Epidemic models with spread of awareness

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Joan Saldaña

Universitat de Girona

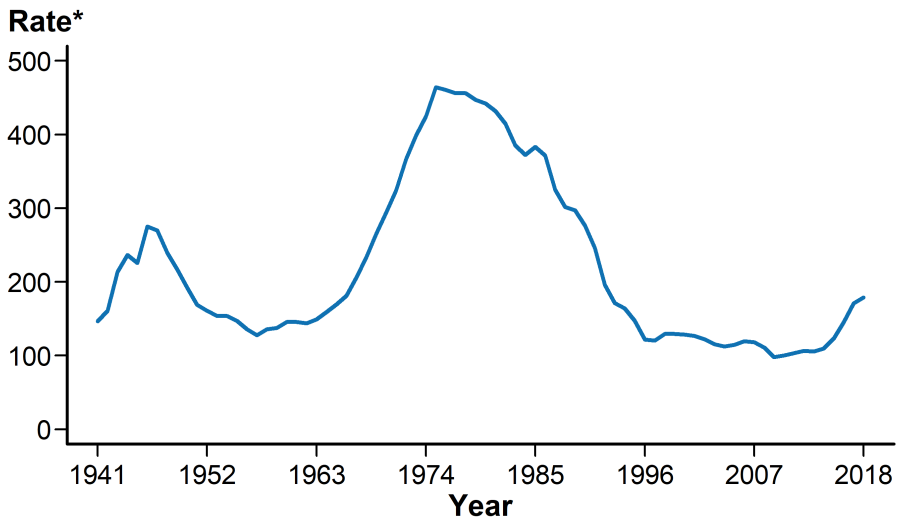
Outline of the talk

1. Introduction
2. SIS models
3. The SAIS model
4. The SAUIS model
5. The SAUIS-ε model

1. Introduction

Sexual transmitted diseases (STDs) are a nice example of the interplay between epidemic spread and human behaviour

Figure 14. Gonorrhea — Rates of Reported Cases by Year, United States, 1941–2018



* Per 100,000.
NOTE: See section A1.3 in the Appendix for more information on gonorrhea case reporting.

high increase in the number of cases up to date (2023)

Their dynamics are modelled by means of an Susceptible-Infected-Susceptible (SIS) epidemic model because there is no immunity after recovery (medical treatment).

Their time evolution has been linked to that of HIV.

Social perception of the risk linked to STDs changed a lot after the appearance of the HIV antiretroviral therapy in the mid 80s

2. SIS models

Basic model:

incidence rate

$$\frac{dS}{dt} = -\beta S\frac{I}{N} + \delta I, \qquad \frac{dI}{dt} = \boxed{\beta S\frac{I}{N}} - \delta I,$$

with $S + I = N$. β: transmission rate, δ: recovery rate.

With birth and death processes (demographics):

$$\frac{dS}{dt} = B - \beta S\frac{I}{N} + \delta I - \mu S, \quad \frac{dI}{dt} = \beta S\frac{I}{N} - \delta I - \mu I,$$

B: recruitment rate of the population.

μ: natural mortality such that $N = \text{const.}$ $\left(\frac{dN}{dt} = B - \mu N = 0\right)$

Neglecting the equation for S (because $S + I = N$) and without demographics, the model reduces to

$$\frac{dI}{dt} = \beta S \frac{I}{N} - \delta I = \left(\beta \frac{S}{N} - \delta \right) I.$$

At the beginning of the epidemic, $S \approx N$:

$$\boxed{\frac{dI}{dt} \approx (R_0 - 1)\, \delta I} \quad \longrightarrow \quad \frac{dI}{dt} > 0 \quad \text{iff} \quad R_0 > 1$$

$R_0 = \dfrac{\beta}{\delta}$: *basic reproduction number*

$=$ mean # of cases directly caused by an infected individual throughout its infectious period in a fully susceptible popul

$\dfrac{1}{\delta}$: mean duration of the infectious period.

Dividing by N, the equation for the *fraction* of infected individuals is

$$\frac{di}{dt} = \left(\frac{\beta}{\delta}\, s - 1\right) \delta\, i = (R_0 s - 1)\, \delta\, i, \quad s + i = 1.$$

$R_e = R_0 s$: effective reproduction number.

Linearising around the DFE[1] $(s^*, i^*) = (1, 0)$, it follows

$$\left.\frac{di}{dt}\right|_{s=1} = (R_0 - 1)\, \delta\, i \quad \longrightarrow \quad i(t) \approx i_0\, e^{(R_0 - 1)\, \delta\, t}.$$

$\hookrightarrow \lambda = (R_0 - 1)\, \delta$: the initial epidemic growth rate.

Endemic equilibrium: $(s^*, i^*) = \left(\dfrac{1}{R_0}, 1 - \dfrac{1}{R_0}\right) \quad \longrightarrow \quad R_0 > 1.$

The instability of the DFE guarantees the existence of an endemic equil.

[1] DFE: Disease-Free Equilibrium

More sophisticated SIS/SIR models

The transmission rate β can be thought as $\beta = c\beta_0$ where c is the contact rate and β_0 is the per-contact probability of transmission.

The number of contacts can change over time (social distancing) as well as the transmission probability β_0 (prophylactic measures).

Different assumptions on the transmission rate β:

β depends on the population size N: $\beta = \beta(N)$.

$\hookrightarrow$ Saturation of the number of contacts per unit of time.

β depends on the prevalence of the disease I: $\beta = \beta(I)$.

$\hookrightarrow$ Introduction of behavioural responses.

