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A STOCHASTIC SIMPLICIAL SIS MODEL DRIVEN BY TWO INDEPENDENT NOISES

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Abstract. We propose a stochastic simplicial SIS model where two independent sources of noise are utilized to perturb the individual and collective infection rates. After proving that the model has a unique global solution, two sets of conditions on the parameters that give exponential stability of the trivial solution are presented. We then find conditions for persistence and show that the solution of the SDE oscillates infinitely often around a point under such requirements. We validate the theoretical statements by performing numerical experiments, as well as simulations on both real and synthetic simplicial networks, with results that align with the theoretical and numerical predictions of the model.

1. Introduction

When using higher-order contagion models to understand the spread of diseases in real-world populations, groups of infectious agents interact with susceptible agents and the probability of infection is thus determined by the interrelations between these groups and susceptible individuals. The interaction topology of these models is usually described by simplicial complexes or hypergraphs [6, 35], and their use has produced numerous advances in a variety of domains [21, 15, 16, 28, 29, 11, 12]. While classical epidemiological studies have traditionally used deterministic compartmental systems [22, 18], recent research has shown the relevance of studying epidemics spreading over hypergraphs or simplicial complexes [13, 19, 3, 5, 27, 7, 4].

Stochastic models are also important as they capture the inherent randomness and unknown control measures in real-world networks modelled by graphs [14, 10, 23, 25, 34, 20, 2, 26, 38, 33]. In [32, 17], we have presented a single white noise

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stochastic model of social contagion in simplicial networks based on the mean-field approach of [19]. We used a specific Itô stochastic differential equation (SDE) to simulate the dynamics of a network and extended a model proposed in [14] to account for simultaneous contagion scenarios.

The novelty of the present work lies in introducing two independent noise sources (each with its own intensity) to represent stochastic fluctuations in individual and group-level contagion, which perturb the transmission parameters of the simplicial contagion model. This modeling choice is motivated by real-world situations where these two mechanisms operate asynchronously, even within the same environment. For instance, in a closed bus where a large group has just disembarked, the risk of group-based transmission may remain high due to poor ventilation, but the actual randomness of group contagion at that moment is low. Meanwhile, individual-level contagion remains possible, for example, if a passenger sits near an infectious person. This decoupling between sources of randomness justifies treating them as independent and allows for a more nuanced representation of epidemic dynamics. The assumption allows us to separate and analyze their respective contributions at a first order approximation, and is a common approach in stochastic modeling when multiple, non-synchronized sources of uncertainty are present. The introduction of stochasticity on a deterministic model over usual networks and using several independent white noises can be found in other contributions; many of them assume that the noises are directly proportional to the number of the corresponding population (see, e.g., [30, 31, 36, 37]), whereas in [8] the authors use a diffusion coefficient in square root form. We show here that, under a couple of appropriate conditions, if the stochastic basic reproductive number is smaller than one, then the disease dies out with probability one; otherwise the solution of the SDE oscillates infinitely often around a point which can be explicitly computed. Moreover, we define a second threshold whose value, if less than one, leads to the almost sure extinction of the disease using only one of the former conditions. We illustrate the theoretical results by performing numerical experiments, and we generalise the stochastic simplicial contagion model of [17] to the case of two independent noises to carry out simulations on a real simplicial network, which show a high degree of agreement with the theoretical and numerical predictions of the model's stochastic differential equation.

2. A stochastic simplicial SIS type model with two independent noises

Given a simplicial complex of dimension 2, [19] introduces a mean field (MF) approach for a simplicial SIS (susceptible–infected–susceptible) model:

$$\frac{dI(t)}{dt} = \beta(1 - I(t))I(t) + \beta_\Delta(1 - I(t))I(t)^2 - \mu I(t), \tag{2.1}$$

where

- μ denotes the recovery probability;
- $\beta := \beta_1\langle k_1\rangle$, $\beta_\Delta := \beta_2\langle k_2\rangle$;

- β_1 is the probability that a node belonging to a 1-simplex (edge) is infected by the remaining vertice assuming that this vertex is infectious, and β_2 is the probability that a node belonging to a 2-simplex (filled triangle) is infected by the two remaining vertices assuming that both vertices are infectious;
- k_1 denotes the number of 1-simplices (edges) a node belongs to; similarly, k_2 denotes the number of 2-simplices (filled triangles) a node belongs to; and $\langle k_i \rangle$ $(i = 1, 2)$ stand for their means.

The introduction of randomness into a compartmental system has been proposed by perturbing the variables of the system (see [9] or [36]) or, more commonly, by considering the natural unpredictability of the parameters of the model (see, e.g., [14, 20, 23, 24, 33, 34]). Notice, for example, that the stochastic system considered in [14] was achieved by replacing in the deterministic model the parameter β by the random term $\beta + \sigma\xi_t$, with ξ_t a white Gaussian noise and σ a coefficient that controls the degree of the perturbation, leading to the SDE

$$dI(t) = \beta\,(1 - I(t) - \mu)I(t)dt + \sigma\,(1 - I(t))I(t)\,dW(t), \tag{2.2}$$

where $W(t)$ stands for a scalar Wiener process. On the other hand, in [32] the authors study the SDE obtained from (2.1) by perturbing both the disease transmission coefficients β and β_Δ with the same standard Wiener process and different intensities σ and σ_Δ, respectively.

In the present work we use two independent white noises ξ_t^1, ξ_t^2 with intensities σ and σ_Δ to perturb, respectively, the parameters β and β_Δ in the simplicial model (2.1). In other words, we are replacing β by the random variable $\beta + \sigma\xi_t^1$ and β_Δ by $\beta_\Delta + \sigma_\Delta\xi_t^2$. The coefficients σ and σ_Δ modulate the intensity of the stochastic fluctuations and are interpreted as context-specific risk factors (for instance, σ_Δ would be higher in crowded or enclosed spaces, reflecting higher exposure to group-based contagion). The term ξ_t^1 represents random individual-level fluctuations and typically arises from direct unpredicted person-to-person contact (such as standing next to an infectious individual). The term ξ_t^2 accounts for random fluctuations in simultaneous group interactions (such as speaking in a group or shared space) and also can involve indirect exposure through accumulated viral load in the environment (e.g., airborne particles persisting after an infected group disembarks from a bus). Although both noise sources may be associated with the same setting (e.g., a closed bus), the actual stochastic fluctuations in individual and group contagion can occur asynchronously. For example, after a group of passengers disembarks, the randomness associated with group-based contagion, ξ_t^2, may be low (even though the structural risk factor σ_Δ remains high due to poor ventilation) while individual-level randomness, ξ_t^1, may still be significant due to remaining nearby contacts. This temporal decoupling between contagion mechanisms supports the assumption that the two noise processes are independent.

