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Analysis of Stability and Sensitivity of Deterministic and Stochastic Models for the Spread of the New Corona Virus SARS-CoV-2

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Abstract. Basic reproduction number for deterministic SEIPHAR model and its stochastic counterpart for the spread of SARS-CoV-2 virus are analyzed and compared. For deterministic version of the model, conditions for stability of the disease-free equilibrium are derived and, in addition, conditions for existence of bifurcation state related to endemic equilibrium are established. For stochastic model, conditions for extinction and persistence in mean of the disease are derived. Complete sensitivity analysis of thresholds between the extinction and mean-persistence are performed for both the deterministic and the stochastic version of the model. Influence of variation in parameter values is illustrated for epidemics in Wuhan in early 2020.

1. Introduction

Coronavirus infections in populations of humans are not new because there were several epidemics during the last two decades including Severe Acute Respiratory Syndrome Coronavirus (SARS-CoV) in China in 2002 and the Middle East Respiratory Syndrome Coronavirus (MERS-CoV) in Saudi Arabia in 2012. In November 2019, the first confirmed case of Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) was reported after which SARS-CoV-2 infection had spread very rapidly throughout the world. The disease that cause SARS-CoV-2 virus is commonly known as Covid-19. The symptoms of COVID-19 disease can be very diverse, ranging from mild to serious, including fatal outcomes. Because of that, a pandemic of Covid-19 has been declared a global emergency by the World Health Organization [1].

Research papers shows that the main mode of transmission of SARS-CoV-2 virus is close contact with a infected person during which the virus is transmitted through the respiratory organs (see [2]). Generally, individuals infected with SARS-CoV-2 virus showing symptoms of the disease will spread the disease to those in close contact. However, many infected individuals are asymptomatic and can serve as carriers

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and unknowingly transmit the virus (see [2]). According to [3], pre-symptomatic ($1-2$ days before the appearance of symptoms) and symptomatic individuals have a greater importance in the spreading of the infection than the asymptomatic carriers. For more information on asymptomatic carriers and the estimations of their proportion within the infected population we refer to [4] and [5]. Superspreader status of an infected individual includes one or more of the following factors: high viral load due the immunity issues, underlying diseases, existing infectious co-factors and/or elevated social activity.

From an epidemiological point of view, mathematical modeling of infectious diseases has a significant role. Number of epidemic models for modeling the spread of SARS-CoV-2 virus, with various characteristics, have been described and investigated. Many classical deterministic SIR, SEIR and refined version of SEIR compartmental epidemiological models are set up and analyze. For different refinements of the SEIR model we refer to [6], [7], [8] and [9]. Besides, influence of environmental uncertainty (e.g. such as temperature, precipitation, absolute humidity, ...) and the behavior of infectious agents (e.g. viruses) in such environment, suggests introduction of the stochastic noise into deterministic biological models to expose the environmental variability effect. There are many ways of introducing environmental variability into epidemiological models. First approach assumed that random noise is introduced into the differential systems proportional to the distances of compartments from their steady state (see [10], for instance). In the second approach assumption that environmental fluctuations mainly affect model parameters results in modeling model parameters with an appropriate diffusion process. For example, in [11] authors assumed that death rate μ can be modeled with an Ornstain-Uhlenbeck process. Third approach assume that environmental fluctuations mainly affect model parameters such as birth rate, death rate or transmission rate, which indicate to introduce noise into the model by perturbation of the certain model parameter by the additive noise. For example, in [12], [13], [14] and [15], perturbation of the transmission rate β by the additive noise gives

$$\beta \mapsto \beta + \sigma dB(t),$$

where $B(t)$ is the standard Brownian motion and σ intensity of the noise. Besides the white noise, epidemic models may be disturbed by telegraph noise which can lead the system to switch from one environmental regime to another (see [16], for instance). On the other hand, epidemic models may be affected by sudden and severe environmental perturbations, such as tsunami, volcanoes, avian influenza, SARS, toxic pollutants, etc. These environmental shocks would lead to jumps in population size. To describe these phenomena, many researchers use stochastic differential equations driven by jump processes (see e.g. [17] and [18]).

This paper is structured as follows. In Section 2, stochastic SARS-CoV-2 epidemic model is described. In Section 3, the deterministic counterpart of the stochastic SARS-CoV-2 epidemic model is introduced, its basic reproductive number is calculated and existence and stability of an endemic equilibrium point is examined. In Section 4, the sufficient conditions for extinction and persistence of the disease are establish. In addition to the results proven in [19], we have introduced a threshold parameter R_0^S, the counterpart of the basic reproduction number, which can be used to utilize in identifying the stochastic extinction and persistence for the stochastic epidemic model. In Section 5, reproduction numbers for deterministic and corresponding stochastic epidemic models for the spread of SARS-CoV-2 virus are compared. Besides, we performed the sensitivity analysis of the deterministic basic reproduction number and the corresponding threshold within the stochastic model.

2. Stochastic SARS-CoV-2 epidemic model

In this paper we observe the SEIPHAR epidemic model for modeling the spread of the new corona virus SARS-CoV-2, introduced in the recent paper [19]. They assumed that the human population is divided into seven mutually exclusive compartments:

- S - susceptible individuals,
- E - individuals exposed to the virus SARS-CoV-2, but not yet infectious to others (they may become infectious after a certain incubation period),

- I - symptomatic infectious individuals,
- P - superspreaders,
- A - asymptomatic infectious individuals,
- H - hospitalized individuals,
- R - recovered individuals.

The total population size at time t is given by

$$N(t) = S(t) + E(t) + I(t) + P(t) + A(t) + H(t) + R(t), \quad t \geq 0.$$

System of stochastic differential equations (SDEs) is of the following form:

$$\begin{cases} dS(t) &= \left(\Lambda - \left(\frac{\beta}{N(t)}\left(I(t) + lH(t)\right) + \frac{\beta'}{N(t)}P(t) + \mu\right)S(t)\right)dt \\ & \quad -\frac{\sigma_1}{N(t)}\left(I(t) + lH(t)\right)S(t)\,dB_1(t) - \frac{\sigma_2}{N(t)}P(t)S(t)\,dB_2(t), \\ dE(t) &= \left(\frac{\beta}{N(t)}\left(I(t) + lH(t)\right)S(t) + \frac{\beta'}{N(t)}P(t)S(t) - (\kappa + \mu)E(t)\right)dt \\ & \quad +\frac{\sigma_1}{N(t)}\left(I(t) + lH(t)\right)S(t)\,dB_1(t) + \frac{\sigma_2}{N(t)}P(t)S(t)\,dB_2(t), \\ dI(t) &= \left(\kappa\rho_1 E(t) - (\gamma_a + k_1\gamma_i + \delta_i)I(t)\right)dt, \\ dP(t) &= \left(\kappa\rho_2 E(t) - (\gamma_a + k_2\gamma_i + \delta_p)P(t)\right)dt, \\ dH(t) &= \left(\gamma_a(I(t) + P(t)) - (\gamma_r + \delta_h)H(t)\right)dt, \\ dA(t) &= \left(\kappa(1 - \rho_1 - \rho_2)E(t) - (\gamma_i + \mu)A(t)\right)dt, \\ dR(t) &= \left(\gamma_i(A(t) + k_1 I(t) + k_2 P(t)) + \gamma_r H(t) - \mu R(t)\right)dt, \end{cases} \tag{1}$$

where k_1, k_2 are constants such that $k_2 < k_1 < 1$, while $B_1 = (B_1(t),\, t \geq 0)$ and $B_2 = (B_2(t),\, t \geq 0)$ are independent Brownian motions with intensities $\sigma_1 > 0$ and $\sigma_2 > 0$ driving the stochastic nature of the transmission coefficients β and β', respectively. Description and values of the model parameters are given in Table (1).

Symbol	Description	Value	Units
Λ	Estimated daily number of newborns in Wuhan in 2019	310 [20]	per day
β	Transmission coefficient due to infected individuals	2.55 [6]	per day
l	Relative transmissibility from hospitalized individuals	1.56 [6]	dimensionless
β'	Transmission coefficient due to superspreaders	7.65 [6]	per day
κ	Rate at which exposed individuals become infectious	0.25 [6]	per day
ρ_1	Proportion of transitions from exposed do infected class	0.58 [6]	dimensionless
ρ_2	Proportion of transitions from exposed to superspreaders	0.001 [6]	dimensionless
γ_a	Hospitalization rate	0.94 [6]	per day
γ_r	Recovery rate for hospitalized patients	0.5 [6]	per day
γ_i	Recovery rate for non-hospitalized patients	0.27 [6]	per day
k_1	Weight for recovery raze due to infected class	0.85 [a]	dimensionless
k_2	Weight for recovery raze due to superspreaders	0.95 [a]	dimensionless
δ_i	Disease induced death rate for infected class	1/23 [21]	per day
δ_p	Disease induced death rate for superspreaders	1/23 [21]	per day
δ_h	Disease induced death rate for hospitalized class	1/23 [21]	per day
μ	Natural death rate	0.00714 [20]	per day

Table 1: Parameters values, either based on the epidemics in Wuhan in the period January 4 - March 9, 2020, or rationally assumed (k_1, k_2).