Two examples of incidence rates

Capasso & Serio (1978): SIR model

$$\frac{dS}{dt} = -g(I)S, \quad \frac{dI}{dt} = g(I)S - \delta I, \quad \frac{dR}{dt} = \delta I,$$

$$(S + I + R = N)$$

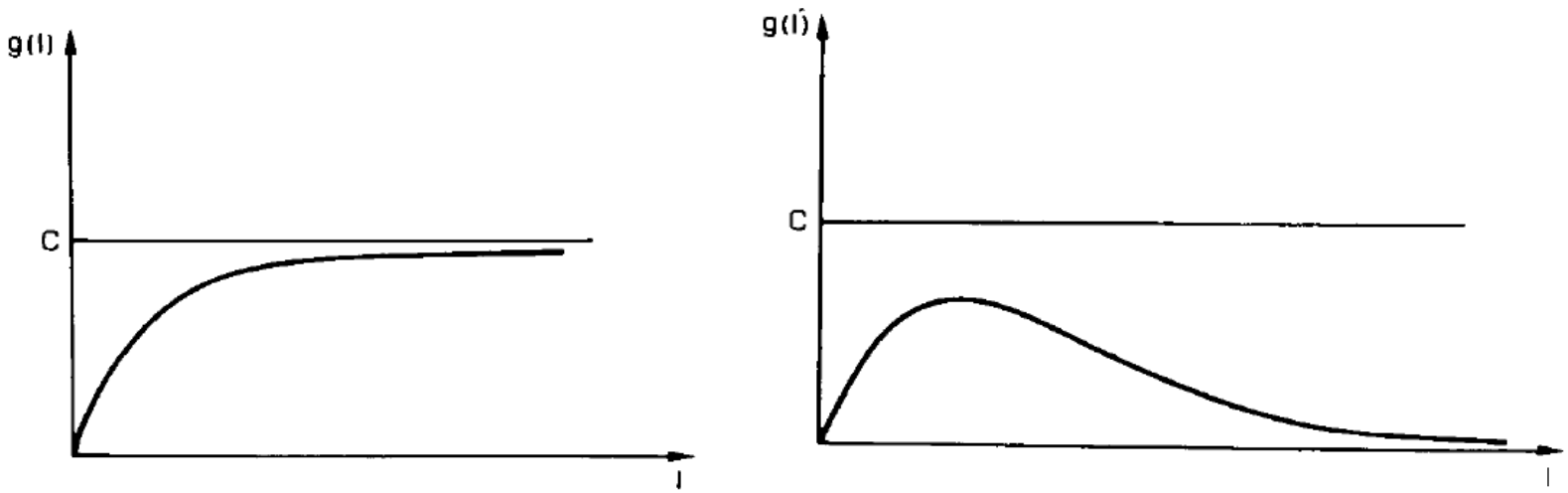


FIG. 1 (a) An asymptotically saturating g is illustrated. (b) A function g which takes into account "psychological" effects is illustrated.

Ruan & Wang (2003): SIR model with demographics

$$\frac{dS}{dt} = B - \frac{kI^2}{1+\alpha I^2} S + vR - \mu S, \qquad \frac{dI}{dt} = \frac{kI^2}{1+\alpha I^2} S - (\mu + \delta) I,$$

$$\frac{dR}{dt} = \delta I - (\mu + v) R, \qquad \frac{dN}{dt} = B - \mu N, \quad N = S + I + R,$$

v: rate of immunity loss; $\alpha \geqslant 0$.

Rescaling the equations for I and R, it follows

$$\frac{dX}{d\theta} = \frac{X^2}{1+pX^2}(A - X - Y) - mX, \quad \frac{dY}{d\theta} = qX - Y,$$

Theorem 2.9. *There are at least two limit cycles in* (1.3) *for some parameters.*

SIS at the level of nodes

Van Mieghem et al.(2009): *The N-intertwined SIS model*

The equation for the *probability* $p_i(t)$ for the node i of being *infected* at time t under the usual assumption of statistical independence among the state of its neighbours ($p(S_i \cap I_j) = p_{S_i} \cdot p_{I_j} = (1 - p_i)\, p_j$) is given by

$$\frac{dp_i}{dt} = \beta_0 (1 - p_i) \sum_{j=1}^{N} a_{ij} p_j - \delta p_i$$

where $A = (a_{ij})$ is the $N \times N$ adjacency matrix of the contact network, and β_0 is the per-contact transmission rate.

$\hookrightarrow$ $I(t) = \sum_{i=1}^{N} p_i(t)$ is the mean number of infected nodes at time t.

If all the nodes have the same number k of neighbours (regular random network) and $p_i(0) = p_0 > 0 \ \forall \, i$ (the same probability of being infected at time 0), then the solution of the previous system is $p_i(t) = p(t) \ \forall \, i$ with $p(t)$ being the solution of

$$\frac{dp}{dt} = k\beta_0(1-p)p - \delta p$$

endowed with the initial condition $p(0) = p_0$.

The mean number of infected nodes at time t is then given by $I(t) = \sum_{i=1}^{N} p_i(t) = Np(t) \rightarrow i(t) = I(t)/N$: *fraction* of infected nodes

So, the faction $i(t)$ is the solution of

$$\frac{di}{dt} = \beta(1-i)i - \delta i$$

with $\beta = k\beta_0$ is the per-node transmission rate.

Multiplying by N (total number of nodes) we recover the standard SIS model:

$$\frac{dI}{dt} = \beta S\frac{I}{N} - \delta I$$

with $S = N(1-i)$.

