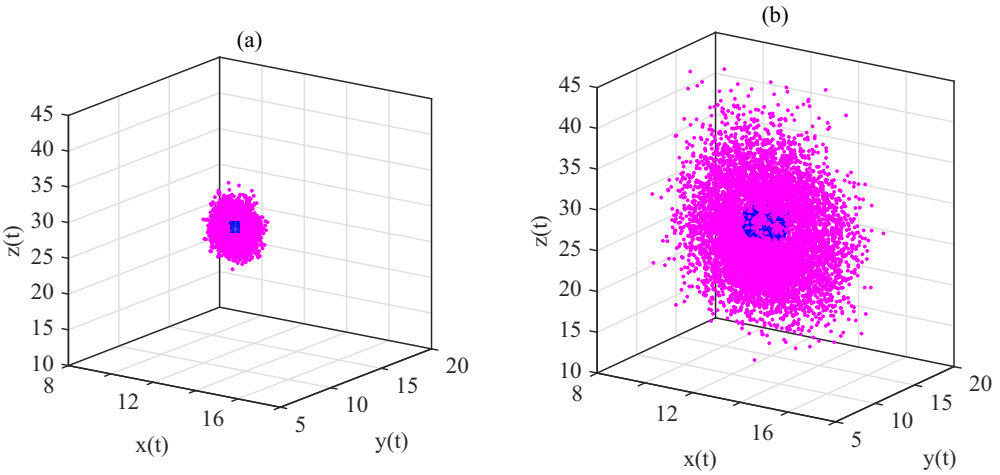

FIG. 3. Stationary distribution of populations of the stochastic system (3) at $t = 100$ is plotted (in pink dots) around the deterministic steady state (in blue dot). It shows how population densities are distributed around the deterministic equilibrium value for lower and higher values of noise. Left panel: $\sigma_1 = 0.03, \sigma_2 = 0.03, \sigma_3 = 0.03$. Right panel: $\sigma_1 = 0.1, \sigma_2 = 0.1, \sigma_3 = 0.1$. Parameters are as in Fig. 1.

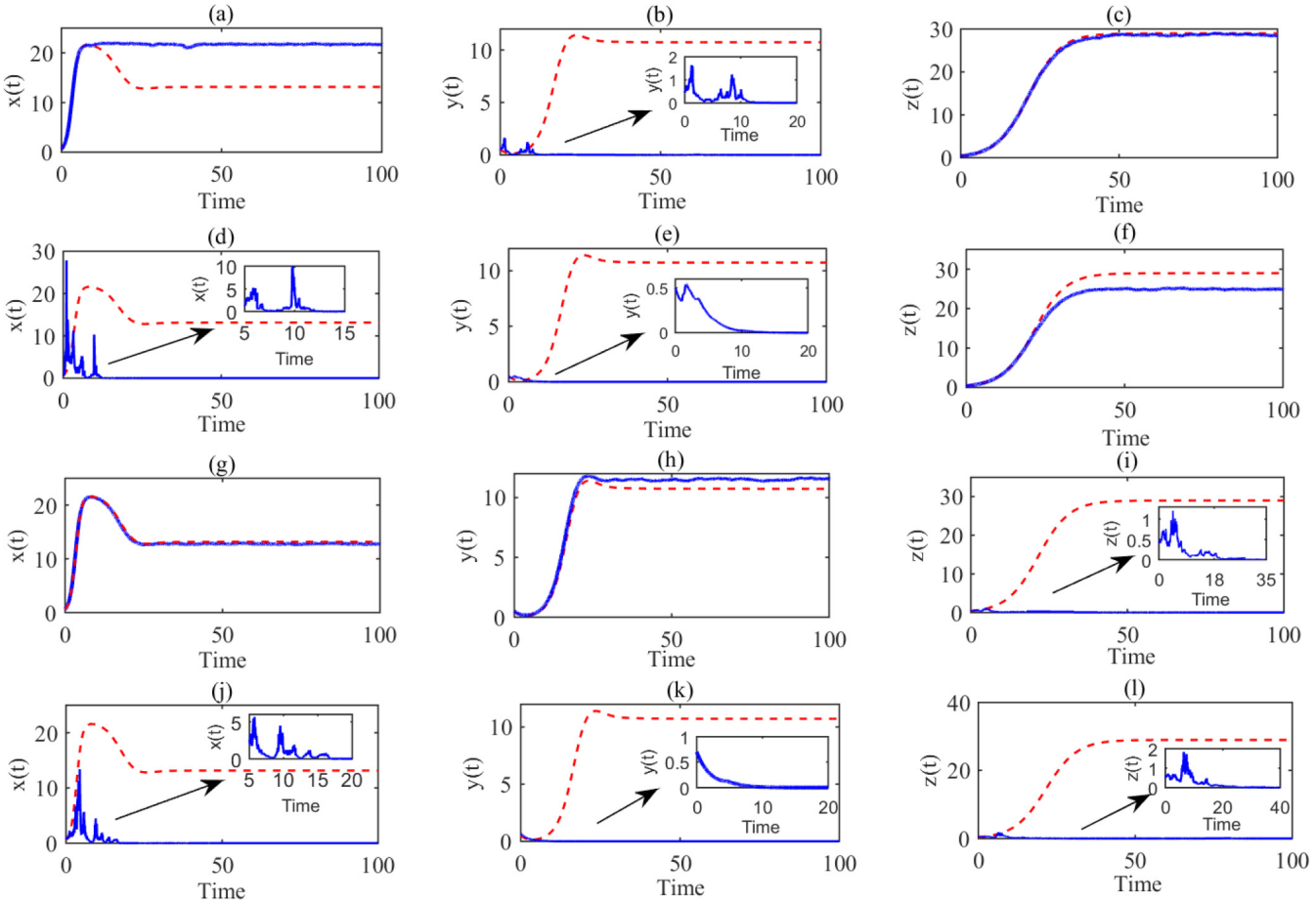

FIG. 4. Solution of the stochastic system (3) with different noises. First row: y population goes to extinction within a short period when $\sigma_1 = 0.01$, $\sigma_2 = 0.95$, $\sigma_3 = 0.01$. Second row: x and y populations are extinct but $z(t)$ survive at a lower density when $\sigma_1 = 1.49$, $\sigma_2 = 0.01$, $\sigma_3 = 0.01$. Third row: z population goes to extinction while x and y populations survive when $\sigma_1 = 0.01$, $\sigma_2 = 0.01$, $\sigma_3 = 0.74$. Last row: All species go to extinction due to higher environmental noise $\sigma_1 = 1.49$, $\sigma_2 = 0.01$, $\sigma_3 = 0.74$. Deterministic solution (dash line) of the system, however, reaches to the coexistence equilibrium value in each case. Parameters are as in Fig. 1.

(Fig. 4, third row) due to higher environmental noise on predator population. If the noise intensity on the predator, σ_3, is kept high ($\sigma_3 = 0.74$) with $\sigma_1 = 1.49$ and $\sigma_2 = 0.01$, then the extinction criteria of predator population [Theorem III.3(i)] as well as the extinction criteria of prey population [Theorem III.1(i)] are satisfied. In such a case, all populations die out due to environmental noise; however, populations of the deterministic system coexist in a stable state (Fig. 4, last row). It is mentionable that population persists and solutions (dashed line) of the deterministic system in each case reach the coexistence equilibrium value, implying that stochasticity can destroy deterministic persistency and stability results. All these results support the fact that environmental noise has a profound influence on the persistence and extinction scenario of interacting species.

In Fig. 4, we have deciphered the various extinction scenarios of the population taking the average of 1000 runs of the stochastic system with the same parameter set and initial value. It is obvious that the extinction time of the individual run of these 1000 simulations is different. It will be, therefore, interesting to see the probability distribution of extinction time. In the upper two panels of Fig. 5, we have presented the average extinction time distribution for susceptible, infected, and predator populations for different fixed noise intensities (as in the Fig. 4) and the corresponding probability distribution curve. Figure 5(e) indicates that there is no extinction of the infected prey population before ten units of time. In population ecology, it is a common phenomenon that extinction time of a large number of individuals of a population is more or less similar, but some others may survive a long time. It is also not unusual to observe that some infected individuals survive for a long time whereas most of the individuals infected with the same parasites die within some average time. In such a case, the data have a low mean and large variance. Data of such skewed distributions often fit log-normal distribution [63]. We also observed here that the log-normal curve fits well the extinction time probability distributions. In the last row of Fig. 5, the average extinction times of 1000 simulations are estimated with varying noise intensity, showing that the extinction time of population decreases with increasing noise intensity. This is in accordance with our analytical result (see Appendix E, Remark E.1). Similar monotonic decreasing behavior of extinction time was also observed in other studies [18,64]. However, nonmonotonic behavior of the extinction time as a function of noise intensity was also observed in two competing species with stochastic

