

ESTIMATING WHITE NOISE INTENSITY REGIONS FOR COMPARABLE PROPERTIES OF A CLASS OF SEIRS STOCHASTIC AND DETERMINISTIC EPIDEMIC MODELS

Divine Wanduku¹

Abstract A comparative stochastic and deterministic study of a family of epidemic models for vector-borne diseases e.g. malaria and dengue fever etc. is presented. The family type is determined by a general nonlinear incidence rate of the disease. Two major sources of environmental white noises are considered: disease transmission and natural death rates. The impacts of each source of noise on the disease dynamics are examined. The basic reproduction numbers and other threshold values for the disease in the stochastic and deterministic settings are determined and compared to determine the impacts of the noises on the dynamics. The question about the extend that stability conditions for steady states in the noise-free disease dynamics, remain valid for the stochastic stability of the steady state is answered in this paper. Moreover, noise intensity regions are computed, within which all stability conditions for both systems are the same, and both systems behave similarly.

Keywords Stochastic delay differential equations, stochastic stability in probability, functional Itô differential operator, deterministic delay differential equations, white noise intensity.

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1. Introduction

Vector-borne diseases such as malaria, and dengue fever etc. rank amongst the top most widely spread infectious diseases of humans in the world today with very high global mortality rates, and heavy economic burdens upon many nations in the world. In fact, according to the WHO report [71] released in December 2016, it is estimated that about 212 million cases of malaria occurred in 2015 resulting in a large-scale death count of about 429 thousand people. Dengue fever on the other hand prevails globally among about 400 million people annually, and approximated 22,000 of the 100 million infected cases die from the disease [71, 73].

Certain biological characteristics are unique to vector-borne diseases such as dengue fever, malaria, yellow fever, zika fever, lymphatic filariasis, and the different types of encephalitis etc. For instance, the incubation of the diseases require two hosts-the vector and human hosts, which may be either involved in one full life cycle of the infectious agent consisting of two separate and independent segments

Emails: dwanduku@georgiasouthern.edu; wandukudivine@yahoo.com

¹Department of Mathematical Sciences, Georgia Southern University, 65 Georgia Ave, Room 3309, Statesboro, Georgia, 30460, U.S.A

of sub-life cycles, where each segment is completed separately inside the two hosts (e.g. malaria), or the vector and the human constitute different forms of behavior of the infectious agent in two separate and independent half-life cycles in each host (e.g. dengue fever). Therefore, there exists a total latent time lapse of disease incubation which extends over the two segments of delay incubation times namely: (1) the incubation period of the infectious agent (or the first half-life cycle) inside the vector, and (2) the incubation period of the infectious agent (or the second half-life cycle) inside the human being.

For example, the dengue fever virus transmitted primarily by the *Aedes aegypti* and *Aedes albopictus* mosquitos undergoes two delay incubation periods: (1) about 8-12 days incubation period inside the female mosquito vector, which starts immediately after the ingestion of an infected blood meal collected, after biting a dengue fever infectious person, and (2) another delay incubation period of about 2-7 days inside a human, whenever the infected female vector leaves its resting place for another blood meal, and bites a susceptible person, and successfully transmits the virus to the person [71, 73].

The following facts about malaria appear in the earlier study [56]. The malaria plasmodium undergoes the first developmental half-life cycle called the *sporogonic cycle* inside the female *Anopheles* mosquito lasting approximately 10–18 days, following a successful blood meal obtained from an infectious human being through a mosquito bite. Moreover, the mosquito becomes infectious. The parasite completes the second developmental half-life cycle called the *exo-erythrocytic cycle* lasting about 7–30 days inside the exposed human being, whenever the parasite is transferred to the human being in the process of the infectious mosquito foraging for another blood meal. See the references [16, 71, 72].

The exposure and successful recovery from a malaria parasite, for example, *falciparum vivax* induces natural immunity against the disease which can protect against subsequent severe outbreaks of the disease. Moreover, the effectiveness and duration of the naturally acquired immunity against malaria is determined by several factors such as the species and the frequency of exposure to the parasites. Furthermore, it has been determined that other biological factors such as the genetics of the human being, for instance, sickle-cell anaemia, duffy negative blood types have bearings on the naturally acquired immunity against different species of malaria. See the references [17, 23, 72]. Similarly, the exposure and successful recovery from one dengue fever viral strain confers lifelong immunity against the particular viral serotype [73].

Various types of mathematical models have been proposed for malaria with the earliest studies by Ross [50] who studied mosquito control in 1911. Many other authors have addressed different aspects of the malaria dynamics (cf. [2, 12, 24, 39, 43–45, 55]). Dengue fever has also been studied mathematically (cf. [53]).

Some studies have shown the presence of noise in the dynamics of malaria. Noise can be seen in seasonal variations of the malaria incidence rates over yearly data, and over spatial disparities of malaria prevalence rates. In fact, some authors such as [49] studying the seasonality of *P. falciparum* transmission have shown that there are several climatic drivers responsible for the temporal variation and spatial distribution of malaria transmission rates, for instance, temperature, rainfall, and vegetation indices etc. The randomness in the malaria incidence rates over time, and spatially is a good reason to consider stochastic representations of the disease dynamics.

There are several different ways to introduce white noise into infectious disease dynamic systems, for example, as random perturbation of the driving parameters of the infectious system known as environmental white noise (see [19, 46, 59, 60, 63]), or random perturbation of the state of the system also known demographic white noise (see [5]). Some authors such as [1, 7] have suggested a mean-reverting process technique to include white noise processes. Also, some stochastic models for malaria involving white noise perturbations include [27, 32].

A stochastic white noise driven system exhibits more complex behavior in the disease dynamics than the corresponding deterministic version. For instance, the presence of noise in the disease dynamics may destabilize a disease-free steady state. The occurrence of noise with high intensity may cause massive oscillations over time in the population state, which may decrease the population size over time, and lead to extinctions depending on the source of the noise e.g. high intensity noise in natural death rates can lead to extinction etc. (cf. [35, 59, 60, 63, 69, 70]).

Cooke [14] presented a deterministic epidemic dynamic model for vector-borne diseases, where the bilinear incidence rate defined as $\beta S(t)I(t - T)$ represents the number of new infections occurring per unit time during the disease transmission process. It is assumed in the formulation of this incidence rate that the number of infectious vectors $V_i(t)$ at time t interacting and effectively transmitting infection to susceptible human beings, $S(t)$, after β number of effective contacts per unit time per infective is proportional to the infectious human population, $I(t - T)$, at earlier time $t - T$. Cook’s method of studying the dynamics of a vector-borne disease in a human population without directly including the vector population dynamics has been utilized by several other authors, for example [35, 40, 54, 56, 59]. Some criticism of the Cooke model concerns the absence of a rationale for the assumption about the proportionality between $V_i(t)$ and $I(t - T)$ used as an approximation for the force of infection. Furthermore, there is the question whether the Cooke model emerges from the combined vector-host dynamics. Takeuchi et. al. [54] answered this question and presented an extension of the Cooke model, with a joint dynamics for the vector-host populations, and under the assumption of a large constant vector population present, the proportionality between the states $V_i(t)$ and $I(t - T)$ is justified.

Recently, applying similar reasoning of the Cooke model, Wanduku [56, 57] developed and studied a novel family of SEIRS dynamic models for malaria with three distributed delays given in (1.1)–(1.4) below. The model in [56, 57] was simplified by omitting the vector dynamics (similarly as [14]), and applying other assumptions about the death rates of the vectors and humans. Moreover, time scales for the model parameters were not well specified. Thus, to improve understanding and usefulness of [56, 57], it is necessary to add more epidemiologically sound assumptions, and define time scales for the model parameters in (1.1)–(1.4), and derive the model [56, 57] from the vector vs. human dynamics.

The new modified family of SEIRS dynamic models is given in (1.1)–(1.4). Note, the modified assumptions for (1.1)–(1.4), and complete derivation of the model (1.1)–(1.4) are given in 8.

$$\begin{aligned}
 dS(t) = & \left[B - \beta S(t) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_v s} G(I(t - s)) ds - \mu S(t) \right. \\
 & \left. + \alpha \int_{t_0}^{\infty} f_{T_3}(r) I(t - r) e^{-\mu r} dr \right] dt,
 \end{aligned}
 \tag{1.1}$$

$$dE(t) = \left[\beta S(t) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_v s} G(I(t-s)) ds - \mu E(t) - \beta \int_{t_0}^{h_2} f_{T_2}(u) S(t-u) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_v s - \mu u} G(I(t-s-u)) ds du \right] dt, \quad (1.2)$$

$$dI(t) = \left[\beta \int_{t_0}^{h_2} f_{T_2}(u) S(t-u) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_v s - \mu u} G(I(t-s-u)) ds du - (\mu + d + \alpha) I(t) \right] dt, \quad (1.3)$$

$$dR(t) = \left[\alpha I(t) - \mu R(t) - \alpha \int_{t_0}^{\infty} f_{T_3}(r) I(t-r) e^{-\mu r} dr \right] dt, \quad (1.4)$$

where the initial conditions are given in the following: let $h = h_1 + h_2$ and define

$$(S(t), E(t), I(t), R(t)) = (\varphi_1(t), \varphi_2(t), \varphi_3(t), \varphi_4(t)), t \in (-\infty, t_0], \quad (1.5)$$

$$\varphi_k \in UC_g \subset \mathcal{C}((-\infty, t_0], \mathbb{R}_+), \forall k = 1, 2, 3, 4, \quad \varphi_k(t_0) > 0, \forall k = 1, 2, 3, 4,$$

where UC_g is some fading memory sub Banach space of the Banach space $\mathcal{C}((-\infty, t_0], \mathbb{R}_+)$ endowed with the norm

$$\|\varphi\|_g = \sup_{t \leq t_0} \frac{|\varphi(t)|}{g(t)}, \quad (1.6)$$

and g is some continuous function with the following properties: (P1.) $g((-\infty, t_0]) \subseteq [1, \infty)$, non-increasing, and $g(t_0) = 1$; (P2.) $\lim_{u \rightarrow t_0^-} \frac{g(t+u)}{g(t)} = 1$, uniformly on $[t_0, \infty)$; $\lim_{t \rightarrow -\infty} g(t) = \infty$. An example of such a function is $g(t) = e^{-at}$, $a > 0$ (cf. [28]). Note that for any g satisfying (P1.)–(P2.) the Banach space $\mathcal{C}((-\infty, t_0], \mathbb{R}_+)$ is continuously embedded in UC_g which allows structural properties for $\mathcal{C}((-\infty, t_0], \mathbb{R}_+)$ with the uniform norm to hold in UC_g with $\|\cdot\|_g$ norm. Moreover, $\varphi \in UC_g$, $\exists g$ if and only if $\|\varphi\|_g < \infty$ and $\frac{|\varphi(t)|}{g(t)}$ is uniformly continuous on $(-\infty, t_0]$. Also, the function G satisfies the conditions of Assumption 1.1.

In (1.1)–(1.4), the disease spreads in the human population of total size $N(t) = S(t) + E(t) + I(t) + R(t)$, where $S(t)$, $E(t)$, $I(t)$ and $R(t)$ represent the susceptible, exposed, infectious and naturally acquired immunity classes at time t , respectively. The positive constants B , and μ represent the constant birth and natural death rates, respectively. Furthermore, the disease related deathrate is denoted d . The rate β is the average effective contact rate per infected mosquito per unit time. The recovery rate from the disease with acquired immunity is α . All parameters of (1.1)–(1.4) are dimensionless and defined in (8.42).

Also, the incubation delays inside the mosquito and human hosts are denoted T_1 and T_2 , respectively, and the period of effective naturally acquired immunity is denoted T_3 . Moreover, the delays are random variables with arbitrary densities denoted f_{T_1} , f_{T_2} and f_{T_3} , and their supports given as $T_1 \in [t_0, h_1]$, $T_2 \in [t_0, h_1]$ and $T_3 \in [t_0, +\infty)$. The nonlinear incidence function G which signifies the response to disease transmission by the susceptible class as disease increases in the population, satisfies the following assumptions

Assumption 1.1. A1: $G(0) = 0$; A2: $G(I)$ is strictly monotonic on $[0, \infty)$; A3: $G \in \mathcal{C}^2(\mathbb{R}_+, \mathbb{R}_+)$ and $G''(I) < 0$; A4: $\lim_{I \rightarrow \infty} G(I) = C$, $0 \leq C < \infty$; and A5: $G(I) \leq I$, $\forall I > 0$.

Deterministic models represent disease dynamics in ideal situations, and are simply first approximations. Realistically, the occurrence of noise is inevitable in disease dynamics. As emphasized in the introduction, the occurrence of noise in a disease dynamics can destabilize a steady state, or cause the steady state to cease to exist (c.f. [35, 59, 60, 63, 69, 70]).

The question remains about the extend that stability conditions for a steady state of the noise-free dynamics will continue to suffice for the stochastic stability of the steady state when noise occurs in the dynamics. In other words, to what extend will disease eradication conditions for the deterministic system remain sufficient for the ensuing stochastic system? This question is addressed in this paper via a comparative analysis of the deterministic and corresponding stochastic systems: (1.1)–(1.4) and (1.8)–(1.10), and with attention given to elucidate the impacts of noises and delays in the disease dynamics, on the existence and stability of equilibria, which create several interesting features of the disease dynamics near the infection-free equilibrium.

Apart from having a focus on both deterministic and stochastic systems, this work differs considerably from [57] in (a) characterizing noise intensity regions, where behavior of both stochastic vs deterministic systems contrast or are similar; (b) intensifying the importance of delays in the system in defining parameter regions that delineate the behaviors of the stochastic and deterministic systems. And (c) more appropriate stochastic Lyapunov functionals are constructed, and properly estimated, whenever self-invariant spaces exist, or do not exist. Obviously, (a)–(c) could not be obtained in [57].

This work is presented as follows. In 8, the stochastic and deterministic epidemic dynamic models for the disease are derived. In section 2, the model validation results are presented for both the deterministic and stochastic systems. In section 3, the effects of the noises on the existence of equilibria of the systems are investigated. In Section 4, stochastic stability results with noise only from the disease transmission rate is presented. In Section 5, the stochastic stability results with noises from both the disease transmission rate and natural death rates are presented. In section 6, a parameter region for the intensities of noises in the system within which the stability conditions of the deterministic system remain sufficient for the stochastic stability of equilibria is presented. In Section 7, the stochastic system is characterized when noise occurs from the natural death rate of the susceptible state.

Observe that the system (1.1)–(1.4) is similarly structured as [56], and the model parameters are well defined in (8.42). Thus, the deterministic results in [56] can be translated into (1.1)–(1.4).

It is assumed that the effects of random environmental fluctuations lead to variability in the disease transmission and natural death rates. For $t \geq t_0$, let $(\Omega, \mathfrak{F}, P)$ be a complete probability space, and \mathfrak{F}_t be a filtration (that is, sub σ -algebra \mathfrak{F}_t that satisfies the following: given $t_1 \leq t_2 \Rightarrow \mathfrak{F}_{t_1} \subset \mathfrak{F}_{t_2}$; \mathfrak{F}_0 contains all null sets in \mathfrak{F}_t). Indeed, the variability in the disease transmission and natural death rates are represented by the white noise processes as follows:

$$\mu \rightarrow \mu + \sigma_i \xi_i(t), \quad \xi_i(t)dt = dw_i(t), \quad i = S, E, I, R, \quad \beta \rightarrow \beta + \sigma_\beta \xi_\beta(t), \quad \xi_\beta(t)dt = dw_\beta(t), \quad (1.7)$$

where $\xi_i(t)$ and $w_i(t)$ represent the standard white noise and normalized Wiener processes for the i^{th} state at time t , with the following properties: $w(0) = 0$, $\mathbb{E}(w(t)) = 0$, $\text{Var}(w(t)) = t$. Furthermore, observe from (1.7), $\text{Var}(\mu dt + \sigma_i dw_i(t)) = \sigma_i^2 dt$, $i = S, E, I, R$, where σ_i^2 , $i = S, E, I, R$ represents the intensity of the environmental

white noise in the natural death rate of the i^{th} state, and $\text{Var}(\beta dt + \sigma_\beta dw_\beta(t)) = \sigma_\beta^2 dt$, where σ_β^2 is the intensity of the white noise in the disease transmission rate.