Existence and uniqueness of a positive global solution of system of stochastic differential equations (1) is proven in [19].

Remark 2.1. *According to [19], note that the set*

$$\Gamma^\star = \{(S(t), E(t), I(t), P(t), H(t), A(t), R(t)) :$$
$$S(t) > 0, E(t) > 0, I(t) > 0, P(t) > 0, H(t) > 0, A(t) > 0, R(t) > 0, N(t) \le \Lambda/\mu\}$$

is a positively invariant set of the system (1) *for every* $t > 0$, *i.e. if the system starts from* $\Gamma^\star$, *it never leaves* $\Gamma^\star$.

3. Analysis of the deterministic model

The deterministic counterpart of the system of stochastic differential equations (1) is the system of ordinary differential equations (ODE) of the following form:

$$\begin{cases} dS(t) &= \left(\Lambda - \left(\frac{\beta}{N(t)}\left(I(t) + lH(t)\right) + \frac{\beta'}{N(t)}P(t) + \mu\right)S(t)\right)dt \\ dE(t) &= \left(\frac{\beta}{N(t)}\left(I(t) + lH(t)\right)S(t) + \frac{\beta'}{N(t)}P(t)S(t) - (\kappa + \mu)E(t)\right)dt \\ dI(t) &= \left(\kappa\rho_1 E(t) - (\gamma_a + k_1\gamma_i + \delta_i)I(t)\right)dt, \\ dP(t) &= \left(\kappa\rho_2 E(t) - (\gamma_a + k_2\gamma_i + \delta_p)P(t)\right)dt, \\ dH(t) &= \left(\gamma_a(I(t) + P(t)) - (\gamma_r + \delta_h)H(t)\right)dt, \\ dA(t) &= \left(\kappa(1 - \rho_1 - \rho_2)E(t) - (\gamma_i + \mu)A(t)\right)dt, \\ dR(t) &= \left(\gamma_i(A(t) + k_1 I(t) + k_2 P(t)) + \gamma_r H(t) - \mu R(t)\right)dt, \end{cases} \tag{2}$$

where description of the model parameters are given in the Table (1).

3.1. The basic reproduction number

The basic reproduction number R_0 is a threshold value that is epidemiologically significant and determines the potential of an infectious disease to enter a population. In fact, the basic reproduction number R_0 is defined as the expected number of secondary cases generated by one infected individual during its lifespan as infectious in a fully susceptible population. To obtain the basic reproduction number R_0 of the system (2), we apply the next generation matrix approach introduced by van den Driessche and Watmough [22]. The system has a disease-free equilibrium given by

$$\varepsilon_0\Big(\frac{\Lambda}{\mu}, 0, 0, 0, 0, 0, 0\Big)$$

The infected compartments of the model (2) consist of $(E(t), I(t), P(t), H(t))$ classes. Following the next generation matrix method, the matrix F of the transmission terms and the matrix V of the transition terms, calculated at ε_0 are

$$F = \begin{pmatrix} 0 & \beta & \beta' & l\beta \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} \kappa + \mu & 0 & 0 & 0 \\ -\kappa\rho_1 & \omega_i & 0 & 0 \\ -\kappa\rho_2 & 0 & \omega_p & 0 \\ 0 & -\gamma_a & -\gamma_a & \omega_h \end{pmatrix}$$

with

$$\omega_i = \gamma_a + k_1\gamma_i + \delta_i, \quad \omega_p = \gamma_a + k_2\gamma_i + \delta_p, \quad \omega_h = \gamma_r + \delta_h \tag{3}$$

Calculating the dominant eigenvalue of the next generation matrix $F \cdot V^{-1}$, we obtain the basic reproductive number as follows:

$$R_0^D = \frac{\kappa}{\kappa + \mu}\,\frac{\omega_h(\beta\rho_1\omega_p + \beta'\rho_2\omega_i) + \ell\,\beta\gamma_a(\rho_1\omega_p + \rho_2\omega_i)}{\omega_h\omega_i\omega_p}$$

From the next-generation method, if $R_0^D < 1$, then the disease-free equilibrium point is locally asymptotically stable and if $R_0^D > 1$, then it is unstable. It is well known that extinction of the epidemics appears under conditions for asymptotic stability of the disease-free equilibrium, while persistence occurs when $R_0^D > 1$.

3.2. Existence and stability of an endemic equilibrium point

We are now exploring the existence and stability of endemic equilibrium. Let

$$\varepsilon^\star = \left(S^\star, E^\star, I^\star, P^\star, H^\star, A^\star, R^\star\right)$$

be any endemic equilibrium of system. Let us denote the force of COVID-19 infection

$$F^\star(t) = \beta \frac{I^\star(t)}{N^\star(t)} + l\beta \frac{H^\star(t)}{N^\star(t)} + \beta' \frac{P^\star(t)}{N^\star(t)}. \tag{4}$$

To find conditions for the existence of an equilibrium for which COVID-19 is endemic in the population (i.e., at least one of E, I, P, H, A is non-zero), the equations in (2) are solved in terms of the force of infection at steady-state. Setting the right-hand sides of the model to zero (at steady-state) gives

$$S^\star = \frac{\Lambda}{F^\star + \mu}, \quad E^\star = \frac{\Lambda F^\star}{(F^\star + \mu)(\kappa + \mu)}, \quad I^\star = \frac{\Lambda F^\star \kappa \rho_1}{(F^\star + \mu)(\kappa + \mu)\omega_i},$$

$$P^\star = \frac{\Lambda F^\star \kappa \rho_2}{(F^\star + \mu)(\kappa + \mu)\omega_p}, \quad H^\star = \frac{\Lambda F^\star \kappa \gamma_a(\rho_2\omega_i + \rho_1\omega_p)}{(F^\star + \mu)(\kappa + \mu)\omega_h\omega_i\omega_p}, \quad A^\star = \frac{\Lambda F^\star \kappa(1 - \rho_1 - \rho_2)}{(F^\star + \mu)(\kappa + \mu)\sigma_a},$$

$$R^\star = F^\star \kappa \Lambda \Big[\gamma_a\gamma_r\sigma_a(\rho_1\omega_p + \rho_2\omega_i) + \gamma_i\omega_h\sigma_a(k_1\rho_1\omega_p + k_2\rho_2\omega_i) + \gamma_i(1 - \rho_1 - \rho_2)\omega_h\omega_i\omega_p\Big]$$

where $\sigma_a = \gamma_i + \mu$ and $\omega_h, \omega_i, \omega_p$ are given in (3). Plugging the above expression into (4), we obtain the nonzero equilibrium of the system (2) satisfying the linear equation, in terms of $F^\star$, as follows:

$$\nu F^\star = \mu\sigma_a\eta$$

where

$$\eta = (\kappa + \mu)(R_0^D - 1)\omega_h\omega_i\omega_p,$$

$$\nu = \sigma_a\kappa\Big(\rho_2 m_2\omega_i + \rho_1 m_1\omega_p\Big) + \sigma_a\omega_h\omega_i\omega_p\Big(\kappa(1 - \rho_1 - \rho_2) + \mu\Big),$$

$$m_1 = \gamma_a(\gamma_r + \mu) + \omega_h(\mu + k_1\gamma_i), \quad m_2 = \gamma_a(\gamma_r + \mu) + \omega_h(\mu + k_2\gamma_i).$$

Clearly, $\nu > 0$, while $\eta > 0$ only if $R_0^D > 1$. Hence we can say that the system (2) has a unique positive endemic equilibrium point whenever $R_0^D > 1$ and no positive equilibrium point whenever $R_0^D < 1$.

Theorem 3.1. *The model* (2) *has an endemic equilibrium $\varepsilon^\star$ with all positive components provided $R_0^D > 1$.*

Therefore, the endemic equilibrium exists if and only if $R_0^D > 1$, i.e., if and only if the disease-free equilibrium point is unstable. Next, we show that the endemic equilibrium point $\varepsilon^\star$ is locally asymptotically stable for $R_0^D > 1$. In fact, at $R_0^D = 1$ our model undergoes transcritical bifurcation, with $\beta' = \beta'_c$ as a critical bifurcation parameter, defined by

$$\beta' = \beta'_c = \frac{(\kappa + \mu)\omega_h\omega_p\omega_i - l\beta\,\kappa\gamma_a(\rho_2\omega_i + \rho_1\omega_p) - \beta\kappa\rho_1\omega_h\omega_p}{\kappa\rho_2\omega_h\omega_i}, \tag{5}$$

whenever

$$\beta < \beta_c = \frac{\kappa + \mu}{\kappa} \cdot \frac{\omega_h\omega_i\omega_p}{l\gamma_a(\rho_1\omega_p + \rho_2\omega_i) + \rho_1\omega_h\omega_p} \tag{6}$$

Theorem 3.2. *The model* (2) *undergoes transcritical bifurcation, with $\beta' = \beta'_c$, defined by* (5)*, as a critical bifurcation parameter, whenever transmission coefficient due the regular infected individuals β satisfies* (6)*. Moreover, the endemic equilibrium $\varepsilon^\star$ of the system* (2) *is locally asymptotically stable if $R_0^D > 1$.*