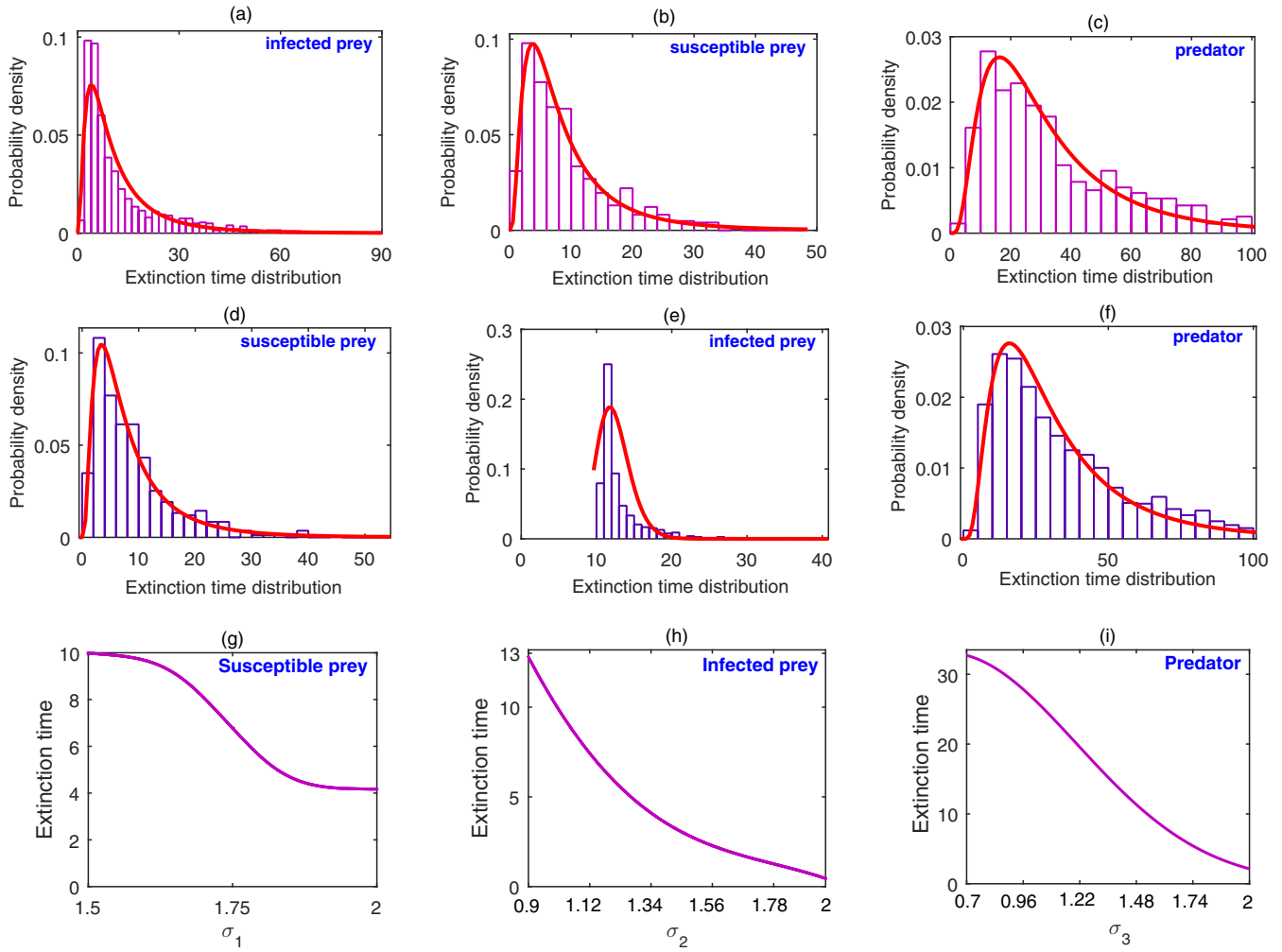

FIG. 5. Probability density and the density curve of extinction time when the stochastic system was run 1000 times with the same set of parameter values of Fig. 4. Upper row: (a) Noise intensity as in the first row of Fig. 4. (b) Noise intensity as in the second row of Fig. 4. (c) Noise intensity as in the third row of Fig. 4. The red curve in all figures denotes the corresponding probability density curve, which satisfies the log-normal distribution function with p value less than 0.0001. Middle row: Probability density of extinction times of (a) susceptible prey, (b) infected prey, and (c) predator when noise intensities are as in the last row of Fig. 4 (where all populations are extinct due to the noise). The red curve is the fitted log-normal probability distribution function. Here mean (μ) and standard deviation (σ) of the fitted log-normal probability distribution functions are (a) $\mu = 1.951\,79$, $\sigma = 0.797\,037$, (b) $\mu = 2.174\,52$, $\sigma = 0.882\,333$, (c) $\mu = 3.292\,99$, $\sigma = 0.709\,178$, (d) $\mu = 1.877\,99$, $\sigma = 0.805\,53$, (e) $\mu = 2.494\,11$, $\sigma = 0.173\,672$, (f) $\mu = 3.265\,33$, $\sigma = 0.708\,356$. Lower row: Extinction time plotted against the varying noise intensity for all three populations. When one noise intensity is varied, then the other two noise intensities remain fixed at 0.01.

resonance [60] and in the Verhulst model with Levy white noise [65].

B. Red grouse: A case study

The red grouse *Lagopus lagopus scoticus*, predominantly observed in heather dominated moorlands of upland Britain, has contributed largely to the long term study of population ecology [66]. Many private estates cultivate red grouse to use them as a game bird [67] and employ gamekeepers to maximize their number. The red grouse population is very much unstable and shows frequent fluctuations over time [68,69]. Long time field data of red grouse clearly show such cyclic and quasicycle behavior [69,70]. Fox is the main predator of the grouse population. Though red grouse is the focal prey of fox in the moorlands of Scotland, it also feeds on other species, like vole [71,72]. These ground-nesting birds are frequently infected by the parasites *trichostrongylus tenuis* [5]. Even though red grouse develops various adaptation for its defense, detection and predation become easier for the fox as the grouse emits a particular scent while the parasite burden is higher in their bodies [73]. Thus our model assumptions perfectly match with the empirical examples of red grouse-fox interaction in presence of infection. We here examined how our stochastic model can effectively predict the long term dynamics of this PPP system.

The method for estimation of parameters of the deterministic system (2) and noise intensities for stochastic system (3) using the grouse and fox data set of the United Kingdom taken from the British Trust for Ornithology [74] for the period 1995

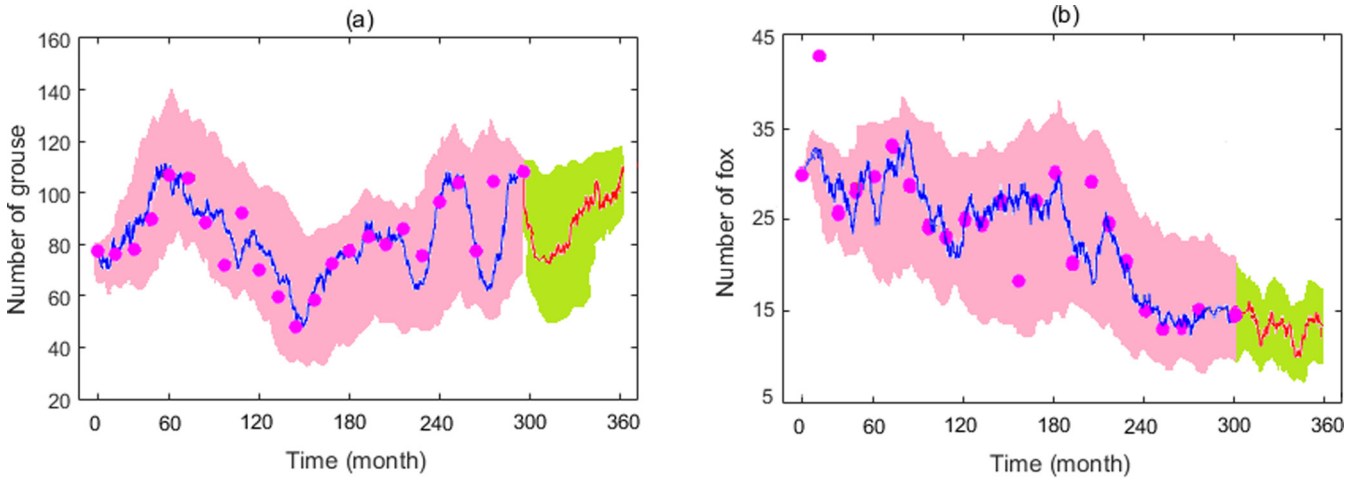

FIG. 6. Comparison of time series solutions of the stochastic system (3) with the field data of red grouse and fox population. Left panel: Actual red grouse data for the period 1995 to 2019 are presented by solid circles in magenta color. Simulated stochastic time series data of total grouse population are presented by a solid line in blue color. Right panel: Actual fox population data are presented by solid circles and simulated stochastic time series data of fox density are presented by the blue line. Parameters are $a = 0.56$, $b = 0.001\,44$, $\lambda = 0.036$, $e = 5.12$, $\gamma = 0.0875$, $c = 0.21$, $f = 0.0886$, $m_1 = 101$, $m = 0.0428$, $r = 0.032$, $m_2 = 41$ and initial value is (50,27,30). Noise strengths are $\sigma_1 = 0.1$, $\sigma_2 = 0.05$, $\sigma_3 = 0.09$. In both figures, the red color curve is the predicted population densities for the next five years. The shaded region represents the 95% confidence interval. The r-squared values for red grouse and fox data are, respectively, 0.7426 and 0.7231.