Substituting (1.7) into the deterministic system (1.1)–(1.4) leads to the following generalized system of Itô-Doob stochastic differential equations describing the dynamics of vector-borne diseases in the human population.

$$\begin{aligned} dS(t) = & \left[B - \beta S(t) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s} G(I(t-s)) ds - \mu S(t) \right. \\ & \left. + \alpha \int_{t_0}^{\infty} f_{T_3}(r) I(t-r) e^{-\mu r} dr \right] dt \\ & - \sigma_S S(t) dw_S(t) - \sigma_\beta S(t) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s} G(I(t-s)) ds dw_\beta(t) \end{aligned} \quad (1.8)$$

$$\begin{aligned} dE(t) = & \left[\beta S(t) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s} G(I(t-s)) ds - \mu E(t) \right. \\ & \left. - \beta \int_{t_0}^{h_2} f_{T_2}(u) S(t-u) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s - \mu u} G(I(t-s-u)) ds du \right] dt \\ & - \sigma_E E(t) dw_E(t) + \sigma_\beta S(t) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s} G(I(t-s)) ds dw_\beta(t) \\ & - \sigma_\beta \int_{t_0}^{h_2} f_{T_2}(u) S(t-u) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s - \mu u} G(I(t-s-u)) ds du dw_\beta(t) \end{aligned} \quad (1.9)$$

$$\begin{aligned} dI(t) = & \left[\beta \int_{t_0}^{h_2} f_{T_2}(u) S(t-u) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s - \mu u} G(I(t-s-u)) ds du \right. \\ & \left. - (\mu + d + \alpha) I(t) \right] dt - \sigma_I I(t) dw_I(t) \end{aligned} \quad (1.10)$$

$$+ \sigma_\beta \int_{t_0}^{h_2} f_{T_2}(u) S(t-u) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s - \mu u} G(I(t-s-u)) ds du dw_\beta(t)$$

$$dR(t) = \left[\alpha I(t) - \mu R(t) - \alpha \int_{t_0}^{\infty} f_{T_3}(r) I(t-r) e^{-\mu r} dr \right] dt - \sigma_R R(t) dw_R(t), \quad (1.11)$$

where the initial conditions are given in the following: let $h = h_1 + h_2$ and define

$$\begin{aligned} (S(t), E(t), I(t), R(t)) &= (\varphi_1(t), \varphi_2(t), \varphi_3(t), \varphi_4(t)), t \in (-\infty, t_0], \\ \varphi_k &\in UC_g \subset \mathcal{C}((-\infty, t_0], \mathbb{R}_+), \forall k = 1, 2, 3, 4, \quad \varphi_k(t_0) > 0, \forall k = 1, 2, 3, 4, \end{aligned} \quad (1.12)$$

where UC_g is some fading memory sub Banach space of the Banach space $\mathcal{C}((-\infty, t_0], \mathbb{R}_+)$ endowed with the norm

$$\|\varphi\|_g = \sup_{t \leq t_0} \frac{|\varphi(t)|}{g(t)}, \quad (1.13)$$

and g is some continuous function with the following properties: (P1.) $g((-\infty, t_0]) \subseteq [1, \infty)$, non-increasing, and $g(t_0) = 1$; (P2.) $\lim_{u \rightarrow t_0^-} \frac{g(t+u)}{g(t)} = 1$, uniformly on $[t_0, \infty)$; $\lim_{t \rightarrow -\infty} g(t) = \infty$. Furthermore, the random continuous functions $\varphi_k, k = 1, 2, 3, 4$ are \mathfrak{F}_0 -measurable, or independent of $w(t)$ for all $t \geq t_0$.

Observe that (1.9) and (1.11), and the corresponding equations (1.2) and (1.4) all decouple from the other two equations in their respective systems: (1.8)–(1.11) and (1.1)–(1.4). Nevertheless, for convenience most of the results in this paper related to the systems (1.8)–(1.11) and (1.1)–(1.4) will be shown for the vector $X(t) = (S(t), E(t), I(t))^T$. The following notations are utilized:

$$\begin{aligned} Y(t) &= (S(t), E(t), I(t), R(t))^T, \quad X(t) = (S(t), E(t), I(t))^T, \\ N(t) &= S(t) + E(t) + I(t) + R(t). \end{aligned} \tag{1.14}$$

Also, observe from the dimensionless formulas (8.42) that

$$\frac{B}{\mu} = \frac{\left[\frac{\hat{B}}{\left(\frac{\hat{B}}{\mu}\right)^2 \Lambda} \right]}{\left[\frac{\hat{\mu}}{\left(\frac{\hat{B}}{\mu}\right) \Lambda} \right]} = 1. \tag{1.15}$$

However, in this paper, the notation $\frac{B}{\mu} \equiv 1$ will be used to emphasize the origin of the dimensionless quantity “1” in (1.15).

2. Model Validation Results

Observe from the deterministic and stochastic systems (1.1)–(1.4) and (1.8)–(1.11), respectively, and (1.14), that when the intensities $\sigma_i = 0, i \in \{S, E, I, R\}$, both systems are the same. The following result shows that there exists a unique positive self-invariant space for both systems, whenever the intensities of the noises are infinitesimally small.

Theorem 2.1. *Given the initial conditions (1.5)–(1.6), there exists a unique solution $Y(t) = (S(t), E(t), I(t), R(t))^T$ satisfying (1.1)–(1.4), for all $t \geq t_0$. Moreover, the solution is nonnegative for all $t \geq t_0$ and also lies in $D(\infty)$. That is, $S(t) > 0, E(t) > 0, I(t) > 0, R(t) > 0, \forall t \geq t_0$ and*

$$\limsup_{t \rightarrow \infty} N(t) \leq S_0^* = \frac{B}{\mu} \equiv 1, \tag{2.1}$$

for $N(t) = S(t) + E(t) + I(t) + R(t)$, and $Y(t) \in D(\infty) = \bar{B}_{\mathbb{R}_+^4}^{(-\infty, \infty)} \left(0, \frac{B}{\mu} \equiv 1 \right)$, where $D(\infty)$ is defined in (2.3).

Proof. Observe from the deterministic and stochastic systems (1.1)–(1.4) and (1.8)–(1.11), respectively, and (1.14), that when the intensities $\sigma_i = 0, i \in \{S, E, I, R\}$, both systems satisfy

$$dN(t) = [B - \mu N(t) - dI(t)]dt. \tag{2.2}$$

It follows that for $Y(t) \in \mathbb{R}_+^4$, the equation (2.2) leads to $N(t) \leq \frac{B}{\mu} - \frac{B}{\mu} e^{-\mu(t-t_0)} + N(t_0)e^{-\mu(t-t_0)}$. And under the assumption that $N(t_0) \leq \frac{B}{\mu}$, it follows that the set

$$\begin{aligned} D(\infty) &= \left\{ Y(t) \in \mathbb{R}_+^4 : N(t) = \|Y(t)\|_1 \leq \frac{B}{\mu}, \forall t \in (-\infty, \infty) \right\} \\ &\equiv \bar{B}_{\mathbb{R}_+^4}^{(-\infty, \infty)} \left(0, \frac{B}{\mu} \equiv 1 \right), \end{aligned} \tag{2.3}$$

representing the closed unit ball in \mathbb{R}_+^4 centered at the origin and radius $\frac{B}{\mu} \equiv 1$, with norm $\|\cdot\|_1$ is almost surely self-invariant with respect to the deterministic and stochastic systems, provided that $\sigma_i = 0, i \in \{S, E, I, R\}$ in (1.8)–(1.11). \square

Theorem 2.2. *Given the initial conditions (1.12) and (1.13), there exists a unique solution process $X(t, w) = (S(t, w), E(t, w), I(t, w))^T, \forall w \in \Omega$ satisfying (1.8)–(1.11), for all $t \geq t_0$. Moreover,*

- (a.) *the solution process is positive for all $t \geq t_0$ a.s. and lies in $D(\infty)$, whenever the intensities of the independent white noise processes in the system satisfy $\sigma_i = 0, i \in \{S, E, I\}$ and $\sigma_\beta \geq 0$. That is, $S(t, w) > 0, E(t, w) > 0, I(t, w) > 0, \forall t \geq t_0$ a.s. and $X(t, w) \in D(\infty) = \bar{B}_{\mathbb{R}_+^4}^{(-\infty, \infty)}\left(0, \frac{B}{\mu}\right)$, where $D(\infty)$ is defined in (2.3).*
- (b.) *Also, the solution process is positive for all $t \geq t_0$ a.s. and lies in \mathbb{R}_+^4 , whenever the intensities of the independent white noise processes in the system satisfy $\sigma_i > 0, i \in \{S, E, I\}$ and $\sigma_\beta \geq 0$. That is, $S(t, w) > 0, E(t, w) > 0, I(t, w) > 0, \forall t \geq t_0$ a.s. and $X(t, w) \in \mathbb{R}_+^4$.*

Proof. Observe that when $\sigma_i = 0, i \in \{S, E, I\}$ and $\sigma_\beta = 0$, the result of (a) follows immediately from Theorem 2.1. Also, when $\sigma_i = 0, i \in \{S, E, I\}$ and $\sigma_\beta > 0$, observe that the ensuing stochastic system (1.8)–(1.11), still satisfies (2.2), and the results follow from Theorem 2.1.

For (b), when at least one of $\sigma_i > 0, i \in \{S, E, I, R\}$, the equation (2.2) is no longer satisfied, and the closed unit ball $D(\infty)$ is no longer self-invariant for the system (1.8)–(1.11). Nevertheless, all sample paths for the stochastic system remain in \mathbb{R}_+^4 (cf. [59–62]). \square

Remark 2.1.

1. Theorem 2.2 signifies that the stochastic system (1.8)–(1.11) almost surely has a unique global positive solution process $Y(t) \in \mathbb{R}_+^4$, for all $t \in (-\infty, \infty)$. Furthermore, it follows that a positive solution of the system that starts in the closed ball centered at the origin with a radius of $\frac{B}{\mu} \equiv 1$, given by $D(\infty) = \bar{B}_{\mathbb{R}_+^4}^{(-\infty, \infty)}\left(0, \frac{B}{\mu}\right)$, will continue to oscillate in the closed unit ball for all time $t \geq t_0$, whenever the intensities of the noises from the natural death rates are zero, that is, $\sigma_i = 0, i \in \{S, E, I, R\}$. Hence, the unit ball $D(\infty) = \bar{B}_{\mathbb{R}_+^4}^{(-\infty, \infty)}\left(0, \frac{B}{\mu}\right)$ is a positive self-invariant set for the stochastic system (1.8)–(1.11), whenever $\sigma_i = 0, i \in \{S, E, I, R\}$. In other words, the trajectories of the system (1.8)–(1.11) are "well-behaved" whenever the only major source of variability in the system is the disease transmission rate $\sigma_\beta > 0$.

When at least one of the intensities of the noises from the natural death rates is positive, that is, $\sigma_i > 0, i \in \{S, E, I, R\}$, all trajectories that start in the unbounded positive space \mathbb{R}_+^4 continue to oscillate in the space \mathbb{R}_+^4 for all time $t \geq t_0$. This suggests that the paths of the stochastic system are inflated out of bounds in $D(\infty)$ by the additional source of noise, the natural death rates, but they continue to oscillate unpredictably in \mathbb{R}_+^4 . Thus, in this scenario, various complex behaviors are possible, for example, extinction of the population.

2. Theorem 2.1 also signifies that the deterministic system (1.1)–(1.4) has a unique global positive solution denoted by $Y(t) \in \mathbb{R}_+^4$, for all $t \in (-\infty, \infty)$. Furthermore, it follows that any positive solution of the deterministic system that starts in the closed unit ball $D(\infty)$, grows and becomes bounded within the closed unit ball

for all time $t \geq t_0$, as signified by (2.1). In other words, $D(\infty) = \bar{B}_{\mathbb{R}_+^4}^{(-\infty, \infty)} \left(0, \frac{B}{\mu}\right)$ is also positive self-invariant space for the deterministic system (1.1)–(1.4).

The Remark 2.1 suggests that the character of the stochastic disease dynamics in this paper is more profound than [56]. For instance, there is tendency for the noises in the dynamics to drive the population to extinction etc. Moreover, some nontrivial factors influencing the behavior of the disease dynamics are identified namely: (1) the presence or absence of noises in the dynamics, (2) the major source of noise in the dynamics: disease transmission and/or natural death rates, and (3) the magnitude of the intensities of the noises in the disease dynamics etc. Given limited space, (1)&(2) are given full treatment in this paper.

3. Existence of infection-free equilibrium

Let the equilibria of the two delayed systems (1.1)–(1.4) and (1.8)–(1.11) be denoted generally by $E = (S^*, E^*, I^*)$. For infection free steady state, $E = I = R = 0$. Note that the existence of a disease free steady state solution for the stochastic system is determined by the intensities of the white noises in the system $\sigma_i, i = S, E, I, \beta$. For easy reference, the following result characterizes the existence of the disease-free steady solution of the systems: (1.1)–(1.4) and (1.8)–(1.11).

Theorem 3.1.

1. *There exists a disease-free steady state $E_0 = (S_0^*, 0, 0)$ for the deterministic system (1.1)–(1.4), where $S_0^* = \frac{B}{\mu} \equiv 1$.*
2. *When $\sigma_i \geq 0, i = E, I, \beta$ and $\sigma_S = 0$, there exists a disease-free steady state solution $E_0 = (S_0^*, 0, 0)$, for the stochastic system (1.8)–(1.11), where $S_0^* = \frac{B}{\mu} \equiv 1$.*
3. *When $\sigma_i \geq 0, i = E, I, \beta$ and $\sigma_S > 0$, the system (1.8)–(1.11) does not have a disease-free steady state solution.*

Proof. The results follow immediately by applying standard methods of finding equilibria for stochastic systems. □

Remark 3.1. Theorem 3.1[1.] signifies that the deterministic system (1.1)–(1.4) always has a disease free equilibrium given by E_0 . Theorem 3.1[2.] and Theorem 3.1[3.] signify that regardless of the intensities $\sigma_i \geq 0, i = E, I, \beta$ of the noises in the natural death rates of the exposed, infectious and removal states, and also from the disease transmission rate, there exists a steady state disease-free population E_0 , which is exactly the same as that of the deterministic system, provided the intensity of the white noise in the natural death rate of the susceptible state is zero. That is, $\sigma_S = 0$.

These observations suggest that the source: disease transmission rate or natural death rates, and also the magnitude of the intensities of the noises in the stochastic system (1.8)–(1.11) have bearings on the asymptotic behavior of the paths of the stochastic system (1.8)–(1.11) near the infection-free steady state E_0 .

In the following, the asymptotic stability of the disease free equilibrium, E_0 , of the deterministic system (1.1)–(1.4) and the stochastic system (1.8)–(1.11), whenever $\sigma_S = 0$ are investigated and compared. The deterministic and stochastic versions of the Lyapunov functionals techniques [59, 60, 62] are utilized to establish

the stability results. The paths of the systems: (1.1)–(1.4) and (1.8)–(1.11) are transformed using $E_0 = (S_0^*, 0, 0)$, $S_0^* = \frac{B}{\mu} \equiv 1$ as follows:

$$U(t) = S(t) - S_0^*, \quad V(t) = E(t), \quad W(t) = I(t). \quad (3.1)$$

By employing the transformation in (3.1) to the system (1.8)–(1.10), the following system is obtained:

$$\begin{aligned} dU(t) = & \left[-\beta U(t) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu s} G(W(t-s)) ds - \mu U(t) \right. \\ & \left. + \alpha \int_{t_0}^{\infty} f_{T_3}(r) W(t-r) e^{-\mu r} dr \right] dt - \sigma_S (S_0^* + U(t)) dw_S(t) \\ & - \sigma_\beta (S_0^* + U(t)) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s} G(W(t-s)) ds dw_\beta(t), \\ dV(t) = & \left[\beta (S_0^* + U(t)) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu s} G(W(t-s)) ds - \mu V(t) \right. \\ & \left. - \beta \int_{t_0}^{h_2} f_{T_2}(u) (S_0^* + U(t-u)) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_e s - \mu u} G(W(t-s-u)) ds du \right] dt \\ & - \sigma_E V(t) dw_E(t) + \sigma_\beta (S_0^* + U(t)) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s} G(W(t-s)) ds dw_\beta(t) \\ & - \sigma_\beta \int_{t_0}^{h_2} f_{T_2}(u) (S_0^* + U(t-u)) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_e s - \mu u} G(W(t-s-u)) ds du dw_\beta(t), \end{aligned} \quad (3.2)$$

$$(3.3)$$

and

$$\begin{aligned} dW(t) = & \left[\beta \int_{t_0}^{h_2} f_{T_2}(u) (S_0^* + U(t-u)) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s - \mu u} G(W(t-s-u)) ds du \right. \\ & \left. - (\mu + d + \alpha) W(t) \right] dt - \sigma_I W(t) dw_I(t) \\ & + \sigma_\beta \int_{t_0}^{h_2} f_{T_2}(u) (S_0^* + U(t-u)) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s - \mu u} G(W(t-s-u)) ds du dw_\beta(t). \end{aligned} \quad (3.4)$$

The lemmas that follow in this section will be utilized to establish the asymptotic results for the system (1.8)–(1.11) with respect to the steady state solution E_0 . Note that from Assumption 1.1 the nonlinear function G is bounded. Therefore, suppose

$$G^* = \sup_{z>0} G(z), \quad (3.5)$$

then it is easy to see that $0 \leq G(z) \leq G^*$. It follows further from Assumption 1.1 that given $\lim_{I \rightarrow \infty} G(I) = C$, if G is strictly monotonic increasing then $G^* \leq C$. Also, if G is strictly monotonic decreasing then $G^* \geq C$.

Recall the following lemma in the earlier study [63, Lemma 4.1]. Also, from (1.14) and (3.1), we organize all notations used in the subsequent sections of this paper as follows.

Notation 3.1.

