



Capturing Memory and Randomness in Epidemic Dynamics

Bojana Jovanović

Faculty of Sciences and Mathematics, University of Niš

bojana.jovanovic@pmf.edu.rs

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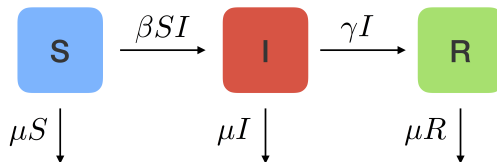
Joint work with prof. dr Jasmina Đorđević and prof. dr Nenad Šuvak

Deterministic SIR model – a system of ordinary differential equations

$$S' = \Lambda - \beta SI - \mu S,$$

$$I' = \beta SI - (\gamma + \mu)I,$$

$$R' = \gamma I - \mu R.$$



- A more realistic assumption is that the model parameters vary over time,

$$\beta \longrightarrow \beta(t), \quad t \geq 0.$$

This leads to a deterministic model that provides a more realistic description of the epidemic dynamics, but is also more complex.

- Environmental conditions have a significant impact on the behaviour of population groups and on the transmission of infectious diseases. However, **random environmental effects** cannot be described by deterministic models. For this reason, epidemic models described by different types of stochastic differential equations (SDEs) have been considered.

References:

- X. Mao, *Stochastic Differential Equations and Applications*, Woodhead Publishing Limited, 2011.
- X. Mao, C. Yuan. *Stochastic Differential Equations With Markovian Switching*. Imperial College Press, 2006.
- B. Øksendal, A. Sulem. *Applied Stochastic Control of Jump Diffusions*. Heidelberg: Springer-Verlag, 2005.
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There are **several ways** to introduce environmental variability into epidemiological models.

1. One approach consists of perturbing the parameters of the deterministic epidemic model,

$$\beta \longrightarrow \beta + \text{"noise"} \approx \beta + \sigma \frac{dB(t)}{dt}, \quad \sigma > 0.$$

where $\{B(t), t \geq 0\}$ is a Brownian motion, $dB(t)$ is its increment with distribution $\mathcal{N}(0, dt)$, and σ is the noise intensity.

Substituting this perturbation into the deterministic SIR model yields the following system of SDEs

$$\begin{aligned} dS(t) &= (\Lambda - \beta S(t)I(t) - \mu S(t)) dt - \sigma S(t)I(t) dB(t), \\ dI(t) &= (\beta S(t)I(t) - (\rho + \mu)I(t)) dt + \sigma S(t)I(t) dB(t), \\ dR(t) &= (\rho I(t) - \mu R(t)) dt. \end{aligned}$$

Reference: B. Jovanović, J. Đorđević, J. Manojlović, N. Šuvak. *Analysis of stability and sensitivity of deterministic and stochastic models for the spread of the new corona virus SARS-CoV-2. Filomat*, 35 (3):1045–1063.

2. Incorporation of random noise into the deterministic model so that its intensity depends on the population size. This leads to the following system of SDEs

$$\begin{aligned}dS(t) &= (\Lambda - \beta S(t)I(t) - \mu S(t)) dt + \sigma_1 S(t) dB_1(t), \\dI(t) &= (\beta S(t)I(t) - (\rho + \mu)I(t)) dt + \sigma_2 I(t) dB_2(t), \\dR(t) &= (\rho I(t) - \mu R(t)) dt + \sigma_3 R(t) dB_3(t),\end{aligned}$$

where $B_1(t)$, $B_2(t)$ and $B_3(t)$ are mutually independent Brownian motions.

Reference: J. Đorđević, B. Jovanović. Optimal Control of a Stochastic SVIR Model with Logistic Growth and Saturated Incidence Function. In A. A. Elsadany, W. Adel, Y. Sabbar (Eds). Biology and Sustainable Development Goals, Mathematics for Sustainable Developments. Springer, Singapore, 2025.

3. Incorporation of random noises of Brownian and Lévy type into the deterministic model, so that its intensity depends on the population size.

$$\begin{aligned}dS(t) &= (\Lambda - \beta S(t)I(t) - \mu S(t)) dt + \sigma_1 S(t) dB_1(t) + \int_R \eta_1(u) S(t^-) \tilde{N}(dt, du), \\dI(t) &= (\beta S(t)I(t) - (\rho + \mu)I(t)) dt + \sigma_2 I(t) dB_2(t) + \int_R \eta_2(u) I(t^-) \tilde{N}(dt, du), \\dR(t) &= (\rho I(t) - \mu R(t)) dt + \sigma_3 R(t) dB_3(t) + \int_R \eta_3(u) R(t^-) \tilde{N}(dt, du),\end{aligned}$$

where $B_1(t)$, $B_2(t)$ and $B_3(t)$ are mutually independent Brownian motions, $\tilde{N}$ the compensator of a Poisson process that is independent of B_i , $i = 1, 2, 3$.

Reference: B. Jovanović. Dynamical analysis and stationary distribution of a stochastic delayed epidemic model with Lévy jump, Mathematical Communications, 30, 2025, 1-25.