Proof. Let $x = (x_1, x_2, x_3, x_4, x_5, x_6, x_7)^T = (S, E, I, P, H, A, R)^T$. Thus, the model (2) can be rewritten in the form

$$\frac{dx}{dt} = f(x), \quad \text{with} \quad f(x) = (f_1(x), \ldots, f_7(x)),$$

as follows:

$$\begin{aligned}
\frac{dx_1}{dt} &= \lambda - \beta\frac{x_3}{X} - \beta'\frac{x_4}{X} - l\beta\frac{x_5}{X} - \mu x_1 \\
\frac{dx_2}{dt} &= \beta\frac{x_3}{X} + \beta'\frac{x_4}{X} + l\beta\frac{x_5}{X} - \sigma_e x_2 \\
\frac{dx_3}{dt} &= \kappa\rho_1 x_2 - \omega_i x_3 \qquad \frac{dx_4}{dt} = \kappa\rho_2 x_2 - \omega_p x_4 \\
\frac{dx_5}{dt} &= \gamma_a x_3 + \gamma_a x_4 - \omega_h x_5 \qquad \frac{dx_6}{dt} = \kappa(1-\rho_1-\rho_2)x_2 - \sigma_a x_6 \\
\frac{dx_7}{dt} &= k_1\gamma_i x_3 + k_2\gamma_i x_4 + \gamma_r x_5 + \gamma_i x_6 - \mu x_7
\end{aligned} \tag{7}$$

where $X = x_1 + \cdots + x_7$, $\sigma_e = \kappa + \mu$, $\sigma_a = \gamma_i + \mu$ and $\omega_h, \omega_i, \omega_p$ are given in (3). The Jacobian $\mathbb{J}^\star$ of the system (7) at the DFE ε_0 is given as

$$\mathbb{J}^\star = \begin{pmatrix}
-\mu & 0 & -\beta & -\beta' & -l\beta & 0 & 0 \\
0 & -\sigma_e & \beta & \beta' & l\beta & 0 & 0 \\
0 & \kappa\rho_1 & -\omega_i & 0 & 0 & 0 & 0 \\
0 & \kappa\rho_2 & 0 & -\omega_p & 0 & 0 & 0 \\
0 & 0 & \gamma_a & \gamma_a & -\omega_h & 0 & 0 \\
0 & \kappa(1-\rho_1-\rho_2) & 0 & 0 & 0 & -\sigma_a & 0 \\
0 & 0 & k_1\gamma_i & k_2\gamma_i & \gamma_r & \gamma_i & -\mu
\end{pmatrix}$$

The characteristic equation of $\mathbb{J}^\star$ corresponding to the eigenvalue λ is $\det(\mathbb{J}^\star - \lambda\,\mathbb{I}_4) = 0$. From the characteristics equation, three eigenvalues of $\mathbb{J}^\star$ are real and negative, that is, $\lambda_1 = \lambda_2 = -\mu$ and $\lambda_3 = -\sigma_a = -\gamma_i - \mu$. The other four eigenvalues can be obtained from the following equation

$$Q(\lambda) = \lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0\,,$$

where

$$\begin{aligned}
a_4 &= \omega_h\omega_i\omega_p\sigma_e(1 - R_0^D) \\
a_3 &= \sigma_e(\omega_h\omega_i + \omega_h\omega_p + \omega_i\omega_p) + \omega_h\omega_i\omega_p \\
&\quad -l\beta\kappa\gamma_a(\rho_1 + \rho_2) - \kappa\omega_h(\beta\rho_1 + \beta'\rho_2) - \kappa(\beta\rho_1\omega_p + \beta'\rho_2\omega_i) \\
a_2 &= \sigma_e(\omega_h + \omega_i + \omega_p) + \omega_h\omega_i + \omega_h\omega_p + \omega_i\omega_p - \kappa(\beta\rho_1 + \beta'\rho_2) \\
a_1 &= \sigma_e + \omega_h + \omega_i + \omega_p
\end{aligned} \tag{8}$$

Next, consider the case when $R_0^D = 1$ and choose β' as a bifurcation parameter. Solving $R_0^D = 1$ by β', we obtain (6). Considering assumption (6) β_c' is positive. Since with $\beta' = \beta_c'$, we have from (8) that $a_4 = 0$ and $a_1 > 0$, $a_1a_2 - a_3 > 0$, so that $\lambda = 0$ is a root of the polynomial $Q(\lambda)$ and Routh-Hurwitz stability criterion implies that all of its other three roots have strictly negative real part. Therefore, the transformed system (7), with $\beta' = \beta_c'$, has a hyperbolic equilibrium point i.e. the Jacobian $\mathbb{J}^\star$ evaluated for $\beta = \beta_c'$, denoted by $\mathbb{J}^\star\big|_{\beta'=\beta_c'}$, has a simple eigenvalue with zero real part and all other eigenvalues have negative real part. Hence, the Centre manifold theory [23, Theorem 4.1] can be used to analyze the dynamics of the model (7) near $\beta' = \beta_c'$.

The Jacobian $J^\star\big|_{\beta=\beta_c'}$ has a right eigenvector (corresponding to the zero eigenvalue) given by $v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)^T$, where

$$v_1 = -\sigma_a\sigma_e\omega_h\omega_i\omega_p,\ v_2 = \mu\sigma_a\omega_h\omega_i\omega_p,\ v_3 = \kappa\mu\rho_1\sigma_a\omega_h\omega_p,\ v_4 = \kappa\mu\rho_2\sigma_a\omega_h\omega_i,$$

$$v_5 = \kappa\mu\gamma_a\sigma_a(\rho_1\omega_p + \rho_2\omega_i),\ v_6 = \kappa\mu(1-\rho_1-\rho_2)\omega_h\omega_i\omega_p,\ v_7 = \kappa\,\Omega$$

$$\Omega = \gamma_a\gamma_r\sigma_a(\rho_1\omega_p + \rho_2\omega_i) + \gamma_i\omega_h\Big(\sigma_a(k_2\rho_2\omega_i + k_1\rho_1\omega_p) + (1-\rho_1-\rho_2)\omega_i\omega_p\Big)$$

and a left eigenvector (corresponding to the zero eigenvalue) given by $u = (u_1, u_2, u_3, u_4, u_5, u_6, u_7)$, where

$$u_1 = u_6 = u_7 = 0,\ u_2 = \kappa\rho_2\omega_h\omega_i,\ u_3 = \beta\kappa\rho_2(l\,\gamma_a + \omega_h),$$

$$u_4 = \sigma_e\omega_h\omega_i - \beta\kappa\rho_1(l\,\gamma_a + \omega_h),\ u_5 = l\,\beta\kappa\rho_2\omega_i$$

Hence, we have

$$\begin{aligned} a &= \sum_{k,i,j=1}^{7} u_k v_i v_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(\varepsilon_0)\Big|_{\beta'=\beta'_c} \\ &= -\frac{2\kappa\sigma_a\sigma_e\rho_2\mu^2\omega_h^2\omega_i^2\omega_p}{\Lambda} \\ &\quad \times \Big[\kappa\,\Omega + \kappa\sigma_a\mu(\gamma_a+\omega_h)(\rho_2\omega_i+\rho_1) + \mu\omega_h\omega_i\omega_p(\kappa(1-\rho_1-\rho_2)+\sigma_a)\Big] < 0 \\ b &= \sum_{k,i=1}^{7} u_k v_i \frac{\partial^2 f_k}{\partial x_i \partial \beta'}(\varepsilon_0)\Big|_{\beta'=\beta'_c} = \mu\sigma_a(\kappa\rho_2\omega_h\omega_i)^2 > 0 \end{aligned}$$

Thus, using Theorem 4.1 in [23], a transcritical bifurcation occurs at $R_0^D = 1$ and unique endemic equilibrium is locally asymptotically stable for $R_0^D > 1$. □

The results in Theorem 3.2 is numerically illustrated by simulating the model (2) using the following set of parameter values

$$\begin{cases} \rho_1 = 0.58;\ \rho_2 = 0.001;\ \gamma_a = 0.94;\ \gamma_i = 0.27;\ \gamma_r = 0.5;\ \delta_i = \delta_p = \delta_h = \frac{1}{23}; \\ k_1 = 0.85;\ k_2 = 0.95;\ \mu = 0.00714;\ \kappa = 0.25\,. \end{cases} \quad \leftarrowtail \tag{9}$$

The result obtained is depicted in Fig. 1, by plotting the force of COVID-19 infection $F^\star$ as a function of the basic reproduction number $R_0^D \in [0, 2]$.

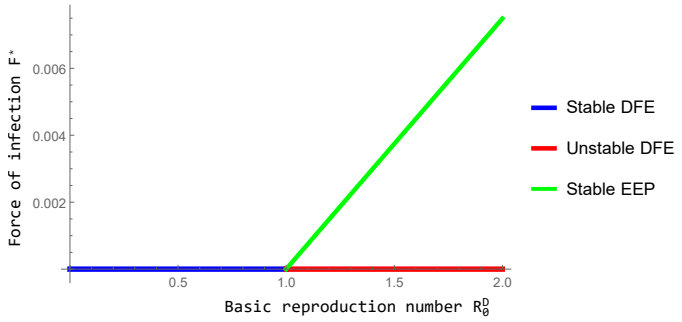


Figure 1: Transcritical bifurcation for the force of infection $F^\star$ against basic reproduction number R_0^D of the model (2), using the parameter values in (9)

Taking $l = 0.97$, so that $\beta_c = 0.802476$ and to satisfy the condition (6) we take $\beta = 0.8$, giving that the value of a critical bifurcation parameter is $\beta'_c = 3.9353$. Thus, the force of COVID-19 infection $F^\star$ as a function of superspreaders transmission coefficient $\beta' \in [3.6, 4.3]$ is displayed in Fig. 2.