(1.) We denote by

$$\begin{aligned} Y(t) &= (S(t), E(t), I(t), R(t))^T, X(t) = (S(t), E(t), I(t))^T, \\ N(t) &= S(t) + E(t) + I(t) + R(t). \\ x(t) &= (U(t), V(t), W(t))^T = (S(t) - S_0^*, E(t), I(t))^T. \end{aligned} \tag{3.6}$$

(2.) Note from (3.6) that for each $t \in \mathbb{R}_+$, $x(t)$ is the transformed or shifted value of $X(t)$. Also, $x = (U, V, W)^T = (S - S_0^*, E, I)^T$ denotes a point on the functions vector space $[\mathcal{C}^1(\mathbb{R} \times \mathbb{R}_+, \mathbb{R}_+)]^3 \equiv \mathcal{C}^1(\mathbb{R} \times \mathbb{R}_+, \mathbb{R}_+) \times \mathcal{C}^1(\mathbb{R} \times \mathbb{R}_+, \mathbb{R}_+) \times \mathcal{C}^1(\mathbb{R} \times \mathbb{R}_+, \mathbb{R}_+)$. That is, $x = (U, V, W)^T = (S - S_0^*, E, I)^T \in [\mathcal{C}^1(\mathbb{R} \times \mathbb{R}_+, \mathbb{R}_+)]^3$.

(3.) For convenience, the notations “ $x(t)$ ” and “ x ” are commonly used interchangeable throughout this paper. To prevent “abuse” of notation, an explicit note will be made, whenever x refers to a point in the functions vector space, and it is used to define a Lyapunov functional.

Lemma 3.1. *Let $V_1 \in \mathcal{C}^{2,1}(\mathbb{R}^3 \times \mathbb{R}_+, \mathbb{R}_+)$, defined by*

$$\begin{aligned} V_1(x(t), t) &= (S(t) - S^* + E(t))^2 + c(E(t))^2 + (I(t))^2, \\ x(t) &= (S(t) - S^*, E(t), I(t))^T, \end{aligned} \tag{3.7}$$

where c is a positive constant. There exists two increasing positive real valued functions ϕ_1 , and ϕ_2 , such that V_1 satisfies the inequality

$$\phi_1(\|x(t)\|^2) \leq V_1(x(t), t) \leq \phi_2(\|x(t)\|^2). \tag{3.8}$$

Proof. Using the notations in $X(t) - E_0 = (U(t), V(t), W(t))$, observe that V_1 can be expressed as follows

$$V_1(x, t) = \left(\frac{c}{2+c}\right)U^2(t) + \left(\frac{1}{\sqrt{\frac{c+2}{2}}}U(t) + \sqrt{\frac{c+2}{2}}V(t)\right)^2 + \frac{c}{2}V^2(t) + W^2(t). \tag{3.9}$$

It is easy to see from (3.9) and using (1.14) that

$$V_1(x, t) \geq \left(\frac{c}{2+c}\right)\|X(t) - E_0\|^2 \equiv \phi_1(\|X(t) - E_0\|^2). \tag{3.10}$$

Also, from (3.9) it is easy to see that

$$V_1(x, t) \leq (2+c)\|X(t) - E_0\|^2 \equiv \phi_2(\|X(t) - E_0\|^2). \tag{3.11}$$

□

Note that Lemma 3.1, (3.8) signifies that the function $V(x, t)$ in (3.7) is positive definite and decrescent. This function will be used to create Lyapunov functionals, and also to examine stochastic stability in probability, of the equilibria of the system (1.8)–(1.11). See [67].

4. Stochastic stability in the absence of noise in the natural deathrate of all states

Recall Theorem 2.2(a) asserts that the unit ball $D(\infty)$ is a positive self-invariant space for the stochastic system (1.8)–(1.11), whenever the intensities $0 < \sigma_\beta < \infty$ and $\sigma_i = 0, i = S, E, I$. In this section, the stochastic stability of E_0 in $D(\infty)$ is investigated, whenever $0 < \sigma_\beta < \infty$ and $\sigma_i = 0, i = S, E, I, \beta$. The following result estimates the stochastic derivative of the Lyapunov function in (3.7) in $D(\infty)$, whenever $0 < \sigma_\beta < \infty$ and $\sigma_i = 0, i = S, E, I, \beta$.

Lemma 4.1. *Let the hypothesis of Theorem 2.2(a) be satisfied, i.e. $0 < \sigma_\beta < \infty$ and $\sigma_i = 0, i = S, E, I, \beta$. The differential operator [62, 63] applied to the Lyapunov function V_1 in (3.7) with respect to the system of stochastic differential equation (1.8)–(1.11) is given by*

$$\begin{aligned} dV_1(x, t) &= LV_1(x, t)dt - 2\sigma_S(U(t) + V(t))(S_0^* + U(t))dw_S(t) \\ &\quad - 2\sigma_E(U(t)V(t) + (c+1)V^2(t))dw_E(t) - 2\sigma_I W^2(t)dw_I(t) \\ &\quad - 2c\sigma_\beta(S_0^* + U(t))V(t) \int_{t_0}^{h_1} f_{T_1}(s)e^{-\mu v s}G(W(t-s))dsdw_\beta \\ &\quad - 2\sigma_E[U(t) + (c+1)V(t) + W(t)] \\ &\quad \times \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u)f_{T_1}(s)e^{-(\mu v s + \mu u)}(S_0^* + U(t-u))G(W(t-s-u))dsdudw_\beta(t), \end{aligned} \tag{4.1}$$

where for some positive valued function $\tilde{K}(\mu)$ that depends on μ , the drift part LV_1 of dV_1 in (4.1), satisfies the inequality

$$\begin{aligned} LV_1(x, t) &\leq (2\beta S_0^* + \beta + \alpha + 2\frac{\mu}{\tilde{K}(\mu)^2} - 2\mu)U^2(t) \\ &\quad + [2\mu\tilde{K}(\mu)^2 + \alpha + \beta(2S_0^* + 1) + c\beta(3S_0^* + 1) - 2(1+c)\mu] V^2(t) \\ &\quad + 2[\beta S_0^* - (\mu + d + \alpha)]W^2(t) \\ &\quad + 2\alpha \int_{t_0}^{\infty} f_{T_3}(r)e^{-2\mu r}W^2(t-r)dr \\ &\quad + [2\beta S_0^*(1+c) + \sigma_\beta^2(S_0^*)^2(4c+2(1-c)^2)] \int_{t_0}^{h_1} f_{T_1}(s)e^{-2\mu s}G^2(W(t-s))ds \\ &\quad + [\beta S_0^*(4+c) + \beta(S_0^*)^2(2+c) + \sigma_\beta^2(S_0^*)^2(4c+10)] \\ &\quad \times \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u)f_{T_1}(s)e^{-2\mu(s+u)}G^2(W(t-s-u))dsdu. \end{aligned} \tag{4.2}$$

Proof. The computation of the drift part LV (see the references [60, 62]) of the differential operator dV applied to the Lyapunov function V_1 in (3.7) with respect to the system of stochastic differential equation (1.8)–(1.11) gives the following:

$$LV_1(x, t)$$

$$\begin{aligned}
 &= -4\mu U(t)V(t) - 2\mu U^2(t) - 2(1+c)\mu V^2(t) - 2(\mu+d+\alpha)W^2(t) \\
 &\quad + 2\alpha(U(t)+V(t)) \int_{t_0}^{\infty} f_{T_3}(r)e^{-\mu r}W(t-r)dr \\
 &\quad + 2\beta[S_0^*U(t)+(1+c)S_0^*V(t)+cV(t)U(t)] \int_{t_0}^{h_1} f_{T_1}(s)e^{-\mu_v s}G(W(t-s))ds \\
 &\quad - 2\beta[U(t)+(1+c)V(t)-W(t)] \\
 &\quad \times \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u)f_{T_1}(s)e^{-(\mu_v s+\mu u)}(S_0^*+U(t-u))G(W(t-s-u))dsdu \\
 &\quad + \sigma_{\beta}^2 c(S_0^*+U(t))^2 \left(\int_{t_0}^{h_1} f_{T_1}(s)e^{-\mu_v s}G(W(t-s))ds \right)^2 \\
 &\quad + \sigma_{\beta}^2 (c+2) \left(\int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u)f_{T_1}(s)e^{-(\mu_v s+\mu u)}(S_0^*+U(t-u))G(W(t-s-u))dsdu \right)^2 \\
 &\quad + \sigma_{\beta}^2 (1-c)(S_0^*+U(t)) \left(\int_{t_0}^{h_1} f_{T_1}(s)e^{-\mu_v s}G(W(t-s))ds \right) \\
 &\quad \times \left(\int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u)f_{T_1}(s)e^{-(\mu_v s+\mu u)}(S_0^*+U(t-u))G(W(t-s-u))dsdu \right).
 \end{aligned} \tag{4.3}$$

Applying Theorem 2.2, *Cauchy – Schwarz*, *Hölder* inequalities, (8.1) and the following algebraic inequality

$$2ab \leq \frac{a^2}{g(c)} + b^2g(c), \tag{4.4}$$

where $a, b, c \in \mathbb{R}$, and the function g is such that $g(c) > 0$, to estimate the terms with integral signs in (4.3), one can see the following:

$$\begin{aligned}
 &2\alpha(U(t)+V(t)) \int_{t_0}^{\infty} f_{T_3}(r)e^{-\mu r}W(t-r)dr \\
 &\leq \alpha U^2(t) + \alpha V^2(t) + 2\alpha \int_{t_0}^{\infty} f_{T_3}(r)e^{-2\mu r}W^2(t-r)dr,
 \end{aligned} \tag{4.5}$$

$$\begin{aligned}
 &2\beta[S_0^*U(t)+(1+c)S_0^*V(t)+cV(t)U(t)] \int_{t_0}^{h_1} f_{T_1}(s)e^{-\mu s}G(W(t-s))ds \\
 &\leq \beta S_0^*U^2(t) + \beta S_0^*(1+2c)V^2(t) + 2\beta S_0^*(1+c) \int_{t_0}^{h_1} f_{T_1}(s)e^{-2\mu s}G^2(W(t-s))ds,
 \end{aligned} \tag{4.6}$$

$$\begin{aligned}
 &-2\beta[U(t)+(1+c)V(t)-W(t)] \\
 &\quad \times \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u)f_{T_1}(s)e^{-(\mu_v s+\mu u)}(S_0^*+U(t-u))G(W(t-s-u))dsdu \\
 &\leq \beta(S_0^*+1)U^2(t) + (1+c)\beta(S_0^*+1)V^2(t) + 2\beta S_0^*W^2(t) \\
 &\quad + [\beta S_0^*(4+c) + \beta(S_0^*)^2(2+c)] \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u)f_{T_1}(s)e^{-2\mu(s+u)}G^2(W(t-s-u))dsdu,
 \end{aligned} \tag{4.7}$$

$$\begin{aligned} & \sigma_\beta^2 c (S_0^* + U(t))^2 \left(\int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_v s} G(W(t-s)) ds \right)^2 \\ & \leq 4c \sigma_\beta^2 (S_0^*)^2 \int_{t_0}^{h_1} f_{T_1}(s) e^{-2\mu s} G^2(W(t-s)) ds, \end{aligned} \quad (4.8)$$

$$\begin{aligned} & \sigma_\beta^2 (c+2) \left(\int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-(\mu_v s + \mu u)} (S_0^* + U(t-u)) G(W(t-s-u)) ds du \right)^2 \\ & \leq 4(c+2) \sigma_\beta^2 (S_0^*)^2 \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-2\mu(s+u)} G^2(W(t-s-u)) ds du, \end{aligned} \quad (4.9)$$

$$\begin{aligned} & \sigma_\beta^2 (1-c) (S_0^* + U(t)) \left(\int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_v s} G(W(t-s)) ds \right) \\ & \quad \times \left(\int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-(\mu_v s + \mu u)} (S_0^* + U(t-u)) G(W(t-s-u)) ds du \right) \\ & \leq 2\sigma_\beta^2 (1-c)^2 (S_0^*)^2 \int_{t_0}^{h_1} f_{T_1}(s) e^{-2\mu s} G^2(W(t-s)) ds \\ & \quad + 2\sigma_\beta^2 (S_0^*)^2 \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-2\mu(s+u)} G^2(W(t-s-u)) ds du. \end{aligned} \quad (4.10)$$

The result (4.2) follows by applying (4.6)–(4.10) and the inequality (4.4) into (4.3). That is, $LV_1(x, t)$ becomes

$$\begin{aligned} & LV_1(x, t) \\ & \leq (2\beta S_0^* + \beta + \alpha + 2 \frac{\mu}{\tilde{K}(\mu)^2} - 2\mu) U^2(t) \\ & \quad + [2\mu \tilde{K}(\mu)^2 + \alpha + \beta(2S_0^* + 1) + c\beta(3S_0^* + 1) - 2(1+c)\mu] V^2(t) \\ & \quad + 2[\beta S_0^* - (\mu + d + \alpha)] W^2(t) \\ & \quad + 2\alpha \int_{t_0}^{\infty} f_{T_3}(r) e^{-2\mu r} W^2(t-r) dr \\ & \quad + [2\beta S_0^* (1+c) + \sigma_\beta^2 (S_0^*)^2 (4c + 2(1-c)^2)] \int_{t_0}^{h_1} f_{T_1}(s) e^{-2\mu s} G^2(W(t-s)) ds \\ & \quad + [\beta S_0^* (4+c) + \beta (S_0^*)^2 (2+c) + \sigma_\beta^2 (S_0^*)^2 (4c+10)] \\ & \quad \times \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-2\mu(s+u)} G^2(W(t-s-u)) ds du, \end{aligned} \quad (4.11)$$

where $\tilde{K}(\mu) = g(\mu)$ and g is defined in (4.4). \square

The following set of lemmas characterize the stochastic asymptotic stability of E_0 in the absence of noises from the natural death rates μ in the susceptible, exposed, infectious, and removal states. That is, whenever $0 < \sigma_\beta < \infty$ and $\sigma_i = 0, i = S, E, I, \beta$. In this case, the only source of variability is the disease transmission rate.

One other major objective of this study is to understand the role of the delays in the stochastic system on the stability of equilibria. This goal is not apparent when the delays $T_i, i = 1, 2, 3$ are distributed, and averaged in the system (1.8)–(1.11)(cf. [57]). Thus, the system (1.8)–(1.11) will be transformed into a finite constant delay system, using a centering density function for the random variables $T_i, i = 1, 2, 3$ in (1.8)–(1.11).

Assume that the incubation delays T_1 and T_2 , and the immunity delay period T_3 are constants and finite. This is equivalent to the special case of letting the probability density functions $f_{T_i}, i = 1, 2, 3$ of the random variables T_1, T_2 and T_3 in 8 (A) be the dirac-delta function*. That is,

$$f_{T_i}(s) = \delta(s - T_i) = \begin{cases} +\infty, s = T_i, \\ 0, \text{otherwise,} \end{cases}, i = 1, 2, 3. \tag{4.12}$$

Moreover, under the assumption that $T_1 \geq 0, T_2 \geq 0$ and $T_3 \geq 0$ are constant, the following expectations can be written as $E(e^{-2\mu(T_1+T_2)}) = e^{-2\mu(T_1+T_2)}, E(e^{-2\mu T_1}) = e^{-2\mu T_1}$ and $E(e^{-2\mu T_3}) = e^{-2\mu T_3}$.

Theorem 4.1. *Let the hypotheses of Theorem 2.2, Theorem 3.1[2.] and Lemma 4.1 be satisfied, where $0 < \sigma_\beta < \infty$ and $\sigma_i = 0, i = S, E, I$. Also, let T_1, T_2 and T_3 be constant positive values. There exists a Lyapunov functional*

$$V(x, t) = V_1(x, t) + V_{12}(x, t), \tag{4.13}$$

where $V_1 \in C^{2,1}(\mathbb{R}^3 \times \mathbb{R}_+, \mathbb{R}_+)$ is defined by (3.7), and the functional component V_{12} is defined over the functions vector space $V_{12} : [C^1(\mathbb{R}, \mathbb{R}_+)]^3 \rightarrow \mathbb{R}_+$, such that $x = (S - S^*, E, I) \rightarrow V_{12}(x, t) \in \mathbb{R}_+, \forall t \in \mathbb{R}_+$. Moreover, V_{12} is given as follows:

$$\begin{aligned} V_{12}(x, t) = & 2\alpha e^{-2\mu T_3} \int_{t-r}^t I^2(v) dv \\ & + [2\beta S_0^* (1 + c) + \sigma_\beta^2 (S_0^*)^2 (4c + 2(1 - c)^2)] e^{-2\mu T_1} \int_{t-s}^t G^2(I(v)) dv \\ & + [\beta S_0^* (4 + c) + \beta (S_0^*)^2 (2 + c) + \sigma_\beta^2 (S_0^*)^2 (4c + 10)] \\ & \times e^{-2\mu(T_1+T_2)} \int_{t-(T_1+T_2)}^t G^2(I(v)) dv. \end{aligned} \tag{4.14}$$

Furthermore, there exists threshold values R_1^*, R_0^*, U_0 and V_0 defined as follows:

$$R_1^* = 4R_0^* + \frac{\beta (S_0^*)^2}{(\mu + d + \alpha)} + \frac{\alpha}{(\mu + d + \alpha)} + 6 \frac{\sigma_\beta^2 (S_0^*)^2}{(\mu + d + \alpha)}, \tag{4.15}$$

$$R_0^* = \frac{\beta S_0^*}{(\mu + d + \alpha)}, \tag{4.16}$$

$$U_0 = \frac{2\beta S_0^* + \beta + \alpha + 2 \frac{\mu}{K(\mu)^2}}{2\mu}, \tag{4.17}$$

*Note that to minimize notations $T_i, i = 1, 2, 3$, are abusively used as the random variables and the single observations of the random variable.

and

$$V_0 = \frac{(2\mu\tilde{K}(\mu)^2 + \alpha + \beta(2S_0^* + 1))}{2\mu}, \tag{4.18}$$

and some positive constants ϕ , ψ , and φ , such that under the assumptions that $R_0^* < 1$, $U_0 \leq 1$, and $V_0 \leq 1$, and

$$T_{min} \geq \frac{1}{2\mu} \log \frac{R_1^*}{1 - R_0^*}, \tag{4.19}$$

where

$$T_{min} = \min(T_1, T_1 + T_2, T_3), \tag{4.20}$$

the drift part LV of the functional Itô differential operator (cf. [13]) dV applied to V with respect to the stochastic dynamic system (1.8)–(1.11) satisfies the following inequality:

$$LV(x, t) \leq -(\phi U^2(t) + \psi V^2(t) + \varphi W^2(t)). \tag{4.21}$$

In addition, the infection-free equilibrium E_0 of the stochastic dynamic system (1.8)–(1.11) is stochastically asymptotically stable in the large in the unit ball $D(\infty)$. Moreover, the steady state E_0 is exponentially mean square stable.