to 2019 is given in Appendix G. The initial values of grouse and fox populations were considered as 77 and 30 per square kilometer, which were their respective values in the year 1995. An adult female grouse lays 6 to 12 eggs per year [75] and two-thirds of the grouse chicks survive [76,77]. Thus, the new recruitment of red grouse is between 4 and 9 per year per adult female grouse. In our estimation, we found the birth rate of grouse a as 0.56 per month, i.e., 6.7 newborn grouse per year. The estimated value of intraspecies competition coefficient b is 0.001 44. The death rate of grouse (γ) is estimated to be 0.0864, which is very close to the field estimated value, 0.0875 [78]. Fox predation of red grouse (e) has been observed to vary from one to two grouse per week [5], giving the average predation 4 to 8 in a month. We estimated the predation rate parameter e as 5.12 per month, which lies within the experimental range. Parasitic infection rate (λ) in red grouse has been reported as 0.16 to 0.6 per year [79] (i.e, 0.0133 to 0.5 per month) and our estimated value is 0.036. The other parameters estimated through curve fitting are $c = 0.21$, $f = 0.0886$, $m_1 = 101$, $m = 0.0428$, $r = 0.032$, $m_2 = 41$. Using these parameter values, we plotted (blue curve) the total red grouse population (susceptible and infected) [Fig. 6(a)] and fox population [Fig. 6(b)] obtained from the average of 1000 simulations of the stochastic system (3) with noise intensities $\sigma_1 = 0.1, \sigma_2 = 0.05, \sigma_3 = 0.09$. It shows that the stochastic model solutions well match the 25 years (1995 to 2019) of field data. Furthermore, we extended our simulation results for another five years to predict the red grouse and fox population (red curve) beyond the study period. It shows that both the red grouse and fox populations coexist and will continue to do so if vital parameters and environmental noise are not perturbed significantly. Culling of foxes is an old practice in Great Britain. It was estimated that 190 000 foxes were collectively killed annually by hounds, gamekeepers, and farmers [80]. Such culling can significantly reduce fox population on a regional scale [81] and can eventually lower the intrinsic growth rate of the fox population below some critical level, which can be determined from Theorem III.3. The effect of such a reduced growth rate may send the fox population to extinction on a local scale.

V. DISCUSSION

The ubiquitous ecological phenomena predator-prey interaction is frequently influenced by parasites. Environmental stochasticity, on the other hand, may play a critical role in the persistence or extinction of any biological species. Study of such predator-prey models in presence of infection is extremely important because it encapsulates both the ecological and epidemiological issues simultaneously. Population extinction is a serious issue both from the theoretical and practical points of view. Interacting populations in a natural system may go to extinction in a variety of ways. Such extinction routes have been shown in single-species discrete systems [82] and two species continuous predator-prey systems by defining a master equation [83]. It is therefore interesting to know the routes to extinction in the higher-dimensional systems. In this paper, we considered a predator–prey-parasite model, where the interaction between prey and predator follows the modified Leslie-Gower (or Holling-Tanner) type model with a type II functional response. A parasite infects the prey population and the predator feeds on both the susceptible and infected preys. Environmental stochasticity was incorporated into the system by considering random perturbation, where an error term is added with the average value of a parameter in which perturbation has to be introduced. The error term, in general, follows a normal distribution and, therefore, can be approximated by a white noise [84]. Reproduction and death are frequently affected by the environmental noise [85] and, consequently, the random perturbation was considered in

the intrinsic growth rate of susceptible prey, the death rate of infected prey, and growth rate of predator population.

The main objective of this paper is to explore the population extinction routes in a PPP system due to environmental stochasticity even when the populations remain stable in its deterministic counterpart. We, therefore, restricted our deterministic analysis to the local stability of the coexisting or interior equilibrium point only. For the stochastic model, we first showed the non-negativity and global existence of a solution and proved its boundedness to mean that interacting populations will not grow abruptly for a long time and each population density will have some upper limit. It is also shown that there exists a stationary distribution of the populations under some parametric restrictions. We have proved that the asymptotic behavior of the stochastic solution can be made very close to the coexistence equilibrium solution of the deterministic solution by choosing noise intensity small. Some sufficient conditions have been prescribed on some important parameters as well as on the noise intensities so that both the prey and predator populations persist together or in isolation for a long time. For example, Theorems III.1(i) and III.2(a) say that both the susceptible and infected preys cannot persist if the susceptible prey growth rate is lower than some critical value, measured by half of the corresponding noise intensity. This restriction may be satisfied in two ways: (i) by increasing the noise intensity of the system, keeping the other system parameters unaffected, or (ii) by decreasing the intrinsic birth rate, leaving the noise intensity unchanged. The susceptible prey can surely persist if its growth rate is significantly higher than the critical value [Theorem III.1(iii)].

Eradication of infection from a system is an important issue in epidemiology and always a challenging task to the system manager. It is very helpful if the system manager obtains some insights, possibly by analyzing the disease dynamics of the system, regarding various avenues of disease eradication mechanisms. Our results show that the extinction of the susceptible prey (as stated above) always leads to the extinction of the infected prey, causing resolution of infection from the system. This may be one of the possible ways of removing the infection from the system, which is straightforward but maybe, in many cases, unrealistic. Our analysis also prescribes some alternative way of disease eradication even when the susceptible prey growth is sufficiently high. In this case, the infection can be removed from the prey species and a healthy predator-prey system can be established, following the result of Theorem III.2 b(i), if the noise intensity on the infected prey is significantly high and/or the death or removal rate of the infected prey (γ) is high and/or the intraspecies competition of the infected prey (b) is high. Infection can also be removed through parasites burden reduction [86] so that force of infection (λ) becomes low and the corresponding extinction criterion is satisfied [see Theorem III.2 b(i)]. One can relate these eradication criteria with the basic reproduction number of epidemic theory, which determines whether an infection will spread in a population or not. This threshold quantity may be used as a measure of intervention strategy and therefore has very important practical utility. It has been shown that the stochastic system (3) will be disease free if $R_0^S < 1$, where $R_0^S = \frac{\lambda(a-\frac{\sigma_1^2}{2})}{b(\gamma+\frac{\sigma_2^2}{2})}$ and the corresponding threshold for the deterministic system (2) is $R_0^D < 1$, where $R_0^D = \frac{a\lambda}{b\gamma}$ [39]. Thus, $R_0^D > R_0^S$ for any nonzero value of the noises, implying that environmental noise plays a positive role in disease extinction. The parasitic infection can be eradicated even at higher infection rate with the right environmental noises (see Fig. 7).

The disease will always persist if the susceptible prey has a high growth rate, or the disease has high infectivity [see Theorem III.2 b(iii)]. On the other hand, predator population can not survive if its growth rate is lower than some critical value, where the critical predator's growth rate is defined by half of the corresponding noise intensity, even when its focal prey strongly persists [see Theorem III.3(i)]. The predator, however, almost surely persists if its growth rate exceeds the critical value. The predator can survive in absence of its focal prey at a lower density by consuming the nonpreferred secondary prey. It is interesting to observe that all these extinction scenarios occur in the stochastic system when the corresponding deterministic system shows stable persistence of all three populations. The average extinction time decreases with the increasing noise intensity and the probability distribution of the extinction time follows the log-normal density curve. Thus, environmental noise may play a critical role in population persistency as well as infection removal process by changing the physical property of the system.

The considered eco-epidemiological situation on which the model is based has similarity with the red grouse-fox interaction in presence of the parasites *trichostrongylus tenuis*. We, therefore, verified the field data of red grouse and fox populations with the time series solutions of our stochastic model. The solution of our model well fit the experimental data. Furthermore, the population densities of red grouse and fox populations have been predicted for the extended periods. Though both species have been coexisting for a long period and expected to do so in future if the environmental noises do not vary significantly, extinction of species cannot be ruled out. For instance, foxes are regularly killed to maintain grouse population [86]. This may be a potential threat to the fox population and may even send it to extinction if the fox killing rate increases and the intrinsic growth rate falls below the corresponding critical noise intensity.

This study, however, has not taken into account two important natural processes but may be considered in a future study, e.g., Allee effect, which generally enhances the extinction possibility [87], and immigration, which enhances the persistence of species, which is at the verge of extinction [88]. Despite these shortcomings, this paper shows that environmental variability has significant influences on the persistence and extinction of interacting species in the natural environment. It also points out different routes to extinction, which may be beneficial to the system manager to take various control measures to prevent species extinction.