4. Analysis of the stochastic model

Suppose that independent Brownian motions $B_1 = \{B_1(t),\ t \geq 0\}$ and $B_2 = \{B_2(t),\ t \geq 0\}$, that govern the SDE system (1), are defined on the complete probability space $(\Omega, \mathcal{F}, \mathbb{F}, \mathbb{P})$ with filtration $\mathbb{F} = \{\mathcal{F}_t,\ t \geq 0\}$, where $\mathcal{F}_t := \mathcal{F}_t^{B_1} \cup \mathcal{F}_t^{B_2}$, where $\mathcal{F}_t^{B_i}$, $i = 1, 2$, are σ-algebras derived from natural filtrations of Brownian motions B_1 and B_2 and they contain all $\mathbb{P}$-null sets.

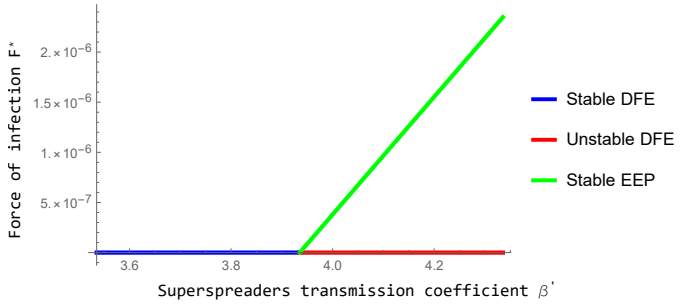


Figure 2: Transcritical bifurcation for the force of infection $F^\star$ against superspreaders transmission coefficient β' of the model (2), using the parameter values in (9) and $l = 0.97$, $\beta = 0.8$.

Let us define

$$R_0^S = \frac{(\beta + \beta')\frac{\Lambda}{\mu}}{\kappa + \mu + \frac{1}{2}\left(\sigma_1^2 + \sigma_2^2\right)\frac{\Lambda^2}{\mu^2}}. \tag{10}$$

4.1. Extinction

Theorem 4.1. *For any initial value* $(S(0), E(0), I(0), P(0), A(0), H(0), R(0)) \in \Gamma^\star$ *such that the solution of the system* (1) *is in* $\Gamma^\star$*, if one of the following conditions is satisfied*

1. $\sigma_1^2 \le \beta\frac{4\mu}{\Lambda}\max\{1, l\}$, $\sigma_2^2 \le \beta'\frac{4\mu}{\Lambda}$ *and* $R_0^S < 1$, *where* R_0^S *is defined with* (10),
2. $\frac{\frac{\beta^2}{\sigma_1^2} + \frac{(\beta')^2}{\sigma_2^2}}{2(\kappa+\mu)} < 1$,

then the disease goes to extinction with probability one i.e.,

$$E(t) + I(t) + P(t) + H(t) + A(t) \to 0 \quad \mathbb{P} - a.s.\ as\ t \to \infty,$$

while

$$\limsup_{t\to\infty} S(t) = \frac{\Lambda}{\mu} \quad \mathbb{P} - a.s.$$

Proof.

1. The proof of the first part of the theorem is derived following the idea from [24]. Let us assume that $\sigma_1^2 \le \beta\frac{4\mu}{\Lambda}\max\{1, l\}$, $\sigma_2^2 \le \beta'\frac{4\mu}{\Lambda}$ and $R_0^S < 1$. Applying the Itô formula (see e.g. [25]) to $\log E(t)$ we obtain

$$\begin{aligned} d(\log E(t)) &= \frac{1}{E(t)}\left(\frac{\beta}{N(t)}(I(t) + lH(t))S(t) + \frac{\beta'}{N(t)}P(t)S(t) - (\kappa+\mu)E(t)\right)dt \\ &\quad - \frac{S^2(t)}{2E^2(t)N^2(t)}\left(\sigma_1^2(I(t) + lH(t))^2 + \sigma_2^2P^2(t)\right)dt \\ &\quad + \frac{S(t)}{E(t)N(t)}(\sigma_1(I(t) + lH(t))dB_1(t) + \sigma_2P(t)dB_2(t)) \\ &\le \left(\beta\frac{\Lambda}{\mu} + \beta'\frac{\Lambda}{\mu} - (\kappa+\mu) - \frac{1}{2}\left(\sigma_1^2 + \sigma_2^2\right)\frac{\Lambda^2}{\mu^2}\right)dt + \sigma_1(1+l)dB_1(t) + \sigma_2dB_2(t) \\ &=: R^*dt + \sigma_1(1+l)dB_1(t) + \sigma_2dB_2(t), \end{aligned} \tag{11}$$

where we have used the fact that functions $f_1(x) = \beta x - \frac{1}{2}\sigma_1^2x^2$ (where $x = \frac{S(t)(I(t)+lH(t))}{E(t)N(t)}$) and $f_2(y) = \beta' y - \frac{1}{2}\sigma_2^2y^2$ (where $y = \frac{S(t)P(t)}{E(t)N(t)}$) are increasing on $\left[0, \frac{\beta}{\sigma_1^2}\right]$ and $\left[0, \frac{\beta'}{\sigma_2^2}\right]$ and boundedness of the solution. Integrating the last expression from 0 to t and dividing by t yield the following result

$$\frac{\log E(t) - \log E(0)}{t} \le \frac{1}{t}\int_0^t R^*dt + \frac{M_1(t)}{t} + \frac{M_2(t)}{t} \tag{12}$$

where

$$M_1(t) = \sigma_1 \int_0^t (1+l)\, dB_1(s), \quad M_2(t) = \sigma_2 \int_0^t dB_2(s).$$

are continuous local martingales with values 0 at time $t = 0$ and

$$\limsup_{t\to\infty} \frac{\langle M_1, M_1\rangle_t}{t} < \infty, \qquad \limsup_{t\to\infty} \frac{\langle M_2, M_2\rangle_t}{t} < \infty.$$

Therefore,

$$\frac{M_1(t)}{t} \to 0 \quad \text{and} \quad \frac{M_2(t)}{t} \to 0 \quad \mathbb{P}-\text{a.s. as } t \to \infty. \tag{13}$$

Taking the superior limit on the both sides of expression (12) we obtain

$$\begin{aligned} \limsup_{t\to\infty} \tfrac{\log E(t)}{t} &\leq R^* = (\beta+\beta')\tfrac{\Lambda}{\mu} - \left(\kappa+\mu+\tfrac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\tfrac{\Lambda^2}{\mu^2}\right) \\ &= \left(\kappa+\mu+\tfrac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\tfrac{\Lambda^2}{\mu^2}\right)\left(R_0^S - 1\right) < 0, \ a.s. \end{aligned} \tag{14}$$

which implies

$$\lim_{t\to\infty} E(t) = 0, \ a.s.$$

To verify that $I(t), P(t), H(t), A(t), R(t) \to 0$, $\mathbb{P}$-a.s. as $t \to \infty$ we solve equations $3-7$ from the system (1) explicitly:

$$\begin{aligned} I(t) &= e^{-\left(\gamma_a+k_1\gamma_i+\delta_i\right)t} I(0) + \kappa\rho_1[E(t)], \\ P(t) &= e^{-\left(\gamma_a+k_2\gamma_i+\delta_p\right)t} P(0) + \kappa\rho_2[E(t)], \\ H(t) &= e^{-\left(\gamma_r+\delta_h\right)t} H(0) + \gamma_a[I(t)+P(t)], \\ A(t) &= e^{-\left(\gamma_i+\mu\right)t} A(0) + \kappa\left(1-\rho_1-\rho_2\right)[E(t)], \\ R(t) &= e^{-\mu t} R(0) + \gamma_i[I(t)+P(t)+A(t)] + \gamma_r[H(t)]. \end{aligned} \tag{15}$$

Since $E(t) \to 0$, $\mathbb{P}$-a.s. as $t \to \infty$, from previous solutions it follows that $I(t) \to 0$, $P(t) \to 0$ and $A(t) \to 0$, $\mathbb{P}$- a.s. as $t \to \infty$. Furthermore, it follows that $H(t) \to 0$ and $R(t) \to 0$, $\mathbb{P}$-a.s. as $t \to \infty$.
Recall that

$$N(t) = S(t) + E(t) + I(t) + P(t) + H(t) + A(t) + R(t),$$

and since $E(t) + I(t) + P(t) + H(t) + A(t) + R(t) \to 0$, it follows that

$$\limsup_{t\to\infty} S(t) = \limsup_{t\to\infty} N(t) = \frac{\Lambda}{\mu}, \quad \mathbb{P}-\text{a.s.},$$

where we applied the results of Theorem 1 in [19].

2. The proof of the second part of the theorem can be found in [19].

This completes the proof.

