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Epidemiological Model with Fluctuations

Michael Bestehorn¹ & Thomas Michelitsch²

¹ Institut für Physik, Brandenburgische Technische Universität Cottbus-Senftenberg
Erich-Weinert-Str. 1, 03046 Cottbus, Germany

² Sorbonne Université, Institut Jean le Rond d'Alembert CNRS UMR 7190
4 place Jussieu, 75252 Paris cedex 05, France

bestehorn@b-tu.de

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Abstract

We study a three-compartment system based on the SIR model [1]. From the view of dynamical systems, the inherent property of SIR and SIRS models is the change of stability. A stable fixed point (healthy state) turns into an unstable saddle if the infection rate reaches a critical value and the trajectories end in a newborn (stable) fixed point, the endemic equilibrium.

In our model, noise effects from the environment that allow for a fluctuating infection rate play a crucial role. This results in multiplicative noise terms and our model turns into a set of stochastic nonlinear differential equations (SDEs). A periodic behavior of the infection number is observed that becomes more and more irregular if the noise is increasing. For rather large fluctuations repeated disease outbreaks in the form of sharp pulses with a variety of frequencies and amplitudes are found. The results based on SDEs are confirmed by numerical and approximate analytical solutions of the corresponding Fokker-Planck equation.

1 Introduction

Since the advent of the Covid-19 pandemic in 2020 the interest in the mathematical modulation of the development of infectious diseases has grown enormously. Nevertheless, the simplest model serving nowadays as the basic for numerous extensions and specialisations goes back to the work of Kermak and McKendrick [1] in 1927, nowadays called SIR model. The acronym SIR stands for **S**usceptible, **I**nfectious, **R**ecovered and the model consists of three nonlinearly coupled ODEs describing the time behavior of the three relative concentrations $s = S/N$, $j = I/N$, $r = r/N$ where $N = S + I + R$ is the constant total number of individuals:

$$\frac{ds}{dt} = -\beta_0 s j + \nu r \quad (1a)$$

$$\frac{dj}{dt} = \beta_0 s j - \gamma j \quad (1b)$$

$$\frac{dr}{dt} = \gamma j - \nu r . \quad (1c)$$

Here, β_0 denotes the infection rate and $1/\gamma$ is the average time of being infectious or the time of recovery. the time constant $1/\nu$ describes the loss of immunity where ν is zero in the original SIR model.

Numerical solutions [2] of (1) show the instability of the healthy state $s_h = 1$, $j_h = r_h = 0$, if $\beta_0/\gamma > 1$ and the asymptotic solution

$$s_e = \frac{\gamma}{\beta_0}, \quad j_e = \frac{\nu(\beta_0 - \gamma)}{\beta_0(\nu + \gamma)} \quad r_e = \frac{\gamma(\beta_0 - \gamma)}{\beta_0(\nu + \gamma)} \quad (2)$$

is reached for $t \rightarrow \infty$. However, looking on real time data of epidemics such as COVID, the infection comes in pronounced waves and the long time behavior should rather periodic than approaching a fixed point (fig. 1).