4. Perturbing the parameters of the deterministic epidemic model

$$dS(t) = (\Lambda(a(t)) - \beta(a(t))S(t)I(t) - \mu(a(t))S(t)) dt - \sigma(a(t))S(t)I(t)dB(t) \\ - \int_R \eta(u, (a(t)))S(t^-)I(t^-)\tilde{N}(dt, du),$$

$$dI(t) = (\beta(a(t))S(t)I(t) - (\rho(a(t)) + \mu(a(t)))I(t)) dt + \sigma(a(t))S(t)I(t)dB(t) \\ + \int_R \eta(u, (a(t)))S(t^-)I(t^-)\tilde{N}(dt, du),$$

$$dR(t) = (\rho(a(t))I(t) - \mu(a(t))R(t)) dt,$$

Changes in the values of the model parameters are modeled by an irreducible, right-continuous **Markov chain** $a(t)$, $t \geq -\tau$, with values in the finite state space $A = \{1, \dots, n\}$ and generator matrix $\Upsilon = (\gamma_{ij})_{n \times n}$, where

$$\mathbb{P}\{a(t + \Delta t) = j \mid a(t) = i\} = \begin{cases} \gamma_{ij}\Delta t + o(\Delta t), & i \neq j \\ 1 + \gamma_{ii}\Delta t + o(\Delta t), & i = j \end{cases}$$

where $\Delta t > 0$, $\gamma_{ij} \geq 0$ are the transition rates from state i to state j , $i \neq j$, and $\gamma_{ii} \leq 0$, such that $\sum_{i=1}^n \gamma_{ij} = 0$, and $\gamma_{ij} > 0$ for $i \neq j$.

The Brownian motion $B(t)$, the Markov chain $a(t)$, and the Poisson process $\tilde{N}(t, \omega)$ are **mutually independent**.

Reference: J. Đorđević, B. Jovanović, Dynamical analysis of a stochastic delayed epidemic model with Lévy jumps and regime switching. Journal of the Franklin Institute 360 (2023) 1252-1283.

Why Volterra stochastic integral equations?

Classical stochastic differential equation (SDE) models are **Markovian**: the future evolution depends only on the current state.

In contrast, Volterra stochastic integral equations naturally describe **non-Markovian** epidemic dynamics by incorporating the memory of the infection process.

In our model, the Volterra kernels represent the distributions of

- incubation period,
- infectious period,
- duration of naturally acquired immunity,
- duration of vaccine-induced immunity.

Consequently, the model allows **general probability distributions** instead of assuming exponentially distributed transition times.

Motivation

- R. Forien, G. Pang, and E. Pardoux. Estimating the state of the COVID-19 epidemic in France using a non-Markovian model. Royal Society Open Science, 8:202327, 2021.
- R. Forien, G. Pang, and E. Pardoux. Epidemic models with varying infectivity. SIAM J. Applied Math., 81:1893–1930, 2021.
- R. Forien and G. Pang. Functional central limit theorems for epidemic models with varying infectivity. Stochastics, to appear.
- R. Forien, G. Pang, and E. Pardoux. Multi-patch multi-group epidemic model with varying infectivity. Probab. Uncertain. Quant. Risk 7, 4:333–364, 2022.
- G. Pang and E. Pardoux. Multi-patch epidemic models with general exposed and infectious periods. ESAIM P & S, to appear.
- R.E. Forien, G. Pang, E. Pardoux, and A.B. Zotsa-Ngoufack. Stochastic epidemic models with varying infectivity and susceptibility. Arhiv, 2210.04667,.
- R. Forien, G. Pang, and E. Pardoux. Recent advances in epidemic modeling: NonMarkov Stochastic Models and their Scaling Limits. Graduate J. Math., 7:19–75, 2022.

SDEs models vs Volterra SDEs models

Advantages over classical SDE models

- incorporates memory effects through convolution integrals;
- models realistic non-exponential epidemiological periods;
- allows infection-age dependent infectivity;
- captures delayed loss of natural and vaccine immunity;
- provides a more realistic description of epidemic dynamics.

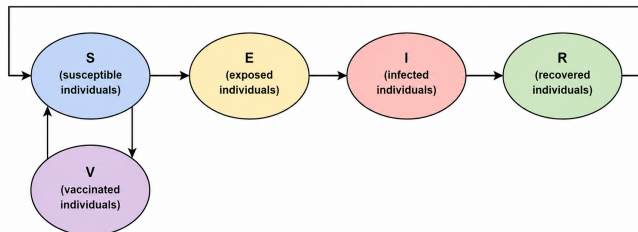
Reference: J. Đorđević, B. Jovanović, N. Šuvak, Volterra-type integral model for epidemic of COVID-19. Stochastics, 98(2), 2026, 184–210.

Population structure

The population of size n is divided into five compartments ($n = S^n(t) + V^n(t) + E^n(t) + I^n(t) + R^n(t)$, $t \geq 0$):

- $S^n(t)$ susceptible individuals,
- $V^n(t)$ vaccinated individuals,
- $E^n(t)$ exposed (latent) individuals,
- $I^n(t)$ infectious individuals,
- $R^n(t)$ recovered individuals.

The disease evolution follows



Counting process of new infections

The cumulative number of newly infected individuals is

$$A_E^n(t) = A^* \left(n \int_0^t \beta(s) \frac{S^n(s)}{n} \frac{I^n(s)}{n} ds \right),$$

where

- A^* is a unit-rate Poisson process,
- $\beta(t)$ is the deterministic contact (transmission) rate.

The Poisson process determines the random arrival times of new infections.

Disease history of an infected individual

Each infected individual follows the path

$$S \longrightarrow E \longrightarrow I \longrightarrow R \longrightarrow S.$$

The successive disease stages are:

- τ_i – the time of becoming exposed,
- $\tau_i + \mathcal{E}_i$ – the time of becoming infectious,
- $\tau_i + \mathcal{E}_i + \mathcal{I}_i$ – the time of becoming immune,
- $\tau_i + \mathcal{E}_i + \mathcal{I}_i + \mathcal{W}_i$ – the time of becoming susceptible again,

where

- $\mathcal{E}_i$ – exposure (latent) period,
- $\mathcal{I}_i$ – infectious period,
- $\mathcal{W}_i$ – duration of naturally acquired immunity.