■

4.2. Persistence in mean

The virus remains persistent in population if at least one of the classes of symptomatic infectious, asymptomatic infectious, hospitalized infectious or super-spreader is not empty. From the mathematical point of view, in accordance with [19], we say that system (1) is persistent in mean if

$$\lim_{t\to\infty}\frac{1}{t}\int_0^t (I(s)+P(s)+A(s)+H(s))\,ds>0, \qquad \mathbb{P}-\text{a.s.}\,. \tag{16}$$

Let us introduce the notation

$$[x(t)]=\frac{1}{t}\int_0^t x(s)\,ds.$$

Theorem 4.2. *Let initial value* $(S(0),E(0),I(0),P(0),A(0),H(0),R(0))\in\Gamma^\star$, *such that the solution of the system* (1) *is in* $\Gamma^\star$. *Assume that one of the following conditions is satisfied*

1. $R_0^S>1$, *where* R_0^S *is defined with* (10),
2. μ,β,β' *and* l *satisfy the relation*

$$\Lambda>\left(\frac{\beta}{N(t)}\left(I(t)+lH(t)\right)+\frac{\beta'}{N(t)}P(t)+\mu\right)S(t),\quad \forall t\geq 0,$$

$\inf_{t\geq 0}E(t)/N(t)\geq c$ *where* c *is a small fixed constant and*

$$\sigma_1^2+\sigma_2^2<c\kappa\left(\rho_1\frac{\gamma_r+\gamma_a+\delta_p}{(\gamma_a+k_1\gamma_i+\delta_i)(\gamma_r+\delta_p)}+\rho_2\frac{\gamma_r+\gamma_a+\delta_p}{(\gamma_a+k_2\gamma_i+\delta_p)(\gamma_r+\delta_p)}+\frac{1-\rho_1-\rho_2}{\gamma_i+\mu}\right),$$

then the solution $(S(t),E(t),I(t),P(t),A(t),H(t),R(t))$ *of the system* (1) *is persistent in mean.*
More precisely, if

1. *condition* $R_0^S>1$, *is satisfied, than the solution* $(S(t),E(t),I(t),P(t),A(t),H(t),R(t))$ *has the property*

$$\begin{aligned}
\liminf_{t\to\infty}\tfrac{1}{t}\textstyle\int_0^t E(t)\,dt &\geq C\left(R_0^S-1\right)>0,\ a.s.\\
\liminf_{t\to\infty}\tfrac{1}{t}\textstyle\int_0^t I(t)\,dt &\geq C\frac{\kappa\rho_1}{\gamma_a+k_1\gamma_i+\delta_i}\left(R_0^S-1\right)>0,\ a.s.\\
\liminf_{t\to\infty}\tfrac{1}{t}\textstyle\int_0^t P(t)\,dt &\geq C\frac{\kappa\rho_2}{\gamma_a+k_2\gamma_i+\delta_p}\left(R_0^S-1\right)>0,\ a.s.\\
\liminf_{t\to\infty}\tfrac{1}{t}\textstyle\int_0^t H(t)\,dt &\geq C\left(\frac{\kappa\rho_1}{\gamma_a+k_1\gamma_i+\delta_i}+\frac{\kappa\rho_2}{\gamma_a+k_2\gamma_i+\delta_p}\right)\frac{\gamma_a}{\gamma_r+\delta_h}\left(R_0^S-1\right)>0,\ a.s.\\
\liminf_{t\to\infty}\tfrac{1}{t}\textstyle\int_0^t A(t)\,dt &\geq C\frac{\kappa\left(1-\rho_1-\rho_2\right)}{\gamma_i+\mu}\left(R_0^S-1\right)>0,\ a.s.\\
\liminf_{t\to\infty}\tfrac{1}{t}\textstyle\int_0^t R(t)\,dt &\geq C\frac{1}{\mu}\left(\left(\gamma_i k_1+\frac{\gamma_a\gamma_r}{\gamma_r+\delta_h}\right)\frac{\kappa\rho_1}{\gamma_a+k_1\gamma_i+\delta_i}+\left(\gamma_i k_2+\frac{\gamma_a\gamma_r}{\gamma_r+\delta_h}\right)\frac{\kappa\rho_2}{\gamma_a+k_2\gamma_i+\delta_p}+\frac{\kappa\left(1-\rho_1-\rho_2\right)\gamma_i}{\gamma_i+\mu}\right)\\
&\quad\left(R_0^S-1\right)>0,\ a.s.
\end{aligned}$$

where $C=\dfrac{\kappa+\mu+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\frac{\Lambda^2}{\mu^2}}{\left(\frac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\frac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}$ *is a positive constant;*

2. μ,β,β' *and* l *satisfy the relation*

$$\Lambda>\left(\frac{\beta}{N(t)}\left(I(t)+lH(t)\right)+\frac{\beta'}{N(t)}P(t)+\mu\right)S(t),\quad \forall t\geq 0,$$

$\inf_{t\geq 0} E(t)/N(t) \geq c$ *where* c *is a small fixed constant and*

$$\sigma_1^2+\sigma_2^2 < c\kappa\left(\rho_1\frac{\gamma_r+\gamma_a+\delta_p}{(\gamma_a+k_1\gamma_i+\delta_i)(\gamma_r+\delta_p)}+\rho_2\frac{\gamma_r+\gamma_a+\delta_p}{(\gamma_a+k_2\gamma_i+\delta_p)(\gamma_r+\delta_p)}+\frac{1-\rho_1-\rho_2}{\gamma_i+\mu}\right),$$

then the solution $(S(t),E(t),I(t),P(t),A(t),H(t),R(t))$ *has the property*

$$\liminf_{t\to\infty}[I(t)+P(t)+H(t)+A(t)] \geq$$

$$c\left(\kappa\rho_1\frac{\gamma_r+\gamma_a+\delta_p}{(\gamma_a+k_1\gamma_i+\delta_i)(\gamma_r+\delta_p)}+\kappa\rho_2\frac{\gamma_r+\gamma_a+\delta_p}{(\gamma_a+k_2\gamma_i+\delta_p)(\gamma_r+\delta_p)}+\frac{\kappa(1-\rho_1-\rho_2)}{\gamma_i+\mu}-\frac{\left(\sigma_1^2+\sigma_2^2\right)}{c}\right)>0.$$

Proof.

1. The proof of the first part of the theorem is derived following the idea from [26]. Applying the Itô formula to the function $-\log E(t)$ we obtain

$$\begin{aligned}
d\left(-\log E(t)\right) &= \tfrac{1}{E(t)}\left(-\tfrac{\beta}{N(t)}\left(I(t)+lH(t)\right)S(t)-\tfrac{\beta'}{N(t)}P(t)S(t)+(\kappa+\mu)E(t)\right)dt\\
&\quad+\tfrac{S^2(t)}{2E^2(t)N^2(t)}\left(\sigma_1^2\left(I(t)+lH(t)\right)^2+\sigma_2^2P^2(t)\right)dt\\
&\quad-\tfrac{S(t)}{E(t)N(t)}\left(\sigma_1\left(I(t)+lH(t)\right)dB_1(t)+\sigma_2P(t)dB_2(t)\right)\\
&\leq \left(\kappa+\mu+\tfrac{S^2(t)}{2E^2(t)N^2(t)}\sigma_1^2\left(I(t)+lH(t)\right)^2-\tfrac{\beta}{E(t)}\left(I(t)+lH(t)\right)\right.\\
&\quad+\tfrac{\beta(E(t)+I(t)+P(t)+H(t)+A(t)+R(t))}{N(t)E(t)}\left(I(t)+lH(t)\right)\\
&\quad\left.+\tfrac{S^2(t)}{2E^2(t)N^2(t)}\sigma_2^2P^2(t)-\tfrac{\beta'}{E(t)}P(t)+\tfrac{\beta'(E(t)+I(t)+P(t)+H(t)+A(t)+R(t))}{N(t)E(t)}P(t)\right)dt\\
&\quad-\tfrac{S(t)}{E(t)N(t)}\left(\sigma_1\left(I(t)+lH(t)\right)dB_1(t)+\sigma_2P(t)dB_2(t)\right)\\
&\leq \left(\kappa+\mu+\tfrac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\tfrac{\Lambda^2}{\mu^2}-\beta\tfrac{\Lambda}{\mu}-\beta'\tfrac{\Lambda}{\mu}+\beta\left(I(t)+lH(t)\right)+\beta'P(t)\right)dt\\
&\quad+\sigma_1\left(1+l\right)dB_1(t)+\sigma_2dB_2(t),
\end{aligned}$$

where we have used the fact that functions $f_1(x)=-\beta x+\frac{S^2(t)}{2N^2(t)}\sigma_1^2x^2$ (where $x=\frac{I(t)+lH(t)}{E(t)}$) and $f_2(y)=-\beta' y+\frac{S^2(t)}{2N^2(t)}\sigma_2^2y^2$ (where $y=\frac{P(t)}{E(t)}$) are decreasing on $\left[0,\frac{\beta}{\sigma_1^2}\right]$ and $\left[0,\frac{\beta'}{\sigma_2^2}\right]$ and increasing on $\left[\frac{\beta}{\sigma_1^2},\frac{\Lambda}{\mu}\right]$ and $\left[\frac{\beta'}{\sigma_2^2},\frac{\Lambda}{\mu}\right]$. Let us define a Lyapunov function $U:\Gamma^\star\Rightarrow\mathbb{R}$ as follows:

$$U(t)=-\log E(t)+\frac{\beta\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}{\gamma_a+k_1\gamma_i+\delta_i}I(t)+\frac{\beta'\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}{\gamma_a+k_2\gamma_i+\delta_p}P(t)+\frac{\beta l}{\gamma_r+\delta_h}H(t).$$