With this change, the equation (2.1) results in the SDE

$$\begin{aligned} dI(t) = &\left(\beta\,(1 - I(t))I(t) - \mu\,I(t) + \beta_\Delta\,(1 - I(t))I(t)^2\right)dt \\ &+ \sigma\,(1 - I(t))I(t)\,dW^1(t) + \sigma_\Delta\,(1 - I(t))I(t)^2\,dW^2(t), \end{aligned} \tag{2.3}$$

where $W^1(t), W^2(t)$ are independent one-dimensional Wiener processes. So, this stochastic equation generalizes the deterministic simplicial model (2.1) ($\sigma = \sigma_\Delta = 0$) and the stochastic SIS model (2.2) presented in [14] ($\beta_\Delta = \sigma_\Delta = 0$). Notice that, although two independent noises are used as in [8], the rationale of this approach is different and square root diffusion terms do not appear.

The term $\sigma\,(1 - I(t))I(t)\,dW^1(t)$ captures individual-level stochastic transmission, where σ quantifies the structural risk of random interpersonal contacts (e.g., casual encounters in public transport, markets, or workplaces), and $W^1(t)$ introduces randomness due to the unpredictable nature of such contacts over time. The term $\sigma_\Delta\,(1 - I(t))I(t)^2\,dW^2(t)$ corresponds to group-level or collective contagion, where σ_Δ reflects the environmental risk associated with shared spaces (e.g., crowded indoor venues, poorly ventilated rooms), and $W^2(t)$ accounts for stochastic fluctuations in whether or not such group exposure results in transmission.

We start this section by proving an existence and uniqueness theorem for (2.3). Then we analyze the stability behavior of its trivial solution. Finally, we study its persistence.

Theorem 2.1. *The SDE* (2.3) *with initial condition* $I(0) = I_0 \in (0, 1)$ *has a unique global solution* $I(t)$ *satisfying* $0 \le I(t) \le 1$ *for all* $t \ge 0$ *with probability 1.*

Proof. Let us denote by

$$\begin{aligned} f(x) &= \beta\,(1 - x)x - \mu\,x + \beta_\Delta\,(1 - x)x^2, \\ g_1(x) &= \sigma\,(1 - x)x, \\ g_2(x) &= \sigma_\Delta\,(1 - x)x^2 \end{aligned} \tag{2.4}$$

the coefficients of (2.3). Since they satisfy a local Lipschitz condition, for any $I_0 \in (0, 1)$ there is a unique maximal local solution $I(t)$ with $t \in [0, \tau_e)$; see, e.g., [2]. Let $k_0 \in \mathbb{N}$ be such that $1/k_0 < I_0 < 1 - 1/k_0$, and for each $k \in \mathbb{N}$, $k \ge k_0$, consider the stopping time

$$\tau_k = \inf\{t \in [0, \tau_e) : I(t) \notin (1/k, 1 - 1/k)\}.$$

From its definition, τ_k is monotonically increasing, and if we set $\tau_\infty = \lim_{k\to\infty} \tau_k$, then $\tau_\infty \le \tau_e$ almost surely. If we show that $\tau_\infty = \infty$ a.s., then $\tau_e = \infty$ a.s. and $I(t) \in (0, 1)$ a.s. for all $t \ge 0$.

To show that $\tau_\infty = \infty$ a.s. we assume it does not hold. Then there exist $T, \varepsilon > 0$ such that

$$\mathbb{P}\{\tau_\infty \le T\} = \varepsilon.$$

As a consequence, there exists $k_1 \in \mathbb{N}$, $k_1 \ge k_0$, such that for all $k \ge k_1$,

$$\mathbb{P}\,(\Omega_k) \ge \varepsilon,$$

where we have denoted $\Omega_k = \{\tau_k \le T\}$. Consider the function $V : (0, 1) \to \mathbb{R}$ defined by

$$V(x) = -\log(x) - \log(1 - x)$$

with derivatives

$$V_x = -\frac{1}{x} + \frac{1}{1 - x}, \quad V_{xx} = \frac{1}{x^2} + \frac{1}{(1 - x)^2}.$$

If we denote the associated operator by

$$L = f\,\frac{\partial}{\partial x} + \frac{1}{2}(g_1^2 + g_2^2)\frac{\partial^2}{\partial x^2}$$

and use the Itô formula, we obtain

$$\begin{aligned} V(I(t\wedge\tau_k)) = V(I_0) &+ \int_0^{t\wedge\tau_k} LV(I(s))\,ds \\ &+ \int_0^{t\wedge\tau_k}\left(g_1\,\frac{\partial V}{\partial x}\right)(I(s))\,dW_s^1 + \int_0^{t\wedge\tau_k}\left(g_2\,\frac{\partial V}{\partial x}\right)(I(s))\,dW_s^2 \end{aligned}$$

for any $t\in[0,T]$ and $k\geq k_1$. Then

$$\mathbb{E}[V(I(t\wedge\tau_k))] = V(I_0) + \mathbb{E}\int_0^{t\wedge\tau_k} LV(I(s))\,ds. \tag{2.5}$$

If $0<x<1$,

$$\begin{aligned} LV(x) &= f(x)V_x + \frac{1}{2}\left(g_1^2(x)+g_2^2(x)\right)V_{xx} \\ &= -\beta(1-x) + \mu - \beta_\Delta(1-x)x + \beta x - \mu\frac{x}{1-x} + \beta_\Delta x^2 \\ &\quad + \frac{1}{2}\sigma^2(1-x)^2 + \frac{1}{2}\sigma^2x^2 + \frac{1}{2}\sigma_\Delta^2(1-x)^2x^2 + \frac{1}{2}\sigma_\Delta^2x^4 \\ &\leq \mu+\beta+\beta_\Delta+\sigma^2+\sigma_\Delta^2 = C \end{aligned}$$

and, using (2.5),

$$\mathbb{E}[V(I(t\wedge\tau_k))] \leq V(I_0) + \mathbb{E}\int_0^{t\wedge\tau_k} C\,ds \leq V(I_0) + C\,t$$

for any $t\in[0,T]$ and $k\geq k_1$. Then

$$\mathbb{E}[V(I(T\wedge\tau_k))] \leq V(I_0) + C\,T, \quad k\geq k_1. \tag{2.6}$$