ACKNOWLEDGMENTS

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APPENDIX A

Existence of a unique interior equilibrium point $E^*(x^*, y^*, z^*)$ of the system (3) and its stability can be deduced from [39]. The equilibrium population densities are $y^* = \frac{(ae+\gamma c)-(be+\lambda c)x^*}{\lambda e-mc}$, $z^* = \frac{r}{f}(m_2 + x^* + y^*)$, and x^* is the unique positive root of the quadratic equation $B_1 x^{*2} - B_2 x^* - B_3 = 0$ where $B_1 = \frac{be+\lambda c}{\lambda e-mc}[b + \lambda - \frac{\lambda(be+\lambda c)}{\lambda e-mc}] - b = \frac{(\lambda^2+mb)c}{(\lambda e-mc)^2}[\lambda(e-c) - (eb+mc)]$, $B_2 = \frac{be+\lambda c}{\lambda e-mc}(a - \lambda m_1 - \frac{rc}{f}) + (b+\lambda)\frac{ae+\gamma c}{\lambda e-mc} - (a - \lambda m_1 - \frac{rc}{f}) - 2\lambda\frac{(ae+\gamma c)(be+\lambda c)}{(\lambda e-mc)^2}$, $B_3 = -(am_1 - \frac{rcm_2}{f}) - \frac{ae+\gamma c}{\lambda e-mc}(a - \lambda m_1 - \frac{rc}{f}) + \lambda(\frac{ae+\gamma c}{\lambda e-mc})^2 = \frac{c(ma+\gamma\lambda)}{\lambda e-cm}[m_1 + \frac{ae+\gamma c}{\lambda e-cm}] + \frac{rc}{f}[m_2 + \frac{ae+\gamma c}{\lambda e-cm}]$. The last equation will have a unique positive root if $B_1 > 0$ and $B_3 > 0$. One can observe that $B_1 > 0$ holds if $m < \frac{1}{c}\{\lambda(e-c) - be\} = m^*(\text{say})$ and $e > c$. y^* will be positive if $x^* < \frac{ae+\gamma c}{be+\lambda c}$ and $\lambda > \frac{mc}{e} = \lambda^*$ (say). Therefore, a set of sufficient conditions for the existence of a unique equilibrium point of system (3) is

$$\text{(i) } e > c, \text{ (ii) } \lambda > \lambda^*, \text{ (iii) } m < m^*, \text{ and (iv) } x^* < \frac{ae+\gamma c}{be+\lambda c}.$$

The equilibrium E^*, whenever it exists, is locally asymptotically stable if (i) $\frac{z^*(cx^*+ey^*)}{(m_1+x^*+y^*)^2} < bx^* + my^* + \frac{ey^*-cx^*}{m_1+x^*+y^*}$, (ii) $\frac{2cx^*z^*}{(m_1+x^*+y^*)^2} + \frac{r^2}{f} < (b+\lambda)x^*$, (iii) $\frac{2ey^*z^*}{(m_1+x^*+y^*)^2} + (\lambda - m)y^* < r + \frac{r^2}{f}$, and (iv) $\{(\lambda-b)e - (\lambda+m)c\}[-m + \frac{e(m_2-m_1)z^*}{(m_1+x^*+y^*)^2(m_2+x^*+y^*)}] + (\lambda e - mc)(\lambda + m) > 0$.

APPENDIX B

Theorem B.1 For any initial value $(x(0), y(0), z(0)) \in \mathbb{R}^3_+$, there exists a unique solution $(x(t), y(t), z(t)) \in \mathbb{R}^3_+$ of the system (3) for all $t \geqslant 0$ and the solution remains in $\mathbb{R}^3_+$ with probability 1, i.e., $(x(t), y(t), z(t)) \in \mathbb{R}^3_+$ for all $t \geqslant 0$ a.s.

Proof. As the coefficients of system (3) are locally Lipschitz continuous, for any given initial value $(x(0), y(0), z(0)) \in \mathbb{R}^3_+$, there exists a unique local solution $(x(t), y(t), z(t)) \in \mathbb{R}^3_+$ for all $t \in [0, \tau_e)$ [89]. To prove the global existence of the solution, we need to show that $\tau_e = \infty$ a.s.

Let $\kappa_0 > 0$ be sufficiently large so that every initial coordinate $(x(0), y(0), z(0))$ lies within the interval $[\frac{1}{\kappa_0}, \kappa_0]$. For every integer $\kappa > \kappa_0$, we define the stopping time:

$$\tau_\kappa = \inf\left\{t \in [0, \tau_e) : x(t) \notin \left(\frac{1}{\kappa}, \kappa\right) \text{ or } y(t) \notin \left(\frac{1}{\kappa}, \kappa\right) \text{ or } z(t) \notin \left(\frac{1}{\kappa}, \kappa\right)\right\}.$$

Here τ_κ is increasing as $\kappa \to \infty$. Set $\lim_{\kappa\to\infty} \tau_\kappa = \tau_\infty$, when $\tau_\infty \leqslant \tau_e$ a.s. Therefore, if we can show that $\tau_\infty = \infty$, we will obtain $\tau_e = \infty$ and $(x(t), y(t), z(t)) \in \mathbb{R}^3_+$ a.s. for all $t \geqslant 0$. For this, let us assume that the result is not true and there exist two constants $T > 0$ and $\epsilon \in (0, 1)$ such that

$$P(\tau_\infty \leqslant T) > \epsilon, \tag{B1}$$

and then there exists an integer $\kappa_1 \geqslant \kappa_0$ such that

$$P(\tau_\kappa \leqslant T) \geqslant \epsilon, \quad \forall\, \kappa \geqslant \kappa_1. \tag{B2}$$

Define the positive definite function V for all $(x, y, z) \in \mathbb{R}^3$ as

$$V = x + 1 - \ln x + y + 1 - \ln y + z + 1 - \ln z.$$

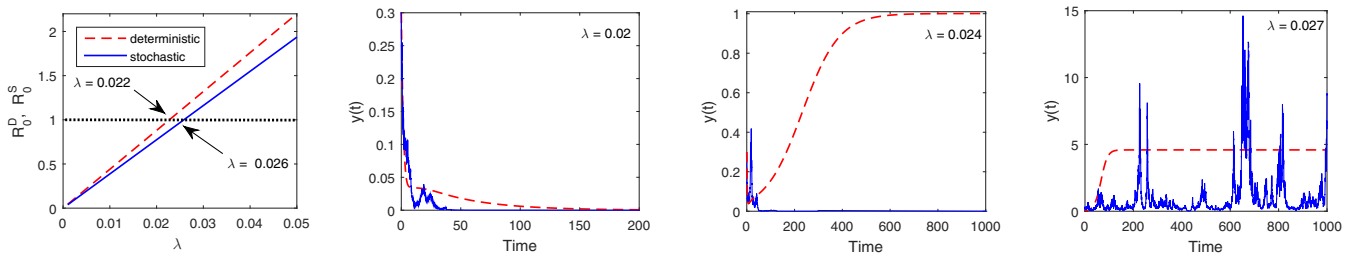

FIG. 7. Comparison between stochastic and deterministic basic reproduction numbers for the varying force of infection. The left figure shows that infection will be eradicated from the deterministic system if $\lambda < \lambda_1 = 0.022$ and for the stochastic system the range is $0 < \lambda < \lambda_2 = 0.026$. Disease persists in both the systems if $\lambda > \lambda_2$. This fact is demonstrated with the time series result for three different values of λ such that $\lambda < \lambda_1$, $\lambda_1 < \lambda < \lambda_2$, and $\lambda > \lambda_2$. It demonstrates that environmental noise can remove infection at higher force of infection. Here $\sigma_1 = 0.3$, $\sigma_2 = 0.3$, $\sigma_3 = 0.01$ and other parameters are as in Fig. 1.

One can then apply Ito's formula to have

$$\begin{aligned}
dV &= \left(1-\frac{1}{x}\right)dx + \frac{1}{2x^2}(dx)^2 + \left(1-\frac{1}{y}\right)dy + \frac{1}{2y^2}(dy)^2 + \left(1-\frac{1}{z}\right)dz + \frac{1}{2z^2}(dz)^2 \\
&= \left[(x-1)\left(a-bx-\lambda y-\frac{cz}{m_1+x+y}\right) + (y-1)\left(\lambda x - my - \gamma - \frac{ez}{m_1+x+y}\right)\right. \\
&\quad \left. + (z-1)\left(r-\frac{fz}{m_2+x+y}\right) + \frac{\sigma_1^2+\sigma_2^2+\sigma_3^2}{2}\right]dt + \sigma_1(x-1)dW_1(t) + \sigma_2(y-1)dW_2(t) \\
&\quad + \sigma_3(z-1)dW_3(t) \\
&\leqslant \left[\gamma + (a+b)x + (b+\lambda+m)y + \left(r+\frac{c+e}{m_1}+\frac{f}{m_2}\right)z + \frac{\sigma_1^2+\sigma_2^2+\sigma_3^2}{2}\right]dt \\
&\quad + \sigma_1(x-1)dW_1(t) + \sigma_2(y-1)dW_2(t) + \sigma_3(z-1)dW_3(t).
\end{aligned}$$

Noting $u \leqslant 2(u+1-\ln u)$ for all $u > 0$, one can write

$$\begin{aligned}
dV &\leqslant \left[\left(\gamma + \frac{\sigma_1^2+\sigma_2^2+\sigma_3^2}{2}\right) + 2(a+b)(x+1-\ln x) + 2(b+\lambda+m)(y+1-\ln y)\right. \\
&\quad \left. + 2\left(r+\frac{c+e}{m_1}+\frac{f}{m_2}\right)(z+1-\ln z)\right]dt + \sigma_1(x-1)dW_1(t) + \sigma_2(y-1)dW_2(t) + \sigma_3(z-1)dW_3(t) \\
&\leqslant (\Delta_1 + \Delta_2 V)dt + \sigma_1(x-1)dW_1(t) + \sigma_2(y-1)dW_2(t) + \sigma_3(z-1)dW_3(t),
\end{aligned}$$

where

$$\Delta_1 = \gamma + \frac{\sigma_1^2+\sigma_2^2+\sigma_3^2}{2}, \ \Delta_2 = \max\left\{2(a+b), 2(b+\lambda+m), 2\left(r+\frac{c+e}{m_1}+\frac{f}{m_2}\right)\right\}.$$

Again, defining $\Delta_3 = \max\{\Delta_1, \Delta_2\}$, we have

$$dV \leqslant \Delta_3(1+V)dt + \sigma_1(x-1)dW_1(t) + \sigma_2(y-1)dW_2(t) + \sigma_3(z-1)dW_3(t). \tag{B3}$$