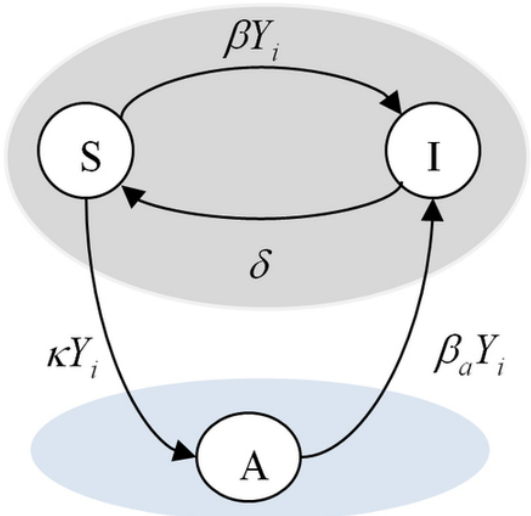
Remark: If we think of k as a contact rate, then β_0 is the transmission probability during an S-I contact.

3. The SAIS model

In the formulations of the SIS model with $\beta = \beta(I)$, all the susceptible individuals become aware of the risk because they have the same β, even if β is not constant. However this is never the case.

$\hookrightarrow$ Alerted individuals with different level of awareness.

In the original N-intertwined SAIS model, the transitions between states are



Epidemic Model

Y_i: number of infected neighbours of node i

Behavioral Response Model

Sahneh et al. (2012)

Equations for the probability $p_i(t)$ for the node i of being infected at time t and the probability $q_i(t)$ of being aware at time t:

$$\frac{dp_i}{dt} = \beta_0(1 - p_i - q_i) \sum_{j=1}^{N} a_{ij} p_j + \beta_0^a q_i \sum_{j=1}^{N} a_{ij} p_j - \delta p_i$$

$$\frac{dq_i}{dt} = \kappa_0(1 - p_i - q_i) \sum_{j=1}^{N} a_{ij} p_j - \beta_0^a q_i \sum_{j=1}^{N} a_{ij} p_j - \delta_a q_i$$

where β_0^a is the transmission rate per $A - I$ contact: $\beta_0^a < \beta_0$,
κ_0 is the alerting rate per $S - I$ contact, and δ_a is the awareness decay rate, and $1 - p_i - q_i$ is the probability that node i is susceptible.

$\hookrightarrow$ Aware indiv are created from a direct interaction with infected indiv.

Remark: In Sahneh et al. (2012), there is no awareness decay ($\delta_a = 0$).

Juher, Kiss & J.S. (2015): The model on regular random ntws, $\delta_a \geqslant 0$.

Lemma 3.1. *Consider the initial value problem (IVP) given by system (2) defined on regular random networks and endowed with the initial conditions* $p_i(0) = p_0 \geq 0$ *and* $q_i(0) = q_0 \geq 0$ *for* $i = 1, 2, \ldots, N$, *and such that* $p_0 + q_0 \leq 1$. *The solution of this IVP is given by* $(p_i(t), q_i(t)) = (p(t), q(t))$ $\forall i$ *with* $(p(t), q(t))$ *being the solution of the system:*

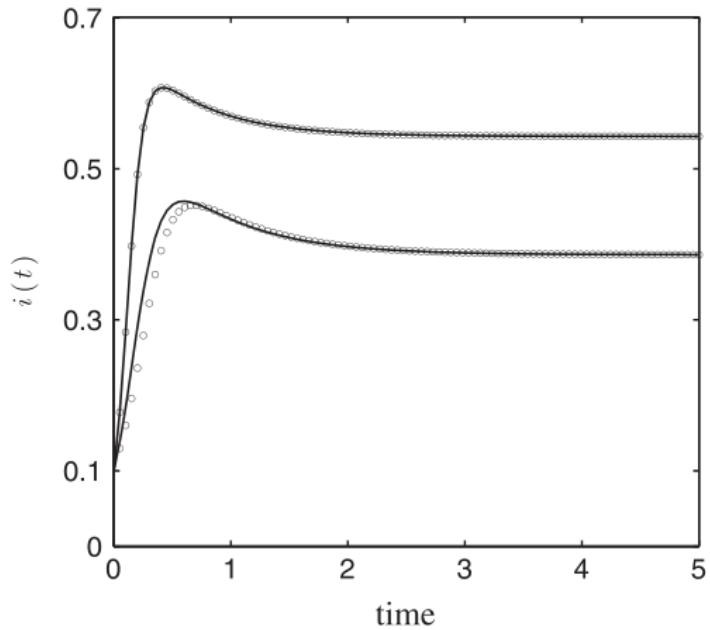
$$\begin{cases} \dfrac{dp}{dt} = k\beta_0(1-p-q)p + k\beta_a^0 pq - \delta p, \\ \dfrac{dq}{dt} = k\kappa_0(1-p-q)p - k\beta_a^0 pq - \delta_a q, \end{cases} \tag{3}$$

endowed with the initial condition $(p(0), q(0)) = (p_0, q_0)$.