Then

$$\begin{aligned}
dU(t) &= d(-\log E(t))+\tfrac{\beta\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}{\gamma_a+k_1\gamma_i+\delta_i}d(I(t))+\tfrac{\beta'\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}{\gamma_a+k_2\gamma_i+\delta_p}d(P(t))+\tfrac{\beta l}{\gamma_r+\delta_h}d(H(t))\\
&\leq \left(\kappa+\mu+\tfrac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\tfrac{\Lambda^2}{\mu^2}-\beta\tfrac{\Lambda}{\mu}-\beta'\tfrac{\Lambda}{\mu}+\left(\tfrac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\tfrac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\tfrac{l\gamma_a}{\gamma_r+\delta_h}\right)E(t)\right)dt\\
&\quad+\sigma_1\left(1+l\right)dB_1(t)+\sigma_2dB_2(t)\\
&= -\left(\kappa+\mu+\tfrac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\tfrac{\Lambda^2}{\mu^2}\right)\left(R_0^S-1\right)dt+\left(\tfrac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\tfrac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\tfrac{l\gamma_a}{\gamma_r+\delta_h}\right)E(t)\,dt\\
&\quad+\sigma_1\left(1+l\right)dB_1(t)+\sigma_2dB_2(t),
\end{aligned}$$

where

$$R_0^S=\frac{\left(\beta+\beta'\right)\frac{\Lambda}{\mu}}{\kappa+\mu+\frac{1}{2}\left(\sigma_1^2+\sigma_1^2\right)\frac{\Lambda^2}{\mu^2}}.$$

And thus

$$\begin{aligned} dU(t) &\leq -\left(\kappa+\mu+\tfrac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\tfrac{\Lambda^2}{\mu^2}\right)\left(R_0^S-1\right)dt+\left(\tfrac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\tfrac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\tfrac{l\gamma_a}{\gamma_r+\delta_h}\right)E(t)\,dt \\ &\quad +\sigma_1\left(1+l\right)dB_1(t)+\sigma_2 dB_2(t). \end{aligned}$$

Integrating the both sides of the last expression from 0 to t and dividing by t we obtain

$$\begin{aligned} \tfrac{U(t)-U(0)}{t} &\leq -\left(\kappa+\mu+\tfrac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\tfrac{\Lambda^2}{\mu^2}\right)\left(R_0^S-1\right)+\left(\tfrac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\tfrac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\tfrac{l\gamma_a}{\gamma_r+\delta_h}\right)\tfrac{1}{t}\int_0^t E(t)\,dt \\ &\quad +\tfrac{1}{t}\int_0^t \sigma_1\left(1+l\right)\,dB_1(t)+\tfrac{1}{t}\int_0^t \sigma_2\,dB_2(t). \end{aligned} \tag{17}$$

Since $N(t)=S(t)+E(t)+I(t)+P(t)+H(t)+A(t)+R(t)\leq\frac{\Lambda}{\mu}$, it follows that

$$U(t)=-\log E(t)+\frac{\beta\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}{\gamma_a+k_1\gamma_i+\delta_i}I(t)+\frac{\beta'\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}{\gamma_a+k_2\gamma_i+\delta_p}P(t)+\frac{\beta l}{\gamma_r+\delta_h}H(t)\geq-\log E(t)\geq-\log\frac{\Lambda}{\mu}. \tag{18}$$

Taking the inferior limit on both sides of (17) and using (18) and (13), we get

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t E(t)\,dt\geq\frac{\left(\kappa+\mu+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\frac{\Lambda^2}{\mu^2}\right)\left(R_0^S-1\right)}{\left(\frac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\frac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}>0,\ a.s. \tag{19}$$

since the condition $R_0^S>1$ holds.
On the other hand, by integrating both sides of the third equation of system (1) from 0 to t and dividing by t we obtain

$$\tfrac{I(t)-I(0)}{t}=\tfrac{1}{t}\int_0^t\kappa\rho_1E(t)\,dt-\tfrac{1}{t}\int_0^t\left(\gamma_a+k_1\gamma_i+\delta_i\right)I(t)\,dt. \tag{20}$$

Taking the inferior limit on both sides of (20) and using (19), we get

$$\liminf_{t\to\infty}\tfrac{1}{t}\int_0^t I(t)\,dt\geq\frac{\kappa\rho_1\left(\kappa+\mu+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\frac{\Lambda^2}{\mu^2}\right)\left(R_0^S-1\right)}{\left(\gamma_a+k_1\gamma_i+\delta_i\right)\left(\frac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\frac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}>0,\ a.s. \tag{21}$$

Similarly we can prove the persistence in the mean of the populations $P(t)$ and $A(t)$. Integrating both sides of the fourth equation of system (1) from 0 to t and dividing by t we obtain

$$\tfrac{P(t)-P(0)}{t}=\tfrac{1}{t}\int_0^t\kappa\rho_2E(t)\,dt-\tfrac{1}{t}\int_0^t\left(\gamma_a+k_2\gamma_i+\delta_p\right)I(t)\,dt. \tag{22}$$

Taking the inferior limit on both sides of (22) and using (19), we get

$$\liminf_{t\to\infty}\tfrac{1}{t}\int_0^t P(t)\,dt\geq\frac{\kappa\rho_2\left(\kappa+\mu+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\frac{\Lambda^2}{\mu^2}\right)\left(R_0^S-1\right)}{\left(\gamma_a+k_2\gamma_i+\delta_p\right)\left(\frac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\frac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}>0,\ a.s. \tag{23}$$

Integrating both sides of the sixth equation of system (1) from 0 to t and dividing by t we obtain

$$\tfrac{A(t)-A(0)}{t}=\tfrac{1}{t}\int_0^t\kappa\left(1-\rho_1-\rho_2\right)E(t)\,dt-\tfrac{1}{t}\int_0^t\left(\gamma_i+\mu\right)A(t)\,dt. \tag{24}$$

Taking the inferior limit on both sides of (24) and using (19), we get

$$\liminf_{t\to\infty}\tfrac{1}{t}\int_0^t A(t)\,dt\geq\frac{\kappa\left(1-\rho_1-\rho_2\right)\left(\kappa+\mu+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\frac{\Lambda^2}{\mu^2}\right)\left(R_0^S-1\right)}{\left(\gamma_i+\mu\right)\left(\frac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\frac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}>0,\ a.s. \tag{25}$$

We can prove that, when the populations I and P are persistent in the mean a.s., then the population H will also be persistent in the mean a.s. Indeed, by integrating both sides of the fifth equation of system (1) from 0 to t and dividing by t we obtain

$$\frac{H(t)-H(0)}{t} = \frac{1}{t}\int_0^t \gamma_a\left(I(t)+P(t)\right)\,dt - \frac{1}{t}\int_0^t \left(\gamma_r+\delta_h\right)H(t)\,dt. \tag{26}$$

Taking the inferior limit on both sides of (26) and using (21) and (23), we get

$$\liminf_{t\to\infty}\frac{1}{t}\int_0^t H(t)\,dt \geq \left(\frac{\kappa\rho_1}{\gamma_a+k_1\gamma_i+\delta_i}+\frac{\kappa\rho_2}{\gamma_a+k_2\gamma_i+\delta_p}\right)\frac{\gamma_a\left(\kappa+\mu+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\frac{\Lambda^2}{\mu^2}\right)\left(R_0^S-1\right)}{\left(\gamma_r+\delta_h\right)\left(\frac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\frac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)} > 0,\ a.s. \tag{27}$$

Persistence in the mean a.s. of the populations I, P, H and A would imply persistence in the mean a.s. of the population R. Indeed, by integrating both sides of the seventh equation of system (1) from 0 to t and dividing by t we obtain

$$\frac{R(t)-R(0)}{t} = \frac{1}{t}\int_0^t \left(\gamma_i\left(A(t)+k_1I(t)+k_2P(t)\right)+\gamma_rH(t)\right)\,dt - \frac{1}{t}\int_0^t \mu R(t)\,dt. \tag{28}$$

Taking the inferior limit on both sides of (28) and using (21), (23), (27) and (25), we get

$$\begin{aligned}\liminf_{t\to\infty}\frac{1}{t}\int_0^t R(t)\,dt \quad \geq \quad & C\frac{1}{\mu}\left(\left(\gamma_ik_1+\frac{\gamma_a\gamma_r}{\gamma_r+\delta_h}\right)\frac{\kappa\rho_1}{\gamma_a+k_1\gamma_i+\delta_i}+\left(\gamma_ik_2+\frac{\gamma_a\gamma_r}{\gamma_r+\delta_h}\right)\frac{\kappa\rho_2}{\gamma_a+k_2\gamma_i+\delta_p}+\frac{\kappa\left(1-\rho_1-\rho_2\right)\gamma_i}{\gamma_i+\mu}\right)\\ & \left(R_0^S-1\right) > 0,\ a.s.\end{aligned}$$

where $C = \dfrac{\kappa+\mu+\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\frac{\Lambda^2}{\mu^2}}{\left(\frac{\beta}{\gamma_a+k_1\gamma_i+\delta_i}+\frac{\beta'}{\gamma_a+k_2\gamma_i+\delta_p}\right)\left(1+\frac{l\gamma_a}{\gamma_r+\delta_h}\right)}$. This completes the proof of the first part of the theorem.