Integrating both sides of (B3) from zero to $t_1 \wedge \tau_\kappa$ for any $t_1 \leqslant T$ and taking the expectation, we obtain

$$\begin{aligned}
&EV(x(t_1\wedge\tau_\kappa), y(t_1\wedge\tau_\kappa), z(t_1\wedge\tau_\kappa)) \\
&\leqslant V(x(0), y(0), z(0)) + \Delta_3 E\int_0^{t_1\wedge\tau_\kappa}(1+V)dt \\
&\leqslant V(x(0), y(0), z(0)) + \Delta_3 t_1 + \Delta_3 E\int_0^{t_1\wedge\tau_\kappa} V dt \\
&\leqslant V(x(0), y(0), z(0)) + \Delta_3 T + \Delta_3 E\int_0^{t_1} V(x(\tau_\kappa\wedge t), y(\tau_\kappa\wedge t), z(\tau_\kappa\wedge t))dt \\
&= V(x(0), y(0), z(0)) + \Delta_3 T + \Delta_3 \int_0^{t_1} EV(x(\tau_\kappa\wedge t), y(\tau_\kappa\wedge t), z(\tau_\kappa\wedge t))dt.
\end{aligned}$$

Applying Gronwall's inequality, we get

$$EV(x(t_1\wedge\tau_\kappa), y(t_1\wedge\tau_\kappa), z(t_1\wedge\tau_\kappa)) \leqslant (V(x(0), y(0), z(0)) + \Delta_3 T)e^{\Delta_3(t_1\wedge\tau_\kappa)} = \Delta_4 \text{ (say)}. \tag{B4}$$

Set $\Omega_\kappa = \{\tau_\kappa \leqslant T\}$ for all $\kappa \geqslant \kappa_1$. Thus, following (B2), we get $P(\Omega_\kappa) \geqslant \epsilon$ for all $\omega \in \Omega_\kappa$. Clearly, at least one of $x(\tau_\kappa, \omega)$, $y(\tau_\kappa, \omega)$, $z(\tau_\kappa, \omega)$ is equal to either κ or $\frac{1}{\kappa}$. Hence $V(x(\tau_\kappa), y(\tau_\kappa), z(\tau_\kappa))$ is no less than $\min\{\kappa + 1 - \ln\kappa, \frac{1}{\kappa} + 1 + \ln\kappa\}$. Form (B1) and (B4), we then obtain

$$\Delta_4 \geqslant E[1_{\Omega_\kappa} V(x(\tau_\kappa, \omega), y(\tau_\kappa, \omega), z(\tau_\kappa, \omega))] \geqslant \epsilon\left[(\kappa+1-\ln\kappa) \wedge \left(\frac{1}{\kappa}+1+\ln\kappa\right)\right],$$

where 1_{Ω_κ} is the indicator function of Ω_κ. Therefore, letting $\kappa \to \infty$, we get $\infty > \Delta_4 = \infty$, a contradiction and hence $\tau_\infty = \infty$ a.s. This implies the global existence of the solution of system (3). ■

APPENDIX C

Theorem C.1 For any initial value $(x(0), y(0), z(0)) \in \mathbb{R}^3_+$, there exists some bound $(\bar{x}, \bar{y}, \bar{z})$ of the solution $(x(t), y(t), z(t)) \in \mathbb{R}^3_+$ of the system (3) for all $t \geqslant 0$.

Proof. To prove this result, we will use the following well known lemma.

Lemma C.1 [90] Let $\Phi(t)$ be a solution of the following system:

$$\begin{cases} d\Phi(t) = \Phi(t)(a - b\phi(t))dt + \sigma_1 dW_1(t), \\ \Phi(0) = x(0). \end{cases} \tag{C1}$$

Then $\limsup_{t\to\infty} E[\Phi(t)] \leqslant \frac{a}{b}$.

We now prove that the solutions of system (3) are stochastically ultimately bounded for any positive initial value. First we show that any solution $(x(t), y(t), z(t))$ of system (3) with any positive initial value $(x(0), y(0), z(0)) \in \mathbb{R}^3_+$ is uniformly bounded in the mean. Observe that

$$dx(t) \leqslant x(t)(a - bx(t))dt + \sigma_1 x(t)dW_1(t).$$

Let

$$\Phi(t) = \frac{e^{(a-\frac{\sigma_1^2}{2})t+\sigma_1 W_1(t)}}{\frac{1}{x(0)} + b\int_0^t e^{(a-\frac{\sigma_1^2}{2})\theta+\sigma_1 W_1(\theta)}d\theta}.$$

Then $\Phi(t)$ is the unique solution of (C1).

By the comparison theorem of the stochastic equation, we get $x(t) \leqslant \Phi(t)$ a.s. for all $t \in [0, \tau_e)$. Following Lemma C.1, we have

$$\limsup_{t\to\infty} E[x(t)] \leqslant \frac{a}{b} \text{ a.s.} \tag{C2}$$

Let $G(t) = x(t) + y(t)$. The time derivative of $G(t)$ along the system (3) is given by

$$\begin{aligned} dG(t) &= \left[x(t)(a - bx(t)) - my^2(t) - \gamma y(t) - \frac{cx(t)z(t)}{m_1 + x(t) + y(t)} - \frac{ey(t)z(t)}{m_1 + x(t) + y(t)}\right]dt \\ &\quad + x(t)\sigma_1 dW_1(t) + y(t)\sigma_2 dW_2(t) \\ &\leqslant [x(t)(a - bx(t)) - \gamma y(t)]dt + x(t)\sigma_1 dW_1(t) + y(t)\sigma_2 dW_2(t) \\ &\leqslant [2ax(t) - bx^2(t) - \xi(x(t) + y(t))]dt + x(t)\sigma_1 dW_1(t) + y(t)\sigma_2 dW_2(t), \text{ where } \xi = \min\{a, \gamma\} \\ &= [2ax(t) - bx^2(t) - \xi G(t)]dt + x(t)\sigma_1 dW_1(t) + y(t)\sigma_2 dW_2(t). \end{aligned}$$

Integration of both sides from zero to t gives

$$G(t) \leqslant G(0) + \int_0^t [2ax(t))(\theta) - bx^2(\theta) - \xi G(\theta)]d\theta + \sigma_1 \int_0^t x(\theta)dW_1(\theta) + \sigma_2 \int_0^t y(\theta)dW_2(\theta).$$

Taking the expectation, one gets

$$E[G(t)] \leqslant G(0) + \int_0^t E[2ax(\theta) - bx^2(\theta) - \xi G(\theta)]d\theta.$$

On differentiation, we have

$$\begin{aligned} \frac{dE[G(t)]}{dt} &\leqslant 2aE[x(t)] - bE[x^2(t)] - \xi E[G(t)] \\ &\leqslant 2aE[x(t)] - b(E[x(t)])^2 - \xi E[G(t)]. \end{aligned}$$

As $\max\{2aE[x(t)] - b(E[x(t)])^2\} = \frac{a^2}{b}$,

$$\frac{dE[G(t)]}{dt} \leqslant \frac{a^2}{b} - \xi E[G(t)] \Rightarrow 0 \leqslant \limsup_{t\Rightarrow\infty} E[G(t)] \leqslant \frac{a^2}{b\xi} \Rightarrow \limsup_{t\to\infty} E[x(t) + y(t)] \leqslant \frac{a^2}{b\xi} \text{ a.s.}$$

Hence, $y(t)$ is also uniformly bounded in the mean a.s. Now, following Markov's inequality, for any positive constant α there exists $\beta > 0$ such that $P(x > \alpha) \leqslant \frac{E(x)}{\beta}$. Following (C2), we then have

$$\limsup_{t\to\infty} P(x > \alpha) \leqslant \delta_1 \text{ a.s., where } \delta_1 = \frac{a}{b\beta}.$$

Therefore, for any positive constant $\alpha > 0$, there is a $\delta_1 > 0$ such that

$$\limsup_{t\to\infty} P(x > \alpha) \leqslant \delta_1 \text{ a.s.}$$

Hence, $x(t)$ of system (3) is stochastically ultimately bounded and there exists a positive constant, say $\bar{x}$ (>0), such that for all $t \in [0, \tau_e)$

$$\limsup_{t\to\infty} x(t) \leqslant \bar{x} \text{ a.s.}$$

In a similar manner, we can show that $y(t)$ is also stochastically ultimately bounded and there exists a positive constant, say $\bar{y}$ (>0), such that for all $t \in [0, \tau_e)$

$$\limsup_{t\to\infty} y(t) \leqslant \bar{y} \text{ a.s.}$$

To show $z(t)$ is also uniformly bounded in the mean, we observe that

$$dz(t) = z\left(r - \frac{fz}{m_2 + x + y}\right) + \sigma_3 z dW_3(t) \leqslant z\left(r - \frac{fz}{m_2 + \bar{x} + \bar{y}}\right) + \sigma_3 z dW_3(t).$$

Proceeding as before, we then obtain

$$\limsup_{t\to\infty} E[z(t)] \leqslant \frac{r(m_2 + \bar{x} + \bar{y})}{f} \text{ a.s.}$$

Hence $z(t)$ of system (3) is stochastically ultimately bounded and there exists a positive constant $\bar{z} > 0$ such that for all $t \in [0, \tau_e)$

$$\limsup_{t\to\infty} z(t) \leqslant \bar{z} \text{ a.s.}$$

Hence the theorem is proven.

APPENDIX D: PROOF OF THEOREM III.1

The following definition is well known [61].