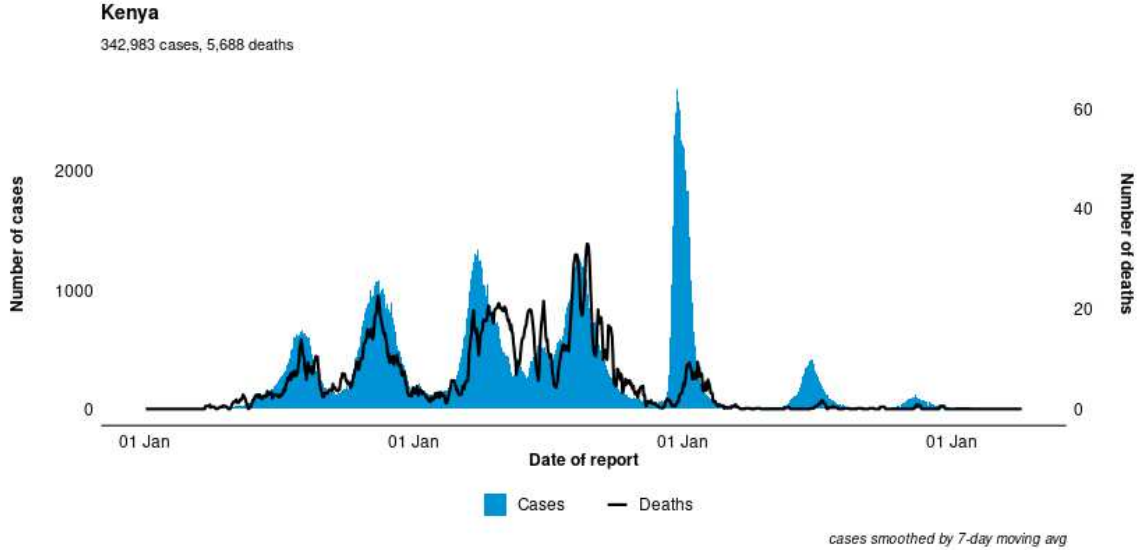


Fig. 1. Time series of the absolute number of cases in Kenya during the years 2020-23 [3].

The question we try to explore in the following is how we must extend the SIRS model to account for the more or less periodic outbreaks of infection waves. Earlier work [4,5,6] considered time delay effects and showed rather regular oscillations if certain critical delay times were exceeded. Here we shall lay the emphasis on fluctuations and show that (strong) fluctuations may cause oscillating behavior and also outbreaks in form of spikes. Even a total suppression of the epidemics and extinction can be observed.

2 The system

In the SIRS model (1) there are three parameters, one of them can be scaled in the time. If we choose $t = \tau \tilde{t}$ with $\tau = 1/\gamma$, the two parameters $R_0 = \beta_0/\gamma$ and $\mu = \nu/\gamma$ are left. R_0 is the basic reproduction number. Since we are interested in fluctuations, we allow for a noisy reproduction number $R_0 + \sigma \xi(t)$ with the random variable $\xi(t)$ and $\langle \xi \rangle = 0$, $\langle \xi^2 \rangle = 1$. Finally, r can be eliminated with $r = 1 - s - j$ and the system considered further is constituted by two coupled nonlinear stochastic differential equations [7]

$$\frac{ds}{dt} = -R_0 s j + \mu(1 - s - j) - \sigma \xi s j \quad (3a)$$

$$\frac{dj}{dt} = R_0 s j - j + \sigma \xi s j . \quad (3b)$$

The noise is multiplicative. For $\sigma = 0$, the endemic state (2) is a stable node for $1 < R_0 < 1 + \mu/4 + O(\mu^2)$. For $R_0 > 1 + \mu/4 + O(\mu^2)$ it turns into a stable focus and small perturbations from (2) decay in time in form of damped oscillations with the frequency

$$\omega = \left[R_0 j_e - \frac{1}{4}(R_0 j_e - \mu)^2 \right]^{1/2} = \sqrt{\mu(R_0 - 1)} + O(\mu) , \quad (4)$$

see fig. 2.

3 Numerical results

Fig. 3 shows a numerical solution of (3) in form of a time series of the infection number $j(t)$. Here, the parameters are $R_0 = 1.3$, $\mu = 0.02$, $\sigma = 0.1$. It can be seen that periodic waves occur. Fig. 4, left frame,