Proof. By applying the translation properties of the Dirac-Delata function (4.12), it can be seen from Lemma 4.1 that the drift part LV of the functional Itô differential operator (cf. [13]) dV applied to the Lyapunov functional defined in (4.13), (3.7) and (4.14) with respect to system (1.8)–(1.11) leads to the following:

$$\begin{aligned} LV(x, t) = & LV_1(x, t) \\ & + 2\alpha e^{-2\mu T_3} W^2(t) \\ & + [2\beta S_0^*(1 + c) + \sigma_\beta^2(S_0^*)^2(4c + 2(1 - c)^2)] e^{-2\mu T_1} G^2(W(t)) \\ & + [\beta S_0^*(4 + c) + \beta(S_0^*)^2(2 + c) + \sigma_\beta^2(S_0^*)^2(4c + 10)] e^{-2\mu(T_1+T_2)} G^2(W(t)) \\ & - 2\alpha e^{-2\mu T_3} W^2(t - T_3) \\ & - [2\beta S_0^*(1 + c) + \sigma_\beta^2(S_0^*)^2(4c + 2(1 - c)^2)] e^{-2\mu T_1} G^2(W(t - T_1)) \\ & - [\beta S_0^*(4 + c) + \beta(S_0^*)^2(2 + c) + \sigma_\beta^2(S_0^*)^2(4c + 10)] \\ & \times e^{-2\mu(T_1+T_2)} G^2(W(t - T_1 - T_2)). \end{aligned} \tag{4.22}$$

It follows that under the assumptions for $0 < \sigma_\beta < \infty$ and $\sigma_i = 0, i = S, E, I$ in Theorem 3.1[2.], and for some suitable choice of the positive constant c , it is easy to see from (4.2), (4.22), the statements of Assumption 1.1, A5 (i.e. $G^2(x) \leq x^2, x \geq 0$) and some further algebraic manipulations and simplifications that

$$LV(x, t) \leq -(\phi U^2(t) + \psi V^2(t) + \varphi W^2(t)), \tag{4.23}$$

where,

$$\phi = 2\mu(1 - U_0), \tag{4.24}$$

$$\psi = 2\mu(1 - V_0) - 2\mu c \left(1 - \frac{\beta(3S_0^* + 1) + \sigma_E^2}{2\mu}\right), \tag{4.25}$$

$$\varphi = 2(\mu + d + \alpha) - [2\beta S_0^* + \sigma_I^2 + 2\alpha e^{-2\mu T_3} + 2(\beta S_0^* + \sigma_\beta^2(S_0^*)^2) e^{-2\mu T_3}]$$

$$\begin{aligned}
 & + (4\beta S_0^* + 2\beta(S_0^*)^2 + 10\sigma_\beta^2(S_0^*)^2) e^{-2\mu(T_1+T_2)} - c(3\beta S_0^* + \beta(S_0^*)^2 + 4\sigma_\beta^2(S_0^*)^2) \\
 & - 2c^2\sigma_\beta^2(S_0^*)^2, \\
 \geq & 2(\mu + d + \alpha) [1 - R_0^* - R_1^* e^{-2\mu T_{max}}] - c(3\beta S_0^* + \beta(S_0^*)^2 + 4\sigma_\beta^2(S_0^*)^2) \\
 & - 2c^2\sigma_\beta^2(S_0^*)^2.
 \end{aligned} \tag{4.26}$$

and R_0^* and R_1^* are defined in (4.15)–(4.16). It is now easy to see that under the assumptions of R_0^* , R_1^* , U_0 , and V_0 in the hypothesis and also for a suitable choice of the positive constant c it follows that ϕ , ψ , and φ are positive constants and (4.21) follows immediately. Also, the stochastic stability results follows very easily by applying the comparison stability results (cf. [60, 67]). \square

The following result for the deterministic system (1.1)–(1.4) will be useful to compare and obtain insight about the influence of the noises and delays in the stochastic system (1.8)–(1.11), whenever the delays $T_i, i = 1, 2, 3$ in both systems are constant, and $\sigma_i = 0, i = S, E, I, R$, and $\sigma_\beta > 0$.

Theorem 4.2. *Let the hypotheses of Theorem 2.2, Theorem 3.1[1.] and Lemma 4.1 be satisfied. Also, let T_1, T_2 and T_3 be constant positive values. There exists a Lyapunov functional*

$$V(x, t) = V_1(x, t) + V_{13}(x, t), \tag{4.27}$$

where $V_1 \in C^{2,1}(\mathbb{R}^3 \times \mathbb{R}_+, \mathbb{R}_+)$ is defined by (3.7) and the functional component V_{13} is defined over the functions vector space $V_{13} : [C^1(\mathbb{R}, \mathbb{R}_+)]^3 \rightarrow \mathbb{R}_+$, such that $x = (S - S^*, E, I) \rightarrow V_{13}(x, t) \in \mathbb{R}_+$. Moreover, V_{13} is given as follows:

$$\begin{aligned}
 V_{13}(x, t) = & 2\alpha e^{-2\mu T_3} \int_{t-T_3}^t I^2(v) dv \\
 & + [2\beta S_0^* (1 + c)] e^{-2\mu T_1} \int_{t-T_1}^t G^2(I(v)) dv \\
 & + [\beta S_0^* (4 + c) + \beta(S_0^*)^2 (2 + c)] e^{-2\mu(T_1+T_2)} \int_{t-(T_1+T_2)}^t G^2(I(v)) dv.
 \end{aligned} \tag{4.28}$$

Furthermore, there exists threshold values \hat{R}_1^* , \hat{R}_0^* , \hat{U}_0 and \hat{V}_0 defined as follows:

$$\hat{R}_1^* = \frac{\beta S_0^* \hat{K}_0^* + \alpha}{(\mu + d + \alpha)}, \tag{4.29}$$

$$\hat{R}_0^* = \frac{\beta S_0^*}{(\mu + d + \alpha)}, \tag{4.30}$$

$$\hat{U}_0 = \frac{2\beta S_0^* + \beta + \alpha + 2\frac{\mu}{\tilde{K}(\mu)^2}}{2\mu}, \tag{4.31}$$

and

$$\hat{V}_0 = \frac{(2\mu \tilde{K}(\mu)^2 + \alpha + \beta(2S_0^* + 1))}{2\mu}, \tag{4.32}$$

and some positive constants ϕ , ψ , and φ , such that, under the assumptions that $\hat{R}_0^* < 1$, $\hat{U}_0 \leq 1$, and $\hat{V}_0 \leq 1$, and

$$T_{min} \geq \frac{1}{2\mu} \log \frac{\hat{R}_1^*}{1 - \hat{R}_0^*}, \tag{4.33}$$

where

$$T_{min} = \min(T_1, T_1 + T_2, T_3), \quad (4.34)$$

the deterministic differential operator \dot{V} applied to V with respect to the deterministic dynamic system (1.1)–(1.4) satisfies the following inequality:

$$\dot{V}(x, t) \leq -(\phi U^2(t) + \psi V^2(t) + \varphi W^2(t)). \quad (4.35)$$

Furthermore, the disease free steady state E_0 is globally uniformly asymptotically stable in $D(\infty)$. Moreover, it is exponentially stable.

Proof. (cf. [56]). □

Remark 4.1.

1. Given $E_0 = (S_0^*, 0, 0) = (1, 0, 0)$, it follows that (4.30) reduces to

$$\hat{R}_0^* = \frac{\beta S_0^*}{(\mu + d + \alpha)} = \frac{\beta}{(\mu + d + \alpha)}. \quad (4.36)$$

The parameter \hat{R}_0^* is called the basic reproduction number (BRN) for the vector-borne disease in the absence of any noise in the system. It is interpreted as the average number of secondary infected cases given by the term $\beta S_0^* = \beta$ that result from one infectious individual placed in a complete infection-free population, $E_0 = (1, 0, 0)$, over the effective average lifespan of an infectious individual given by $\frac{1}{(\mu + d + \alpha)}$. Note $\frac{1}{(\mu + d + \alpha)}$ is the effective average lifespan of a person in the population, where people can either die naturally at rate μ or die from the disease at the rate d , or recover from the disease at rate α .

Observe from (4.16) that when the intensity of noise in the system satisfies $0 < \sigma_\beta < \infty$, then the BRN in (4.36) is exactly the same BRN R_0^* in (4.16). In addition, observe that the other threshold values in Theorem 4.1, (4.15)–(4.18) and Theorem 4.2 (4.29)–(4.32) are also related as follows:

$$U_0 = k_1 \beta S_0^* \frac{1}{\mu} + \frac{1}{2} \alpha \frac{1}{\mu} + \frac{\mu}{\tilde{K}(\mu)^2} \frac{1}{\mu} = \hat{U}_0, \quad (4.37)$$

and

$$\begin{aligned} V_0 &= k_1 \beta S_0^* \frac{1}{\mu} + \frac{1}{2} \alpha \frac{1}{\mu} + \left(\mu \tilde{K}(\mu)^2 \right) \frac{1}{\mu} \\ &= \hat{V}_0 \end{aligned} \quad (4.38)$$

where $k_1 = 1 + \frac{1}{2S_0^*}$.

These observations suggests that the occurrence of noise in the disease transmission rate with intensity $0 < \sigma_\beta < \infty$, does not affect the BRN and some of the other threshold values for disease control or disease eradication.

2. Also, observe from Theorem 4.1 and Theorem 4.2, that when the noise intensity $0 < \sigma_\beta < \infty$, the delay threshold conditions in (4.19) and (4.33), satisfy

$$T_{min} \geq \frac{1}{2\mu} \log \frac{R_1^*}{1 - R_0^*} > \frac{1}{2\mu} \log \frac{\hat{R}_1^*}{1 - \hat{R}_0^*}. \quad (4.39)$$

The relation (4.39), suggests that, the intensity $0 < \sigma_\beta < \infty$ inflates the minimum threshold bounds for the delays in the system, required for the stability of E_0 , and hence for disease eradication.

Thus, from the items (1.) and (2.) above it is easy to see that increasing or decreasing the intensity $0 < \sigma_\beta < \infty$ of the noise in the disease transmission rate will not make much difference to the stability conditions of the infection-free steady state E_0 of the population.

3. The delay condition for stochastic stability of E_0 in (4.19) can be rewritten as follows

$$T_{\min} \geq \frac{1}{2\mu} \log \frac{R_1^*}{1 - R_0^*} = \frac{1}{\mu} \log \left(\frac{R_1^*}{1 - R_0^*} \right)^{\frac{1}{2}}. \tag{4.40}$$

The condition (4.40) signifies that E_0 is stochastically stable, if both the total incubation period $T_1 + T_2$ of the disease inside the vector and human, and the acquired immunity period T_3 of the disease are larger than the fraction $\log \left(\frac{R_1^*}{1 - R_0^*} \right)^{\frac{1}{2}}$ of the average lifespan $\frac{1}{\mu}$ of the human being in the population in the absence of disease. Since in practice $\frac{1}{\mu}$ is significantly larger than either of $T_1 + T_2$ and T_3 , it suffices that

$$0 < \log \left(\frac{R_1^*}{1 - R_0^*} \right)^{\frac{1}{2}} \ll 1. \tag{4.41}$$

4. Theorem 4.1 and Theorem 4.2 signify that in the absence of noise in the natural deathrate of all states (i.e $\sigma_i, i \in \{S, E, I, R\}$), the noise driven system (1.8)–(1.11), and the corresponding deterministic system (1.1)–(1.4) have the same infection-free steady state E_0 . Moreover, all trajectories for both systems that start near E_0 , remain near E_0 , for all time $t \geq t_0$, and almost surely converge to E_0 over sufficiently long time, whenever the threshold conditions for Theorem 4.1 and Theorem 4.2 are satisfied. That is, $1 \geq R_0^* = \hat{R}_0^*, 1 \geq U_0 = \hat{U}_0$ and $1 \geq V_0 = \hat{V}_0, 0 < \sigma_\beta < \infty$.

Since the relation (4.41) holds, whenever $0 < \sigma_\beta < \infty$, suggesting that the delay threshold parameters of the noise driven system are inflated by the intensity $0 < \sigma_\beta < \infty$, it is easy to see that certain magnitudes of the intensity, (i.e. $\exists \sigma_\beta \gg 1$), can lead to very large lower bounds for the delays in the delay threshold condition (4.19), and consequently the condition (4.19) is violated, and E_0 no longer stable.

In fact, using (4.41) and (4.15)–(4.18), a region denoted $D_0(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$, in the space for the intensities $(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4$, can be constructed, where the infection-free steady state E_0 , remains stable with respect to both systems (1.8)–(1.11) and (1.1)–(1.4). This region $D_0(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$ is defined in the following theorem.

Theorem 4.3. *Suppose the hypotheses of Theorem 4.1 and Theorem 4.2 hold. If $0 < \sigma_\beta < \infty$ and $\sigma_i = 0, i \in \{S, E, I, R\}$, then the noise driven system (1.8)–(1.11), and the corresponding deterministic system (1.1)–(1.4) have the same infection-free steady state E_0 . Moreover, all trajectories for both systems that start near E_0 , remain near E_0 , for all time $t \geq t_0$, and almost surely converge to E_0 over sufficiently long time, whenever the threshold conditions in Theorem 4.1 and Theorem 4.2 are satisfied. That is, whenever $1 \geq R_0^* = \hat{R}_0^*, 1 \geq U_0 = \hat{U}_0$ and $1 \geq V_0 = \hat{V}_0$, hold.*

In addition, there exists a region $D_0(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$ in the vector space $[0, \infty)^4$, defined as follows

$$D_0(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) = D_0^{S,E,I}(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \cap D_0^\beta(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta), \tag{4.42}$$

where

$$D_0^{S,E,I}(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) = \{(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 \mid \sigma_i = 0, i = S, E, I\}, \tag{4.43}$$

and

$$\begin{aligned} & D_0^\beta(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \\ &= \left\{ (\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 \mid \frac{1}{6} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) (1 - \hat{R}_0^*) \right. \\ &\quad \left. - \frac{1}{6} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \left(4\hat{R}_0^* + \frac{\beta (S_0^*)^2}{(\mu + d + \alpha)} + \frac{\alpha}{(\mu + d + \alpha)} \right) \right. \\ &\quad \left. < \sigma_\beta^2 \ll e^2 \frac{1}{6} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) (1 - \hat{R}_0^*) \right. \\ &\quad \left. - \frac{1}{6} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \left(4\hat{R}_0^* + \frac{\beta (S_0^*)^2}{(\mu + d + \alpha)} + \frac{\alpha}{(\mu + d + \alpha)} \right) \right\}. \tag{4.44} \end{aligned}$$

And it follows that for any $(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in D_0(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$, the stability conditions for E_0 in Theorem 4.2, given by $\hat{R}_0^* \equiv R_0^* \leq 1, \hat{U}_0 \equiv U_0 \leq 1$ and $\hat{V}_0 \equiv V_0 \leq 1$, remain valid for the stochastic stability of E_0 characterized in Theorem 4.1.

In other words, for any intensities $(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in D_0(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$, the stability conditions for E_0 , in the absence of noise in the system, characterized in Theorem 4.2, and given by $\hat{R}_0^* \equiv R_0^* \leq 1, \hat{U}_0 \equiv U_0 \leq 1$ and $\hat{V}_0 \equiv V_0 \leq 1$, remain valid for the stochastic stability of E_0 characterized in Theorem 4.1.