Definition D.1 Let a function $g(t)$ be such that it represents a population at any time t and $g(t) > 0$ for all $t \geqslant 0$. Then we have the following.

(a) $g(t)$ is said to go to extinction in the mean if $\limsup_{t\to\infty} \frac{1}{t}\int_0^t g(\theta)d\theta < 0$.

(b) $f(t)$ is said to be nonpersistent in the mean if $\limsup_{t\to\infty} \frac{1}{t}\int_0^t g(\theta)d\theta = 0$.

(c) $f(t)$ is said to be weakly persistent in the mean if $\limsup_{t\to\infty} \frac{1}{t}\int_0^t g(\theta)d\theta > 0$.

(d) $f(t)$ is said to be strongly persistent in the mean if $\liminf_{t\to\infty} \frac{1}{t}\int_0^t g(\theta)d\theta > 0$.

We now prove Theorem III.1. (i) From the first equation of system (3), it follows that

$$dx(t) \leqslant x(t)(a - bx(t))dt + \sigma_1 x(t) dW_1(t).$$

If we consider the system

$$dY(t) = Y(t)(a - bY(t))dt + \sigma_1 Y(t) dW_1(t), Y(0) = Y_0$$

then

$$Y(t) = \frac{e^{(a-\frac{\sigma_1^2}{2})t + \sigma_1 W_1(t)}}{\frac{1}{Y_0} + b\int_0^t e^{(a-\frac{\sigma_1^2}{2})s + \sigma_1 W_1(s)} ds}.$$

Obviously $x(t) \leqslant Y(t)\ \ \forall t$ and if $a - \frac{\sigma_1^2}{2} < 0$, then $\lim_{t\to\infty} Y(t) = 0$ and since $x(t)$ is non-negative, we have $\lim_{t\to\infty} x(t) = 0$.

(ii) We have from the first equation of system (3)

$$d(\ln(x)) = \left[a - bx - \lambda y - \frac{cz}{m_1 + x + y} - \frac{\sigma_1^2}{2}\right]dt + \sigma_1 dW_1(t),$$

$$\frac{\ln(x(t))}{t} = \left(a - \frac{\sigma_1^2}{2}\right) - \frac{b}{t}\int_0^t x(s)ds - \frac{\lambda}{t}\int_0^t y(s)ds - \frac{c}{t}\int_0^t \frac{z(s)}{m_1 + x(s) + y(s)} ds + \frac{\int_0^t \sigma_1 dW_1(t)}{t} + \frac{\ln x(0)}{t}$$

$$\therefore \ln x(t) - \ln x(0) \leqslant \left(a - \frac{\sigma_1^2}{2}\right)t - b\int_0^t x(s)ds + M_1, \tag{D1}$$

where $M_1 = \int_0^t \sigma_1 dW_1(t)$. By the strong law a large number for martingales yields

$$\lim_{t\to\infty} \frac{M_1(t)}{t} = 0 \text{ a.s.}$$

From the property of limits, for arbitrary $\epsilon_1 > 0$, $\exists\, T_1 > 0$ such that $\forall\, t \geqslant T_1$, $\frac{M_1(t)}{t} \leqslant \epsilon_1$. From (D1), under the assumption $a - \frac{\sigma_1^2}{2} = 0$, we have

$$\frac{1}{t}\ln\frac{x(t)}{x(0)} \leqslant \epsilon_1 - b\frac{x(0)}{t}\int_0^t \frac{x(s)}{x(0)}ds$$

$$\therefore \limsup_{t\to\infty} \frac{1}{t}\int_0^t x(s)ds \leqslant \epsilon_1.$$

Since ϵ_1 is arbitrary and $x(t)$ is non-negative, we therefore have

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t x(s)ds = 0$$

and $x(t)$ is nonpersistent in the mean.

Before going to the next proof, we define the following well known lemma.

Lemma D.1 [91] *Suppose* $w(t) \in C(\Omega \times [0,\infty), \mathbb{R}_+^0)$, *where* $\mathbb{R}_+^0 = \{p \mid p > 0, p \in \mathbb{R}\}$.

(1) If there exist two positive constants T *and* r_0 *such that*

$$\ln(w(t)) \leqslant rt - r_0\int_0^t w(\theta)d\theta + \sigma_{i=1}^n \alpha_i W_i(t), \quad \forall t \geqslant T,$$

where α_i $(1 < i < n)$ *are constants, then*

$$\begin{cases} \limsup_{t\to\infty} \frac{1}{t}\int_0^t w(\theta)d\theta \leqslant \frac{r}{r_0} \ \ a.s., \ \text{if } r \geqslant 0; \\ \lim_{t\to\infty} w(t) = 0 \ \ a.s., \ \text{if } r < 0. \end{cases}$$

(2) If there exist three positive constants T, r, r_0 *such that*

$$\ln(w(t)) \geqslant rt - r_0\int_0^t w(\theta)d\theta + \sigma_{i=1}^n \alpha_i W_i(t), \quad \forall t \geqslant T,$$

then

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t w(\theta)d\theta \geqslant \frac{r}{r_0} \ \text{a.s.}$$

(iii) Again, from the first equation

$$d\ln x \geqslant \left(a - bx - \lambda y - \frac{cz}{m_1} - \frac{\sigma_1^2}{2}\right)dt + \sigma_1 dW_1(t)$$

$$\Rightarrow \ln\frac{x(t)}{x(0)} \geqslant \left(a - \lambda\bar{y} - \frac{c\bar{z}}{m_1} - \frac{\sigma_1^2}{2}\right)t - b\int_0^t x(\theta)d\theta + \sigma_1 W_1(t).$$

Therefore, if $a - \lambda\bar{y} - \frac{c\bar{z}}{m_1} - \frac{\sigma_1^2}{2} > 0$ then by applying Lemma D.1, we obtain

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t x(t) \geqslant \frac{1}{b}\left(a - \lambda\bar{y} - \frac{c\bar{z}}{m_1} - \frac{\sigma_1^2}{2}\right) > 0.$$

Evidently, $x(t)$ is strongly persistent in the mean if $a > \frac{\sigma_1^2}{2} + \lambda\bar{y} + \frac{c\bar{z}}{m_1}$.

APPENDIX E: PROOF OF THEOREM III.2

(a) Suppose $a - \frac{\sigma_1^2}{2} < 0$. From Theorem III.1, one can easily see that

$$\limsup_{t\to\infty} \frac{1}{t}\int_0^t x(\theta)d\theta < 0.$$

Integration of the second equation of system (3) yields

$$\frac{\ln(y(t))}{t} = \frac{\lambda}{t}\int_0^t x(s)ds - \frac{m}{t}\int_0^t y(s)ds - \gamma - \frac{\sigma_2^2}{2} - \frac{e}{t}\int_0^t \frac{z(s)}{m_1 + x(s) + y(s)}ds + \frac{\int_0^t \sigma_2 dW_2(t)}{t} + \frac{\ln y(0)}{t}. \tag{E1}$$

Then the equation (E1) coupled with the extinction condition of $x(t)$ yields

$$\begin{aligned}\frac{\ln y(t)-\ln y(0)}{t} &\leqslant \left(-\gamma-\frac{\sigma_2^2}{2}\right)+\frac{\lambda}{t}\int_0^t x(\theta)d\theta+\sigma_2\frac{\int_0^t dW_2(t)}{t}\\ &\Rightarrow \limsup_{t\to\infty}\frac{1}{t}\int_0^t y(\theta)d\theta \leqslant \left(-\gamma-\frac{\sigma_2^2}{2}\right)<0\\ &\Rightarrow \lim_{t\to\infty} y(t)=0.\end{aligned}$$

Therefore, extinction of $x(t)$ implies the extinction of $y(t)$.

(b) (i) If we consider $a-\frac{\sigma_1^2}{2}>0$ then from the first equation of (3)

$$\frac{\ln x(t)-\ln x(0)}{t}\leqslant a-\frac{\sigma_1^2}{2}-\frac{b}{t}\int_0^t x(\theta)d\theta+\sigma_1\frac{\int_0^t dW_1(t)}{t}. \tag{E2}$$

Lemma D.1 then leads to

$$\limsup_{t\to\infty}\frac{1}{t}\int_0^t x(\theta)d\theta\leqslant\frac{a-\frac{\sigma_1^2}{2}}{b}. \tag{E3}$$

Again from (3), we have

$$\begin{aligned}&\frac{\ln y(t)-\ln y(0)}{t}\leqslant\left(-\gamma-\frac{\sigma_2^2}{2}\right)+\frac{\lambda}{t}\int_0^t x(\theta)d\theta-\frac{m}{t}\int_0^t y(\theta)d\theta+\sigma_2\frac{W_2(t)}{t}\\ &\therefore\ \limsup_{t\to\infty}\frac{1}{t}\int_0^t y(\theta)d\theta\leqslant\frac{\lambda\left(a-\frac{\sigma_1^2}{2}\right)-b\left(\gamma+\frac{\sigma_2^2}{2}\right)}{mb}.\end{aligned} \tag{E4}$$

Thus, if $\lambda(a-\frac{\sigma_1^2}{2})<b(\gamma+\frac{\sigma_2^2}{2})$ then $\lim_{t\to\infty}y(t)=0$.