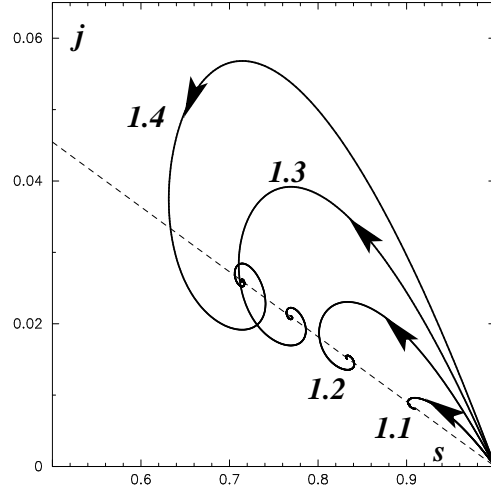


Fig. 2. SIRS model (3) with $\sigma = 0$, trajectories for $\mu = 0.1$ and $R_0 = 1.1, 1.2, 1.3, 1.4$. The unstable fixed point (saddle) ($s = 1, j = 0$, healthy state) is connected with the stable ones (foci) laying on the dashed line ($j_e = \mu(1 - s_e)/(\mu + 1)$, endemic states) by heteroclinic orbits.

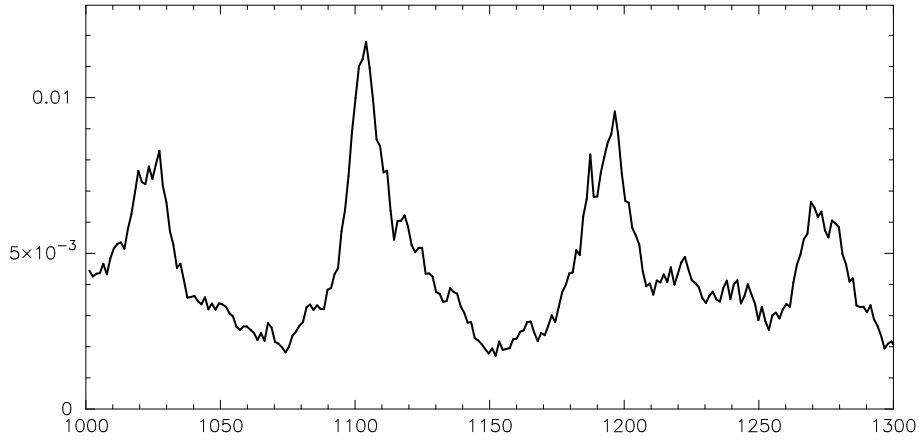


Fig. 3. Time series of j computed from the stochastic SIRS model (3) with $\sigma = 0.1, \mu = 0.02$ and $R_0 = 1.3$. Infection waves occur with a certain mean frequency which is approximately given from (4).

shows the power spectra

$$A(\omega) = |\tilde{j}(\omega)|^2 \quad (5)$$

for several values of R_0 , where $\tilde{j}(\omega)$ denotes the Fourier transform of $j(t)$.

The average frequency of the waves can be computed from

$$\bar{\omega} = \frac{\int d\omega \omega A(\omega)}{\int d\omega A(\omega)} \quad (6)$$

and increases monotonically with R_0 . It shows a rather good agreement with the values computed in (4) for the deterministic case. We may conclude that the, for the deterministic case stable, endemic equilibrium (2) is excited and responds to the fluctuations in its resonance frequency. The agreement of the mean frequency and the resonance (4) is shown in fig. 4, right frame.

