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This is the final peer-reviewed author's accepted manuscript (postprint) of the following publication:

Published Version: Lanconelli A., Mori M. (2022). Itô vs Stratonovich stochastic SIR models. APPLIED MATHEMATICS LETTERS, 134(December), 1-7 [10.1016/j.aml.2022.108368].

Availability:

This version is available at: https://hdl.handle.net/11585/893412 since: 2022-09-02

Published:

DOI: http://doi.org/10.1016/j.aml.2022.108368

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(Article begins on next page)

This is the final peer-reviewed accepted manuscript of:

Alberto Lanconelli, Matteo Mori. (2022). "Itô vs Stratonovich stochastic SIR models". *Applied Mathematics Letters*, Vol. 134, December, Art. No. 108368.

The final published version is available online at: https://doi.org/10.1016/j.aml.2022.108368

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Itô vs Stratonovich stochastic SIR models

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July 28, 2022

Abstract

We prove that the asymptotic behavior of the Stratonovich counterpart of the Itô's type stochastic SIR model investigated in [12], [5] and [6] is ruled by the same threshold as the deterministic system. In other words, in contrast to the Itô's model, the intensity of the noise described through the Stratonovich calculus is not relevant for the extinction of the disease. The Stratonovich interpretation of the model is motivated by the parameter perturbation technique, employed on the disease transmission coefficient and used to implement environmental randomness, in combination with the classical Wong-Zakai approximation argument.

Key words and phrases: SIR epidemic model, Itô and Stratonovich stochastic differential equations, Wong-Zakai approximation, extinction, persistence.

AMS 2000 classification: 60H10, 60H30, 92D30.

1 Introduction and statement of the main result

The susceptible-infected-recovered (SIR) model is a simple mathematical model that describes, under suitable assumptions, the spread of diseases with permanent immunity upon recovery (see e.g. [2]). If S(t), I(t) and R(t) denote the number of susceptible, infected and recovered at time t, respectively, then the differential equations describing the evolution of the disease are

$$\begin{cases} \frac{dS(t)}{dt} = -\beta S(t)I(t) + \mu - \mu S(t), & S(0) = s_0; \\ \frac{dI(t)}{dt} = \beta S(t)I(t) - (\mu + \gamma)I(t), & I(0) = i_0; \\ \frac{dR(t)}{dt} = \gamma I(t) - \mu R(t), & R(0) = r_0. \end{cases}$$
(1.1)

Here, $s_0, i_0, r_0 \ge 0$ with $s_0 + i_0 + r_0 = 1$; μ denotes the birth/death rate, γ is the rate at which infected individuals recover and β stands for the disease transmission coefficient. It is well known that for $\beta \le \gamma + \mu$ the solution of (1.1) converges to the disease free equilibrium (1,0,0); otherwise, the system reaches an endemic equilibrium and the disease persists in the population.

To account for environmental stochasticity, the authors of [12] proposed a perturbed version of (1.1) in which β is formally replaced by β + *Gaussian white noise*; this leads to the system of stochastic differential equations

$$\begin{cases} dS(t) = [-\beta S(t)I(t) + \mu - \mu S(t)] dt - \sigma S(t)I(t)dW(t), & S(0) = s_0; \\ dI(t) = [\beta S(t)I(t) - (\gamma + \mu)I(t)] dt + \sigma S(t)I(t)dW(t), & I(0) = i_0; \\ dR(t) = [\gamma I(t) - \mu R(t)] dt, & R(0) = r_0, \end{cases}$$
(1.2)

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where $\{W(t)\}_{t\geq 0}$ is a one dimensional Brownian motion while the positive parameter σ reflects the intensity of the noise; it is important to remark that system (1.2) is interpreted in [12] in the Itô's sense. The authors investigated the stability of the disease-free equilibrium (1,0,0) of (1.2) identifying a sufficient condition involving β , γ , μ and σ^2 ; this condition was subsequently improved in [5].

A further contribution in this direction is contained in [6]: here the authors found how the intensity of the noise, i.e. σ^2 , affects extinction and persistence of the disease. More precisely, they proved that inequality $\beta - \frac{\sigma^2}{2} < \gamma + \mu$ is sufficient for the extinction of the disease (when $\sigma^2 < \beta$), while $\beta - \frac{\sigma^2}{2} > \gamma + \mu$ is sufficient for its persistence (again for $\sigma^2 < \beta$).