2. The proof of the second part of the theorem can be found in [19].

■

Remark 4.3. *Regarding Theorems* (4.1) *and* (4.2)*, we know that: if* $\sigma_1^2 \leq \beta\frac{4\mu}{\Lambda}\max\{1,l\}$, $\sigma_2^2 \leq \beta'\frac{4\mu}{\Lambda}$ *and* $R_0^S < 1$*, the disease* $I(t)$ *will go to extinction a.s; while if* $R_0^S > 1$*, the disease* $I(t)$ *will be persistent in mean with probability one. That is, for the small values of noises* σ_1 *and* σ_2*,* R_0^S *can be used as a threshold parameter to determines when the infection will vanish or persist in a new host population. Actually, this is the same epidemiological meaning as the basic reproduction number* R_0 *has in deterministic epidemic models. Hence, we can call* R_0^S *the stochastic threshold for the SDE model* (1) *and interpret it as a counterpart of the deterministic basic reproduction number.*

5. Sensitivity analysis of basic reproduction number of deterministic model and its stochastic model counterpart

Deterministic basic reproduction number R_0^D and the stochastic model related threshold R_0^S for models 2 and 1 are compared regarding the values of the respective normalized forward sensitivity indices. Normalized forward sensitivity index measures a relative change in the basic reproduction numbers R_0^i, $i \in \{D, S\}$, with respect to the change in parameter value and gives the overall information regarding the robustness of the model to these changes. Furthermore, it is used to discover parameters that have a high impact on the basic reproduction numbers and that should be targeted by specific epidemiological intervention strategies.

More precisely, normalized forward sensitivity index is the ratio of the relative change in the basic reproduction numbers R_0^i to the relative change in the parameter θ, assuming that R_0^i is differentiable with respect to parameter:

$$\Upsilon_\theta^{R_0^i} = \frac{dR_0^i}{d\theta}\frac{\theta}{R_0^i}, \quad i \in \{D, S\}, \tag{29}$$

where R_0^i is treated as a function of the parameter θ.

Since both reproduction numbers R_0^D and R_0^S are rational functions of model parameters, the normalized forward sensitivity index exists for all model parameters appearing in the explicit formula defining these basic reproduction numbers. In order to calculate the value of sensitivity indices we use parameter values (on the per-person-per-day scale) that are either based on the China demographic data and epidemiological data from Wuhan in February 2020 (see [20], [6] and [21]) or rationally assumed (marked with [a]), see Tables 1 and 2.

Symbol	Description	Value	Units
σ_1	Intensity of Brownian motion B_1 due to infected class	0.0005 [a]	dimensionless
σ_2	Intensity of Brownian motion B_2 due to superspreaders	0.001 [a]	dimensionless

Table 2: Assumed values of intensities σ_1 and σ_2.

The values of the sensitivity index for parameter values from Tables 1 and 2 are given in Table 3.

Parameter	R_0^D - value of sensitivity index	R_0^S - value of sensitivity index
Λ	$+1.0000$	-1.0000
β	0.9986	0.2500
l	0.7289	-0.2500
β'	0.0014	0.7500
κ	0.0001	$-1.5928 \cdot 10^{-9}$
ρ_1	0.9974	-0.2500
ρ_2	0.0026	-0.7500
γ_a	0.0459	-0.2500
γ_r	-0.6706	-0.2500
γ_i	-0.1892	-0.2500
k_1	-0.1887	-0.2500
k_2	-0.0005	-0.2500
δ_i	-0.0358	-0.2500
δ_p	-0.0001	-0.2500
δ_h	-0.0583	-0.2500
μ	-0.0001	1.0000
σ_1	$+0.4000$	-0.4000
σ_2	$+1.6000$	-1.6000

Table 3: Sensitivity of R_0^D and R_0^S for parameter values given in Tables 1 and 2.

Values of the deterministic basic reproduction number and the corresponding stochastic threshold for parameter values given in Tables 1 and 2 are

$$R_0^D = 4.5206,$$

$$R_0^S = 1.0298.$$

However, a moderate decrease of the intensity $\sigma_2 = 0.0001$ of the Brownian motion related to the dynamics of superspreaders results in a huge rise in the value of basic reproduction number:

$$R_0^S = 4.9511,$$

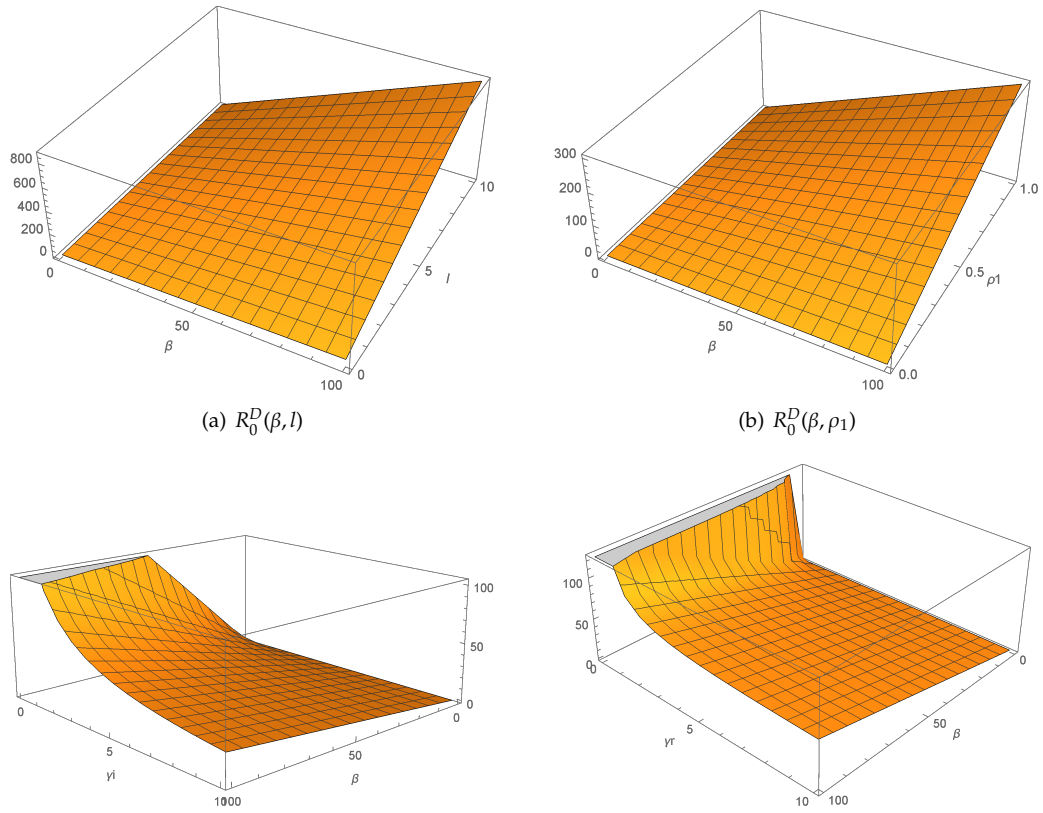
indicating the importance of stochasticity, superspreaders and superspreading events in the dynamics of the epidemics. For more discussion on the role of stochasticity in reducing the basic reproduction number related to a specific infectious disease model we refer to [27] and [11].

From Table 3 we conclude that the deterministic basic reproduction number R_0^D is the most sensitive to change in values of parameters β, ρ_1, l, γ_i and γ_r. The impact of the 10% increase in one parameter value, keeping all other parameters fixed, to the value of R_0^D for these five parameters is given by values in Table 4.

Parameter	Value of R_0^D	Relative change in R_0^D (%)
β	4.9720	+9.98
ρ_1	4.9715	+9.97
l	4.8501	+7.29
γ_i	4.4366	−1.86
γ_r	4.2429	−6.14

Table 4: Change of R_0^D under the 10% increase in values of parameters β, ρ_1, l, γ_i and γ_r.

Note that the signs of the sensitivity indices for parameters with the strongest impact on R_0^D are expected. For example, the increase of transmission coefficient β due to infected individuals for just 10% results in the increase of the value of R_0^D for 9.98%. Furthermore, 10% increase of the recovery rate for hospitalized patients γ_r results in the decrease of R_0^D for 6.14%, which could be interpreted in terms of the contribution of recovered individuals to the population immunity. The behavior of reproduction number R_0^D with respect to parameter values with high sensitivity index is shown in Figure 3.



(a) $R_0^D(\beta, l)$ (b) $R_0^D(\beta, \rho_1)$

(c) $R_0^D(\beta, \gamma_i)$ (d) $R_0^D(\beta, \gamma_r)$

Figure 3: R_0^D as a function of parameters with high sensitivity index.