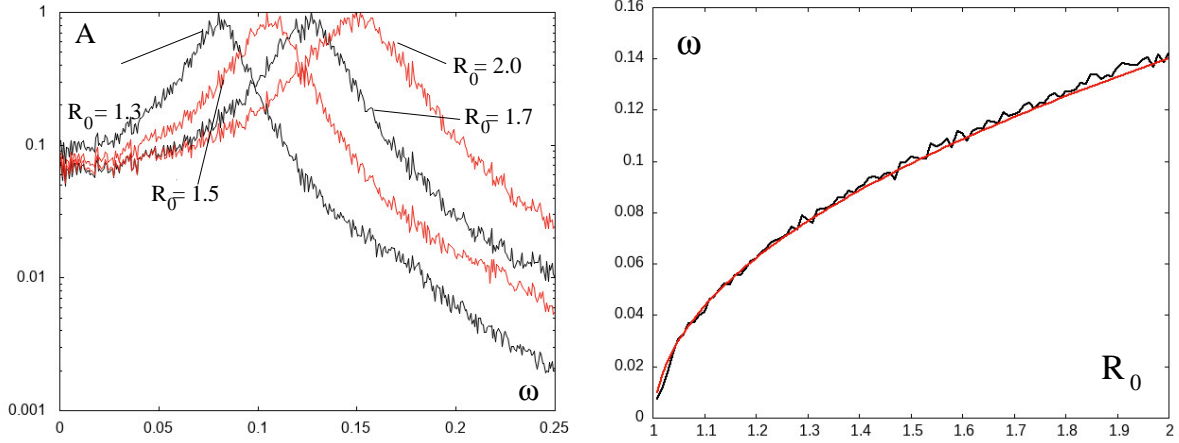


Fig. 4. Left: power spectra of j for different R_0 , each averaged over 100 runs. The maxima coincide with the resonance frequency (4).

Right: mean frequency computed numerically from (6) (black) and resonance frequency from (4) coincide well. Parameters: $\mu = 0.02$, $\sigma = 0.1$.

4 Fokker-Planck equation

It is straightforward to write down the Fokker-Planck equation [8] to the stochastic system (3):

$$\frac{\partial P}{\partial t} = -\frac{\partial}{\partial s} \left[(-R_0 s j + \mu(1-s-j)) P \right] - \frac{\partial}{\partial j} \left[(R_0 s j - j) P \right] + \frac{\sigma^2}{2} \left(\frac{\partial^2}{\partial s^2} - 2 \frac{\partial^2}{\partial s \partial j} + \frac{\partial^2}{\partial j^2} \right) \left[s^2 j^2 P \right]. \quad (7)$$

Here, $P(s, j, t)$ denotes the probability density function (PDF) to find the state s, j at time t .

Before discussing some analytical aspects we solve Eq. (7) numerically, applying a finite difference method on a 100×100 or 200×200 grid with a fixed time step $\Delta t = 0.001$, see [2]. Gaussian distributions for both s and j around the endemic equilibrium (2) serve as initial condition:

$$P(t=0) = N \exp \left(-\frac{(s-s_e)^2}{b_1^2} - \frac{(j-j_e)^2}{b_2^2} \right)$$

with width $b_1 = 5\Delta s$, $b_2 = 5\Delta j$ and $\Delta s, \Delta j$ as the distance between two neighbored grid points. Eq. (7) is a partial differential equation of 2nd order and needs boundary conditions for a unique solution. For simplicity, we assume absorbing boundary conditions $P = 0$ along all boundaries. The solutions for $R_0 = 1.3$, $\mu = 0.02$ and three different σ are shown as time series in fig. 5. After $t \approx 500$ the distribution is stationary. It can be seen that the distributions collapse along the s -axis for increasing σ while their maxima are shifted towards larger s . This reflects the fact that the time series of j resembles more and more a rather flat line close to zero interrupted by a sequence of high and short outbreaks, fig. 6 left frame.

To estimate this shift for the probability distribution we may expand the stationary solution of (7) for small j according to

$$P(s, j) = j^n P_1(s) + j^{n+1} P_2(s) + \dots \quad (8)$$

and find in lowest order from (7)

$$\left(\mu + (n+1)(1-R_0 s) + \frac{1}{2} (n+1)(n+2)\sigma^2 s^2 \right) P_1 = \mu(1-s) \frac{dP_1}{ds}. \quad (9)$$

At the maximum of P_1 the bracket on the l.h.s. must vanish. To estimate the value of the power n in (8), we assume that the maximum is located at $s = 1$. In fact it is smaller (see fig.5) but $s = 1$ may serve as