Distribution functions

An individual can initially be exposed ($\mathcal{E}_i^0$), infected ($\mathcal{I}_i^0$) or recovered ($\mathcal{W}_i^0$), their distributions are given as follows:

$$G^c(t) := P(t < \mathcal{E}_i)$$

$$\Psi(t) := P(\mathcal{E}_i \leq t < \mathcal{E}_i + \mathcal{I}_i),$$

$$\Phi(t) := P(\mathcal{E}_i + \mathcal{I}_i \leq t < \mathcal{E}_i + \mathcal{I}_i + \mathcal{W}_i),$$

$$\Xi(t) := P(\mathcal{E}_i + \mathcal{I}_i + \mathcal{W}_i \leq t)$$

$$G_1^c(t) := P(t < \mathcal{I}_i^0),$$

$$\Phi_1(t) := P(\mathcal{I}_i^0 \leq t < \mathcal{I}_i^0 + \mathcal{W}_i)$$

$$\Xi_1(t) := P(\mathcal{I}_i^0 + \mathcal{W}_i \leq t)$$

$$G_0^c(t) := P(t < \mathcal{E}_i^0)$$

$$\Psi_0(t) := P(\mathcal{E}_i^0 \leq t < \mathcal{E}_i^0 + \mathcal{I}_i)$$

$$\Phi_0(t) := P(\mathcal{E}_i^0 + \mathcal{I}_i \leq t < \mathcal{E}_i^0 + \mathcal{I}_i + \mathcal{W}_i)$$

$$\Xi_0(t) := P(\mathcal{E}_i^0 + \mathcal{I}_i + \mathcal{W}_i \leq t)$$

$$G_2^c(t) := P(t < \mathcal{W}_i^0)$$

$$G_2(t) := P(\mathcal{W}_i^0 \leq t).$$

These kernels determine all transition probabilities in the Volterra model.

Vaccinated

- The class $V^n(t)$ denotes the number of vaccinated individuals at time t in the population of fixed size n .
- The cumulative number of newly vaccinated individuals up to time $t > 0$ is described by the counting process in the population of fixed size n :

$$A_V^n(t) := A_{**} \left(\int_0^t \alpha(s) S^n(s) ds \right), \quad t \geq 0,$$

where A_{**} is a unit-rate Poisson process, independent of the Poisson process A_* .

- The duration of the immunity gained by vaccination for the i -th individual is given by the random time $\mathcal{Y}_i$, i.e. by the random time $\mathcal{Y}_i^0$ for those individuals who are already vaccinated at $t = 0$, where

$$\Upsilon(t) := P(\mathcal{Y}_i \leq t), \quad \Upsilon^c(t) := 1 - \Upsilon(t) = P(t < \mathcal{Y}_i).$$

Distribution function Υ^0 for the random time $\mathcal{Y}_i^0$ is defined analogously.

Counting epidemic model

$$\begin{aligned}
 S^n(t) &:= S^n(\mathbf{0}) + \sum_{i=1}^{V^n(\mathbf{0})} \mathbb{I}\{\mathcal{Y}_i^{\mathbf{0}} \leq t\} + \sum_{i=1}^{E^n(\mathbf{0})} \mathbb{I}\{\varepsilon_i^{\mathbf{0}} + \mathcal{I}_i + \mathcal{W}_i \leq t\} + \sum_{i=1}^{I^n(\mathbf{0})} \mathbb{I}\{\mathcal{I}_i^{\mathbf{0}} + \mathcal{W}_i \leq t\} + \sum_{i=1}^{R^n(\mathbf{0})} \mathbb{I}\{\mathcal{W}_i^{\mathbf{0}} \leq t\} + \\
 &\quad + \sum_{i=1}^{A_V^n(t)} \mathbb{I}\{\tau_i + \mathcal{Y}_i \leq t\} + \sum_{i=1}^{A_E^n(t)} \mathbb{I}\{\tau_i + \varepsilon_i + \mathcal{I}_i + \mathcal{W}_i \leq t\} - A_E^n(t) - A_V^n(t),
 \end{aligned}$$

$$V^n(t) := \sum_{i=1}^{V^n(\mathbf{0})} \mathbb{I}\{\mathcal{Y}_i^{\mathbf{0}} > t\} + \sum_{i=1}^{A_V^n(t)} \mathbb{I}\{\tau_i + \mathcal{Y}_i > t\},$$

$$E^n(t) := \sum_{i=1}^{E^n(\mathbf{0})} \mathbb{I}\{\varepsilon_i^{\mathbf{0}} > t\} + \sum_{i=1}^{A_E^n(t)} \mathbb{I}\{\tau_i + \varepsilon_i > t\},$$

$$I^n(t) := \sum_{i=1}^{I^n(\mathbf{0})} \mathbb{I}\{\mathcal{I}_i^{\mathbf{0}} > t\} + \sum_{i=1}^{E^n(\mathbf{0})} \mathbb{I}\{\varepsilon_i^{\mathbf{0}} \leq t < \varepsilon_i^{\mathbf{0}} + \mathcal{I}_i\} + \sum_{i=1}^{A_E^n(t)} \mathbb{I}\{\tau_i + \varepsilon_i \leq t < \tau_i + \varepsilon_i + \mathcal{I}_i\}$$