Proof. The result follow by applying simple algebraic manipulations using (4.41) and (4.15)–(4.18), to find bounds for $0 < \sigma_\beta < \infty$. \square

5. Stochastic stability in the absence of noise exclusively in the natural deathrate of the susceptible state

Recall Theorem 2.2(b) asserts that when at least one of $0 < \sigma_i < \infty, i = E, I, R, S$, then the unit ball $D(\infty)$ is no longer a positive self-invariant set for the stochastic system (1.8)–(1.11). Thus, the unique positive stochastic solution for (1.8)–(1.11) lies in $X(t) \in \mathbb{R}_+^3$. Also recall Theorem 3.1 (2.) asserts that when $\sigma_S = 0$ and $0 < \sigma_i < \infty, i = E, I, R$, then the infection-free steady state E_0 exists, and it is exactly the same infection-free steady state for the deterministic system (1.1)–(1.4). The following result estimates the stochastic derivative of the Lyapunov function in (3.7) in \mathbb{R}_+^3 , whenever $0 < \sigma_i < \infty, i = S, E, I, R$. The supremum G^* in (3.5) is used.

Lemma 5.1. *Let the hypothesis of Theorem 2.2(b) be satisfied. That is, $0 < \sigma_i < \infty, i = S, E, I, R$. The differential operator [62, 63] applied to the Lyapunov function*

V_1 in (3.7) with respect to the system of stochastic differential equation (1.8)–(1.11) is given by

$$\begin{aligned}
 dV_1 = & LV_1 dt - 2\sigma_S(U(t) + V(t))(S_0^* + U(t))dw_S(t) \\
 & - 2\sigma_E(U(t)V(t) + (c + 1)V^2(t))dw_E(t) - 2\sigma_I W^2(t)dw_I(t) \\
 & - 2c\sigma_\beta(S_0^* + U(t))V(t) \int_{t_0}^{h_1} f_{T_1}(s)e^{-\mu v s}G(W(t - s))dsdw_\beta \\
 & - 2\sigma_E[U(t) + (c + 1)V(t) + W(t)] \\
 & \times \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u)f_{T_1}(s)e^{-(\mu v s + \mu u)}(S_0^* + U(t - u))G(W(t - s - u))dsdudw_\beta(t),
 \end{aligned} \tag{5.1}$$

where for some positive valued function $\tilde{K}(\mu)$ that depends on μ , the drift part $L\tilde{V}_1$ of dV_1 in (5.1), satisfies the inequality

$$\begin{aligned}
 L\tilde{V}_1(x, t) \leq & \left(\beta S_0^* + \beta + \alpha + 2\frac{\mu}{\tilde{K}(\mu)^2} + c\beta(G^*)E(e^{-2\mu v T_1}) \right. \\
 & \left. + \sigma_\beta^2 [2 + (1 - c)^2] (G^*)E(e^{-2\mu v T_1}) - 2\mu \right) U^2(t) \\
 & + [2\mu\tilde{K}(\mu)^2 + \alpha + (1 + c)\beta S_0^* + (1 + 2c)\beta - 2(1 + c)\mu] V^2(t) \\
 & + [\beta S_0^* - 2(\mu + d + \alpha)] W^2(t) \\
 & + 2\alpha \int_{t_0}^\infty f_{T_3}(r)e^{-2\mu r}W^2(t - r)dr \\
 & + [\beta S_0^*(2 + c) + \sigma_\beta^2 S_0^*(1 - c)^2] \int_{t_0}^{h_1} f_{T_1}(s)e^{-2\mu s}G^2(W(t - s))ds \\
 & + \left[2\beta(S_0^*)^2 \left(2 + \frac{1}{S_0^*} + c \right) + \sigma_\beta^2(S_0^*)^2 + \sigma_\beta^2(c + 2) \right] \\
 & \times \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u)f_{T_1}(s)e^{-2\mu(s+u)}G^2(W(t - s - u))dsdu \\
 & + \left[2 \left(2 + \frac{1}{S_0^*} + c \right) \beta(G^*)^2 E(e^{-2\mu T_1}) + (2c + 5)\sigma_\beta^2(G^*)^2 E(e^{-2\mu T_1}) \right] \\
 & \times \int_{t_0}^{h_2} f_{T_2}(u)e^{-2\mu u}U^2(t - u)du \\
 & + \sigma_S^2(S_0^* + U(t))^2 + \sigma_E^2(c + 1)V^2(t) + \sigma_I^2W^2(t).
 \end{aligned} \tag{5.2}$$

Proof. When $0 < \sigma_i < \infty, i = S, E, I, R$, the drift part LV (cf. [60, 62]) of the differential operator dV applied to the Lyapunov function V_1 in (3.7) with respect to the system of stochastic differential equation (1.8)–(1.11) is denoted $L\tilde{V}_1$, and is given by

$$L\tilde{V}_1(x, t) = LV_1 + \sigma_S^2(S_0^* + U(t))^2 + \sigma_E^2(c + 1)V^2(t) + \sigma_I^2W^2(t), \tag{5.3}$$

where LV_1 is given in (4.3) .

In \mathbb{R}_+^3 , we consider a different estimation of (4.3) as follows. Applying Theorem 2.2, *Cauchy–Schwarz*, *Hölder* inequalities, (8.1), and the algebraic inequality (4.4) to estimate the terms with integral signs in (4.3), one can see the following:

$$\begin{aligned} & 2\alpha(U(t) + V(t)) \int_{t_0}^{\infty} f_{T_3}(r) e^{-\mu r} W(t-r) dr \\ & \leq \alpha U^2(t) + \alpha V^2(t) + 2\alpha \int_{t_0}^{\infty} f_{T_3}(r) e^{-2\mu r} W^2(t-r) dr, \end{aligned} \quad (5.4)$$

$$\begin{aligned} & 2\beta [S_0^* U(t) + (1+c)S_0^* V(t) + cV(t)U(t)] \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_v s} G(W(t-s)) ds \\ & \leq [\beta S_0^* + c\beta(G^*)^2 E(e^{-2\mu T_1})] U^2(t) + [\beta S_0^*(1+c) + c\beta] V^2(t) \\ & \quad + (2+c)\beta S_0^* \int_{t_0}^{h_1} f_{T_1}(s) e^{-2\mu s} G^2(W(t-s)) ds, \end{aligned} \quad (5.5)$$

$$\begin{aligned} & -2\beta [U(t) + (1+c)V(t) - W(t)] \\ & \quad \times \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-(\mu_v s + \mu u)} (S_0^* + U(t-u)) G(W(t-s-u)) ds du \\ & \leq \beta U^2(t) + (1+c)\beta V^2(t) + \beta S_0^* W^2(t) \\ & \quad + \left[2 \left(2 + \frac{1}{S_0^*} + c \right) \beta (S_0^*)^2 \right] \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-2\mu(s+u)} G^2(W(t-s-u)) ds du \\ & \quad + 2 \left(2 + \frac{1}{S_0^*} + c \right) \beta (G^*)^2 E(e^{-2\mu T_1}) \int_{t_0}^{h_2} f_{T_2}(u) e^{-2\mu u} U^2(t-u) du, \end{aligned} \quad (5.6)$$

$$\begin{aligned} & \sigma_{\beta}^2 c (S_0^* + U(t))^2 \left(\int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_v s} G(W(t-s)) ds \right)^2 \\ & \leq 2c\sigma_{\beta}^2 (S_0^*)^2 \int_{t_0}^{h_1} f_{T_1}(s) e^{-2\mu s} G^2(W(t-s)) ds + 2c\sigma_{\beta}^2 (G^*)^2 E(e^{-2\mu T_1}) U^2(t), \end{aligned} \quad (5.7)$$

$$\begin{aligned} & \sigma_{\beta}^2 (c+2) \left(\int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-(\mu_v s + \mu u)} (S_0^* + U(t-u)) G(W(t-s-u)) ds du \right)^2 \\ & \leq 2(c+2)\sigma_{\beta}^2 (S_0^*)^2 \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-2\mu(s+u)} G^2(W(t-s-u)) ds du \\ & \quad + 2(c+2)\sigma_{\beta}^2 (G^*)^2 E(e^{-2\mu T_1}) \int_{t_0}^{h_2} f_{T_2}(u) e^{-2\mu u} U^2(t-u) du, \end{aligned} \quad (5.8)$$

$$\sigma_{\beta}^2 (1-c) (S_0^* + U(t)) \left(\int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu_v s} G(W(t-s)) ds \right)$$

$$\begin{aligned}
 & \times \left(\int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-(\mu_v s + \mu u)} (S_0^* + U(t - u)) G(W(t - s - u)) ds du \right) \\
 & \leq \sigma_\beta^2 (1 - c)^2 (S_0^*)^2 \int_{t_0}^{h_1} f_{T_1}(s) e^{-2\mu s} G^2(W(t - s)) ds \\
 & \quad + \sigma_\beta^2 (S_0^*)^2 \int_{t_0}^{h_2} \int_{t_0}^{h_1} f_{T_2}(u) f_{T_1}(s) e^{-2\mu(s+u)} G^2(W(t - s - u)) ds du \\
 & \quad + \sigma_\beta^2 (1 - c)^2 (G^*)^2 E(e^{-2\mu T_1}) U^2(t) \\
 & \quad + \sigma_\beta^2 (1 - c)^2 (G^*)^2 E(e^{-2\mu T_1}) \int_{t_0}^{h_2} f_{T_2}(u) e^{-2\mu u} U^2(t - u) du. \tag{5.9}
 \end{aligned}$$

The result (5.2) follows by combining (5.4)–(5.9), followed by applying the inequality (4.4). \square

The following result characterizes the stochastic stability of E_0 , whenever only the intensity of the noise from the natural death rate of the susceptible state is zero, that is, $\sigma_S = 0$, and $0 < \sigma_i < \infty, i \in \{E, I, \beta\}$. To emphasize the role of the delays on the stability of E_0 , the random variables $T_i, i = 1, 2, 3$ are assumed to have the density functions (4.12). That is, the delays $T_i, i = 1, 2, 3$ are constant. Theorem 5.1 presents the stochastic stability results for the case of constant and finite constant delays in the system.

Theorem 5.1. *Let the hypotheses of Theorem 2.2, Theorem 3.1[2.] and Lemma 5.1 be satisfied, where $0 < \sigma_i < \infty, i = E, I, \beta$, and $\sigma_S = 0$. Also, let T_1, T_2 and T_3 be constant delay values. There exists a Lyapunov functional*

$$\tilde{V}(x, t) = V_1(x, t) + \tilde{V}_{12}(x, t), \tag{5.10}$$

where $V_1 \in \mathcal{C}^{2,1}(\mathbb{R}^3 \times \mathbb{R}_+, \mathbb{R}_+)$ is defined by (3.7) and the functional component \tilde{V}_{12} is defined over the functions vector space $\tilde{V}_{12} : [\mathcal{C}^1(\mathbb{R}, \mathbb{R}_+)]^3 \rightarrow \mathbb{R}_+$, such that $x = (S - S^*, E, I) \rightarrow \tilde{V}_{12}(x, t) \in \mathbb{R}_+$. Moreover, \tilde{V}_{12} is given as follows:

$$\begin{aligned}
 & \tilde{V}_{12}(x, t) \\
 & = 2\alpha e^{-2\mu T_3} \int_{t-r}^t I^2(v) dv \\
 & \quad + \left[\beta S_0^* (2 + c) + \sigma_\beta^2 (S_0^*)^2 (1 - c)^2 \right] e^{-2\mu T_1} \int_{t-T_1}^t G^2(I(v)) dv \\
 & \quad + \left[2 \left(1 + \frac{1}{S_0^*} + c \right) \beta (S_0^*)^2 + 4\sigma_\beta^2 (c + 2) (S_0^*)^2 + \sigma_\beta^2 (S_0^*)^2 \right] \\
 & \quad \times e^{-2\mu(T_1+T_2)} \int_{t-(T_1+T_2)}^t G^2(I(v)) dv \\
 & \quad + \left[2 \left(1 + \frac{1}{S_0^*} + c \right) \beta (G^*)^2 + \sigma_\beta^2 (G^*)^2 (2c + 5) \right] e^{-2\mu(T_1+T_2)} \int_{t-T_2}^t U^2(v) dv. \tag{5.11}
 \end{aligned}$$

Furthermore, let the threshold values $R_0^* \equiv \hat{R}_0^*$, $U_0 \equiv \hat{U}_0$ and $V_0 \equiv \hat{V}_0$ be as defined in (4.15)–(4.18) and (4.29)–(4.32). There exists threshold values R_1, R_0 and U_1

defined as follows:

$$R_1 = \hat{R}_0^* + \left(2 + \frac{1}{S_0^*}\right) \frac{\beta (S_0^*)^2}{(\mu + d + \alpha)} + \frac{\alpha}{(\mu + d + \alpha)} + 9 \frac{\sigma_\beta^2 (S_0^*)^2}{(\mu + d + \alpha)}, \tag{5.12}$$

$$R_0 = \frac{\beta S_0^*}{(\mu + d + \alpha)} + \frac{\frac{1}{2}\sigma_I^2}{(\mu + d + \alpha)} = \hat{R}_0^* + \frac{\frac{1}{2}\sigma_I^2}{(\mu + d + \alpha)}, \tag{5.13}$$

$$U_1 = \frac{\left(2 + \frac{1}{S_0^*}\right) \beta (G^*)^2 + 4\sigma_\beta^2 (G^*)^2}{\mu}, \tag{5.14}$$

$$V_0^* = V_0 + \frac{1}{2} \frac{\sigma_E^2}{\mu}, \tag{5.15}$$

and some positive constants $\phi_1, \psi_1,$ and $\varphi_1,$ such that under the assumptions that $R_0 < 1, U_0 < 1,$ and $V_0^* \leq 1,$ and

$$T_{\min} \geq \frac{1}{2\mu} \log \frac{R_1}{1 - R_0}, \tag{5.16}$$

and

$$T_1 \geq \frac{1}{2\mu} \log \frac{U_1}{1 - U_0}, \tag{5.17}$$

where

$$T_{min} = \min (T_1, T_1 + T_2, T_3), \tag{5.18}$$

the drift part $L\tilde{V}$ of the functional Itô differential operator (cf. [13]) dV applied to V with respect to the stochastic dynamic system (1.8)–(1.11) satisfies the following inequality:

$$L\tilde{V}(x, t) \leq -(\phi_1 U^2(t) + \psi_1 V^2(t) + \varphi_1 W^2(t)). \tag{5.19}$$

In addition, the infection-free equilibrium E_0 of the stochastic dynamic system (1.8)–(1.11) is stochastically asymptotically stable in the large in $\mathbb{R}_+^3.$ Moreover, the steady state E_0 is exponentially mean square stable.

Proof. By applying the translation properties of the Dirac-Delta function (4.12), it can be seen from Lemma 4.1 that the drift part $L\tilde{V}$ of the functional Itô differential operator (cf. [13]) dV applied to the Lyapunov functional defined in (5.10), (3.7) and (5.11), with respect to the system (1.8)–(1.11), leads to the following:

$$\begin{aligned} &L\tilde{V}(x, t) \\ = &L\tilde{V}_1(x, t) + 2\alpha e^{-2\mu T_3} W^2(t) \\ &+ \left[\beta S_0^*(2 + c) + \sigma_\beta^2 (S_0^*)^2 (1 - c)^2\right] e^{-2\mu T_1} G^2(W(t)) \\ &+ \left[2\left(1 + \frac{1}{S_0^*} + c\right) \beta (S_0^*)^2 + 4\sigma_\beta^2 (c + 2) (S_0^*)^2 + \sigma_\beta^2 (S_0^*)^2\right] e^{-2\mu(T_1 + T_2)} G^2(W(t)) \\ &+ \left[2\left(1 + \frac{1}{S_0^*} + c\right) \beta (G^*)^2 + \sigma_\beta^2 (G^*)^2 (2c + 5)\right] e^{-2\mu(T_1 + T_2)} U^2(t) \\ &- 2\alpha e^{-2\mu T_3} W^2(t - T_3) \\ &- \left[\beta S_0^*(2 + c) + \sigma_\beta^2 (S_0^*)^2 (1 - c)^2\right] e^{-2\mu T_1} G^2(W(t - T_1)) \\ &- \left[2\left(1 + \frac{1}{S_0^*} + c\right) \beta (S_0^*)^2 + 4\sigma_\beta^2 (c + 2) (S_0^*)^2 + \sigma_\beta^2 (S_0^*)^2\right] \end{aligned}$$

$$\begin{aligned} & \times e^{-2\mu(T_1+T_2)} G^2(W(t-T_1-T_2)) \\ & - \left[2 \left(1 + \frac{1}{S_0^*} + c \right) \beta (G^*)^2 + \sigma_\beta^2 (G^*)^2 (2c + 5) \right] e^{-2\mu(T_1+T_2)} U^2(t-T_2). \end{aligned} \quad (5.20)$$