Notice now that if $\omega\in\Omega_k$, $k\geq k_1$, then $I(\tau_k)(\omega)$ is equal to either $1/k$ or $1-1/k$ and then

$$V(I(\tau_k)(\omega)) = \log(k) + \log\left(\frac{k}{k-1}\right). \tag{2.7}$$

Then from (2.6) and (2.7), for any $k\geq k_1$

$$\begin{aligned} V(I_0) + C\,T &\geq \mathbb{E}[V(I(T\wedge\tau_k))] \geq \mathbb{E}[\mathbf{1}_{\Omega_k}\cdot V(I(\tau_k))] \\ &= \mathbb{P}(\Omega_k)\,\log\left(\frac{k^2}{k-1}\right) \geq \varepsilon\log\left(\frac{k^2}{k-1}\right), \end{aligned}$$

which leads to a contradiction, since the term on the left is constant. □

From (2.4), the system $f(x)=0$, $g_1(x)=0$, $g_2(x)=0$ has only the solution $x=0$; then $I=0$ is the unique equilibrium of (2.3).

From (2.3),

$$\begin{aligned} d(\log(I(t)) = &\left(\beta\,(1-I(t)) - \mu + \beta_\Delta\,(1-I(t))I(t) - \frac{1}{2}\sigma^2(1-I(t))^2\right.\\ &\left. -\frac{1}{2}\sigma_\Delta^2(1-I(t))^2 I(t)^2\right) dt\\ &+ \sigma(1-I(t))\,dW^1(t) + \sigma_\Delta(1-I(t))I(t)\,dW^2(t), \end{aligned}$$

which can be written as

$$\log(I(t)) = \log(I(0)) + \int_0^t H(I(s))\,ds + \int_0^t G_1(I(s))\,dW^1(s) + \int_0^t G_2(I(s))\,dW^2(s) \tag{2.8}$$

with

$$\begin{aligned} H(x) = &\beta - \mu - \frac{\sigma^2}{2} + \left(\sigma^2 - \beta + \beta_\Delta\right) x\\ &- \left(\frac{\sigma^2}{2} + \frac{\sigma_\Delta^2}{2} + \beta_\Delta\right) x^2 + \sigma_\Delta^2 x^3 - \frac{1}{2}\sigma_\Delta^2 x^4 \end{aligned} \tag{2.9}$$

and

$$G_1(x) = \sigma(1-x), \quad G_2(x) = \sigma_\Delta(1-x)x.$$

Since $-\frac{\sigma_\Delta^2}{2}x^2 + \sigma_\Delta^2 x^3 \le \frac{\sigma_\Delta^2}{2}x^2$,

$$\begin{aligned} H(x) \le &\beta - \mu - \frac{\sigma^2}{2} + \left(\sigma^2 - \beta + \beta_\Delta\right) x\\ &- \left(\frac{\sigma^2}{2} - \frac{\sigma_\Delta^2}{2} + \beta_\Delta\right) x^2 - \frac{1}{2}\sigma_\Delta^2 x^4. \end{aligned}$$

If

$$\sigma^2 - \beta + \beta_\Delta \le 0, \quad \sigma_\Delta^2 - \sigma^2 - 2\beta_\Delta \le 0, \tag{2.10}$$

then $H(I(s)) \le \beta - \mu - \frac{\sigma^2}{2}$ for $s \ge 0$ and

$$\int_0^t H(I(s))\,ds \le \left(\beta - \mu - \frac{\sigma^2}{2}\right) t. \tag{2.11}$$

On the other hand, from the strong law of larger numbers (see Mao [26]),

$$\lim_{t\to\infty} \frac{1}{t}\int_0^t G_i(I(s))\,dW^i(s) = 0 \ \text{ a.s.}, \quad i = 1, 2. \tag{2.12}$$

Using (2.11) and (2.12) in (2.8) gives

$$\limsup_{t\to\infty} \frac{\log(I(t))}{t} \le \beta - \mu - \frac{\sigma^2}{2} \ \text{ a.s.},$$

and we have demonstrated

Theorem 2.2. *If the parameters of the stochastic differential equation* (2.3) *satisfy* (2.10) *and*

$$R_0^S = \frac{\beta - \frac{1}{2}\sigma^2}{\mu} < 1,$$

then the solution $I(t)$ tends to zero exponentially with probability one and the disease dies out almost surely.

Notice that conditions (2.10) can be written

$$\sigma_\Delta^2 - 2\beta_\Delta \leq \sigma^2 \leq \beta - \beta_\Delta$$

or

$$\sigma_\Delta^2 \leq \sigma^2 + 2\beta_\Delta \leq \beta + \beta_\Delta$$

or

$$\frac{1}{2}(\sigma_\Delta^2 - \sigma^2) \leq \beta_\Delta \leq \beta - \sigma^2.$$

These conditions in particular imply that

$$\beta \geq \sigma^2, \quad \beta_\Delta \leq \beta, \quad \frac{1}{2}\frac{\sigma_\Delta^2}{\mu} \leq R_0^S.$$

On the other hand, since $\sigma_\Delta^2 x^3 \leq \sigma_\Delta^2$,

$$\begin{aligned} H(x) &\leq \beta - \mu - \frac{\sigma^2}{2} + \sigma_\Delta^2 + \left(\sigma^2 - \beta + \beta_\Delta\right) x \\ &\quad - \left(\frac{\sigma^2}{2} - \frac{\sigma_\Delta^2}{2} + \beta_\Delta\right) x^2 - \frac{1}{2}\sigma_\Delta^2 x^4 \\ &\leq \beta - \mu - \frac{\sigma^2}{2} + \sigma_\Delta^2 + \left(\sigma^2 - \beta + \beta_\Delta\right) x, \end{aligned}$$

and with a proof similar to that of Theorem 2.2 we obtain

Theorem 2.3. *If the parameters of the stochastic model* (2.3) *satisfy the condition*

$$\sigma^2 - \beta + \beta_\Delta \leq 0 \tag{2.13}$$

and

$$R_0^{SS} = \frac{\beta - \frac{1}{2}\sigma^2 + \sigma_\Delta^2}{\mu} < 1,$$

then the solution $I(t)$ tends to zero exponentially a.s. and the disease dies out with probability one.