$p(t)$: fraction of infected nodes at time t, $i(t)$

$q(t)$: fraction of aware/alerted nodes at time t, $a(t)$

How good is the MF approximation of the node-based SAIS model?



$$\beta = k\beta_0, \qquad \beta_a = k\beta_a^0, \qquad \kappa = k\kappa_0$$

Fig. 1. Evolution of the fraction of infectious nodes $i(t)$ for a smaller epidemic ($\delta = 4$, $\delta_a = 0.5$, $\beta = 12$, $\beta_a = 2$ and $\kappa = 4$) and a larger epidemic ($\delta = 4$, $\delta_a = 0.5$, $\beta = 18$, $\beta_a = 2$ and $\kappa = 4$) on a regular random network with $N = 1000$ nodes of degree $k = 5$. Open circles (○) correspond to the solutions of the node-based model (2), with $\beta_0 = \beta/k$, $\beta_a^0 = \beta_a/k$, $\kappa_0 = \kappa/k$. Continuous lines are the solutions of the mean-field model (4). For the larger epidemic the initial condition is uniform with each node having a probability 0.9, 0.1 and 0 of being susceptible, infectious or aware at time $t = 0$, respectively. For the smaller epidemic, the neighbours of 20 randomly chosen nodes were infected with probability 1.0, resulting in a 10% infectivity at time $t = 0$. As proven in Lemma 3.1, the output from the two models coincide for uniformly random initial conditions. For initially clustered infections, the mean-field model overestimates the initial epidemic growth predicted by the node-based one, although both solutions tend to the same steady state $i^* = 0.38618$.

With a uniform initial condition, the agreement between the solutions to both systems is pretty good on regular random networks (upper curve).

It is not so good if we depart from such an initial condition (clusters of initially infected nodes (lower curve).

The (reactive) SAIS model

Just, J.S. & Xin (2018): Reactive SAIS and SAUIS models

Alerted people can convince others to take preventive measures: $\alpha_a(i)$ is the rate at which a susceptible indiv becomes aware following contact with aware individuals:

$$\begin{aligned} \frac{da}{dt} &= \alpha_i(i)\, s\, i + \overbrace{\alpha_a(i)\, s\, a} + p(i)\, \delta\, i - \beta_a a\, i - \delta_a(i)\, a, \\ \frac{di}{dt} &= (\beta\, s + \beta_a\, a - \delta)\, i, \qquad s + a + i = 1. \end{aligned} \tag{1}$$

Here we assume that $\alpha_i(i)$, $\alpha_a(i)$, $p(i)$, $\delta_a(i)$ are nonnegative differentiable functions in $[0, 1]$, $p(i) \leq 1$, $\delta_a(0) > 0$, and β, β_a, δ are constants such that $0 \leq \beta_a < \beta$ and $\delta > 0$. Moreover, for $i > 0$ we assume that $\alpha_a(i) > 0$ and $\alpha_i(i) + p(i) > 0$.

Equilibria:

Let us define the following basic reproduction numbers for the disease spread (R_0 and R_0^d) and awareness dissemination (R_0^a):

$$\boxed{R_0 = \frac{\beta}{\delta}, \quad R_0^d = \frac{\beta_a}{\delta}, \quad R_0^a = \frac{\alpha_a(0)}{\delta_a(0)}.}$$

DFE: $P_1 = (0,0), \quad P_2 = \left(1 - \frac{1}{R_0^a}, 0\right),$

Endemic equilibria: $P_3 = (a^*, i^*)$ with $i^* = 1 - \frac{\delta}{\beta} - \left(1 - \frac{\beta_a}{\beta}\right) a^* > 0.$

P_2 contains only alerted indiv. It is biologically meaningful if $R_0^a > 1$.

The eigenvalues of the Jacobian matrix at P_2 are:

$$\lambda_1(P_2) = \delta_a(0) - \alpha_a(0), \quad \lambda_2(P_2) = \beta - \delta - (\beta - \beta_a)\left(1 - \frac{1}{R_0^a}\right).$$

Theorem 1 *Assume $\alpha_i(i)$, $\alpha_a(i)$, $p(i)$, $\delta_i(i)$, β satisfy the conditions that were spelled out below (1). Then the global behavior of the solutions of the system (1) depends as follows on the remaining parameters*:

(i) *If $R_0 \leq 1$ and $R_0^a \leq 1$, then P_1 is the only equilibrium point and is globally asymptotically stable.*

(ii) *If $R_0 \leq 1 < R_0^a$, then P_1 and P_2 are the only equilibrium points. P_2 is globally asymptotically stable on $\Omega \backslash \{P_1\}$. When $R_0 < 1$, then P_1 is a saddle point.*

(iii) *If $R_0^a \leq 1 < R_0$, then no equilibrium $P_2 \neq P_1$ exists in Ω. When $R_0^a < 1$, then P_1 is a saddle point. Each trajectory that starts with $i(0) > 0$ will eventually approach an endemic equilibrium of type P_3.*

(iv) *If $R_0 > 1$ and $R_0^a > 1$, then P_1 is an unstable point and system (1) has also the equilibrium P_2. If $\lambda_2(P_2) < 0$, then P_2 is locally asymptotically stable.*

(v) *If instead $\lambda_2(P_2) > 0$, then system (1) has also at least one equilibrium P_3, with P_2 being a saddle point. Each trajectory that starts with $i(0) > 0$ will eventually approach an endemic equilibrium of type P_3.*

Remark: The condition $\lambda_2(P_2) > 0$ can be written as

$$R_0 + (R_0^a - 1)(R_0^d - 1) > 1.$$

There can be more than one endemic equilibrium P_3 even if $\lambda_2(P_2) < 0$:

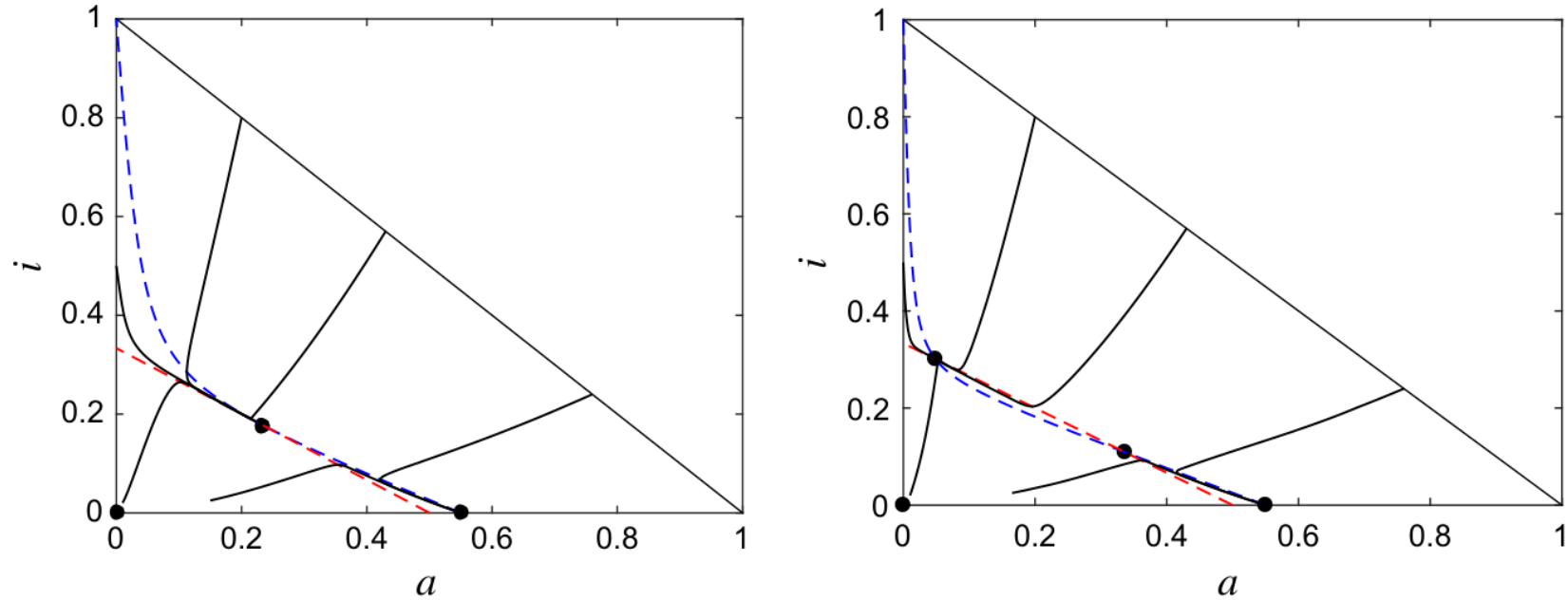


Fig. 2 Phase portrait of the reactive SAIS with constant rates showing the existence of two interior equilibria (*right*) after a saddle-node bifurcation (*left*) using α_i as a tuning parameter. Parameters: $p = 0$, $\beta = 6$, $\delta = 4$, $\beta_a = 2$, $\delta_a = 0.9$, $\alpha_a = 2$, and $\alpha_i = 0.05$ (*right*) and $\alpha_i = 0.1733500838578$ (*left*)

Remark:

$$R_0 + (R_0^a - 1)(R_0^d - 1) = \frac{6}{4} + \left(\frac{2}{0.9} - 1\right)\left(\frac{2}{4} - 1\right) = 0.\hat{8} < 1.$$

But what about oscillatory solutions?

Lemma 3 *Assume $\alpha_i(i)$, $\alpha_a(i)$, $p(i)$, $\delta_i(i)$, β satisfy the conditions that were spelled out below (1). Then the system (1) has no closed orbits inside Ω.*

Proof Let $f_1(a,i)$ and $f_2(a,i)$ denote the functions on the right-hand side of the system. The vector field $(F_1(a,i), F_2(a,i)) = (\frac{1}{a\,i} f_1(a,i), \frac{1}{a\,i} f_2(a,i))$ is C^1 in the interior of Ω, and its divergence is given by

$$\frac{\partial}{\partial a} F_1(a,i) + \frac{\partial}{\partial i} F_2(a,i) = -\frac{\alpha_i(i)}{a}\left(1+\frac{s}{a}\right) - \frac{\alpha_a(i)}{i} - \frac{p(i)\delta}{a^2} - \frac{\beta}{a} < 0$$

for all (a,i) in the interior of Ω. So, the divergence does not change sign and does not take the value 0. Therefore, Dulac's criterion of nonexistence of periodic orbits (Perko 2001) precludes the existence of a closed orbit lying entirely in Ω. □

4. The SAUIS model

Is it possible to have oscillatory solutions from the interplay between disease spread and awareness without demographics or changes in the contact pattern among individuals (Szabó-Solticzky et al. (2016))?

Not all the alerted individuals have the same degree of responsiveness.

The *quality* of the transmitted information has an impact on the creation of aware indiv willing or *unwilling* to pass on the alert to other individuals

$\hookrightarrow$ Idea of degradation of the information introduced by Agliari et al. (2006) and adopted by Funk et al. (2009) for an epidemic context:

"When information is passed from person to person, it loses its quality; in other words, first-hand information about a disease case will lead to much more determined reaction than information that has passed through many people before arriving at a given individual".

The model:

$$\begin{aligned}
\frac{da}{dt} &= \alpha_i s\, i + \alpha_a s\, a + p\,\delta\, i - \beta_a\, a\, i - \delta_a\, a,\\
\frac{du}{dt} &= \boxed{\delta_a\, a} + \boxed{\alpha_u s\, a} + q\,\delta\, i - \beta_u\, u\, i - \delta_u\, u,\\
\frac{di}{dt} &= (\beta\, s + \beta_a a + \beta_u u - \delta)\, i, \quad s + a + u + i = 1.
\end{aligned}$$

with $0 \leqslant \beta_a \leqslant \beta_u < \beta$ and $p + q < 1$.