It follows that under the assumptions for $0 < \sigma_i < \infty, i = E, I, \beta$, and $\sigma_S = 0$ in Theorem 3.1[2.], and for some suitable choice of the positive constant c , it is easy to see from (4.3), (5.20), the statements of Assumption 1.1, A5 (i.e. $G^2(x) \leq x^2, x \geq 0$) and some further algebraic manipulations and simplifications that

$$L\tilde{V}(x, t) \leq -(\phi_1 U^2(t) + \psi_1 V^2(t) + \varphi_1 W^2(t)), \quad (5.21)$$

where,

$$\begin{aligned} \phi_1 = & 2\mu - \left(\beta S_0^* + \beta + \alpha + 2 \frac{\mu}{\tilde{K}(\mu)^2} \right) \\ & - \left[3\sigma_\beta^2 (G^*)^2 + c\beta (G^*)^2 + \sigma_\beta^2 (G^*)^2 (c^2 - 2c) \right] e^{-2\mu T_1} \\ & - \left[2 \left(1 + \frac{1}{S_0^*} + c \right) \beta (G^*)^2 + 5\sigma_\beta^2 (G^*)^2 + 2c \left(\beta (G^*)^2 + \sigma_\beta^2 (G^*)^2 \right) \right] e^{-2\mu(T_1+T_2)} \\ \geq & 2\mu [(1 - U_0) - U_1 e^{-2\mu T_1}] - 2c [\beta (S_0^*)^2 + \sigma_\beta^2 (G^*)^2] e^{-2\mu(T_1+T_2)} \\ & - \left[c\beta (G^*)^2 + \sigma_\beta^2 (G^*)^2 (c^2 - 2c) \right] e^{-2\mu T_1}, \end{aligned} \quad (5.22)$$

$$\begin{aligned} \psi_1 = & 2\mu - \left[2\mu \tilde{K}(\mu)^2 + \alpha + \beta S_0^* + \beta + \sigma_E^2 \right] \\ & + c [2\mu - (2\beta + \beta S_0^* + \sigma_E^2)] \\ \geq & 2\mu [1 - V_0] + 2\mu c \left[1 - \frac{(2\beta + \beta S_0^* + \sigma_E^2)}{2\mu} \right], \end{aligned} \quad (5.23)$$

$$\begin{aligned} \varphi_1 = & 2(\mu + d + \alpha) - (\beta S_0^* + \sigma_I^2) - 2\alpha e^{-2\mu T_3} - [2\beta S_0^* + \sigma_\beta^2 S_0^*] e^{-2\mu T_1} \\ & - \left[2 \left(2 + \frac{1}{S_0^*} \right) \beta (S_0^*)^2 + 9\sigma_\beta^2 (S_0^*)^2 \right] e^{-2\mu(T_1+T_2)} \\ & - [c\beta S_0^* + (c^2 - 2c) \sigma_\beta^2 (S_0^*)] e^{-2\mu T_1} - c [2\beta (S_0^*)^2 + 4\sigma_\beta^2 (S_0^*)^2] e^{-2\mu(T_1+T_2)} \\ \geq & 2(\mu + d + \alpha) [1 - R_0 - R_1 e^{-2\mu T_{min}}] \\ & - [c\beta S_0^* + (c^2 - 2c) \sigma_\beta^2 (S_0^*)] e^{-2\mu T_1} - c [2\beta (S_0^*)^2 + 4\sigma_\beta^2 (S_0^*)^2] e^{-2\mu(T_1+T_2)}. \end{aligned} \quad (5.24)$$

It is now easy to see that under the assumptions of R_0, R_1, U_0 , and V_0^* in the hypothesis, i.e. $R_0 < 1, U_0 < 1$, and $V_0^* \leq 1$, and also for a suitable choice of the positive constant c , it follows that ϕ_1, ψ_1 , and φ_1 are positive constants and (5.18) follows immediately. Also, the stochastic stability result follows very easily by applying the comparison stability results (cf. [60, 67]). \square

Remark 5.1.

1. It follows from (5.13) that when $0 < \sigma_i < \infty, i \in \{E, I, R, \beta\}$ and $\sigma_S = 0$, the BRN in Theorem 4.2, for the deterministic system (1.1)–(1.4) is modified by the

intensity of the intensity of the noise in the natural death rate of the infectious state σ_I . Clearly from (5.13), (4.36) and (4.16), it is easy to see that

$$R_0 \geq R_0^* \equiv \hat{R}_0^*. \quad (5.25)$$

Also, from (5.15) and (4.18), observe that $V_0^* \geq V_0 \equiv \hat{V}_0$, whenever $\sigma_i > 0, i \in \{E, I, R, \beta\}$. These observations suggest that the occurrence of noise in the disease dynamics with significant magnitudes of the intensities $0 < \sigma_i < \infty, i \in \{E, I, R, \beta\}$, inflate the BRN and the threshold values (i) for the stability of the infection-free steady state E_0 , and hence (ii) for disease control, or disease eradication. The inflation of disease control parameters by the noise terms can hinder disease control conditions as explained further below.

2. Theorem 5.1 and Theorem 4.2 signify that in the absence of noise in the natural deathrate of the susceptible state (i.e. $\sigma_S = 0$), the noise driven system (1.8)–(1.11) and the noise-free system (1.1)–(1.4) have the same infection-free steady state E_0 . Moreover, all trajectories of the systems that start near E_0 , tend to remain near E_0 for all time $t \geq t_0$, and almost surely converge to E_0 , over sufficiently long time, whenever the threshold conditions for Theorem 5.1 and Theorem 4.2 are satisfied, i.e. , whenever the following hold $1 > R_0 > \hat{R}_0^*$, $1 > U_0 = \hat{U}_0$, $1 \geq V_0^* > \hat{V}_0 = V_0$, and $0 < \sigma_i < \infty, \forall i = E, I, R, \beta$.

Since the threshold values are related as follows: $R_0 > \hat{R}_0^*$, $U_0 = \hat{U}_0$ and $V_0^* > \hat{V}_0 = V_0$ hold, whenever $0 < \sigma_i < \infty, i \in \{E, I, R, \beta\}$, it is easy to see that certain magnitudes of the intensities, (i.e. $\exists \sigma_i \gg 1, \forall i = E, I, \beta$), can lead to violation of at least one of the following stability conditions for E_0 : $1 > R_0, 1 > U_0$ and $1 \geq V_0$. Thus, it is necessary to characterize a parameter region for the intensities of the noises in the system, $0 < \sigma_i < \infty, \forall i = E, I, R, \beta$, and $\sigma_S = 0$, in which the infection-free steady state E_0 is stochastically stable, and the disease can be eradicated.

3. The delay conditions in (5.16)–(5.17), for the stochastic stability of E_0 , can be written as follows

$$T_{\min} \geq \max \left\{ \frac{1}{\mu} \log \left\{ \frac{R_1}{1 - R_0} \right\}^{\frac{1}{2}}, \frac{1}{\mu} \log \left(\frac{U_1}{1 - U_0} \right)^{\frac{1}{2}} \right\}. \quad (5.26)$$

Applying similar argument in Remark 4.1[3.-4.], it follows that from a practical point of view, the condition (5.26) requires that

$$0 < \log \left(\frac{R_1}{1 - R_0} \right)^{\frac{1}{2}} \ll 1, \quad \text{and} \quad 0 < \log \left(\frac{U_1}{1 - U_0} \right)^{\frac{1}{2}} \ll 1. \quad (5.27)$$

6. Effect of increasing white noise intensity on the stability of the infection-free steady state

From Remark 5.1 , it is apparent that when we are given the threshold conditions $R_0 < 1, U_0 < 1$, and $V_0^* \leq 1$ hold in Theorem 5.1, the steady state E_0 is stochastically stable in the large, for some magnitudes of the intensities $0 < \sigma_i < \infty, \forall i = E, I, R, \beta$, and may become unstable for other values of $0 < \sigma_i < \infty, \forall i = E, I, R, \beta$.

Recall, the behavior of the trajectories of the deterministic system (1.1)–(1.4), near the infection-free steady state E_0 is characterized in Theorem 4.2. The question

of the extend that the white noises, introduced into the ideal deterministic disease dynamics (1.1)–(1.4), influence the behavior of the paths of the ensuing stochastic system (1.8)–(1.11) near E_0 is answered in this section. That is, the question about how much the stability conditions in Theorem 4.2, are affected by the noises introduced into (1.1)–(1.4), is answered in this section.

Using the stability conditions in Theorem 5.1 and Theorem 4.2, a noise intensity region, denoted by $D_1(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$ is determined for the stochastic system (1.8)–(1.11), in which E_0 stable with respect to the deterministic system (1.1)–(1.4), remains stable with respect to the ensuing stochastic system (1.8)–(1.11).

Theorem 6.1. *Suppose the conditions of Theorem 4.2 are satisfied. That is, $\hat{R}_0^* < 1, \hat{U}_0 < 1$ and $\hat{V}_0 \leq 1$, where \hat{R}_0^*, \hat{U}_0 , and \hat{V}_0 are given Theorem 4.2, (4.29)–(4.32). Define the following sets*

$$D_1^S(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) = \{(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 | \sigma_S = 0\}, \tag{6.1}$$

$$D_1^E(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) = \{(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 | 0 < \sigma_E^2 \leq 2(\mu)(1 - \hat{V}_0)\}, \tag{6.2}$$

$$D_1^I(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) = \{(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 | 0 < \sigma_I^2 \leq 2(\mu + d + \alpha)(1 - \hat{R}_0^*)\}, \tag{6.3}$$

and

$$\begin{aligned} & D_1^\beta(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \\ &= \left\{ (\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 \left| \frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha)(1 - \hat{R}_0^*) \right. \right. \\ & \quad \left. \left. - \frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \left[\hat{R}_0^* + \left(2 + \frac{1}{S_0^*} \right) \frac{\beta(S_0^*)^2}{(\mu + d + \alpha)} + \frac{\alpha}{(\mu + d + \alpha)} \right] \right. \right. \\ & \quad \left. \left. - \frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \frac{\frac{1}{2} \sigma_I^2}{(\mu + d + \alpha)} < \sigma_\beta^2 < e^2 \frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha)(1 - \hat{R}_0^*) \right. \right. \\ & \quad \left. \left. - \frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \left[\hat{R}_0^* + \left(2 + \frac{1}{S_0^*} \right) \frac{\beta(S_0^*)^2}{(\mu + d + \alpha)} + \frac{\alpha}{(\mu + d + \alpha)} \right] \right. \right. \\ & \quad \left. \left. - e^2 \frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \frac{\frac{1}{2} \sigma_I^2}{(\mu + d + \alpha)} \right\}. \tag{6.4} \end{aligned}$$

Furthermore, using (6.1)–(6.4), let the region $D_1(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$ be as defined below

$$\begin{aligned} & D_1(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \\ &= D_1^S(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \cap D_1^E(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \cap D_1^I(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \cap D_1^\beta(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta). \tag{6.5} \end{aligned}$$

It follows that the infection-free equilibrium E_0 is both uniformly asymptotically stable with respect to the deterministic system (1.1)–(1.4), and stochastically asymptotically stable in the large, with respect to the stochastic system (1.8)–(1.11), regardless of the source of the noise in the system (1.8)–(1.11), provided that the intensities lie in $(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in D_1(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$.

Proof. Suppose $\hat{R}_0^* < 1, \hat{U}_0 < 1$, and $\hat{V}_0 \leq 1$, where \hat{R}_0^*, \hat{U}_0 , and \hat{V}_0 are given in (4.29)–(4.32). It is easy to see that for $(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in D_1(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$, and (5.27) satisfied, then Theorem 5.1 is also satisfied, and E_0 is stochastically stable in the large in \mathbb{R}_+^3 . \square

Remark 6.1. Theorem 6.1 signifies that the when there is no noise from the natural deathrate of the susceptible state (i.e. $\sigma_S = 0$), the infection-free steady state E_0 continues to exists, regardless of fluctuations from the other sources: natural deathrates of exposed, infectious, removed states, and the disease transmission rate. Moreover, if the intensities of the noises from these sources lie in $(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in D_1(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$, then the disease eradication conditions (i.e. $\hat{R}_0^* < 1$, $\hat{U}_0 \leq 1$, and $\hat{V}_0 \leq 1$) required for the deterministic system (i.e. (1.1)–(1.4)), are still valid for the stochastic dynamics (1.8)–(1.11).

7. Asymptotic behavior of the stochastic system when there is no infection-free steady state

Recall Theorem 3.1[3.] asserts that the stochastic system (1.8)–(1.11) has no infection free steady state for the population, whenever the intensity of the noise in the natural death rate of the susceptible state is positive, i.e. $\sigma_S > 0$. The question remains about the extend that the intensity $\sigma_S > 0$ deviates the paths of the ensuing system (1.8)–(1.11) from the steady state E_0 characterized in Theorem 6.1.

Since the stochastic system has a unique stochastic solution process $X(t) \in \mathbb{R}_+^3, t \geq t_0$, whenever at least one of $\sigma_i > 0, i = S, E, I, R$ (see Theorem 2.2), and the solution has continuous sample paths which are nowhere differentiable, the question above is answered by applying the mean value theorem to examine the average distance of every path of the stochastic solution from the steady state E_0 over sufficiently long time.

Theorem 7.1. *Let the hypothesis of Theorem 3.1[3.] be satisfied. Let $\hat{R}_1^*, \hat{R}_0^*, \hat{U}_0$ and \hat{V}_0 be as defined in Theorem 4.2, (4.29)–(4.32) and R_1, R_0, U_1, U_0 and V_0^* be as defined in Theorem 5.1 (5.12)–(5.15). Also let*

$$U_0^* = U_0 + \frac{\sigma_S^2}{\mu} = \hat{U}_0 + \frac{\sigma_S^2}{\mu}. \tag{7.1}$$

Suppose the following conditions hold: $R_0 < 1, U_0^* < 1$, and $V_0^* \leq 1$, and

$$T_{\min} \geq \left\{ \frac{1}{2\mu} \log \frac{R_1}{1 - R_0}, \frac{1}{2\mu} \log \frac{U_1}{1 - U_0^*} \right\}, \tag{7.2}$$

where

$$T_{\min} = \min (T_1, T_1 + T_2, T_3), \tag{7.3}$$

it follows that there exists a positive constant $\mathbf{m} > 0$, such that the following inequality holds

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \mathbb{E} \int_0^t [\|X(v) - E_0\|_2]^2 dv \leq \frac{2\sigma_S^2 (S_0^*)^2}{\mathbf{m}}, \tag{7.4}$$

where $X(t) = (S(t), E(t), I(t))$, and $\|\cdot\|_2$ is the Euclidean norm on \mathbb{R}_+^3 .

Proof. Let Theorem 3.1[3.] be satisfied, i.e. $\sigma_S > 0$. Applying the functional Itô differential operator (cf. [13]) $d\tilde{V}$ to \tilde{V} defined in (5.10), and utilizing (5.3) and (5.20), it is easy to see that

$$d\tilde{V} = L\tilde{V}dt - 2\sigma_S(U(t) + V(t)) (S_0^* + U(t)) dw_S(t)$$

$$\begin{aligned}
 & -2\sigma_E (U(t)V(t) + (c + 1)V^2(t)) dw_E(t) - 2\sigma_I W^2(t) dw_I(t) \\
 & - 2c\sigma_\beta (S_0^* + U(t)) V(t)e^{-\mu v T_1} G(W(t - T_1)) dw_\beta \\
 & - 2\sigma_E [U(t) + (c + 1)V(t) + W(t)] \\
 & \times e^{-\mu v T_1 - \mu T_2} (S_0^* + U(t - T_2)) G(W(t - T_1 - T_2)) dw_\beta(t), \tag{7.5}
 \end{aligned}$$

where for some positive constant valued function $\tilde{K}(\mu)$, the drift part of (7.5), $L\tilde{V}$, satisfies the inequality

$$L\tilde{V}(x, t) \leq - (\tilde{\phi}_1 U^2(t) + \tilde{\psi}_1 V^2(t) + \tilde{\varphi}_1 W^2(t)), \tag{7.6}$$

where

$$\begin{aligned}
 \tilde{\phi}_1 &= 2\mu - \left(\beta S_0^* + \beta + \alpha + 2 \frac{\mu}{\tilde{K}(\mu)^2} + 2\sigma_S^2 \right) \\
 & - \left[3\sigma_\beta^2 (G^*)^2 + c\beta (G^*)^2 + \sigma_\beta^2 (G^*)^2 (c^2 - 2c) \right] e^{-2\mu T_1} \\
 & - \left[2 \left(1 + \frac{1}{S_0^*} + c \right) \beta (G^*)^2 + 5\sigma_\beta^2 (G^*)^2 + 2c \left(\beta (G^*)^2 + \sigma_\beta^2 (G^*)^2 \right) \right] e^{-2\mu(T_1+T_2)} \\
 & \geq 2\mu \left[(1 - U_0^*) - U_1 e^{-2\mu T_1} \right] - 2c \left[\beta (S_0^*)^2 + \sigma_\beta^2 (G^*)^2 \right] e^{-2\mu(T_1+T_2)} \\
 & - \left[c\beta (G^*)^2 + \sigma_\beta^2 (G^*)^2 (c^2 - 2c) \right] e^{-2\mu T_1}, \tag{7.7}
 \end{aligned}$$

$$\begin{aligned}
 \tilde{\psi}_1 &= 2\mu - \left[2\mu \tilde{K}(\mu)^2 + \alpha + \beta S_0^* + \beta + \sigma_E^2 \right] \\
 & + c \left[2\mu - (2\beta + \beta S_0^* + \sigma_E^2) \right] \\
 & \geq 2\mu [1 - V_0] + 2\mu c \left[1 - \frac{(2\beta + \beta S_0^* + \sigma_E^2)}{2\mu} \right], \tag{7.8}
 \end{aligned}$$

$$\begin{aligned}
 \tilde{\varphi}_1 &= 2(\mu + d + \alpha) - (\beta S_0^* + \sigma_I^2) - 2\alpha e^{-2\mu T_3} - [2\beta S_0^* + \sigma_\beta^2 S_0^*] e^{-2\mu T_1} \\
 & - \left[2 \left(2 + \frac{1}{S_0^*} \right) \beta (S_0^*)^2 + 9\sigma_\beta^2 (S_0^*)^2 \right] e^{-2\mu(T_1+T_2)} \\
 & - [c\beta S_0^* + (c^2 - 2c) \sigma_\beta^2 (S_0^*)] e^{-2\mu T_1} - c [2\beta (S_0^*)^2 + 4\sigma_\beta^2 (S_0^*)^2] e^{-2\mu(T_1+T_2)} \\
 & \geq 2(\mu + d + \alpha) [1 - R_0 - R_1 e^{-2\mu T_{min}}] \\
 & - [c\beta S_0^* + (c^2 - 2c) \sigma_\beta^2 (S_0^*)] e^{-2\mu T_1} - c [2\beta (S_0^*)^2 + 4\sigma_\beta^2 (S_0^*)^2] e^{-2\mu(T_1+T_2)}. \tag{7.9}
 \end{aligned}$$

Under the assumption of R_0 , U_0^* and V_0^* in the hypothesis, and for suitable choice of the positive constant c , it follows that $\tilde{\phi}$, $\tilde{\psi}$, and $\tilde{\varphi}$ are positive constants. Therefore, by integrating (7.5) from 0 to t , and taking expectation, it follows from (7.5)–(7.9) that

$$\mathbb{E}(V(t) - V(0)) \leq -\mathbf{m} \mathbb{E} \int_0^t \left[(S(v) - S_0^*)^2 + E^2(v) + I^2(v) \right] dv + 2\sigma_S^2 (S_0^*)^2 t, \tag{7.10}$$

where $V(0)$ is constant and

$$\mathbf{m} = \min(\tilde{\phi}, \tilde{\psi}, \tilde{\varphi}). \tag{7.11}$$

Hence, diving both sides of (7.10) by t and \mathbf{m} , and taking the limit supremum as $t \rightarrow \infty$, then (7.4) follows immediately. \square

Remark 7.1.