$$R^n(t) := \sum_{i=1}^{R^n(\mathbf{0})} \mathbb{I}\{\mathcal{W}_i^{\mathbf{0}} > t\} + \sum_{i=1}^{I^n(\mathbf{0})} \mathbb{I}\{\mathcal{I}_i^{\mathbf{0}} \leq t < \mathcal{I}_i^{\mathbf{0}} + \mathcal{W}_i\} + \sum_{i=1}^{E^n(\mathbf{0})} \mathbb{I}\{\varepsilon_i^{\mathbf{0}} + \mathcal{I}_i \leq t < \varepsilon_i^{\mathbf{0}} + \mathcal{I}_i + \mathcal{W}_i\} + \sum_{i=1}^{A_E^n(t)} \mathbb{I}\{\tau_i + \varepsilon_i + \mathcal{I}_i \leq t < \tau_i + \varepsilon_i + \mathcal{I}_i + \mathcal{W}_i\}.$$

Random infectivity functions

Further, it is assumed that $\beta: \mathbb{R}_+ \rightarrow \mathbb{R}_+$ is the **time-dependent varying infectivity contact (exposure) rate**, in the sense that for each individual there is an associated infectivity process $\{\beta_i(\cdot)\}_{i \geq 1}$, i.e. is a random right-continuous function defined by

$$\beta_i(t) := \begin{cases} = 0, & 0 \leq t < \mathcal{E}_i, \\ > 0, & \mathcal{E}_i \leq t < \mathcal{E}_i + \mathcal{I}_i, \\ = 0, & \mathcal{E}_i + \mathcal{I}_i \leq t < \mathcal{E}_i + \mathcal{I}_i + \mathcal{W}_i, \\ = 0, & T_i \leq t < T_i + \mathcal{Y}_i, \end{cases}$$

where the last row describes an assumption that vaccinated individuals cannot transmit the disease to others until they lose their immunity and again become susceptible.

The **total infectivity process** is therefore determined by the collection

$$\{\beta_i, \beta_i^0, \beta_i^{0,I}\}_{i \geq 1}.$$

Some **assumptions** are imposed on the functions β_i :

- ① Collection of the functions $\{\beta_i(\cdot)\}_{i \geq 1}$ are i.i.d.

Since

$$\mathcal{E}_i = \inf \{t > 0, \beta_i(t) > 0\} \text{ and } \mathcal{E}_i + \mathcal{I}_i = \inf \{t > \mathcal{E}_i, \beta_i(t) = 0\},$$

$$\mathcal{E}_i + \mathcal{I}_i + \mathcal{W}_i = \inf \{t > \mathcal{E}_i + \mathcal{I}_i, \beta_i(t) = 0\},$$

this assumption implies that **random vectors** $(\mathcal{E}_i, \mathcal{I}_i)_{i \geq 1}$ are also i.i.d.

- ② Each initially exposed individual is associated with an infectivity process $\beta_i^0(\cdot)$, $i = 1, 2, \dots, E^n(0)$ with a càdlàg path; the $\beta_i^0(\cdot)$'s are assumed to be i.i.d. and such that

$$\mathcal{E}_i^0 = \inf \{t > 0, \beta_i^0(t) > 0\} > 0, \text{ a.s. and } \mathcal{E}_i^0 + \mathcal{I}_i^0 = \inf \{t > 0, \beta_i^0(r) = 0, \text{ for each } r \geq t\},$$

$$\mathcal{E}_i^0 + \mathcal{I}_i^0 + \mathcal{W}_i^0 = \inf \{t > \mathcal{E}_i^0 + \mathcal{I}_i^0, \beta_i^0(t) = 0\}.$$

- ③ Each initially infectious individual is associated with an infectivity process $\beta_i^{0,l}(\cdot)$, $i = 1, 2, \dots, I^n(0)$ with a càdlàg path; the $\beta_i^{0,l}(\cdot)$'s are assumed to be i.i.d. and such that

$$\inf \{t > 0, \beta_i^{0,l}(t) > 0\} = 0 \text{ a.s. and } \mathcal{I}_i^{0,l} = \inf \{t > 0, \beta_i^{0,l}(r) = 0, \text{ for each } r \geq t\}.$$

Force of infection

The *total force of infection* which refers to susceptibles in the population of fixed size n at time t , that include varying infectivity rates associated with different population class, can be written as

$$J_E^n(t) := \sum_{j=1}^{E^n(0)} \beta_j^0(t) + \sum_{j=1}^{I^n(0)} \beta_j^{0,I}(t) + \sum_{j=1}^{A_E^n(t)} \beta_j(t - \tau_j^n), \quad t \geq 0.$$

Thus, the *infectivity rate function* in the population of fixed size n at time t is

$$\Upsilon_E^n(t) := J_E^n(t) \frac{S^n(t)}{n}, \quad t \geq 0.$$

The *infection flow* $A_E^n(t)$ can be expressed by

$$A_E^n(t) = \int_0^t \int_0^\infty \mathbb{I}_{\{u \leq \Upsilon_E^n(s^-)\}} Q_1(ds, du), \quad t \geq 0,$$

where Q_1 is a standard Poisson random measure (PRM) on $\mathbb{R}_+^2$.

Force of vaccination

The *total force of vaccination* (which refers to susceptible group) in the population of fixed size n at time t is

$$\Upsilon_V^n(t) := \alpha(t)S^n(t), \quad t \geq 0,$$

where $\alpha(t)$ is rate of vaccination for which it is assumed that $\alpha(t) < \tilde{k}$, $\tilde{k} \in \mathbb{R}_+$.