Equilibria:

DFE: $P_1 = (0, 0, 0)$, $P_2 = (a_0^*, u_0^*, 0)$ with $a_0^*, u_0^* > 0$ iff $R_0^a > 1$,

Endemic equilibria: $P_3 = (a^*, u^*, i^*)$ with

$$i^* = 1 - \frac{\delta}{\beta} - \left(1 - \frac{\beta_a}{\beta}\right) a^* - \left(1 - \frac{\beta_u}{\beta}\right) u^* > 0$$

Transcritical bifurcations at $R_0 = 1$, $R_0^a = 1$, and $\lambda_3(P_2) = 0$ (λ_3 is the only eigenvalue of the Jacobian matrix at P_2 that can be positive).

At $\lambda_3(P_2) = 0$, both forward and backward bifurcations can occur:

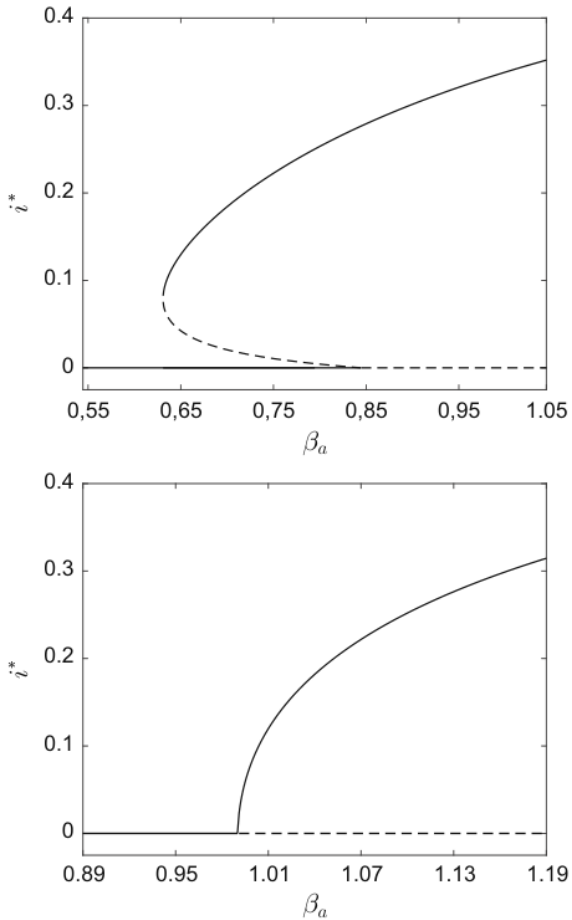


Fig. 3 Transcritical bifurcation diagrams of system (8) for $\beta = 2$, $\beta_u = 1$, $\delta = 1$, $\delta_a = 0.01$, $\delta_u = 0.05$, $\alpha_i = 0.8$, $\alpha_u = 0.1$, $p = q = 0$, $\alpha_a = 0.1$ (*top*) and $\alpha_a = 1$ (*bottom*). The stable (unstable) equilibria are depicted with a *solid* (*dashed*) *line*. Bifurcation values: $\beta_a^c = 0.844$ and $\beta = 0.62$ (for the fold bifurcation) (*upper panel*); $\beta_a^c = 0.988$ (*lower panel*)

Hopf bifurcation (at the endemic equilibrium):

Let P_3 be an endemic equilibrium, $J(P_3)$ be the Jacobian matrix at P_3, and $p(\lambda) = \lambda^3 + c_2\lambda^2 + c_1\lambda + c_0$ its characteristic polynomial ($c_0 = -\det(J)$, c_1 is the sum of the principal minors, $c_2 = -\text{trace}(P_2)$).

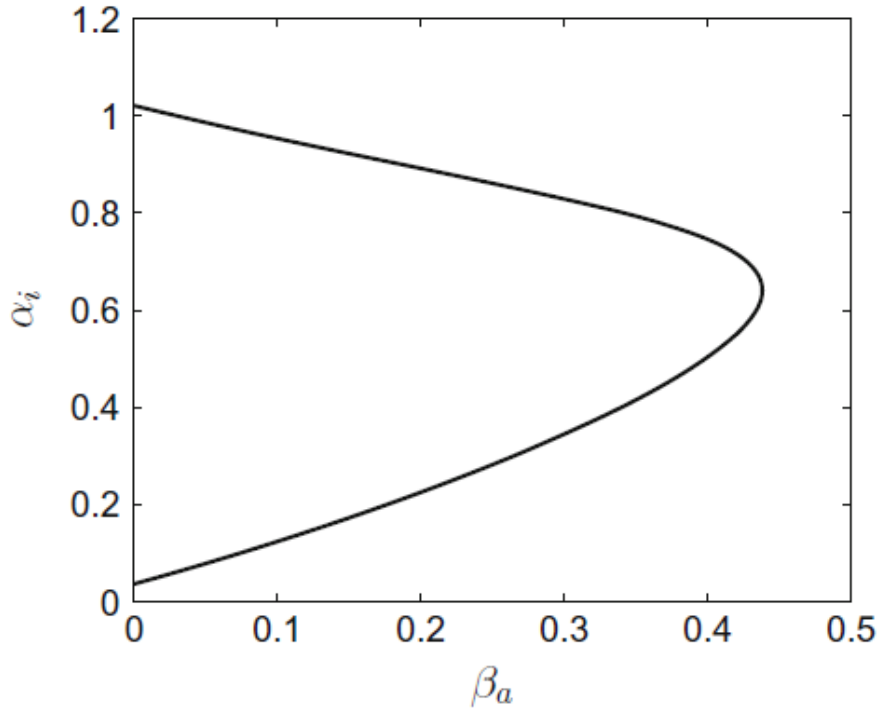
$J(P_3)$ has precisely one pair of pure imaginary eigenvalues iff $c_0 - c_1c_2 = 0$ and $c_1 > 0$ (Guckenheimer et al. (1997)).