The aim of this note is to investigate the asymptotic behavior of the system

which differs from (1.2) for the Stratonovich interpretation (denoted with the symbol $\circ dW(t)$) of the diffusive part of the equation; we will prove that the asymptotic behavior of solution to (1.3) is not influenced by the size of σ^2 .

The reason for considering (1.3) instead of (1.2) is as follows: starting from (1.1) and replacing β with $\beta + \sigma \frac{dW_n(t)}{dt}$ we get

$$\begin{cases} \frac{dS_n(t)}{dt} = -\beta S_n(t)I_n(t) + \mu - \mu S_n(t) - \sigma S_n(t)I_n(t)\frac{dW_n(t)}{dt}, & S_n(0) = s_0; \\ \frac{dI_n(t)}{dt} = \beta S_n(t)I_n(t) - (\mu + \gamma)I_n(t) + \sigma S_n(t)I_n(t)\frac{dW_n(t)}{dt}, & I_n(0) = i_0; \\ \frac{dR_n(t)}{dt} = \gamma I_n(t) - \mu R_n(t), & R_n(0) = r_0; \end{cases}$$
(1.4)

here, $\{W_n(t)\}_{t\geq 0}$ is the polygonal approximation of $\{W(t)\}_{t\geq 0}$ (see [7]). Then, according to the Wong-Zakai theorem [14] the solution of (1.4) converges to the solution of (1.3) as *n* tends to infinity, thus motivating the proposed model. We also mention that the Stratonovich system (1.3) can be rewritten in the Itô's form (see formula (6.1.3) in [11])

$$\begin{cases} d\mathbf{S}(t) = \begin{bmatrix} -\beta \mathbf{S}(t)\mathbf{I}(t) + \mu - \mu \mathbf{S}(t) - \frac{\sigma^2}{2}\mathbf{S}(t)\mathbf{I}(t)(\mathbf{S}(t) - \mathbf{I}(t)) \\ d\mathbf{I}(t) = \begin{bmatrix} \beta \mathbf{S}(t)\mathbf{I}(t) - (\gamma + \mu)\mathbf{I}(t) + \frac{\sigma^2}{2}\mathbf{S}(t)\mathbf{I}(t)(\mathbf{S}(t) - \mathbf{I}(t)) \end{bmatrix} dt - \sigma \mathbf{S}(t)\mathbf{I}(t)dW(t), & \mathbf{I}(0) = s_0; \\ d\mathbf{R}(t) = \begin{bmatrix} \gamma \mathbf{I}(t) - \mu \mathbf{R}(t) \end{bmatrix} dt, & \mathbf{R}(0) = r_0. \end{cases}$$
(1.5)

We are now ready to state our main result.

Theorem 1.1. Equation (1.5) possesses a unique global strong solution that fulfils the conditions

$$\mathbf{S}(t) \ge 0, \quad \mathbf{I}(t) \ge 0, \quad \mathbf{R}(t) \ge 0 \quad and \quad \mathbf{S}(t) + \mathbf{I}(t) + \mathbf{R}(t) = 1, \tag{1.6}$$

for all $t \geq 0$ with probability one. Moreover,

- if $\beta < \gamma + \mu$, then I(t) tends to zero, as t tends to infinity, almost surely;
- if $\beta > \gamma + \mu$, then

$$\liminf_{t \to +\infty} \frac{1}{t} \int_0^t \mathbf{I}(s) ds \ge \frac{\beta - (\mu + \gamma)}{\frac{\beta(\mu + \gamma)}{\mu} + \frac{\sigma^2}{2}} \quad and \quad \limsup_{t \to +\infty} \frac{1}{t} \int_0^t \mathbf{I}(s) ds \le \frac{\beta - (\mu + \gamma)}{\frac{\beta(\mu + \gamma)}{\mu}}, \tag{1.7}$$

almost surely.

Remark 1.2. We observe that, while the size of σ doesn't interfere with the extinction of the disease, it plays a role in the persistence regime. One can in fact see from the inequalities in (1.7) that an increase of the size of σ determines a decrease of the lower bound for the limit and hence larger fluctuations for I(t), as t tends to infinity.

Remark 1.3. The previous theorem asserts that in the Stratonovich model (1.5) the conditions for extinction and persistence of the disease coincide with those of the deterministic system (1.1). This result extends to the SIR model an equivalent feature shown recently for the SIS model: see [4],[1],[9]. It also contributes to the fundamental modelling issues discussed for instance in [3] and [13].