The *vaccination flow* $A_V^n(t)$ can be expressed by

$$A_V^n(t) = \int_0^t \int_0^\infty \mathbb{I}_{\{u \leq \Upsilon_V^n(s-)\}} Q_2(ds, du), \quad t \geq 0,$$

where Q_2 is a standard Poisson random measure (PRM) on $\mathbb{R}_+^2$ independent of Q_1 .

One may observe that besides the PRM's Q_1, Q_2 , the randomness in the epidemic dynamics comes also from the infectivity processes $\{\beta_i(t)\}_{i \geq 1}$, $\{\beta_i^0(t)\}_{i \geq 1}$ and $\{\beta_i^{0,l}(t)\}_{i \geq 1}$ (the infectious periods $\{\mathcal{I}_i(t)\}_{i \geq 1}$, $\{\mathcal{I}_i^0(t)\}_{i \geq 1}$ and $\{\mathcal{I}_i^{0,l}(t)\}_{i \geq 1}$ are induced from them). In the case where $\mathcal{E}_i(t) = 0$ and $\mathcal{E}_i^0(t) = 0$ for each $i \geq 1$, then $E^n(t) = 0$ and the model is a generalized SIR model.

Assumptions

Assumption

Let the random functions $\beta_1(t), \beta_2(t), \dots$, (resp. $\beta_1^0(t), \beta_2^0(t), \dots$, and resp. $\beta_1^{0,l}(t), \beta_2^{0,l}(t), \dots$) be i.i.d. copies of $\beta(t)$ (resp. $\beta^0(t)$ and resp. $\beta^{0,l}(t)$). There exists a constant $\beta^* < +\infty$ such that $\sup_{t \in [0, T]} \max \{ \beta(t), \beta^0(t), \beta^{0,l}(t) \} \leq \beta^*$ almost surely, and in addition there exist a given number $k \geq 1$, a random sequence $0 = \xi^0 < \xi^1 < \xi^2 < \dots < \xi^k = \mathcal{E}$ and random functions $\beta^j(t) \in C(\mathbb{R}_+, \mathbb{R}_+)$, $1 \leq j \leq k$, such that

$$\beta(t) = \sum_{j=1}^k \beta^j(t) \mathbf{I}_{[\xi^{j-1}, \xi^j)}(t).$$

Let $\bar{\beta}(t) = E[\beta(t)]$, $\bar{\beta}^0(t) = E[\beta^0(t)]$ and $\bar{\beta}^{0,l}(t) = E[\beta^{0,l}(t)]$, for $t \geq 0$. It follows that $\bar{\beta}(t)$, $\bar{\beta}^0(t)$ and $\bar{\beta}^{0,l}(t)$ are all càdlàg, and they are also uniformly bounded by β^* .

Let φ_T be defined by

$$\varphi_T(r) := \sup_{1 \leq j \leq k} \sup_{1 \leq s, t \leq T, |t-s| \leq r} |\beta^j(t) - \beta^j(s)|.$$

It is clear that for each $T > 0$, φ_T is continuous and $\varphi_T(0) = 0$.

Let

- $\bar{X}^n := \frac{X^n}{n}$ for any process X^n ,
- $D = D(\mathbb{R}_+; \mathbb{R})$ denote the space of $\mathbb{R}$ -valued càdlàg functions defined on $\mathbb{R}_+$,
- convergence in D means convergence in the Skorokhod J_1 topology,
- D^k stands for the k -fold product equipped with the product topology.

Assumption

Assume that there exist nonnegative deterministic constants $V(0), E(0), I(0), R(0)$ such that $0 < V(0) + E(0) + I(0) + R(0) < 1$ and $(\bar{V}^n(0), \bar{E}^n(0), \bar{I}^n(0), \bar{R}^n(0)) \rightarrow (V(0), E(0), I(0), R(0)) \in \mathbb{R}_+^4$ in probability as $n \rightarrow \infty$.

Assumption

Assume that the triple $(\beta_i(\cdot), i \geq 1; \beta_j^0(\cdot), j \geq 1; \beta_k^{0,I}(\cdot), k \geq 1)$, $(E^n(0); I^n(0))$ and (Q_1, Q_2) are *independent*.

Theorem

Under Assumption 1, Assumption 2 and Assumption 3,

$$(\bar{S}^n(t), \bar{J}_E^n(t), \bar{V}^n(t), \bar{E}^n(t), \bar{I}^n(t), \bar{R}^n(t)) \rightarrow (S(t), J_E(t), V(t), E(t), I(t), R(t)), \text{ as } n \rightarrow \infty,$$

in probability in D^6 , locally uniformly in t , where the limits are the unique solution of the following system of Volterra integral equations

$$\begin{aligned} S(t) &= S(0) + V(0)\Upsilon_0(t) + E(0)\Xi_0(t) + I(0)\Xi_1(t) + R(0)G_2(t) - \int_0^t (S(s)J_E(s)(1 - \Xi(t-s)) + \alpha(s)S(s)\Upsilon(t-s)) ds \\ J_E(t) &= E(0)\bar{\beta}^0(t) + I(0)\bar{\beta}^{0,I}(t) + \int_0^t \bar{\beta}(t-s)S(s)J_E(s) ds \\ V(t) &= V(0)\Upsilon_0^c(t) + \int_0^t (\alpha(s)S(s)\Upsilon^c(t-s)) ds \\ E(t) &= E(0)G_0^c(t) + \int_0^t S(s)J_E(s)G^c(t-s) ds \\ I(t) &= I(0)G_1^c(t) + E(0)\Psi_0(t) + \int_0^t S(s)J_E(s)\Psi(t-s) ds \\ R(t) &= R(0)G_2^c(t) + I(0)\Phi_1(t) + E(0)\Phi_0(t) + \int_0^t (S(s)J_E(s)\Phi(t-s)) ds. \end{aligned}$$