1. Theorem 7.1 signifies that when noise from the natural deathrate of the susceptible state with intensity $\sigma_S > 0$ is introduced into the disease dynamics characterized earlier in Theorem 5.1, the initially stochastically stable steady state E_0 ceases to exist. Nevertheless, the sample paths of the ensuing stochastic system (1.8)–(1.11) (with $\sigma_S > 0$) continue to oscillate near E_0 as shown in (7.4). Moreover, as the intensity $\sigma_S \rightarrow 0$, then the average value of every sample path of the system (1.8)–(1.11) in the phase space \mathbb{R}_+^3 is expected to converge to E_0 .

This result suggests that for small magnitudes of the intensity $0 < \sigma_S \ll 1$, the disease dynamics is still controllable near a potential infection-free state E_0 , provided that all other threshold conditions of Theorem 7.1 are satisfied. However, if the intensity $1 \ll \sigma_S < \infty$, then all paths of the stochastic system (1.8)–(1.11) are expected to deviate further away, on average, from the infection-free steady state E_0 . Deviation from E_0 may imply that the disease becomes endemic in the population, or it may imply that the population is becoming extinct over time.

2. It is also easy to see from Theorem 7.1 that there is another parameter region $D_2(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$ for the intensities of the noises $(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$ in which the paths of the stochastic system are expected to remain in the neighborhood of the E_0 .

It is easy to see that, if the conditions for stability of E_0 in Theorem 4.2 (4.29)–(4.32) for the deterministic system (1.1)–(1.4), are satisfied (i.e. $\hat{R}_0^* < 1, U_0 \equiv \hat{U}_0 \leq 1$ and $\hat{V}_0 \leq 1$), then in the region $D_2(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$ defined in (7.12), the result in Theorem 7.1, (7.4) holds. In other words, the paths of the stochastic solution process of (1.8)–(1.11) will remain near E_0 , depending on the magnitude of the intensity σ_S . This result is stated formally in Theorem 7.2.

Theorem 7.2. *Suppose the conditions in Theorem 4.2 hold. That is, $\hat{R}_0^* < 1, \hat{U}_0 < 1$ and $\hat{V}_0 \leq 1$, where \hat{R}_0^*, \hat{U}_0 , and \hat{V}_0 are given in Theorem 4.2 (4.29)–(4.32). Let the region $D_2(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$ be as defined in (7.12). That is,*

$$\begin{aligned} & D_2(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \\ &= D_2^S(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \cap D_2^E(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \cap D_2^I(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \cap D_2^\beta(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta), \end{aligned} \quad (7.12)$$

where

$$D_2^S(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) = \left\{ (\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 \mid 0 < \sigma_S \leq \mu(1 - \hat{U}_0) \right\}, \quad (7.13)$$

$$D_2^E(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) = \left\{ (\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 \mid 0 < \sigma_E^2 \leq 2(\mu)(1 - \hat{V}_0) \right\}, \quad (7.14)$$

$$D_2^I(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) = \left\{ (\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 \mid 0 < \sigma_I^2 \leq 2(\mu + d + \alpha)(1 - \hat{R}_0^*) \right\}, \quad (7.15)$$

and

$$\begin{aligned} & D_2^\beta(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \\ &= \left\{ (\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in [0, \infty)^4 \mid \frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) (1 - \hat{R}_0^*) \right\} \end{aligned}$$

$$\begin{aligned}
 & -\frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \left(\hat{R}_0^* + \left(2 + \frac{1}{S_0^*} \right) \frac{\beta (S_0^*)^2}{(\mu + d + \alpha)} + \frac{\alpha}{(\mu + d + \alpha)} \right) \\
 & -\frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \frac{\frac{1}{2} \sigma_I^2}{(\mu + d + \alpha)} < \sigma_\beta^2 \ll e^2 \frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) (1 - \hat{R}_0^*) \\
 & -\frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \left(\hat{R}_0^* + \left(2 + \frac{1}{S_0^*} \right) \frac{\beta (S_0^*)^2}{(\mu + d + \alpha)} + \frac{\alpha}{(\mu + d + \alpha)} \right) \\
 & -e^2 \frac{1}{9} \frac{1}{(S_0^*)^2} (\mu + d + \alpha) \frac{\frac{1}{2} \sigma_I^2}{(\mu + d + \alpha)} \Bigg\}, \tag{7.16}
 \end{aligned}$$

and e is the base of the natural logarithm.

It follows that there exists a positive constant $\mathbf{m} > 0$, such that the following inequality holds

$$\limsup_{t \rightarrow \infty} \frac{1}{t} \mathbb{E} \int_0^t [\|X(v) - E_0\|_2]^2 dv \leq \frac{2\sigma_S^2 (S_0^*)^2}{\mathbf{m}}, \tag{7.17}$$

regardless of the source of the noise in the system (1.8)–(1.11), provided that the intensities lie in $(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in D_2(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$, where $X(t) = (S(t), E(t), I(t))$, and $\|\cdot\|_2$ is the Euclidean norm on \mathbb{R}_+^3 .

Proof. Suppose $\hat{R}_0^* < 1$, $\hat{U}_0 \leq 1$, and $\hat{V}_0 \leq 1$, where \hat{R}_0^* , \hat{U}_0 , and \hat{V}_0 are given in (4.29)–(4.32). It is easy to see that for $(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta) \in D_2(\sigma_S, \sigma_E, \sigma_I, \sigma_\beta)$, and (5.27) satisfied, then Theorem 7.1 is also satisfied. \square

8. Conclusion

The presented classes of stochastic and deterministic SEIRS epidemic models with nonlinear incidence rates, and white noise processes characterize the general dynamics of vector-borne diseases such as malaria and dengue fever etc. that are influenced by random environmental fluctuations from (1.) the disease transmission rate, and from (2.) the natural death rate of humans of all states - susceptible, exposed, infectious and removed. The random incubation periods of the parasites or virus in the vectors (e.g. mosquitoes) and humans are considered. Moreover, the random acquired natural immunity period for the disease is also considered.

The model validation results for both stochastic and deterministic systems are presented. The impacts of each source of variability in the stochastic disease dynamics are examined. Comparative threshold conditions for the stability of equilibria of both systems are presented. Moreover, white noise intensity regions within which both systems exhibit similar asymptotic characteristics near the equilibria are computed.

Appendix A.

Derivation of the Model (1.1)–(1.4) from the mosquito-host dynamics

The assumptions for the family of malaria models in [56] are adopted and modified in the following. (A) The delays represent the incubation period of the infectious agents (plasmodium or dengue fever virus etc.) in the vector T_1 , and in the human host T_2 . The third delay represents the naturally acquired immunity period of the disease T_3 , where the delays are random variables with density functions $f_{T_1}, t_0 \leq T_1 \leq h_1, h_1 > 0$, and $f_{T_2}, t_0 \leq T_2 \leq h_2, h_2 > 0$ and $f_{T_3}, t_0 \leq T_3 < \infty$. All other assumptions for T_1, T_2 and T_3 are similar to the study [56].

(B) The vector (e.g. mosquito) population consists of two main classes namely: the susceptible vectors V_s and the infectious vectors V_i . Moreover, it is assumed that the total vector population denoted V_0 is constant at any time, that is, $V_s(t) + V_i(t) = V_0, \forall t \geq t_0$, where $V_0 > 0$ is a positive constant. The susceptible vector population V_s are infected by infectious human beings \hat{I} , and after the incubation period T_1 of the infectious agent, the exposed vector become infectious V_i . Moreover, it is assumed that there is homogenous mixing between the vector-host populations. Therefore, the birth rate and death rate of the vectors must be equal, and denoted $\hat{\mu}_v$. It is further assumed that the turnover of the vector population is very high, and the total number of vectors V_0 at any time t , is a very large, and as a consequence, $\hat{\mu}_v$ is sufficiently large number. In addition, it is assumed that the total number of vectors V_0 is exceedingly larger than the total human population present at any time t , denoted $\hat{N}(t), t \geq t_0$. That is, $V_0 \gg \hat{N}(t), t \geq t_0$.

(C) The human population is similarly defined as in Wanduku [56], and consists of susceptible (\hat{S}), Exposed (\hat{E}), Infectious (\hat{I}) and removed (\hat{R}) classes. The susceptible humans are infected by the infectious vectors V_i , and become exposed (E). The infectious agent incubates for T_2 time units, and the exposed individuals become infectious \hat{I} . The infectious class recovers from the disease with temporary or sufficiently long natural immunity and become (\hat{R}). Therefore, the total population present at time t , $\hat{N}(t) = \hat{S}(t) + \hat{E}(t) + \hat{I}(t) + \hat{R}(t), \forall t \geq t_0$.

Furthermore, it is assumed that the interaction between the infectious vectors V_i and susceptible humans \hat{S} exhibits nonlinear behavior, due to the overcrowding of the vectors as described in (B), and resulting in psychological effects on the susceptible individuals which leads to change of behavior that limits the disease transmission rate, and consequently result in a nonlinear character for the incidence rate characterized by the nonlinear incidence function G . G satisfies the conditions of Assumption 1.1.

(D) There is constant birthrate of human beings \hat{B} in the population, and all births are susceptible individuals. It is also assumed that the natural deathrate of human beings in the population is $\hat{\mu}$ and individuals die additionally due to disease related causes at the rate \hat{d} . From a biological point of view, the average lifespan of vectors $\frac{1}{\hat{\mu}_v}$, is much less than the average lifespan of a human being in the absence of disease $\frac{1}{\hat{\mu}}$. It follows very easily that assuming exponential lifetime for all individuals (both vector and host) in the population, then the survival probability over the time intervals of length $T_1 = s \in [t_0, h_1]$, and $T_2 = s \in [t_0, h_2]$, satisfy

$$e^{-\hat{\mu}_v T_1} \ll e^{-\hat{\mu} T_1} \quad \text{and} \quad e^{-\hat{\mu}_v T_1 - \hat{\mu} T_2} \ll e^{-\hat{\mu}(T_1 + T_2)}. \quad (8.1)$$

That is, (8.1) signifies that the survival chance of the mosquitoes, and consequently the parasites or virus inside the mosquitoes (and inside humans) over the complete life cycle of the parasites lasting for T_1+T_2 time units, is less than the survival chance of human beings over the same period of time. Furthermore, recall [Theorem 5.1, [56]] asserts that it is necessary for the expected survival rate $E(e^{-\hat{\mu}_v T_1 - \hat{\mu} T_2})$ to be significant for the disease to establish a steady endemic population. All other assumptions for the malaria model (1.1)–(1.4) remain the same as in [56].

Applying similar ideas in [54], the vector dynamics from (A)-(D) follows the system

$$dV_s(t) = [-\Lambda e^{-\hat{\mu}_v T_1} \hat{I}(t - T_1) V_s(t - T_1) - \hat{\mu}_v V_s(t) + \hat{\mu}_v (V_s(t) + V_i(t))]dt, \tag{8.2}$$

$$dV_i(t) = [\Lambda e^{-\hat{\mu}_v T_1} \hat{I}(t - T_1) V_s(t - T_1) - \hat{\mu}_v V_i(t)]dt, \tag{8.3}$$

$$V_0 = V_s(t) + V_i(t), \forall t \geq t_0, t_0 \geq 0, \tag{8.4}$$

where Λ is the effective disease transmission rate from an infectious human being to a susceptible vector. Observe that the incidence rate of the disease into the vector population $\Lambda e^{-\hat{\mu}_v T_1} \hat{I}(t - T_1) V_s(t - T_1)$ represents the rate of new infectious vectors occurring at time t , which became exposed at earlier time $t - T_1$ after obtaining an infected blood meal from an infectious person, and surviving over the incubation period T_1 , with the exponential survival probability rate $e^{-\hat{\mu}_v T_1}$, the vectors become infectious at time t . The detailed host population dynamics is derived as follows.

At time t , it follows from (C) that when susceptible humans \hat{S} and infectious vectors V_i interact with $\hat{\beta}$ effective contacts per vector, per unit time, then under the assumption of homogenous mixing, the incidence rate of the disease into the human population is given by the term $\hat{\beta} \hat{S}(t) V_i(t)$. With the assumption of crowding effects of the vector population, it follows from (C) that the incidence rate of the disease can be written as

$$\hat{\beta} \hat{S}(t) G(V_i(t)), \tag{8.5}$$

where G is the nonlinear incidence function satisfying the conditions in Assumption 1.1.

The susceptible individuals \hat{S} who have acquired infection from infectious vectors V_i , but are non infectious form the exposed class \hat{E} . The population of exposed individuals at time t is denoted $\hat{E}(t)$. After the incubation period, $T_2 = u \in [t_0, h_2]$, of the infectious agent in the exposed human host, the individual becomes infectious, $\hat{I}(t)$, at time t . Applying similar reasoning in [15], the exposed population, $\hat{E}(t)$, at time t can be written as follows

$$\hat{E}(t) = \hat{E}(t_0) e^{-\hat{\mu}(t-t_0)} p_1(t - t_0) + \int_{t_0}^t \hat{\beta} \hat{S}(\xi) G(V_i(\xi)) e^{-\hat{\mu}(t-\xi)} p_1(t - \xi) d\xi, \tag{8.6}$$

where

$$p_1(t) = \begin{cases} 0, & t \geq T_2, \\ 1, & t < T_2 \end{cases} \tag{8.7}$$

represents the probability that an individual remains exposed over the time interval $[0, t]$. It is easy to see from (8.6) that under the assumption that the disease has been in the population for at least a time $t > \max_{t_0 \leq T_1 \leq h_1, t_0 \leq T_2 \leq h_2} (T_1 + T_2)$, in fact, $t > h_1 + h_2$, so that all initial perturbations have died out, the number of

exposed individuals at time t is given by

$$\hat{E}(t) = \int_{t-T_2}^t \hat{\beta} \hat{S}(v) G(V_i(v)) e^{-\hat{\mu}(t-T_2)} dv. \quad (8.8)$$

Moreover, since $T_2 = u \in [t_0, h_2]$ is a random variable, it follows from (8.8) that the expected number of exposed individuals at time t is given by

$$\hat{E}(t) = \int_{t_0}^{h_2} f_{T_2}(u) \int_{t-u}^t \hat{\beta} \hat{S}(v) G(V_i(v)) e^{-\hat{\mu}(t-u)} dv du. \quad (8.9)$$

Similarly, for the removal population, $\hat{R}(t)$, at time t , individuals recover from the infectious state $\hat{I}(t)$ at the per capita rate $\hat{\alpha}$ and acquire natural immunity. The natural immunity wanes after the varying immunity period $T_3 = r \in [t_0, \infty]$, and removed individuals become susceptible again to the disease. Therefore, at time t , individuals leave the infectious state at the rate $\hat{\alpha} \hat{I}(t)$ and become part of the removal population $\hat{R}(t)$. Thus, at time t the removed population is given by the following equation

$$\hat{R}(t) = \hat{R}(t_0) e^{-\hat{\mu}(t-t_0)} p_2(t-t_0) + \int_{t_0}^t \hat{\alpha} \hat{I}(\xi) e^{-\hat{\mu}(t-\xi)} p_2(t-\xi) d\xi, \quad (8.10)$$

where

$$p_2(t) = \begin{cases} 0, & t \geq T_3, \\ 1, & t < T_3 \end{cases} \quad (8.11)$$

represents the probability that an individual remains naturally immune to the disease over the time interval $[0, t]$. But it follows from (8.10) that under the assumption that the disease has been in the population for at least a time $t > \max_{t_0 \leq T_1 \leq h_1, t_0 \leq T_2 \leq h_2, T_3 \geq t_0} (T_1 + T_2, T_3) = T_{max} \geq \max_{T_3 \geq t_0} (T_3)$, in fact, the disease has been in the population for sufficiently large amount of time so that all initial perturbations have died out, then the number of removed individuals present at time t from (8.10), is given by

$$\hat{R}(t) = \int_{t-T_3}^t \hat{\alpha} \hat{I}(v) e^{-\hat{\mu}(t-v)} dv. \quad (8.12)$$