2 Proof of the main result

2.1 Existence, uniqueness and conditions (1.6) for the solution to (1.5)

The coefficients of system (1.5) are locally Lipschitz continuous; therefore, according to Theorem 5.2.5 in [7] there exists a unique, possibly local in time, strong solution. To prove that such solution is global, i.e. it is almost surely finite for all $t \ge 0$, we can borrow the technique based on the Lyapunov function employed in [5] where the authors prove an equivalent statement for the Itô's version of (1.3), i.e. system (1.2). Our goal is achieved following step-by-step the proof of Theorem 2.1 in [5]: the only minor modifications needed to recover our case consist in setting $b = \mu$, $\alpha = 0$ and replacing the term $\frac{\sigma^2}{2}I(t)^2 + \frac{\sigma^2}{2}S(t)^2$ from the second-to-last line of page 759 in [5] with $\sigma^2\mathbf{I}(t)\mathbf{S}(t)$. This is due to the presence of the Itô-Stratonovich correction term $\frac{\sigma^2}{2}\mathbf{S}(t)\mathbf{I}(t)(\mathbf{S}(t) - \mathbf{I}(t))$, which distinguishes model (1.3) from (1.2). Nevertheless, using inequality $\sigma^2\mathbf{I}(t)\mathbf{S}(t) \leq \frac{\sigma^2}{2}\mathbf{I}(t)^2 + \frac{\sigma^2}{2}\mathbf{S}(t)^2$, we see that the key ingredients of the proof of Theorem 2.1 in [5] are fully restored and we can conclude without any further change. This proves existence, uniqueness and non negativity for the solution to (1.5) (we remark that Theorem 2.1 from [5] also includes the non negativity of the components of the solution). We are left with the proof of identity $\mathbf{S}(t) + \mathbf{I}(t) + \mathbf{R}(t) = 1$, for all $t \ge 0$; summing the three equations in (1.5) we get $\frac{d}{dt}(\mathbf{S}(t) + \mathbf{I}(t) + \mathbf{R}(t)) = \mu[1 - (\mathbf{S}(t) + \mathbf{I}(t) + \mathbf{R}(t))]$, which, together with $s_0 + i_0 + r_0 = 1$, yields $\frac{d}{dt}(\mathbf{S}(t) + \mathbf{I}(t) + \mathbf{R}(t)) = 0$, and hence the thesis.

2.2 Extinction of the disease for $\beta < \gamma + \mu$

We rewrite the equation for $\{I(t)\}_{t>0}$ in (1.5) as

$$d\mathbf{I}(t) = \mathbf{I}(t) \left[\beta \mathbf{S}(t) - (\gamma + \mu) + \frac{\sigma^2}{2} \mathbf{S}(t)^2 - \frac{\sigma^2}{2} \mathbf{S}(t) \mathbf{I}(t) \right] dt + \sigma \mathbf{S}(t) \mathbf{I}(t) dW(t)$$

= $\mathbf{I}(t) \left[\xi(t) - \eta(t) \mathbf{I}(t) \right] dt + \theta(t) \mathbf{I}(t) dW(t),$ (2.1)

where we set

$$\xi(t) := \beta \mathbf{S}(t) - (\gamma + \mu) + \frac{\sigma^2}{2} \mathbf{S}(t)^2, \quad \eta(t) := \frac{\sigma^2}{2} \mathbf{S}(t) \quad \text{and} \quad \theta(t) := \sigma \mathbf{S}(t).$$

Observe that these stochastic processes are bounded, adapted to the Brownian filtration and $\eta(t)$ is non negative. Therefore, according to (2.1) we can represent I(t) as the solution of a logistic stochastic differential equation with random coefficients:

$$\mathbf{I}(t) = \frac{i_0 e^{\int_0^t (\xi(r) - \theta(r)^2/2) dr + \int_0^t \theta(r) dW_r}}{1 + \int_0^t \eta(s) i_0 e^{\int_0^s (\xi(r) - \theta(r)^2/2) dr + \int_0^s \theta(r) dW_r} ds}.$$
(2.2)