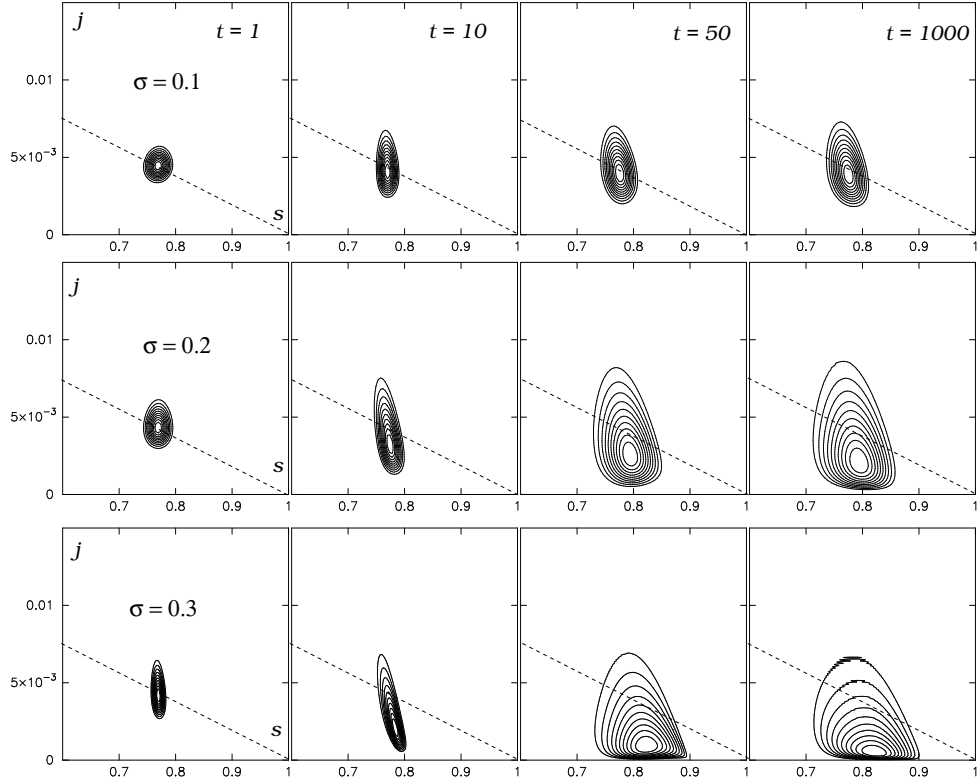


Fig. 5. Numerical solutions of the Fokker-Planck equation (7) for $R_0 = 1.3$, $\mu = 0.02$ and three different σ . The last stages ($t = 1000$) are stationary. The dashed lines mark the location of the endemic equilibrium without fluctuations, cmp. fig. 2.

an upper limit. Then we find from (9)

$$\mu + (n + 1)(1 - R_0) + \frac{1}{2} \sigma^2 (n + 1)(n + 2) = 0$$

and solving for n yields

$$n = \frac{2(R_0 - 1)}{\sigma^2} - 2 + O(\mu) .$$

It is interesting to see that n may become negative if

$$\sigma > \sigma_c = \sqrt{R_0 - 1} . \quad (10)$$

But for $n < 0$, the PDF (8) is singular at $j = 0$ leading to the observed accumulation at $j = 0$. Thus, σ_c can be considered as a critical point from where the PDF collapses to the s -axis. Note that for negative n , the PDF is still normable as long as $n > -1$.

Taking $R_0 = 1.3$ the critical point is at $\sigma_c \approx 0.55$. For collapsing P the numerical code applied for (7) diverges. Alternatively one can compute a histogram solving (3) numerically. Fig. 7 shows the result of 10^{10} iterations with a time step of 0.01. Clearly, P is only significantly different on a narrow stripe around $j = 0$.