The Hopf pairs (β_a, α_i) are found by solving the three equilibrium equations and $c_0 - c_1c_2 = 0$ (with $c_1 > 0$) with α_i as a tuning parameter: $\beta_a = f(a^*(\alpha_i), u^*(\alpha_i), i^*(\alpha_i), \alpha_i)$

$\hookrightarrow$ Algebraic Hopf-bifurcation curve: $H = \{(\beta_a, \alpha_i)\}$

Example:

Fig. 4 Hopf-bifurcation curve H of system (8) for $\delta = 1$, $\delta_a = 0.01$, $\delta_u = 0.05$, $\beta = 3$, $\beta_u = 0.5$, $\alpha_a = 0.01$, $\alpha_u = 1$, and $p = q = 0$. For pairs (β_a, α_i) inside the region bounded by this curve and the α_i-axis system (8) has an unstable endemic equilibrium and a stable periodic orbit



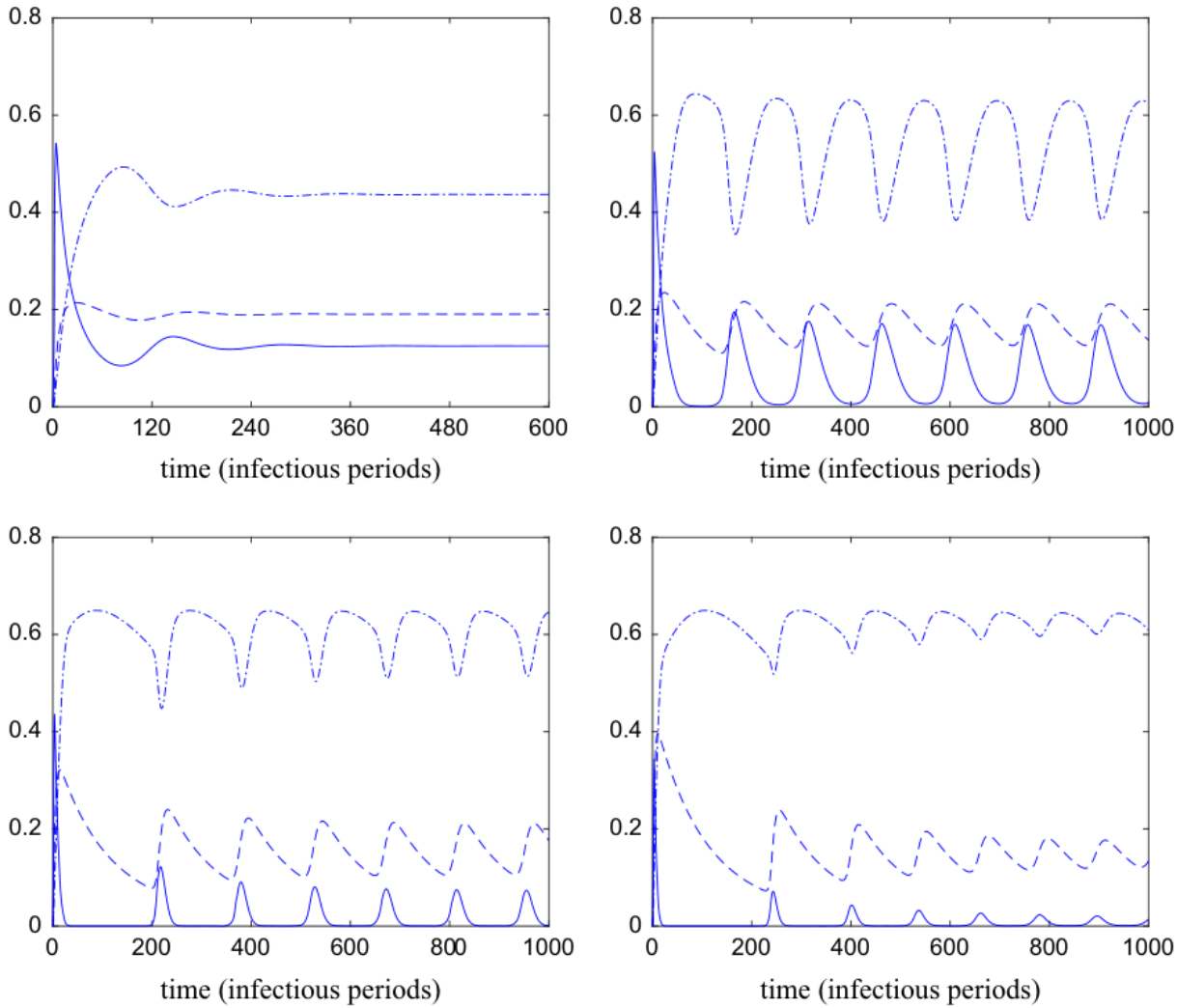


Fig. 6 Evolution of the fraction of infectious (*solid line*), aware (*dashed line*), and unwilling (*dot-dashed line*) hosts according to system (8) for different values of α_i along the vertical section in Fig. 4 corresponding to $\beta_a = 0.2$: $\alpha_i = 0.2$ (*top left*), 0.24 (*top right*), 0.5 (*bottom left*), 0.94 (*bottom right*). Fixed parameters: $\alpha_a = 0.01$, $\alpha_u = 1$, $\delta = 1$, $\delta_a = 0.01$, $\delta_u = 0.05$, $\beta = 3$, $\beta_a = 0.2$, $\beta_u = 0.5$, $p = q = 0$. Initial condition: $a(0) = u(0) = 0$, $i(0) = 0.1$

5. The SAUIS-ε model

How robust are the predicted oscillations when we depart from the underlying assumptions of the mean-field approximation?