Transmission coefficient

Assume that **transmission coefficient** β is described by the **non-stationary Ornstein-Uhlenbeck process** $\beta = (\beta(t), t \geq 0)$ given by the stochastic differential equation (SDE)

$$d\beta(t) = \theta (\beta_e - \beta(t)) dt + \sigma dB(t), \quad t \geq 0,$$

where

- β_e is the mean of the stationary Gaussian distribution with variance $\sigma/\sqrt{2\theta}$,
- $\theta > 0$ determines the speed of the mean reversion,
- σ is the intensity of volatility
- Brownian motion $B = (B(t), t \geq 0)$ is defined on the complete probability space $(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, \mathbf{P})$.

The explicit solution of the SDE (23)

$$\beta(t) = \beta_e + (\beta(0) - \beta_e) e^{-\theta t} + \sigma \int_0^t e^{-\theta(t-s)} dB(s).$$

The expected value of $\beta(t)$ and its variance are given by

$$E[\beta(t)] = \beta_e + (\beta(0) - \beta_e) e^{-\theta t}, \quad \text{Var}(\beta(t)) = \frac{\sigma^2}{2\theta} (1 - e^{-2\theta t}).$$

Stochastic model

Let $\tilde{\beta}(t) = \max\{\beta(t), 0\}$, $\tilde{\beta}^{\mathbf{0}}(t) = \max\{\beta^{\mathbf{0}}(t), 0\}$ and $\tilde{\beta}^{\mathbf{0},I}(t) = \max\{\beta^{\mathbf{0},I}(t), 0\}$. The resulting stochastic model:

$$\begin{aligned}
 S(t) &= S(0) + V(0)\Upsilon_{\mathbf{0}}(t) + E(0)\Xi_{\mathbf{0}}(t) + I(0)\Xi_{\mathbf{1}}(t) + R(0)G_{\mathbf{2}}(t) \\
 &\quad - \int_0^t (S(s)J_E(s)(1 - \Xi(t-s)) + \alpha(s)S(s)\Upsilon^c(t-s) - (\lambda_S - \mu)S(s)) ds, \\
 J_E(t) &= E(0)\tilde{\beta}^{\mathbf{0}}(t) + I(0)\tilde{\beta}^{\mathbf{0},I}(t) + \int_0^t \tilde{\beta}(t-s)S(s)J_E(s) ds, \\
 V(t) &= V(0)\Upsilon_{\mathbf{0}}^c(t) + \int_0^t (\alpha(s)S(s)\Upsilon^c(t-s) + (\lambda_V - \mu)V(s)) ds, \\
 E(t) &= E(0)G_{\mathbf{0}}^c(t) + \int_0^t (S(s)J_E(s)G^c(t-s) + (\lambda_E - \mu)E(s)) ds, \\
 I(t) &= I(0)G_{\mathbf{1}}^c(t) + E(0)\Psi_{\mathbf{0}}(t) + \int_0^t (S(s)J_E(s)\Psi(t-s) - \delta I(s)) ds, \\
 R(t) &= R(0)G_{\mathbf{2}}^c(t) + I(0)\Phi_{\mathbf{1}}(t) + E(0)\Phi_{\mathbf{0}}(t) + \int_0^t (S(s)J_E(s)\Phi(t-s) + (\lambda_R - \mu)R(s)) ds, \\
 d\beta(t) &= \theta(\beta_e - \beta(t))dt + \sigma dB(t).
 \end{aligned}
 \tag{VSDE}$$

Parameters space, range & units

Throughout the paper all random variables and stochastic processes are defined on the filtered complete probability space

$$(\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, P),$$

where $\{\mathcal{F}_t\}_{t \geq 0}$ is the natural filtration which is right continuous and $\mathcal{F}$ contains all P-null sets.

Parameters space involved in the (VSDE)

Symbol	Description	Range	Units
Λ_S	Recruitment rate for susceptible class	$(0, \infty)$	day^{-1}
Λ_E	Recruitment rate for exposed class	$(0, \infty)$	day^{-1}
Λ_V	Recruitment rate for vaccinated class	$(0, \infty)$	day^{-1}
Λ_R	Recruitment rate for recovered class	$(0, \infty)$	day^{-1}
β_e	Transmission coefficient	$(0, 1.5)$	day^{-1}
θ	Speed of the mean reversion	$(0, \infty)$	day^{-1}
α	Vaccination rate	$(0, 1)$	day^{-1}
δ	Disease induced death rate	$(0.000001, 0.1)$	day^{-1}
μ	Natural death rate	$(0.00001, 0.77)$	day^{-1}

The solution of VSDE system is global and positive

Let $\mathbb{R}_+^6 = \{(x_1, x_2, x_3, x_4, x_5, x_6) \mid x_i > 0, i \in \{1, 2, 3, 4, 5, 6\}\}$.