We now upper bound I(t) with the numerator of the right hand side of (2.2), we take the natural logarithm and we divide by t; this gives

$$\frac{1}{t}\ln(\mathbf{I}(t)) \le \frac{\ln(i_0)}{t} + \frac{1}{t} \int_0^t (\xi(r) - \theta(r)^2/2) dr + \frac{1}{t} \int_0^t \theta(r) dW_r$$
$$= \frac{\ln(i_0)}{t} + \frac{1}{t} \int_0^t [\beta \mathbf{S}(r) - (\gamma + \mu)] dr + \frac{1}{t} \int_0^t \sigma \mathbf{S}(r) dW_r$$

$$\leq \frac{\ln(i_0)}{t} + \beta - (\gamma + \mu) + \frac{1}{t} \int_0^t \sigma \mathbf{S}(r) dW_r$$

where in the last inequality we utilized the bound $\mathbf{S}(t) \leq 1$. We remark that the boundedness of $\{\mathbf{S}(t)\}_{t\geq 0}$ implies that the stochastic integral $\int_0^t \sigma \mathbf{S}(r) dW_r$ is a martingale with quadratic variation upper bounded by $\sigma^2 t$; therefore, according to Theorem 3.4 in [10] we get

$$\lim_{t \to +\infty} \frac{1}{t} \int_0^t \sigma \mathbf{S}(r) dW_r = 0, \quad \text{almost surely.}$$

This fact yields

$$\limsup_{t \to +\infty} \frac{1}{t} \ln(\mathbf{I}(t)) \le \beta - (\gamma + \mu) < 0,$$

which corresponds to the exponential convergence to zero of $\{I(t)\}_{t\geq 0}$, as t tends to infinity.

2.3 Persistence of the disease for $\beta > \gamma + \mu$

First of all we observe that

$$S(t) + I(t) = s_0 + i_0 + \int_0^t [\mu - \mu S(s) - (\gamma + \mu)I(s)] ds$$

and hence that

$$\frac{1}{t} \int_0^t \mathbf{S}(s) ds = 1 - \frac{\gamma + \mu}{\mu t} \int_0^t \mathbf{I}(s) ds + \varphi(t),$$
(2.3)

where

$$\varphi(t) := \frac{s_0 + i_0}{\mu t} - \frac{\mathbf{S}(t) + \mathbf{I}(t)}{\mu t}.$$

Now, using the Itô's formula we can write

$$\begin{split} \frac{1}{t}\ln(\mathbf{I}(t)) &= \frac{1}{t}\left(\ln(i_0) + \int_0^t \left[\beta \mathbf{S}(s) - (\gamma + \mu) - \frac{\sigma^2}{2}\mathbf{S}(s)\mathbf{I}(s)\right] ds + \int_0^t \sigma S(s)dW(s)\right) \\ &= \frac{\ln(i_0)}{t} + \frac{\beta}{t} \int_0^t \mathbf{S}(s)ds - (\gamma + \mu) - \frac{1}{t}\frac{\sigma^2}{2} \int_0^t \mathbf{S}(s)\mathbf{I}(s)ds + \frac{1}{t} \int_0^t \sigma S(s)dW(s) \\ &\geq \frac{\ln(i_0)}{t} + \frac{\beta}{t} \int_0^t \mathbf{S}(s)ds - (\gamma + \mu) - \frac{1}{t}\frac{\sigma^2}{2} \int_0^t \mathbf{I}(s)ds + \frac{1}{t} \int_0^t \sigma S(s)dW(s) \\ &= \frac{\ln(i_0)}{t} + \beta - \frac{\beta(\gamma + \mu)}{\mu t} \int_0^t \mathbf{I}(s)ds + \beta\varphi(t) - (\gamma + \mu) - \frac{1}{t}\frac{\sigma^2}{2} \int_0^t \mathbf{I}(s)ds \\ &+ \frac{1}{t} \int_0^t \sigma S(s)dW(s) \\ &= \beta - (\gamma + \mu) - \left(\frac{\beta(\gamma + \mu)}{\mu} + \frac{\sigma^2}{2}\right)\frac{1}{t} \int_0^t \mathbf{I}(s)ds + \tilde{\varphi}(t). \end{split}$$