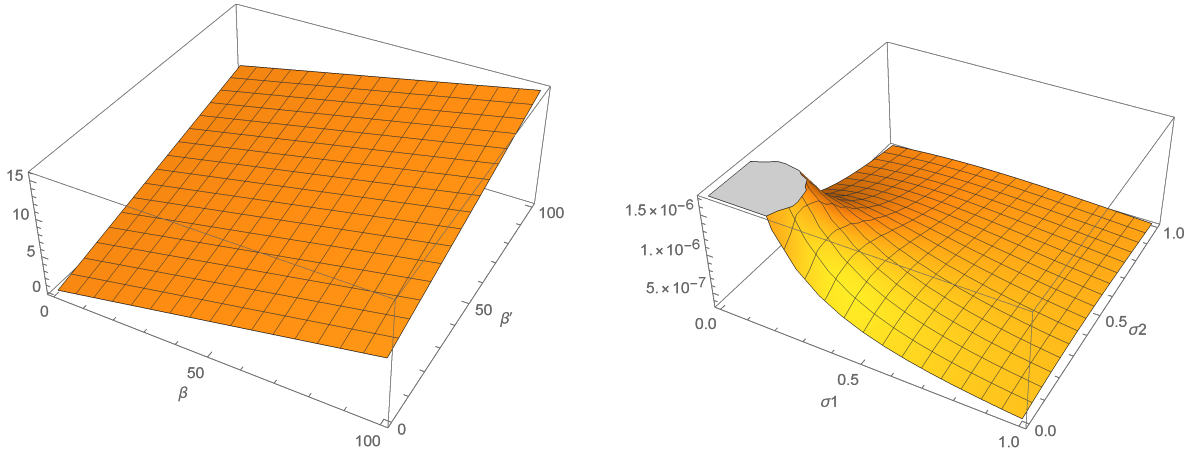
Furthermore, from Table 3 we see that the threshold R_0^S related to the stochastic model is very sensitive to change of all of its parameters, except parameter κ. Here we point out the structure of the sensitivity index with respect to transmission rates β and β'

$$\Upsilon_{\beta}^{s} = \frac{\beta}{\beta + \beta'}, \quad \Upsilon_{\beta'}^{s} = \frac{\beta'}{\beta + \beta'},$$

since

$$\Upsilon_{\beta}^{s} + \Upsilon_{\beta'}^{s} = 1,$$

regardless of the value of parameters β and β'. As expected, sensitivity indices regarding noise intensities σ_1 and σ_2 are analytically the same, reflecting the fact that the value of R_0^S is invariant under the replacement of values of σ_1 and σ_2. This observation is of pure theoretical nature, since the nature of intensities σ_1 (infected class) and σ_2 (superspreaders) implies that $\sigma_1 < \sigma_2$. The behavior of the basic reproduction number R_0^S as a function of parameters σ_1 and σ_2, as well as parameters β and β', is shown in Figure 4.



(a) $R_0^S(\beta, \beta')$ (b) $R_0^S(\sigma_1, \sigma_2)$

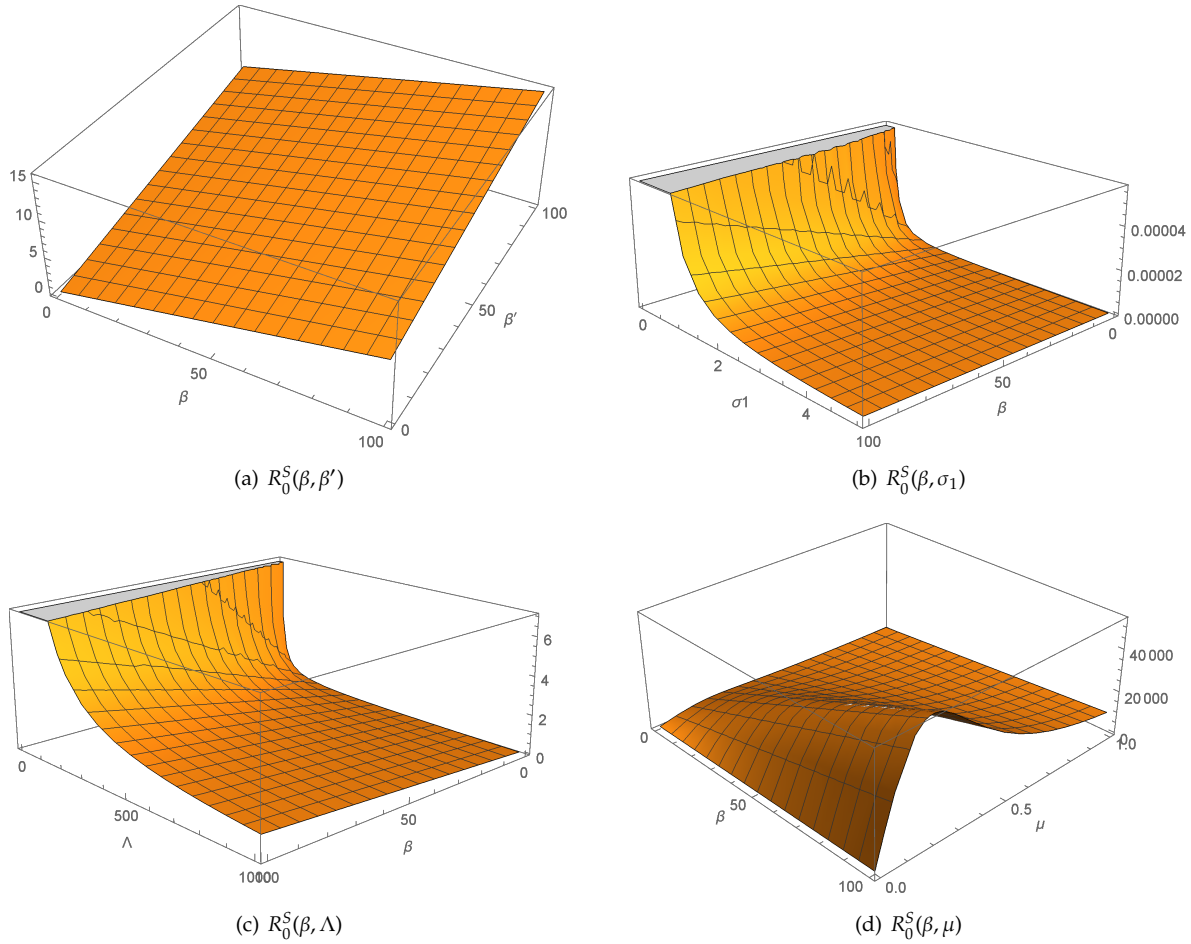
Figure 4: R_0^S as a function of parameters

The impact of the 10% increase in one parameter value, keeping all other parameters fixed, to the value of R_0^S is given in Table 5.

Parameter	Value of R_0^S	Relative change in R_0^S (%)
β'	0.8627	+7.51
β	0.8226	+2.51
σ_1	0.7927	−4.03
σ_2	0.6701	−14.38

Table 5: Change of R_0^S under the 10% increase in values of parameters β', β, σ_1 and σ_2.

Note that, unlike R_0^D, R_0^S depends on the number of births Λ and the natural death rate μ. The value of sensitivity index regarding these two parameters is approximately (-1) and $(+1)$, respectively, meaning that the increase in the number of births during epidemics results in the decrease of R_0^S, while the increase in the natural death rate during the epidemic results in the decrease in R_0^S, which is quite expected observation. The behavior of threshold R_0^S with respect to parameter values with high sensitivity index is shown in Figure 5.



(a) $R_0^S(\beta, \beta')$ (b) $R_0^S(\beta, \sigma_1)$

(c) $R_0^S(\beta, \Lambda)$ (d) $R_0^S(\beta, \mu)$

Figure 5: R_0^S as a function of parameters with high sensitivity index.

Since both basic reproduction numbers R_0^D and R_0^S depend on quite different set of parameters (the parameters present in both are only β, β', κ and μ), their comparison is quite limited. However, a significant conclusion is that the introduction of the noise into the deterministic model 2 via perturbation

$$\beta \mapsto \beta + dB_1(t), \quad \beta' \mapsto \beta' + dB_2(t), \quad t \geq 0,$$

where B_1 and B_2 are independent Brownian motions, results in the decrease of the impact of transmission coefficient β due to infected class and significant increase of the impact of transmission coefficient β' due to superspreaders on the course of the epidemic. Furthermore, threshold R_0^S shows high sensitivity even on the small changes in intensities σ_1 and σ_2 of the noises - a moderate increase in either noise results in the decrease of R_0^S. Note that just a moderate decrease of the intensity σ_2 related to superspreaders results in a significant enlargement of R_0^S, which gives the superspreaders an important role in the dynamic of the stochastic model. For analysis of sensitivity of a different stochastic compartmental model for COVID-19 to the noise level we refer to [28]. These arguments can be seen as a justification for use of stochastic differential models for describing and forecasting the course of the epidemic.

For similar approaches to sensitivity analysis of basic reproduction number regarding different infectious diseases we refer to [29] and [30], and for COVID-19 to the recent papers [31] and [32].

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