Remark 2.4. Observe that $R_0^S = R_0 - \sigma^2/(2\mu) < R_0$. So, if $R_0^S < 1 < R_0$ then the equilibrium of the deterministic problem (2.1) is unstable (see [19]), whereas, from Theorem 2.2, the equilibrium of the stochastic model is asymptotically stable. This result is an example of how the introduction of noise stabilizes the unstable solution of a deterministic problem (see [1, 14, 33]).

When $\beta_\Delta = \sigma_\Delta = 0$, the proposed simplicial model (2.3) reduces to the stochastic SIS model (2.2) presented in [14], both conditions (2.10) and (2.13) reduce to $\sigma^2 - \beta \leq 0$, and Theorem 2.2 and Theorem 2.3 become:

Corollary 2.5 ([14])**.** *If $\sigma^2 \leq \beta < \frac{1}{2}\sigma^2 + \mu$ then the trivial solution of the stochastic SIS model* (2.2) *is almost surely exponentially stable.*

Let us now study its persistence.

Theorem 2.6. *If the parameters of the stochastic differential equation* (2.3) *satisfy* (2.10) *and*

$$R_0^S = \frac{\beta - \frac{1}{2}\sigma^2}{\mu} > 1,$$

then for any $I(0) \in (0,1)$*, the solution* $I(t)$ *oscillates infinitely around* $\xi > 0$*:*

$$\liminf_{t\to\infty} I(t) \leq \xi \leq \limsup_{t\to\infty} I(t) \quad a.s.,$$

where ξ *is the unique root in* $(0,1)$ *of* (2.9)*.*

Proof. We start by proving that the equation $H(x) = 0$, where $H(x)$ is given in (2.9), has a unique solution ξ in $(0,1)$. Existence is clear, due to $H(0) = \beta - \mu - \frac{\sigma^2}{2} > 0$ and $H(1) = -\mu < 0$. Uniqueness is proved if we show that $H'(x) < 0$ for $x \in (0,1)$. Observe that

$$H'(x) = \sigma^2 - \beta + \beta_\Delta - (\sigma^2 + \sigma_\Delta^2 + 2\beta_\Delta)x + 3\sigma_\Delta^2 x^2 - 2\sigma_\Delta^2 x^3.$$

If $\sigma_\Delta = \sigma = \beta_\Delta = 0$, $H'(x) = -\beta < 0$. If $\sigma_\Delta = 0$ and $\sigma \neq 0$ or $\beta_\Delta \neq 0$, then $\sigma^2 + 2\beta_\Delta > 0$ and $H'(x) = \sigma^2 - \beta + \beta_\Delta - (\sigma^2 + 2\beta_\Delta)x < 0$ for $x > 0$, where we have used (2.10). If $\sigma_\Delta \neq 0$ then

$$H''(x) = -(\sigma^2 + \sigma_\Delta^2 + 2\beta_\Delta) + 6\sigma_\Delta^2 x - 6\sigma_\Delta^2 x^2$$

is a downward-opening parabola; since the abscissa of the vertex is $x = 1/2$,

$$H''(x) \leq H''\left(\frac{1}{2}\right) = \frac{1}{2}\sigma_\Delta^2 - \sigma^2 - 2\beta_\Delta < \sigma_\Delta^2 - \sigma^2 - 2\beta_\Delta \leq 0$$

for any $x \in \mathbb{R}$. From here, H' is strictly decreasing in $\mathbb{R}$; since $H'(0) = \sigma^2 - \beta + \beta_\Delta \leq 0$, we conclude that $H'(x) < 0$ for $x > 0$.

We show hereafter that

$$\limsup_{t\to\infty} I(t) \geq \xi. \tag{2.14}$$

If (2.14) does not hold, then there exists $\epsilon \in (0,1)$ such that $\mathbb{P}(\Omega_1) > \epsilon$, where $\Omega_1 = \{\limsup_{t\to\infty} I(t) \leq \xi - 2\epsilon\}$. So, if $\omega \in \Omega_1$, there exists $T(\omega) > 0$ such that

$$I(t,\omega) \leq \xi - \epsilon \quad \text{for } t \geq T(\omega). \tag{2.15}$$

Since H is a decreasing function in $(0,1)$, we can select $\epsilon \in (0,\xi)$ so that $H(0) > F(\xi - \epsilon)$. Then, if $\omega \in \Omega_1$ and $t \geq T(\omega)$,

$$H(I(t,\omega)) \geq H(\xi - \epsilon). \tag{2.16}$$

Moreover, by the law of large numbers, there exists Ω_2 such that $\mathbb{P}(\Omega_2) = 1$ and, for every $\omega \in \Omega_2$,

$$\lim_{t\to\infty} \frac{1}{t}\int_0^t G_1(I(s,\omega))\, dW^1(s) = 0 = \lim_{t\to\infty} \frac{1}{t}\int_0^t G_2(I(s,\omega))\, dW^2(s).$$

If $\omega \in \Omega_1 \cap \Omega_2$ and $t \geq T(\omega)$, from (2.8) and (2.16) we obtain

$$\begin{aligned}\log(I(t)) &= \log(I(0)) + \int_0^{T(\omega)} H(I(s,\omega))\,ds + \int_{T(\omega)}^{t} H(I(s,\omega))\,ds \\ &\quad + \int_0^t G_1(I(s,\omega))\,dW^1(s) + \int_0^t G_2(I(s,\omega))\,dW^2(s) \\ &\geq \log(I(0)) + \int_0^{T(\omega)} H(I(s,\omega))\,ds + H(\xi-\epsilon)(t-T(\omega)) \\ &\quad + \int_0^t G_1(I(s,\omega))\,dW^1(s) + \int_0^t G_2(I(s,\omega))\,dW^2(s).\end{aligned}$$

Then, if $\omega \in \Omega_1 \cap \Omega_2$,

$$\liminf_{t\to\infty} \frac{\log(I(t,\omega))}{t} \geq H(\xi-\epsilon) > 0$$

and

$$\lim_{t\to\infty} I(t,\omega) = \infty,$$

in contradiction with (2.15). We deduce that (2.14) is true.