(ii) Assume $\limsup_{t\to\infty}\frac{1}{t}\int_0^t y(\theta)d\theta\geqslant 0$. For sufficiently small $\eta>0$, there exists $T>0$ such that for all $t>T$

$$\frac{\lambda}{t}\int_0^t x(\theta)d\theta<\limsup_{t\to\infty}\frac{\lambda}{t}\int_0^t x(\theta)d\theta+\eta.$$

The second equation of (3) then yields

$$\begin{aligned}\frac{\ln y(t)-\ln y(0)}{t}&\leqslant\left(-\gamma-\frac{\sigma_2^2}{2}\right)+\frac{\lambda}{t}\int_0^t x(\theta)d\theta-\frac{m}{t}\int_0^t y(\theta)d\theta+\sigma_2\frac{W_2(t)}{t}\\ &\leqslant\left(-\gamma-\frac{\sigma_2^2}{2}\right)+\limsup_{t\to\infty}\frac{\lambda}{t}\int_0^t x(\theta)d\theta+\eta-\frac{m}{t}\int_0^t y(\theta)d\theta+\sigma_2\frac{W_2(t)}{t}.\end{aligned}$$

By Lemma D.1, one have

$$\limsup_{t\to\infty}\frac{1}{t}\int_0^t y(\theta)d\theta\leqslant\frac{\left(-\gamma-\frac{\sigma_2^2}{2}\right)+\limsup_{t\to\infty}\frac{\lambda}{t}\int_0^t x(\theta)d\theta+\eta}{m}.$$

If $\lambda(a-\frac{\sigma_1^2}{2})=b(\gamma+\frac{\sigma_2^2}{2})$ then we must have $a>\frac{\sigma_1^2}{2}$. As η is arbitrary, we get from (E3)

$$\limsup_{t\to\infty}\frac{1}{t}\int_0^t y(\theta)d\theta\leqslant\frac{-b\left(\gamma+\frac{\sigma_2^2}{2}\right)+\lambda\left(a-\frac{\sigma_1^2}{2}\right)}{bm}=0,\quad\text{provided }\lambda\left(a-\frac{\sigma_1^2}{2}\right)=b\left(\gamma+\frac{\sigma_2^2}{2}\right).$$

Thus, $y(t)$ is nonpersistent in the mean a.s. if $\lambda(a-\frac{\sigma_1^2}{2})=b(\gamma+\frac{\sigma_2^2}{2})$.

(iii) From the second equations of (3), we have

$$\begin{aligned}\frac{1}{t}\ln\frac{y(t)}{y(0)}&=-\left(\gamma+\frac{\sigma_2^2}{2}\right)+\frac{\lambda}{t}\int_0^t x(\theta)d\theta-\frac{m}{t}\int_0^t y(\theta)d\theta-\frac{1}{t}\int_0^t\frac{ez(\theta)}{m_1+x(\theta)+y(\theta)}d\theta+\sigma_2\frac{W_2(t)}{t}\\ &\geqslant-\left(\gamma+\frac{\sigma_2^2}{2}\right)+\liminf_{t\to\infty}\frac{\lambda}{t}\int_0^t x(\theta)d\theta-\frac{m}{t}\int_0^t y(\theta)d\theta-\frac{1}{t}\int_0^t\frac{ez(\theta)}{m_1+x(\theta)+y(\theta)}d\theta+\sigma_2\frac{W_2(t)}{t}\\ &\geqslant-\left(\gamma+\frac{\sigma_2^2}{2}\right)+\frac{\lambda}{b}\left(a-\lambda\bar{y}-\frac{e\bar{z}}{m_1}-\frac{\sigma_1^2}{2}\right)-\frac{e\bar{z}}{m_1}-\frac{m}{t}\int_0^t y(\theta)d\theta+\sigma_2\frac{W_2(t)}{t}.\end{aligned}$$

Assuming $\lambda(a-\frac{\sigma_1^2}{2}) > b(\gamma+\frac{\sigma_2^2}{2}) + \lambda^2\bar{y} + (\lambda c + be)\frac{\bar{z}}{m_1}$, from Lemma D.1, it follows that

$$\liminf_{t\to\infty} \frac{1}{t}\int_0^t y(\theta)d\theta \geqslant \frac{\lambda\left(a-\frac{\sigma_1^2}{2}\right) - b\left(\gamma+\frac{\sigma_2^2}{2}\right) - \left(\lambda^2\bar{y} + (\lambda c + be)\frac{\bar{z}}{m_1}\right)}{mb}.$$

Clearly, $y(t)$ is strongly persistent in the mean if $\lambda(a-\frac{\sigma_1^2}{2}) > b(\gamma+\frac{\sigma_2^2}{2}) + \lambda^2\bar{y} + (\lambda c + be)\frac{\bar{z}}{m_1}$.

The proof of III.3 is similar to that of Theorem III.1 and hence omitted.

Remark E.1 From (E2), we have $\limsup_{t\to\infty} \frac{\ln x(t)}{t} \leqslant a - \frac{\sigma_1^2}{2}$. Then there exists a sufficiently large $T_3 > 0$ such that $\frac{\ln x(t)}{t} < a - \frac{\sigma_1^2}{2} \;\; \forall\, t > T_3$. Therefore, $x(t) < e^{(a-\frac{\sigma_1^2}{2})t} \;\; \forall\, t > T_3$. It shows that x is a monotonic decreasing function of time under the restriction $a < \frac{\sigma_1^2}{2}$ and the extinction of the susceptible prey will be faster as the noise, σ_1, becomes larger. Similarly, one can show from (E4) that the infected prey y goes extinct monotonically under the restriction $\lambda(a-\frac{\sigma_1^2}{2}) < b(\gamma+\frac{\sigma_2^2}{2})$ and the extinction will be quicker if the corresponding noise σ_2 grows faster. From (3), it is straightforward to show that increasing noise in predator population also ushers quicker extinction.

APPENDIX F: PROOF OF THEOREM III.4

System (3) can be written as

$$d\begin{pmatrix} x(t)\\ y(t)\\ z(t)\end{pmatrix} = \begin{pmatrix} x(a-bx) - \lambda xy - \frac{cxz}{m_1+x+y}\\ \lambda xy - my^2 - \gamma y - \frac{eyz}{m_1+x+y}\\ r - \frac{fz}{m_2+x+y}\end{pmatrix}dt + \begin{pmatrix}\sigma_1 x(t)\\ 0\\ 0\end{pmatrix}dW_1(t) + \begin{pmatrix}0\\ \sigma_2 y(t)\\ 0\end{pmatrix}dW_2(t) + \begin{pmatrix}0\\ 0\\ \sigma_3 z(t)\end{pmatrix}dW_3(t)$$

and the diffusion matrix is

$$A' = \begin{pmatrix}\sigma_1^2x^2 & 0 & 0\\ 0 & \sigma_2^2y^2 & 0\\ 0 & 0 & \sigma_3^2z^2\end{pmatrix}.$$

Define $\bar{V}(x,y,z) = V_1(x,y,z) + V_2(x,y,z) + V_3(x,y,z)$, where

$$V_1 = A\left[x - x^* - x^*\ln\frac{x}{x^*}\right], V_2 = A\left[y - y^* - y^*\ln\frac{y}{y^*}\right], V_3 = \frac{1}{r}\left[z - z^* - z^*\ln\frac{z}{z^*}\right].$$

At E^*, we have

$$a = bx^* + \lambda y^* + \frac{cz^*}{m_1+x^*+y^*}, \;\; \gamma = \lambda x^* - my^* - \frac{ez^*}{m_1+x^*+y^*}, \;\; \frac{f}{r}\left(\frac{z^*}{m_2+x^*+y^*}\right) = 1. \tag{F1}$$

Using (F1), one can calculate

$$\begin{aligned} d\bar{V} &= A\left[1-\frac{x^*}{x}\right]\left\{\left[ax - bx^2 - \lambda xy - \frac{cxz}{m_1+x+y}\right]dt + \sigma_1 x dW_1(t)\right\}\\ &\quad + A\left[1-\frac{y^*}{y}\right]\left\{\left[\lambda xy - my^2 - \frac{eyz}{m_1+x+y} - \gamma y\right]dt + \sigma_2 y dW_2(t)\right\}\\ &\quad + \frac{1}{r}\left[1-\frac{z^*}{z}\right]\left\{\left[rz - \frac{fz^2}{m_2+x+y}\right]dt + \sigma_3 z dW_3(t)\right\}\\ &= L\bar{V}dt + A(x-x^*)dW_1(t) + A(y-y^*)dW_2(t) + \frac{1}{r}(z-z^*)dW_3(t), \end{aligned}$$

where

$$
\begin{aligned}
L\bar{V} &= -\left(Ab - \frac{cz^*}{m_1+x+y}\right)(x-x^*)^2 - \left(m - \frac{ez^*}{m_1+x+y}\right)(y-y^*)^2 - \frac{f(z-z^*)^2}{r(m_2+x+y)} \\
&\quad + \left[\frac{fz^*}{r(m_2+x^*+y^*)(m_2+x+y)} - \frac{Ac}{m_1+x+y}\right](z-z^*)(x-x^*) \\
&\quad + \left[\frac{fz^*}{r(m_2+x^*+y^*)(m_2+x+y)} - \frac{Ae}{m_1+x+y}\right](z-z^*)(y-y^*) \\
&\quad + \frac{(c+e)z^*(x-x^*)(y-y^*)}{m_1+x+y} + \Theta \\
&\leqslant -\left(Ab - \frac{cz^*}{m_1+x+y}\right)(x-x^*)^2 - \left(m - \frac{ez^*}{m_1+x+y}\right)(y-y^*)^2 - \frac{f(z-z^*)^2}{r(m_2+x+y)} \\
&\quad + \left[\frac{fz^*}{rm_2^2} - \frac{Ac}{m_1+x+y}\right](z-z^*)(x-x^*) + \left[\frac{fz^*}{rm_2^2} - \frac{Ae}{m_1+x+y}\right](z-z^*)(y-y^*) \\
&\quad + \frac{(c+e)z^*(x-x^*)(y-y^*)}{m_1+x+y} + \Theta \\
&\leqslant -\left(Ab - \frac{cz^*}{m_1+x+y}\right)(x-x^*)^2 - \left(m - \frac{ez^*}{m_1+x+y}\right)(y-y^*)^2 - \frac{f(z-z^*)^2}{r(m_2+x+y)} \\
&\quad + \left[\frac{fz^*}{rm_2^2} - \frac{Ac}{m_1+\bar{x}+\bar{y}}\right](z-z^*)(x-x^*) + \left[\frac{fz^*}{rm_2^2} - \frac{Ae}{m_1+\bar{x}+\bar{y}}\right](z-z^*)(y-y^*) \\
&\quad + \frac{(c+e)z^*(x-x^*)(y-y^*)}{m_1+x+y} + \Theta.
\end{aligned}
$$