Theorem

Let Assumption 1, Assumption 2 and Assumption 3 hold. For any $t \geq 0$ and any initial value $(S(0), J_E(0), V(0), E(0), I(0), R(0), \beta(0)) \in \mathbb{R}_+^6 \times \mathbb{R}$, *there is a unique solution* $(S(t), J_E(t), V(t), E(t), I(t), R(t), \beta(t))$ to the system VSDEs and the solution remains in $\mathbb{R}_+^6 \times \mathbb{R}$ with probability one. Moreover,

$$\limsup_{t \rightarrow \infty} N(t) = \begin{cases} \kappa(K), & \lambda_{\max} > \mu, \\ N(0), & \lambda_{\max} = \mu, \\ 0, & \lambda_{\max} < \mu, \end{cases}$$

where $\lambda_{\max} := \max\{\lambda_S, \lambda_V, \lambda_E, \lambda_R\}$.

Extinction of the disease

Theorem

Let Assumption 1, Assumption 2 and Assumption 3 hold and let $(S(t), J_E(t), V(t), E(t), I(t), R(t), \beta(t), t \geq 0)$, be the solution of the system (VSDE) with any initial value $(S(0), J_E(0), V(0), E(0), I(0), R(0), \beta(0)) \in \mathbb{R}_+^6 \times \mathbb{R}$. If

$$R_0^{S^*} := \frac{4\lambda_E + 4\kappa^3(K)K + \theta\beta_e^2 + 2\sigma^2}{4(\mu + \delta)} < 1$$

then *the disease goes to extinction*, that is,

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

Persistence in mean of the disease

System (VSDE) is said to be strong persistent in mean if

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \left(\int_0^t E(s) ds + \int_0^t I(t) ds \right) > 0, \text{ a.s.}$$

Theorem

Let Assumption 1, Assumption 2 and Assumption 3 hold and let $(S(t), J_E(t), V(t), E(t), I(t), R(t), \beta(t)), t \geq 0$ be the solution of the system (VSDE) with any initial value $(S(0), J_E(0), V(0), E(0), I(0), R(0), \beta(0)) \in \mathbb{R}_+^6 \times \mathbb{R}$. If

$$R_0^S := \frac{\sqrt[3]{\frac{\theta \beta_e}{2} \mu - \frac{\sigma^2}{2a_1^2}}}{\frac{a_1}{2} K + \theta} > 1$$

for some $a_1 > 0$, then the **system (VSDE) is strong persistent in mean**, that is,

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \left(\int_0^t E(s) ds + \int_0^t I(t) ds \right) \geq \left(\frac{a_1}{2} K + 1 \right) (R_0^S - 1) > 0, \text{ a.s.}$$

Numerical examples - Chosen distribution functions:

$G(t) = 1 - P(t < \mathcal{E}_i),$ $G_0(t) = 1 - P(t < \mathcal{E}_i^0)$	Exponential distribution with parameter $\frac{1}{7}$
$\Psi(t) = P(\mathcal{E}_i \leq t < \mathcal{E}_i + \mathcal{I}_i),$ $\Psi_0(t) = P(\mathcal{E}_i^0 \leq t < \mathcal{E}_i^0 + \mathcal{I}_i)$	Exponential $(\frac{1}{7})$ - Hypoexponential $(\frac{1}{7}, \frac{1}{30})$
$\Phi(t) = P(\mathcal{E}_i + \mathcal{I}_i \leq t < \mathcal{E}_i + \mathcal{I}_i + \mathcal{W}_i),$ $\Phi_0(t) = P(\mathcal{E}_i^0 + \mathcal{I}_i \leq t < \mathcal{E}_i^0 + \mathcal{I}_i + \mathcal{W}_i)$	Hypoexponential $(\frac{1}{7}, \frac{1}{30})$ - Hypoexponential $(\frac{1}{7}, \frac{1}{30}, \frac{1}{180})$
$\Xi(t) = P(\mathcal{E}_i + \mathcal{I}_i + \mathcal{W}_i \leq t),$ $\Xi_0(t) = P(\mathcal{E}_i^0 + \mathcal{I}_i + \mathcal{W}_i \leq t)$	Hypoexponential $(\frac{1}{7}, \frac{1}{30}, \frac{1}{180})$
$\Upsilon(t) = P(\mathcal{Y}_i \leq t),$ $\Upsilon_0(t) = P(t < \mathcal{Y}_i^0)$	Gamma (2, 90)
$G_1(t) = 1 - P(t < \mathcal{I}_i^0),$	Exponential $(\frac{1}{30})$
$\Phi_1(t) = P(\mathcal{I}_i^0 \leq t < \mathcal{I}_i^0 + \mathcal{W}_i)$	Exponential $(\frac{1}{30})$ - Hypoexponential $(\frac{1}{30}, \frac{1}{180})$
$\Xi_1(t) = P(\mathcal{I}_i^0 + \mathcal{W}_i \leq t)$	Hypoexponential $(\frac{1}{30}, \frac{1}{180})$
$G_2(t) = P(\mathcal{W}_i^0 \leq t)$	Exponential $(\frac{1}{180})$

1st example - Extinction of the disease

Parameter and initial values taken in simulations:

Λ_S	Λ_V	Λ_E	Λ_R	β_e	θ	μ	α	δ	σ
0.024	0.024	0.001	0.024	0.0004	0.5	0.024	0.0005	0.02401	0.00003
$S(0)$	$V(0)$	$E(0)$	$I(0)$	$R(0)$	$\beta(0)$				
0.988	0.004	0.003	0.005	0	0.00039				