We now show that $\liminf_{t\to\infty} I(t) \leq \xi$. Suppose that it does not hold. Then there exists $\delta \in (0,1)$ such that $\mathbb{P}(\Omega_3) > \delta$, where $\Omega_3 = \{\liminf_{t\to\infty} I(t) \geq \xi + 2\delta\}$. So, if $\omega \in \Omega_3$ there is a $\tau(\omega) > 0$ such that

$$I(t,\omega) \geq \xi + \delta \quad \text{for } t \geq \tau(\omega). \tag{2.17}$$

Since H is a decreasing function in $(0,1)$, if $\omega \in \Omega_3$ and $t \geq \tau(\omega)$ then

$$H(I(t,\omega)) \leq H(\xi+\delta). \tag{2.18}$$

Taking $\omega \in \Omega_2 \cap \Omega_3$, if $t \geq \tau(\omega)$, from (2.8) and (2.18)

$$\begin{aligned}\log(I(t))) &= \log(I(0)) + \int_0^{\tau(\omega)} H(I(s,\omega))\,ds + \int_{\tau(\omega)}^{t} H(I(s,\omega))\,ds \\ &\quad + \int_0^t G_1(I(s,\omega))\,dW^1(s) + \int_0^t G_2(I(s,\omega))\,dW^2(s) \\ &\leq \log(I(0)) + \int_0^{\tau(\omega)} H(I(s,\omega))\,ds + H(\xi+\delta)(t-\tau(\omega)) \\ &\quad + \int_0^t G_1(I(s,\omega))\,dW^1(s) + \int_0^t G_2(I(s,\omega))\,dW^2(s).\end{aligned}$$

Then, if $\omega \in \Omega_2 \cap \Omega_3$,

$$\limsup_{t\to\infty} \frac{\log(I(t,\omega))}{t} \leq H(\xi+\delta) < 0$$

and

$$\lim_{t\to\infty} I(t,\omega) = 0,$$

in contradiction with (2.17). This completes the proof. □

3. Numerical simulations

In this section we conduct numerical experiments to confirm the results presented in the previous section.

Let us recall that the parameters σ and σ_Δ represent the intensity of the noise affecting individual and group-level infections, respectively. Although the present article does not use specific empirical data to calibrate these values (e.g., from real-world contact tracing or Bayesian inference), the selection of parameters in the experiments is guided by a consistent epidemiological rationale, based on the following criteria:

- Exploring different epidemiological regimes: we selected combinations of $(\beta, \beta_\Delta, \sigma, \sigma_\Delta)$ that produce values of R_0, R_0^S, and R_0^{SS} below or above 1, allowing us to observe the transition between extinction and persistence, and to validate the theoretical results.
- Contextual interpretation of risk: in scenarios with high σ_Δ we simulate settings with high collective infection risk (such as parties, poorly ventilated indoor spaces, or large events). In cases with high σ but low σ_Δ, we focus on uncertainty arising from individual contacts (e.g., essential workers or public transportation users).
- Comparison with previous work: some values replicate or extend parameter settings already used in the single-noise model ([32, 17]), enabling consistent comparisons.

3.1. **Simulations of the SDE.** Experiments 1 and 2 are devoted to testing the stability of the equilibrium of the stochastic differential equation (2.3), whereas experiment 3 is related to the persistence statement of Theorem 2.6. The ordinary (2.1) and stochastic (2.3) equations were solved numerically using the Euler and Euler–Maruyama methods, respectively, with step size $\Delta = 1/10$ over the interval $[0, 50]$ in experiment 1 and over $[0, 500]$ in experiments 2 and 3.

Experiment 1. We select two sets of parameters:

$$\mu = 0.7,\ \beta = 0.5,\ \beta_\Delta = 0.25,\ \sigma = 0.35,\ \sigma_\Delta = 0.25; \tag{3.1}$$

$$\mu = 0.9,\ \beta = 0.3,\ \beta_\Delta = 0.2,\ \sigma = 0.3,\ \sigma_\Delta = 0.8. \tag{3.2}$$

With both sets, $R_0 < 1$; therefore, the disease-free equilibrium of the SIS model (2.1) is asymptotically stable. The set of parameters (3.1) satisfies conditions (2.10) (in particular, (2.13)) and with it, $R_0^S < 1$ and $R_0^{SS} < 1$. Then both Theorem 2.2 and Theorem 2.3 give exponential stability of the equilibrium of the stochastic model (2.3) with probability 1. The results of the numerical experiments with parameters (3.1) are shown in Figure 1, which shows ten trajectories of the solution of the stochastic equation (2.3) together with the exact solution of the deterministic equation (2.1) starting at $I(0) = 0.8$ (left) and $I(0) = 0.2$ (right).

On the other hand, the set (3.2) does not satisfy the condition on the right of (2.10) and, in consequence, Theorem 2.2 is not applicable. In this case, we can apply Theorem 2.3 to obtain almost sure exponential stability of the stochastic simplicial equation (2.3) because the parameters satisfy condition (2.13) together

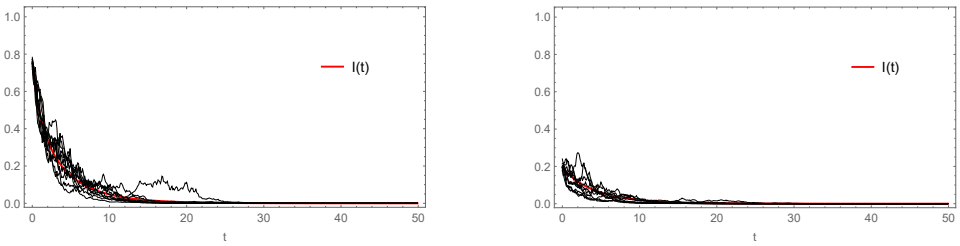

FIGURE 1. Solution of the deterministic equation (2.1) (colored thick line) together with 10 trajectories of the solution of the stochastic equation (2.3) (black thin lines), with initial values $I(0) = 0.8$ (left) and $I(0) = 0.2$ (right), in both cases with the parameters (3.1).

with $R_0^{SS} < 1$. The results of the numerical experiments, shown in Figure 2, confirm the exponential stability of the equilibrium.

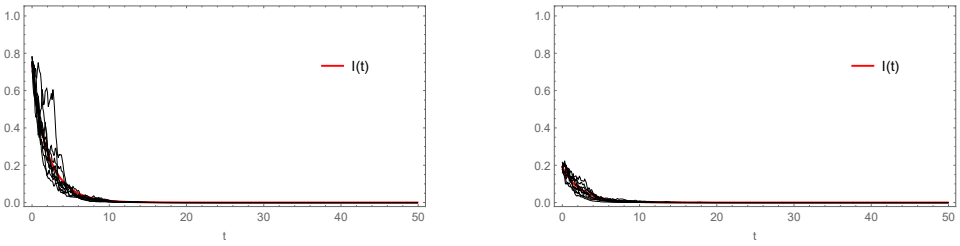

FIGURE 2. Solution of the deterministic equation (2.1) (colored thick line) together with 10 trajectories of the solution of the stochastic equation (2.3) (black thin lines), with initial values $I(0) = 0.8$ (left) and $I(0) = 0.2$ (right), in both cases with the parameters (3.2).