Here, we set

$$\tilde{\varphi}(t) := \beta \varphi(t) + \frac{\ln(i_0)}{t} + \frac{1}{t} \int_0^t \sigma S(s) dW(s);$$

the inequality above is a consequence of the bound $S(s) \leq 1$ for all $s \geq 0$ while in the second-to-last equality we employed identity (2.3). This proves that

$$\frac{1}{t}\ln(\mathtt{I}(t)) \ge \beta - (\gamma + \mu) - \left(\frac{\beta(\gamma + \mu)}{\mu} + \frac{\sigma^2}{2}\right) \frac{1}{t} \int_0^t \mathtt{I}(s) ds + \tilde{\varphi}(t),$$

with $\tilde{\varphi}(t) \to 0$ as $t \to +\infty$, almost surely. According to Lemma 5.1 in [6] the last inequality implies

$$\liminf_{t \to +\infty} \frac{1}{t} \int_0^t \mathbf{I}(s) ds \ge \frac{\beta - (\gamma + \mu)}{\frac{\beta(\gamma + \mu)}{\mu} + \frac{\sigma^2}{2}} > 0, \quad \text{almost surely.}$$

The statement about the limsup is obtained similarly writing

$$\begin{split} \frac{1}{t}\ln(\mathtt{I}(t)) = & \frac{\ln(i_0)}{t} + \frac{\beta}{t} \int_0^t \mathtt{S}(s)ds - (\gamma + \mu) - \frac{1}{t}\frac{\sigma^2}{2} \int_0^t \mathtt{S}(s)\mathtt{I}(s)ds + \frac{1}{t} \int_0^t \sigma S(s)dW(s) \\ \leq & \frac{\ln(i_0)}{t} + \frac{\beta}{t} \int_0^t \mathtt{S}(s)ds - (\gamma + \mu) + \frac{1}{t} \int_0^t \sigma S(s)dW(s), \end{split}$$

using equality (2.3) and concluding with Lemma 5.2 from [6].

3 Numerical illustrations

We illustrate in the figures below our theoretical findings through some simulations performed with the software R. We utilized a Milstein scheme (see e.g. [8]) to numerically approximate the solutions to (1.1), (1.2) and (1.5) with step size 0.01. We plotted the number of infected against time for the systems (1.1),(1.2) and (1.5) with $\gamma = 0.4$, $\mu = 0.3$ and I(0) = I(0) = 0.4.

Figures (a) and (b), in case of extinction, and figures (c) and (d), in case of persistence, illustrate how the process I(t) from our model (1.5) behaves like the function I(t) from (1.1) for different values of σ . In figures (e) and (f) we see how the behavior of the solution to (1.2) may agree or disagree with those of (1.5) and (1.1).

4 Discussion

In this paper we studied the asymptotic behavior of the solution to system (1.5). This system is rigorously derived from the classical deterministic SIR model (1.1) through a stochastic perturbation of the disease transmission coefficient β . This procedure is carried out via an approximation argument that allows to handle the singularities of the Gaussian white noise. Our system differs from (1.2), which is widely studied in the literature, for the stochastic calculus utilized to model the noise. We proved that the threshold for extinction and persistence of our model agrees with the deterministic one; this feature is not shared with the model (1.2) where the size of the noise condition the asymptotic regimes of the disease. Such striking difference emphasizes once more the subtleties connected with the choice of the model. Our approach can in principle be utilized to derive other stochastic models from deterministic ones with the aim of adding environmental stochasticity to the description of the investigated phenomena.

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(a) Three simulated paths of (1.5) (black) tend to zero like the solution to (1.1) with $\frac{\beta}{\gamma+\mu}=0.86$ and $\sigma=0.2$



(b) Three simulated paths of (1.5) for different values of σ tend to zero like the solution to (1.1) (black) with $\frac{\beta}{\gamma+\mu}=0.86$



(c) Three simulated paths of (1.5) (black) persist like (d) Three simulated paths of (1.5) for different values of the solution to (1.1) with $\frac{\beta}{\gamma+\mu} = 2$ and $\sigma = 0.2$

 σ persist like the solution to (1.1) (black) with $\frac{\beta}{\gamma+\mu}=2$



(e) One simulated path of (1.5) (red), one of (1.2) (f) One simulated path of (1.5) (red), one of (1.2)(blue) and the solution to (1.1) (black): here $\sigma = 0.75$ (blue) and the solution to (1.1) (black): here $\sigma = 0.75$ and $\frac{\beta}{\gamma + \mu} = 1.71$ imply persistence for (1.2) (see [6]) and $\frac{\beta}{\gamma + \mu} = 1.29$ imply extinction for (1.2) (see [6]) and $\frac{\beta}{\gamma+\mu} = 1.71$ imply persistence for (1.2) (see [6])

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