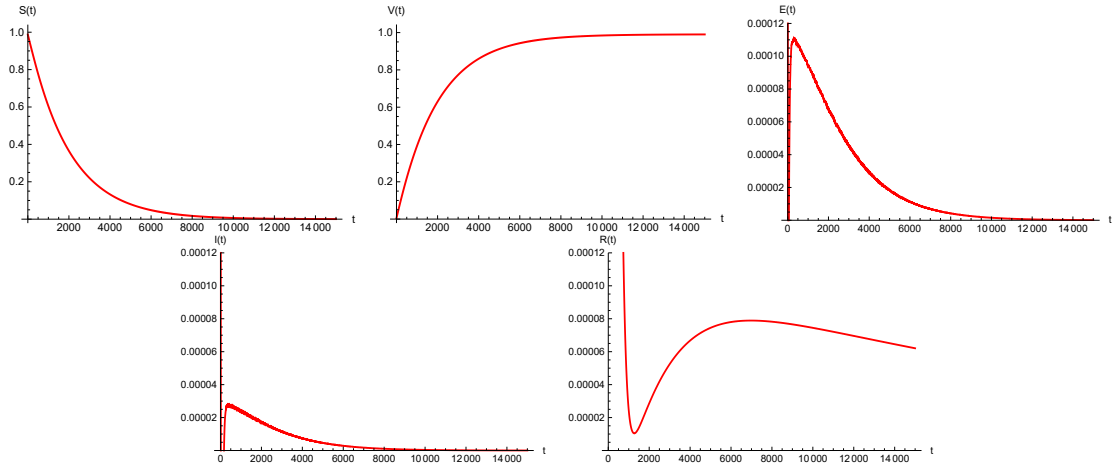
With such choice of model parameters, we obtain

$$R_0^{S*} = \frac{4\lambda_E + 4\kappa^3(K)K + \theta\beta_e^2 + 2\sigma^2}{4(\mu + \delta)} = 0.703 < 1.$$

According to Theorem 3, [the disease goes to extinction](#), that is,

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

1st example - Extinction of the disease



2nd example - Persistence in mean of the disease

Parameter and initial values taken in simulations:

Λ_S	Λ_V	Λ_E	Λ_R	β_e	θ	μ	α	δ	σ
0.00001	0.00001	0.00001	0.00001	0.4	0.005	0.00001	0.0001	0.00001	0.003
$S(0)$	$V(0)$	$E(0)$	$I(0)$	$R(0)$	$\beta(0)$				
0.988	0.004	0.003	0.005	0	0.39				

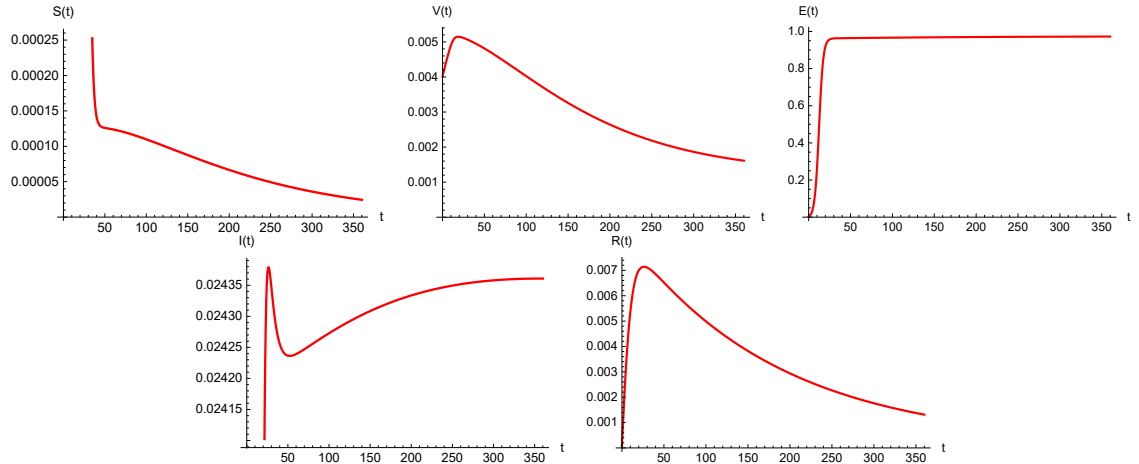
With such choice of model parameters, we obtain

$$R_0^S = \frac{\sqrt[3]{\frac{\theta\beta_e}{2}\mu} - \frac{\sigma^2}{2a_1^2}}{\frac{a_1}{2}K + \theta} = 1.18 > 1,$$

for $a_1 = 1$. According to Theorem 4, the [system \(VSDE\)](#) is [strong persistent in mean](#), that is,

$$\liminf_{t \rightarrow \infty} \frac{1}{t} \left(\int_0^t E(s) ds + \int_0^t I(t) ds \right) \geq \left(\frac{1}{2}K + 1 \right) (R_0^S - 1) = 0.27 > 0, \text{ a.s.}$$

2nd example - Persistence in mean of the disease



Conclusions

- A stochastic epidemic model with a **general contact rate** was developed using Volterra stochastic integral equations.
- The model incorporates
 - general distributions of the latent, infectious, natural immunity and vaccine-induced immunity periods,
 - infection-age dependent infectivity,
 - deterministic or stochastic transmission rate.
- The existence and uniqueness of the stochastic model were established.
- The proposed framework extends classical Markovian epidemic models by incorporating memory effects and allowing realistic non-exponential epidemiological periods.

The proposed model provides a flexible mathematical framework for modelling realistic epidemic dynamics with memory.

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Thank you for your attention!