Since T_3 is distributed, the expected number of removal individuals at time t can be written as

$$\hat{R}(t) = \int_{t_0}^{\infty} f_{T_3}(r) \int_{t-r}^t \hat{\alpha} \hat{I}(v) e^{-\hat{\mu}(t-v)} dv dr. \quad (8.13)$$

It follows from the assumptions (A)-(D), (8.5), (8.8), (8.9), and (8.13) that for $T_j, j = 1, 2, 3$ fixed in the population, the dynamics of malaria in the human population is given by the system

$$d\hat{S}(t) = \left[\hat{B} - \hat{\beta} \hat{S}(t) G(V_i(t)) - \hat{\mu} \hat{S}(t) + \hat{\alpha} \hat{I}(t - T_3) e^{-\hat{\mu} T_3} \right] dt, \quad (8.14)$$

$$d\hat{E}(t) = \left[\hat{\beta} \hat{S}(t) G(V_i(t)) - \hat{\mu} \hat{E}(t) - \hat{\beta} \hat{S}(t - T_2) e^{-\hat{\mu} T_2} G(V_i(t - T_2)) \right] dt, \quad (8.15)$$

$$d\hat{I}(t) = \left[\hat{\beta} \hat{S}(t - T_2) e^{-\hat{\mu} T_2} G(V_i(t - T_2)) - (\hat{\mu} + \hat{d} + \hat{\alpha}) \hat{I}(t) \right] dt, \quad (8.16)$$

$$d\hat{R}(t) = \left[\hat{\alpha}\hat{I}(t) - \hat{\mu}\hat{R}(t) - \hat{\alpha}\hat{I}(t - T_3)e^{-\hat{\mu}T_3} \right] dt. \tag{8.17}$$

Furthermore, the incidence function G satisfies the conditions in Assumption 1.1. And the initial conditions are given in the following:

$$\begin{aligned} (\hat{S}(t), \hat{E}(t), \hat{I}(t), \hat{R}(t)) &= (\varphi_1(t), \varphi_2(t), \varphi_3(t), \varphi_4(t)), t \in (-T_{max}, t_0], \\ \varphi_k &\in \mathcal{C}((-T_{max}, t_0], \mathbb{R}_+), \forall k = 1, 2, 3, 4, \\ \varphi_k(t_0) &> 0, \forall k = 1, 2, 3, 4, \quad \text{and} \quad \max_{\substack{t_0 \leq T_1 \leq h_1, \\ t_0 \leq T_2 \leq h_2, \\ T_3 \geq t_0}} (T_1 + T_2, T_3) = T_{max} \end{aligned} \tag{8.18}$$

where $\mathcal{C}((-T_{max}, t_0], \mathbb{R}_+)$ is the space of continuous functions with the supremum norm

$$\|\varphi\|_\infty = \sup_{t \leq t_0} |\varphi(t)|. \tag{8.19}$$

It is shown in the following that the vector-host dynamics in (8.2)–(8.4) and (8.14)–(8.18) lead to the model (1.1)–(1.4), which omits the dynamics of the vector population, under the assumptions (A)–(D).

Firstly, observe that the system (8.14)–(8.18) satisfies [56, Theorem 3.1], and the total human population $\hat{N}(t) = \hat{S}(t) + \hat{E}(t) + \hat{I}(t) + \hat{R}(t), \forall t \geq t_0$ obtained from system (8.14)–(8.18) with initially condition that satisfies $N(t_0) \leq \frac{\hat{B}}{\hat{\mu}}$, must satisfy

$$\limsup_{t \rightarrow \infty} \hat{N}(t) = \frac{\hat{B}}{\hat{\mu}}. \tag{8.20}$$

Therefore, the assumption (B) above, interpreted as $\frac{\hat{N}(t)}{V_0} \ll 1, \forall t \geq t_0$ implies that

$$\limsup_{t \rightarrow \infty} \hat{N}(t) = \frac{\hat{B}}{\hat{\mu}}, \quad \text{and} \quad \left(\frac{\hat{B}}{\hat{\mu}} \right) \ll 1. \tag{8.21}$$

Define

$$\epsilon = \frac{\left(\frac{\hat{B}}{\hat{\mu}} \right)}{V_0}, \tag{8.22}$$

then from (8.21)–(8.22), it follows that $\epsilon = \frac{\left(\frac{\hat{B}}{\hat{\mu}} \right)}{V_0} \ll 1$.

Employing similar reason in [54], define two natural dimensionless time scales η and ϱ for the joint vector-host dynamics (8.2)–(8.4) and (8.14)–(8.18) in the following.

$$\eta = \left(\frac{\hat{B}}{\hat{\mu}} \right) \Delta t, \tag{8.23}$$

$$\varrho = V_0 \Delta t. \tag{8.24}$$

Note that since the total vector population V_0 from (B) above is constant, that is, $V_s(t) + V_i(t) = V_0, \forall t \geq t_0$, and from (8.20) and [56, Theorem 3.1] the total human $0 < \hat{N}(t) \leq \frac{\hat{B}}{\hat{\mu}}, \forall t \geq t_0$, whenever $\hat{N}(t_0) \leq \frac{\hat{B}}{\hat{\mu}}$, then the time scales η and ϱ arise naturally to rescale the total vector and maximum total human populations V_0 and $\left(\frac{\hat{B}}{\hat{\mu}} \right)$, respectively, at any time.

The time scales (8.23)–(8.24) can be distinguished as “fast” and “slow” using the following example. A particle’s movement on the ϱ time scale covers one unit of time $\varrho = 1$ at much early time $t_\varrho = \frac{1}{V_0\Lambda}$ on the t time scale, compared to the particle’s movement on the η time scale, where the particle covers one unit of time $\eta = 1$ at much later time $t_\eta = \frac{1}{(\frac{\hat{B}}{\hat{\mu}})\Lambda} \gg t_\varrho$, since (8.21) holds. Thus, movement on the time scale ϱ is “fast”, and on η is “slow”. See [54] for more information.

Therefore, from above, let

$$\hat{V}_i(t) = \frac{V_i(t)}{V_0}, \quad \text{and} \quad \hat{V}_s(t) = \frac{V_s(t)}{V_0}, \quad (8.25)$$

be the dimensionless vector variables, and

$$S(t) = \frac{\hat{S}(t)}{\left(\frac{\hat{B}}{\hat{\mu}}\right)}, I(t) = \frac{\hat{I}(t)}{\left(\frac{\hat{B}}{\hat{\mu}}\right)}, E(t) = \frac{\hat{E}(t)}{\left(\frac{\hat{B}}{\hat{\mu}}\right)}, R(t) = \frac{\hat{R}(t)}{\left(\frac{\hat{B}}{\hat{\mu}}\right)} \quad \text{and} \quad N(t) = \frac{\hat{N}(t)}{\left(\frac{\hat{B}}{\hat{\mu}}\right)}, \quad (8.26)$$

be the dimensionless human variables. And since $0 < \hat{N}(t) \leq \frac{\hat{B}}{\hat{\mu}}, \forall t \geq t_0$, whenever $\hat{N}(t_0) \leq \frac{\hat{B}}{\hat{\mu}}$, it follows from (8.26) that

$$0 < S(t) + E(t) + I(t) + R(t) = N(t) \leq 1, \forall t \geq t_0. \quad (8.27)$$

Applying (8.25)–(8.26) to (8.2)–(8.4) leads to the following

$$d\hat{V}_i(t) = \epsilon \left[e^{-\hat{\mu}_v T_1} I(t - T_1) \hat{V}_s(t - T_1) - \frac{\hat{\mu}_v}{\Lambda \left(\frac{\hat{B}}{\hat{\mu}}\right)} \hat{V}_i(t) \right] d\varrho, \quad (8.28)$$

$$d\hat{V}_s(t) = -d\hat{V}_i(t), \quad (8.29)$$

$$1 = \hat{V}_s(t) + \hat{V}_i(t), \forall t \geq t_0, t_0 \geq 0. \quad (8.30)$$

Observe from (8.27)–(8.30) that for nonnegative values for the vector variables $\hat{V}_i(t) \geq 0, \hat{V}_s(t) \geq 0, \forall t \geq t_0$, and positive values for the human variables $S(t), E(t), I(t), R(t) > 0, \forall t \geq t_0$, it follows that

$$-\epsilon \frac{\hat{\mu}_v}{\Lambda \left(\frac{\hat{B}}{\hat{\mu}}\right)} \leq \frac{d\hat{V}_i(t)}{d\varrho} \leq \epsilon e^{-\hat{\mu}_v T_1}. \quad (8.31)$$

Thus, on the time scale ϱ which is “fast”, it is easy to see from (8.28)–(8.31), that under the assumption that ϵ from (8.22) is infinitesimally small, that is $\epsilon \rightarrow 0$, then

$$\frac{d\hat{V}_i(t)}{d\varrho} = -\frac{d\hat{V}_s(t)}{d\varrho} = 0, \quad (8.32)$$

which implies that the dynamics of \hat{V}_i and \hat{V}_s behaves as in steady state. And thus, it follows from (8.28)–(8.32) that

$$\hat{V}_i(t) = \frac{e^{-\hat{\mu}_v T_1}}{\hat{\mu}_v} \Lambda \left(\frac{\hat{B}}{\hat{\mu}}\right) I(t - T_1) \hat{V}_s(t - T_1),$$

$$1 = \hat{V}_s(t) + \hat{V}_i(t). \quad (8.33)$$

It follows further from (8.33) that

$$\hat{V}_s(t) = \frac{1}{1 + \frac{e^{-\hat{\mu}_v T_1}}{\hat{\mu}_v} \Lambda \left(\frac{\hat{B}}{\hat{\mu}} \right) I(t - T_1)}. \tag{8.34}$$

For sufficiently large value of the birth-death rate $\hat{\mu}_v$ (see assumption (B)), such that $\hat{\mu}_v e^{\hat{\mu}_v T_1} \gg \Lambda \left(\frac{\hat{B}}{\hat{\mu}} \right)$, then it follows from (8.34) that $\hat{V}_s(t) \approx 1$, and consequently from (8.30) and (8.25), $V_s(t) \approx V_0$. Moreover, it follows further from (8.33) that

$$\hat{V}_i(t) \approx \frac{e^{-\hat{\mu}_v T_1}}{\hat{\mu}_v} \Lambda \left(\frac{\hat{B}}{\hat{\mu}} \right) I(t - T_1), \tag{8.35}$$

and equivalently from (8.25)–(8.26) that (8.35) can be rewritten as follows

$$V_i(t) \approx \frac{e^{-\hat{\mu}_v T_1}}{\hat{\mu}_v} \Lambda V_0 \hat{I}(t - T_1). \tag{8.36}$$

While on the fast scale ϱ the term $\hat{I}(t - T_1)$ behaves as the steady state, on the slow scale η , it is expected to still be evolving. In the following, using (8.25)–(8.26), the dynamics for the human population in (8.14)–(8.18) is nondimensionalized with respect to the slow time scale η in (8.23).

Without loss of generality (as it is usually the case e.g. $G(x) = \frac{x}{1+\alpha x}$, $G(x) = \frac{x}{1+\alpha x^2}$), it is assumed that on the η timescale, the nonlinear term $G(V_i(t))$ expressed as $G(V_0 \hat{V}_i(\eta))$, can be rewritten from (8.36) as

$$G(V_0 \hat{V}_i(\eta)) \equiv \frac{\Lambda V_0 \left(\frac{\hat{B}}{\hat{\mu}} \right)}{\hat{\mu}_v} \hat{G}(\hat{V}_i(\eta)) e^{-\hat{\mu}_v T_1}, \tag{8.37}$$

by factoring a constant term $\frac{\Lambda V_0 \left(\frac{\hat{B}}{\hat{\mu}} \right)}{\hat{\mu}_v}$, and the function \hat{G} carries all the properties of Assumption 1.1. Thus, from the above and (8.36), the system (8.14)–(8.18) is rewritten in dimensionless form as follows:

$$dS(\eta) = [B - \beta S(\eta) \hat{G}(I(\eta - T_{1\eta})) e^{-\mu_v T_{1\eta}} - \mu S(\eta) + \alpha I(\eta - T_{3\eta}) e^{-\mu T_{3\eta}}] d\eta, \tag{8.38}$$

$$dE(\eta) = [\beta S(\eta) \hat{G}(I(\eta - T_{1\eta})) e^{-\mu_v T_{1\eta}} - \mu E(\eta) - \beta S(\eta - T_{2\eta}) \hat{G}(I(\eta - T_{1\eta} - T_{2\eta})) e^{-\mu_v T_{1\eta} - \mu T_{2\eta}}] d\eta, \tag{8.39}$$

$$dI(\eta) = [\beta S(\eta - T_{2\eta}) \hat{G}(I(\eta - T_{1\eta} - T_{2\eta})) e^{-\mu_v T_{1\eta} - \mu T_{2\eta}} - \mu I(\eta) - (\mu + d + \alpha) I(\eta)] d\eta, \tag{8.40}$$

$$dR(\eta) = [\alpha I(\eta) - \mu R(\eta) - \alpha I(\eta - T_{3\eta}) e^{-\mu T_{3\eta}}] d\eta, \tag{8.41}$$

where

$$B = \frac{\hat{B}}{\left(\frac{\hat{B}}{\hat{\mu}} \right)^2 \Lambda}, \quad \beta = \frac{\hat{\beta} V_0}{\hat{\mu}_v}, \quad \mu = \frac{\hat{\mu}}{\left(\frac{\hat{B}}{\hat{\mu}} \right) \Lambda}, \quad \alpha = \frac{\hat{\alpha}}{\left(\frac{\hat{B}}{\hat{\mu}} \right) \Lambda}$$

$$\mu_v = \frac{\hat{\mu}_v}{\left(\frac{\hat{B}}{\hat{\mu}} \right) \Lambda}, \quad d = \frac{\hat{d}}{\left(\frac{\hat{B}}{\hat{\mu}} \right) \Lambda}, \quad T_{j\eta} = \left(\frac{\hat{B}}{\hat{\mu}} \right) \Lambda T_j, \forall j = 1, 2, 3. \tag{8.42}$$

The system (8.38)–(8.41) describes the dynamics of malaria on the slow scale η . Furthermore, moving forward, the analysis of the model (8.38)–(8.41) is considered only on the η timescale. To reduce heavy notation, the following substitutions are made. Substitute t for η , and the delays $T_j, \forall j = 1, 2, 3$ will substitute $T_j\eta, \forall j = 1, 2, 3$. Moreover, since the delays are distributed with density functions $f_{T_j}, \forall j = 1, 2, 3$, it follows from (A)–(D), (8.6)–(8.13), (8.38)–(8.41) and (8.18) that the expected SEIRS model for malaria is given as follows:

$$dS(t) = \left[B - \beta S(t) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s} G(I(t-s)) ds - \mu S(t) + \alpha \int_{t_0}^{\infty} f_{T_3}(r) I(t-r) e^{-\mu r} dr \right] dt, \quad (8.43)$$

$$dE(t) = \left[\beta S(t) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s} G(I(t-s)) ds - \mu E(t) - \beta \int_{t_0}^{h_2} f_{T_2}(u) S(t-u) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s - \mu u} G(I(t-s-u)) ds du \right] dt, \quad (8.44)$$

$$dI(t) = \left[\beta \int_{t_0}^{h_2} f_{T_2}(u) S(t-u) \int_{t_0}^{h_1} f_{T_1}(s) e^{-\mu v s - \mu u} G(I(t-s-u)) ds du - (\mu + d + \alpha) I(t) \right] dt, \quad (8.45)$$

$$dR(t) = \left[\alpha I(t) - \mu R(t) - \alpha \int_{t_0}^{\infty} f_{T_3}(r) I(t-r) e^{-\mu r} dr \right] dt, \quad (8.46)$$

where the initial conditions are given in the following: let $h = h_1 + h_2$ and define

$$(S(t), E(t), I(t), R(t)) = (\varphi_1(t), \varphi_2(t), \varphi_3(t), \varphi_4(t)), t \in (-\infty, t_0], \quad (8.47)$$

$$\varphi_k \in \mathcal{C}((-\infty, t_0], \mathbb{R}_+), \forall k = 1, 2, 3, 4, \quad \varphi_k(t_0) > 0, \forall k = 1, 2, 3, 4,$$

where $\mathcal{C}((-\infty, t_0], \mathbb{R}_+)$ is the space of continuous functions with the supremum norm

$$\|\varphi\|_{\infty} = \sup_{t \leq t_0} |\varphi(t)|. \quad (8.48)$$

Also, the function G satisfies the conditions of Assumption 1.1.

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