Experiment 2. We choose

$$\mu = 0.5,\ \beta = 0.6,\ \beta_\Delta = 0.2,\ \sigma = 0.5,\ \sigma_\Delta = 0.1, \tag{3.3}$$

which satisfy conditions (2.10). Observe that in this case $R_0^S = 0.95 < 1$ and then, according to Theorem 2.2, a.s. exponential stability of the equilibrium of (2.3) is achieved. Moreover, $R_0 = 6/5 > 1$, and the solution of the deterministic s-SIS equation tends, as t tends to infinity, to the endemic equilibrium

$$\lim_{t\to\infty} I(t) \approx 0.22474.$$

Figure 3, which shows the computer simulations of this experiment, fully supports these results. This example illustrates how in some cases the introduction

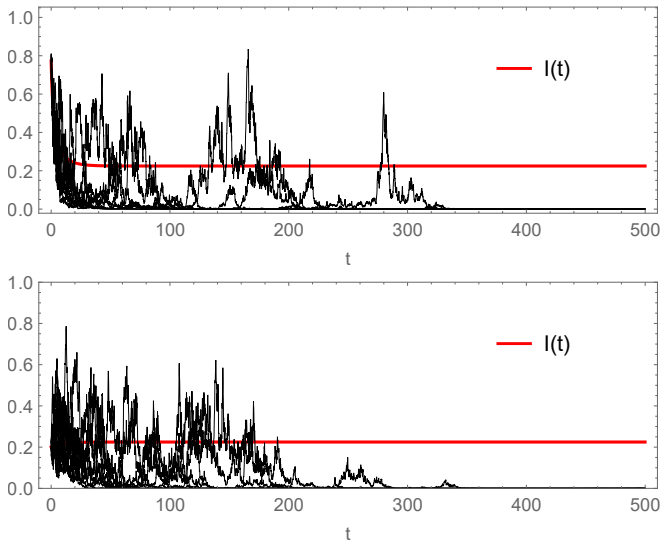

FIGURE 3. Solution of the deterministic equation (2.1) (colored thick line) together with 10 trajectories of the solution of the stochastic equation (2.3) (black thin lines), with initial values $I(0) = 0.8$ (top) and $I(0) = 0.2$ (bottom), in both cases with the parameters (3.3).

of noise in a equation can stabilize the unstable trivial solution of a deterministic system, see Remark 2.4.

Experiment 3. We choose

$$\mu = 0.7,\ \beta = 0.8,\ \beta_\Delta = 0.5,\ \sigma = 0.2,\ \sigma_\Delta = 0.3, \tag{3.4}$$

which satisfy conditions (2.10). Observe that in this case $R_0^S = 1.142$ and $R_0^{SS} = 1.24286$. Although the exact solution of $H(x) = 0$ is not available, it can be approximated as

$$\xi \approx 0.21251.$$

The solution $I(t)$ oscillates infinitely often around this value. Moreover, $R_0 > 1$ and the solution of the deterministic s-SIS equation satisfies

$$\lim_{t\to\infty} I(t) \approx 0.238516.$$

Figure 4 shows that the computer simulations fully support the above theoretical statements: the solution of the deterministic equation (2.1) (thick red line) tends to 0.238, whereas the trajectories of the solution oscillate infinitely often around the value $\xi = 0.212$, represented by the thick dashed black line.

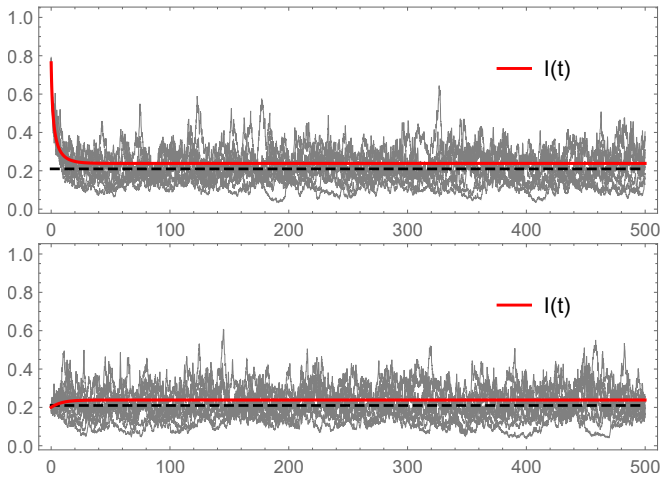

FIGURE 4. Solution of the deterministic equation (2.1) (colored thick line) together with 10 trajectories of the solution of the stochastic equation (2.3) (gray thin lines), with initial values $I(0) = 0.8$ (top) and $I(0) = 0.2$ (bottom), in both cases with the parameters (3.4).

3.2. **Simulations on a real simplicial network.** We use a variant of the stochastic simplicial contagion model (SSCM) of [17], driven by two independent noises, to run simulations in a real-world simplicial network. We model this type of situation using two independent white Gaussian noises ξ_t^1 and ξ_t^2 to respectively perturb infection parameters β_1 and β_2 with different intensities σ_1 and σ_2 (see Figure 5). All the code used to perform the simulations in this section is available in the public repository https://github.com/juanelas/sscm. Instructions for running the experiments can be found in the `README.md` file, under the section titled "A stochastic simplicial SIS model driven by two independent noises". We use the dataset InVS15 of face-to-face interactions collected from a workplace and obtained from [19] as input for the two independent noises SSCM.

According to equation (2.3), we have $\beta = \beta_1\langle k_1\rangle$, $\beta_\Delta = \beta_2\langle k_2\rangle$, where β_i are the infection probabilities of equation (2.1), and we denote $\sigma = \sigma_1\langle k_1\rangle$ and $\sigma_\Delta = \sigma_2\langle k_2\rangle$.

Experiment 1. Fix the following sets of parameters:

$$\mu = 0.7,\ \beta_1 = 0.014,\ \beta_2 = 0.019,\ \sigma_1 = 0.0187,\ \sigma_2 = 0.0094; \tag{3.5}$$

$$\mu = 0.7,\ \beta_1 = 0.014,\ \beta_2 = 0.01,\ \sigma_1 = 0.0187,\ \sigma_2 = 0.867. \tag{3.6}$$

The set (3.5) satisfies conditions (2.10). With these parameters, $R_0 = 0.4885 < 1$, $R_0^S = 0.3394 < 1$ and $R_0^{SS} = 0.3455 < 1$. The simulation results are shown in Figure 6.