Let us assume $\frac{fz^*}{rm_2^2} > \max\{\frac{Ac}{m_1+\bar{x}+\bar{y}}, \frac{Ae}{m_1+\bar{x}+\bar{y}}\}$. Hence

$$
\begin{aligned}
L\bar{V} &\leqslant -\left(Ab - \frac{cz^*}{m_1}\right)(x-x^*)^2 - \left(m - \frac{ez^*}{m_1}\right)(y-y^*)^2 - \frac{f(z-z^*)^2}{r(m_2+x+y)} + \left[\frac{fz^*}{rm_2^2} - \frac{Ac}{m_1+\bar{x}+\bar{y}}\right]|z-z^*||x-x^*| \\
&\quad + \left[\frac{fz^*}{rm_2^2} - \frac{Ae}{m_1+\bar{x}+\bar{y}}\right]|z-z^*||y-y^*| + \frac{(c+e)z^*|x-x^*||y-y^*|}{m_1} + \Theta \\
&\leqslant -\left[Ab - \frac{cz^*}{m_1} - \frac{fz^*}{2rm_2^2} - \frac{(c+e)z^*}{2m_1}\right](x-x^*)^2 - \left[m - \frac{ez^*}{m_1} - \frac{fz^*}{2rm_2^2} - \frac{(c+e)z^*}{2m_1} + \frac{Ae}{2(m_1+\bar{x}+\bar{y})}\right](y-y^*)^2 \\
&\quad - \left[\frac{f}{r(m_2+\bar{x}+\bar{y})} + \frac{A(e+c)}{2(m_1+\bar{x}+\bar{y})} - \frac{fz^*}{rm_2^2}\right](z-z^*)^2 + \Theta.
\end{aligned}
$$

Therefore, if $Ab > \frac{cz^*}{m_1} + \frac{fz^*}{2rm_2^2} + \frac{(c+e)z^*}{2m_1}$, $m + \frac{Ae}{2(m_1+\bar{x}+\bar{y})} > \frac{ez^*}{m_1} + \frac{fz^*}{2rm_2^2} + \frac{(c+e)z^*}{2m_1}$, and $\frac{f}{r(m_2+\bar{x}+\bar{y})} + \frac{A(e+c)}{2(m_1+\bar{x}+\bar{y}} > \frac{fz^*}{rm_2^2}$ hold, we then have

$$
\begin{aligned}
d\bar{V} &\leqslant -[K(x-x^*)^2 + Q(y-y^*)^2 + T(z-z^*)^2 - \Theta]dt + A(x-x^*)dW_1(t) + A(y-y^*)dW_2(t) + \frac{1}{r}(z-z^*)dW_3(t) \\
&\leqslant -[\min\{K,Q,T\}((x-x^*)^2 + (y-y^*)^2 + (z-z^*)^2) - \Theta]dt + A(x-x^*)dW_1(t) \\
&\quad + A(y-y^*)dW_2(t) + \frac{1}{r}(z-z^*)dW_3(t).
\end{aligned}
$$

Integrating it from zero to t, we obtain

$$
\begin{aligned}
\bar{V}(t) - \bar{V}(0) &\leqslant -\min\{K,Q,T\}\int_0^t [(x(s)-x^*)^2 + (y(s)-y^*)^2 + (z(s)-z^*)^2]ds + \Theta t \\
&\quad + \int_0^t \left[A(x(s)-x^*)dW_1(s) + A(y(s)-y^*)dW_2(s) + \frac{1}{r}(z(s)-z^*)dW_3(s)\right].
\end{aligned}
$$

Therefore,

$$\int_0^t [(x(s)-x^*)^2+(y(s)-y^*)^2+(z(s)-z^*)^2]ds$$
$$\leqslant \frac{\bar{V}(0)}{\min\{K,Q,T\}}+\frac{\Theta t}{\min\{K,Q,T\}}$$
$$+\frac{1}{\min\{K,Q,T\}}\int_0^t\left[A(x(s)-x^*)dW_1(s)+A(y(s)-y^*)dW_2(s)+\frac{1}{r}(z(s)-z^*)dW_3(s)\right]. \quad \text{(F2)}$$

Assume $M(t)=\int_0^t [A(x(s)-x^*)dW_1(s)+A(y(s)-y^*)dW_2(s)+\frac{1}{r}(z(s)-z^*)dW_3(s)]$. M is a continuous martingale and $M(0)=0$. Moreover,

$$\langle M,M\rangle_t = \left(\int_0^t\left[A(x(s)-x^*)dW_1(s)+A(y(s)-y^*)dW_2(s)+\frac{1}{r}(z(s)-z^*)dW_3(s)\right]\right)^2$$
$$=\left(\int_0^t\left[A^2(x(s)-x^*)^2ds+A^2(y(s)-y^*)^2ds+\frac{1}{r^2}(z(s)-z^*)^2ds\right]\right)$$
$$\leqslant\left(A^2(\bar{x}^2+\bar{y}^2)+\frac{1}{r^2}\bar{z}^2\right)t$$

and

$$\limsup_{t\to\infty}\frac{\langle M,M\rangle_t}{t}\leqslant A^2(\bar{x}^2+\bar{y}^2)+\frac{1}{r^2}\bar{z}^2<\infty \text{ a.s.}$$

We now use the following lemma.

Lemma F.1 Let $N=\{N_t\}_{t\geqslant 0}$ be a real-valued continuous local martingale with $N(0)=0$. Then

$$\limsup_{t\to\infty}\frac{\langle N,N\rangle_t}{t}<\infty \text{ a.s.} \Rightarrow \lim_{t\to\infty}\frac{N_t}{t}=0 \text{ a.s.}$$

Using this lemma, one gets

$$\limsup_{t\to\infty}\frac{M(t)}{t}=0 \text{ a.s.}$$

Therefore, from (F2), we obtain

$$\limsup_{t\to\infty}\frac{1}{t}\int_0^t[(x(s)-x^*)^2+(y(s)-y^*)^2+(z(s)-z^*)^2]ds\leqslant G\Theta \text{ a.s.}$$

where $G=\frac{1}{\min\{K,Q,T\}}$.

APPENDIX G: PARAMETER ESTIMATION

For the estimation of the parameter set, we first find the best fit parameters for the deterministic system through least square method such that the sum of the squared difference of deterministic model output and experimental data would be minimized. Consider the multidimensional parameter set $\theta=(\theta_1,\theta_2,...,\theta_n)$ and $(t_1,y_1),(t_2,y_2),...,(t_n,y_n)$ to be the given set of experimental data points. If we assume $h(t_j,\theta)$ to be the model output at t_j time step, then our objective is to minimize the squared sum of errors (SSE):

$$\text{SSE}(\theta)=\sum_{i=1}^{n}(h(t_i,\theta)-y_i)^2.$$

Starting from an initial guess of θ, we will find the best parameter set iteratively using the FMINSEARCH algorithm toolbox of MATLAB. FMINSEARCH is a derivative-free method [92] which finds the minimum of an unconstrained multivariable function using the Nelder-Mead simplex algorithm as described in Langarias *et al.* [93]. After the estimation of parameters for the deterministic system, we search for a proper noise intensity of the stochastic system (3) to obtain a good agreement between stochastic system output and experimental data. In the quest of suitable noise strength, we compute the SSE, the total sum of squares (SST), and the corresponding r-squared value for stochastic simulation data and experimental data. The statistical measure of fit r squared was computed from the relation [94]

$$r \text{ squared}=1-\frac{\text{SSE}}{\text{SST}}.$$

We considered 10 000 different random values of noise intensities $\sigma_1,\sigma_2,\sigma_3$ between 0 and 1 through Latin hypercube sampling. Then for each of these 10 000 tuples $(\sigma_1,\sigma_2,\sigma_3)$, the stochastic system was simulated 1000 times. Taking the mean of the

1000 simulations, r squared was computed between the average stochastic simulation output and the experimental data. During the parameter estimation, we intended to keep the values of a, λ, b, e, γ in the observed range of field data, and the rest parameters (c, f, m_1, m, r, m_2) were selected such that the existence and stability conditions of the interior equilibrium E^* hold simultaneously.

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