5 Conclusion

We showed that fluctuations added to the simple SIRS model may show irregular oscillations with the characteristic frequency of the endemic equilibrium. For large fluctuations, the solutions turn into spikes

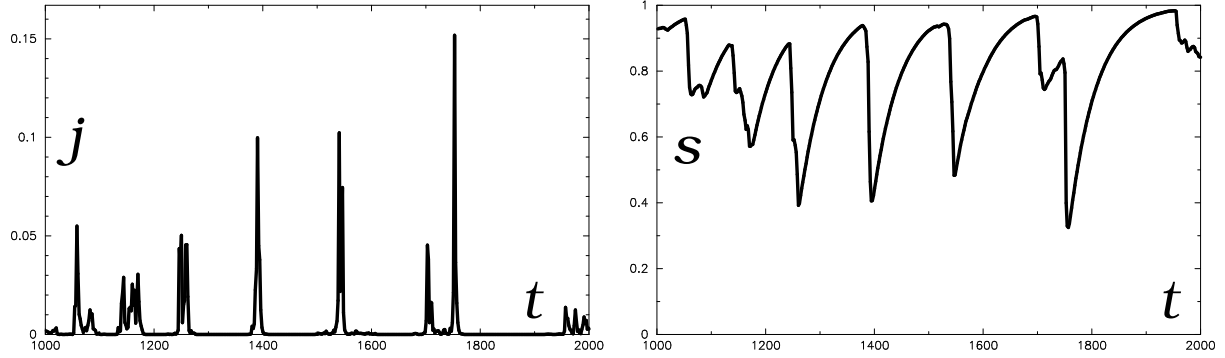


Fig. 6. Time series of j and s computed from the stochastic SIRS model (3) with $\sigma = 1$, $\mu = 0.02$ and $R_0 = 1.3$. Infection waves occur now in form of short and high outbreaks. Note that s is still varying on a broad region, j is more or less zero except for very short times.

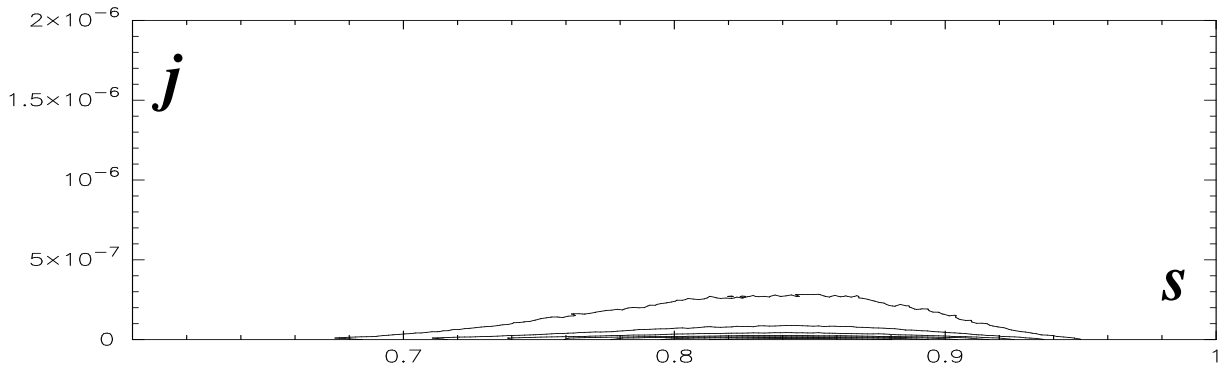


Fig. 7. PDF for large $\sigma = 0.6$, $R_0 = 1.3$ above threshold (10). The PDF is zero almost everywhere except on a narrow stripe around $j = 0$.

with almost zero infection rate interrupted by short outbreaks. This is confirmed by solutions of the corresponding Fokker-Planck equation that shows a collapsing PDF if a certain critical fluctuation is exceeded.

If j becomes smaller than $1/N$ ($N =$ total size of population) the number of infected individuals $j \times N$ would be smaller than one. Therefore, a period of very small values of j can lead to extinction of the pandemic.

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