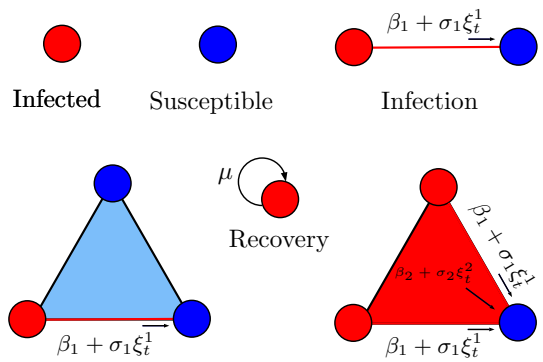

FIGURE 5. Channels of infection of the two independent noises SSCM.

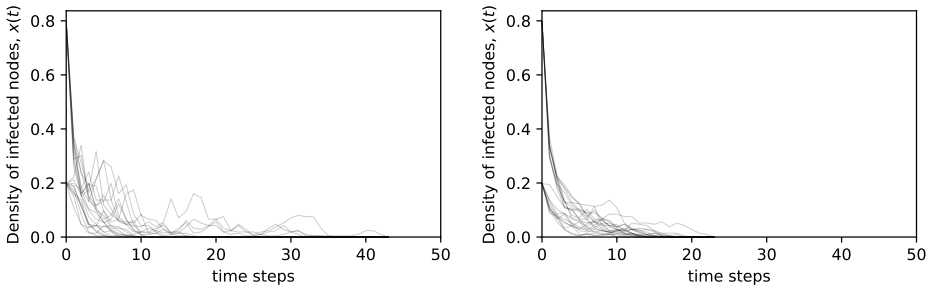

FIGURE 6. 20 trajectories resulting from running the SSCM driven by two independent noise terms (left) and the SCM (right) on the real simplicial network InVS15 (10 with initial value 0.2 and another 10 with 0.8) and having $R_0 < 1$, $R_0^S < 1$, and $R_0^{SS} < 1$ with the set of parameters (3.5).

The left panel shows that the disease dies out on this network, validating both Theorem 2.2 and Theorem 2.3. The right one confirms that the disease-free equilibrium of the deterministic SIS equation (2.1) is asymptotically stable, as shown in [19].

On the other hand, with the set of parameters (3.6) we have $R_0 = 0.4885 < 1$, $R_0^S = 0.3394 < 1$, and $R_0^{SS} = 0.8536 < 1$, but these parameters do not satisfy (2.10); thus, Theorem 2.2 does not apply. Nonetheless, this set of parameters satisfy both (2.13) and $R_0^{SS} < 1$. See Figure 7 for the simulation.

The left panel validates both Theorem 2.2 and Theorem 2.3, and the right one provides the same information as Figure 6 (right).

Experiment 2. We fix the parameters

$$\mu = 0.6,\ \beta_1 = 0.0319,\ \beta_2 = 0.0087,\ \sigma_1 = 0.0347,\ \sigma_2 = 0.0174, \tag{3.7}$$

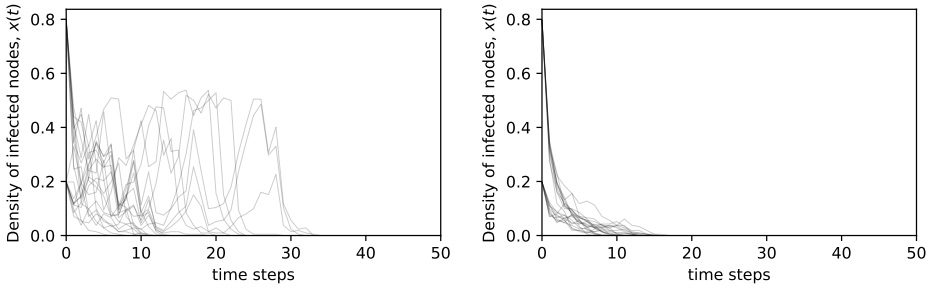

FIGURE 7. 20 trajectories (10 with initial value 0.2 and another 10 with 0.8) resulting from running the SSCM driven by two independent noise terms (left) and the SCM (right) on the real simplicial network InVS15 with the set of parameters (3.6).

which satisfy conditions (2.10). With them, $R_0 = 1.3 > 1$ and $R_0^S = 0.7 < 1$. The results are shown in Figure 8.

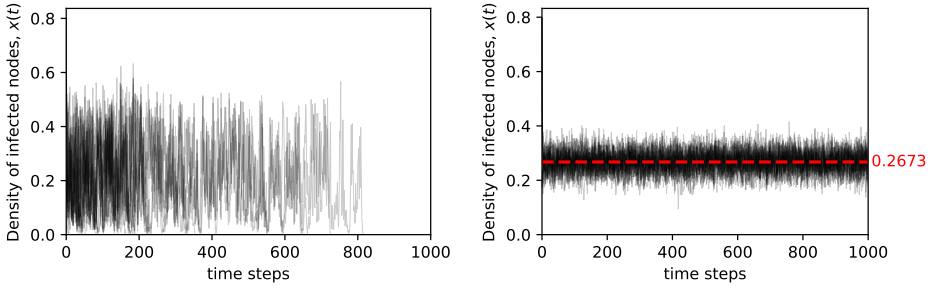

FIGURE 8. Trajectories resulting from running the SSCM driven by two independent noise terms (left) and the SCM (right) on the real simplicial network InVS15 for different initial values and parameters (3.7).

The left panel validates Theorem 2.2, and the right one proves that an endemic equilibrium is reached in the deterministic SIS equation (2.1) (as shown in [19]). The red dashed line represents the average of the values of the last 500 time steps. This confirms how introducing noise in a contagion process can stabilize the unstable trivial solution of a deterministic system (as predicted by the simulations of Section 3.1).

Experiment 3. We fix the parameters

$$\mu = 0.5,\ \beta_1 = 0.0375,\ \beta_2 = 0.0578,\ \sigma_1 = 0.0274,\ \sigma_2 = 0.0137, \tag{3.8}$$

which satisfy conditions (2.10). With them, $R_0 = 1.8318 > 1$, $R_0^S = 1.3844 > 1$ and $R_0^{SS} = 1.4024 > 1$. The results are shown in Figure 9.

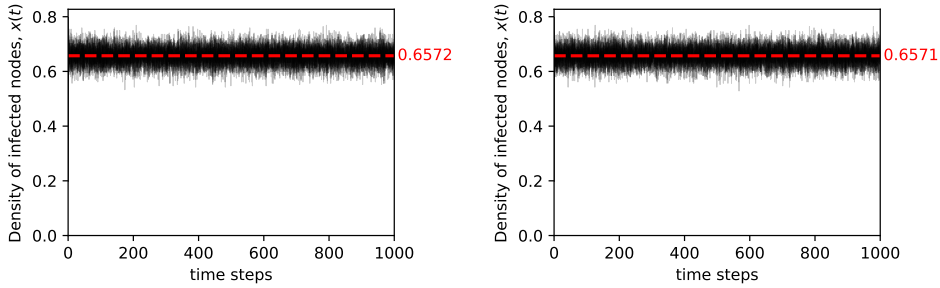

FIGURE 9. Trajectories resulting from running the SSCM driven by two independent noise terms (left) and the SCM (right) on the real simplicial network InVS15 for different initial values and parameters (3.8).

The left panel shows that an endemic equilibrium point is reached, which is consistent with Theorem 2.6, and the right one confirms that an endemic equilibrium is achieved in the deterministic SIS equation (2.1) (as shown in [19]).

We have also obtained similar results by running the SSCM with two independent noises on a synthetically generated network using the random simplicial complex (RSC) model of dimension 2 proposed by [19]. The simulations have been carried out with parameters complying with the three previous experiments and taking $\langle k_1 \rangle = 20$ and $\langle k_2 \rangle = 6$ as the input parameters for the RSC model (they consist of a generalization of the simulations of [32] to the case of two independent noises, and are available on demand).

4. Limitations and future work

While the present model offers a refined approach to stochastic epidemic dynamics by incorporating two independent sources of noise, several limitations and avenues for future research remain.

Independence of noise sources. We have modified the stochastic simplicial SIS model proposed in [32], where the same noise was used to perturb both individual and collective infection rates. Since there are contexts where individual and group noise independence is epidemiologically reasonable, in this work we have proposed a stochastic model driven by two independent noises, one for each infection rate. For example, as we mentioned earlier, after a group disembarks from a closed bus, the randomness associated with group-level transmission may decrease sharply, while the randomness in individual-level contagion may persist if a passenger is seated next to an infectious individual. This is why we assume that the random fluctuations affecting individual and group-level contagion are independent. This assumption not only simplifies the mathematical analysis but also enables us to isolate and better understand the distinct roles played by these two contagion mechanisms. However, it may not fully capture possible couplings observed in

real-world settings. For instance, in a crowded indoor event such as a concert or a nightclub, both the likelihood of close interpersonal contact (individual contagion) and the accumulation of airborne viral particles (group-level contagion) are simultaneously elevated due to shared environmental and behavioral factors—such as crowd density, poor ventilation, or loud talking and singing. In such cases, fluctuations in one contagion mode may be statistically linked to fluctuations in the other. Future extensions of the model could consider correlated noise processes to account for environments or scenarios where contagion pathways are more tightly coupled. For example, $Z_1 = W^1$ and $Z_2 = \rho W^1 + \sqrt{1-\rho^2}W^2$, where $0 \leq \rho \leq 1$ and $W = (W^1, W^2)$ is a 2-dimensional Wiener process, could be used as correlated noise sources to perturb the parameters β and β_Δ, thus obtaining a generalization of the models presented here and in [32]; these correspond to the cases $\rho = 0$ and $\rho = 1$, respectively.

Mean-field approximation. The model relies on a mean-field framework, which assumes a homogeneous mixing of individuals and uniform interaction patterns across the network. That is, it essentially assumes that the contact network topology follows a degree distribution of Poisson or exponential type. This assumption facilitates analytical tractability and provides general insights into the global behavior of the system. However, it may not adequately reflect the structural heterogeneity found in many real-world networks, particularly those with scale-free degree distributions, where a few nodes or simplices (so-called hubs) have disproportionately high connectivity. Such heterogeneity can give rise to phenomena like community clustering, local outbreaks and super-spreader events, features that are not captured by the mean-field approach. To address these limitations, future studies could incorporate more realistic network topologies, including modular, small-world, or scale-free structures on higher order networks, and assess how these influence the effect of stochastic perturbations. Alternatively, agent-based simulations could be employed to evaluate how individual-level interactions and network variability impact the thresholds and dynamical regimes described in this work.

Disease-specific applicability. The proposed framework is particularly suited for modeling infectious diseases characterized by continuous transmission, short infectious periods, and no long-term immunity—such as certain respiratory or endemic viral infections (e.g., common cold, influenza, or early phases of COVID-19). These conditions align well with the SIS structure of the model, where individuals can become reinfected repeatedly after recovery. However, for diseases with more complex progression patterns, such as those involving latent periods, long-lasting immunity, or structured interventions, the current model would need to be extended to include additional compartments (e.g., exposed, recovered, vaccinated, quarantined). For instance, incorporating an SEIR compartimental structure (susceptible–exposed–infected–recovered) would be necessary to model incubation delays, while adding a vaccinated or quarantined class would allow for the

study of public health interventions and control strategies. Moreover, these extensions would likely require more elaborate stochastic dynamics, potentially involving non-Markovian effects, time-dependent parameters, or correlated noise terms. Exploring such generalizations would provide a more comprehensive framework applicable to a wider range of infectious diseases and policy scenarios.

These limitations suggest several directions for future research, which would contribute to a more realistic and comprehensive understanding of epidemic dynamics under uncertainty.

5. Conclusions

In this study, we have modified the stochastic simplicial SIS model proposed in [32] to include two independent sources of noise that affect the individual and collective infection rates, respectively. Our analysis shows that when the stochastic basic reproductive number $R_0^S < 1$ and the additional conditions (2.10) are satisfied, the disease will die out with probability one. On the other hand, when $R_0^S > 1$, the solution of the SDE will oscillate infinitely around a point that can be explicitly calculated. We have also introduced a new stochastic basic reproductive number R_0^{SS} and have shown that if $R_0^{SS} < 1$, the disease will still die out with probability one, even if one of the conditions in (2.10) is not met. To validate our theoretical results, we have conducted numerical experiments using various methods of analysis. We have also proposed a generalization of the stochastic simplicial contagion model [17] that includes two independent sources of noise and applied it to simulations on a real and synthetic simplicial network. Our empirical results confirm the theoretical findings presented in this study.

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