Different network architectures (not only regular random networks)

$\hookrightarrow$ Integrate the N-intertwined epidemic model to include adjacency matrices corresponding to different networks.

Gillespie algorithm for performing stochastic simulations of the model.

To avoid the stochastic epidemic extinction when the prevalence is very low, we consider a very low rate ε of imported cases from individuals who have been infected abroad (Juher, Rojas & J.S. (2020)).

The SAUIS-ε model:

$$\begin{aligned}
\frac{da}{dt} &= \alpha_i s\, i + \alpha_a s\, a + p\, \delta\, i - \beta_a\, a\, i - \delta_a\, a\, - \boxed{\varepsilon a}, \\
\frac{du}{dt} &= \delta_a\, a + \alpha_u s\, a + q\, \delta\, i - \beta_u\, u\, i - \delta_u\, u\, - \boxed{\varepsilon u}, \\
\frac{di}{dt} &= (\beta\, s + \beta_a a + \beta_u u - \delta)\, i + \boxed{(1-i)\varepsilon},
\end{aligned}$$

with $s + a + u + i = 1$ and $\varepsilon \geqslant 0$.

Very low values of ε will not alter the underlying deterministic dynamic of the epidemic.

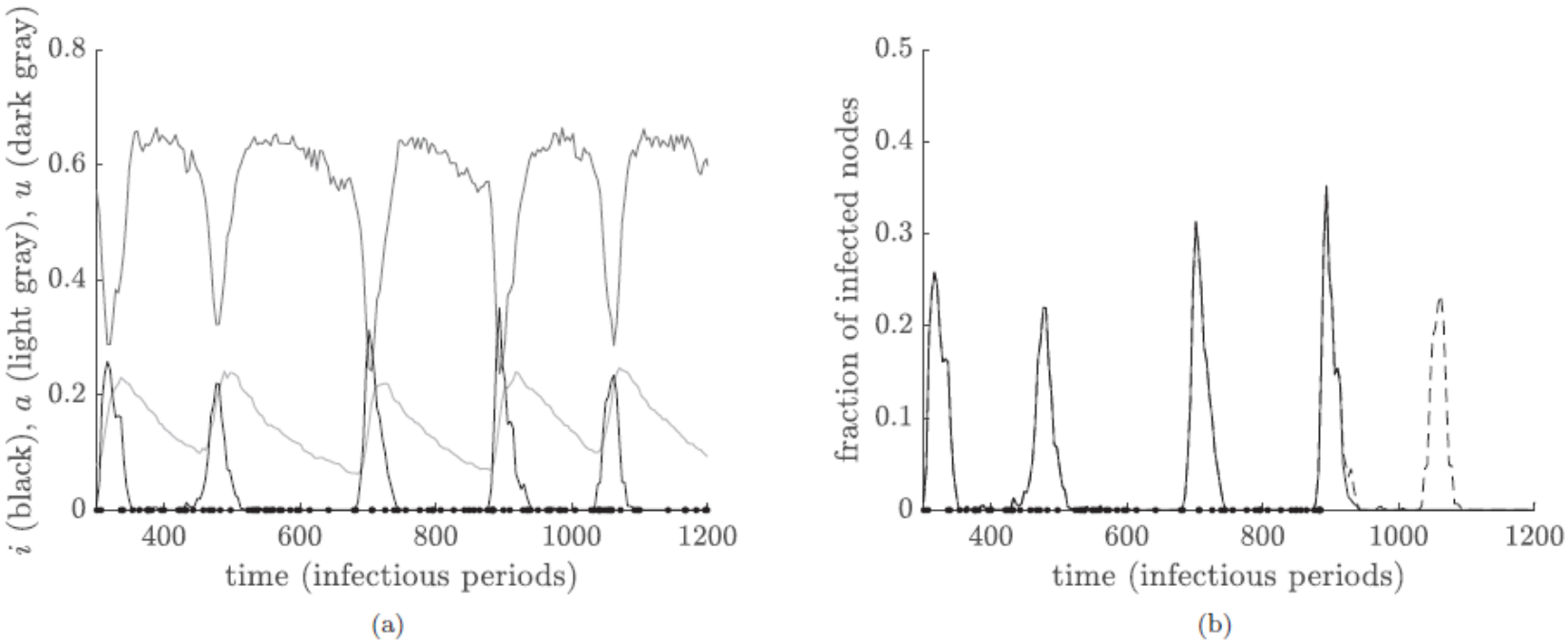


FIG. 6. (a) Time evolution of a stochastic simulation of an epidemic on a random regular network of size $N = 1000$ and degree $k = 100$ showing the input of imported cases (infections from abroad) for $\varepsilon = 10^{-4}$ (black dots on the time axis). (b) Fraction of infected nodes in the same simulation until the beginning of the fourth flare-up where $\varepsilon = 0$ (*lockdown*). Dashed line: Fraction of infected nodes without lockdown. Parameters: $\delta = 1$, $\delta_a = 0.01$, $\delta_u = 0.05$, $\beta = 3$, $\beta_a = 0.05$, $\beta_u = 0.5$, $\alpha_i = 0.15$, $\alpha_a = 0.01$, $\nu_a = 1$, $i(0) = 0.1$, $a(0) = u(0) = 0.2$.

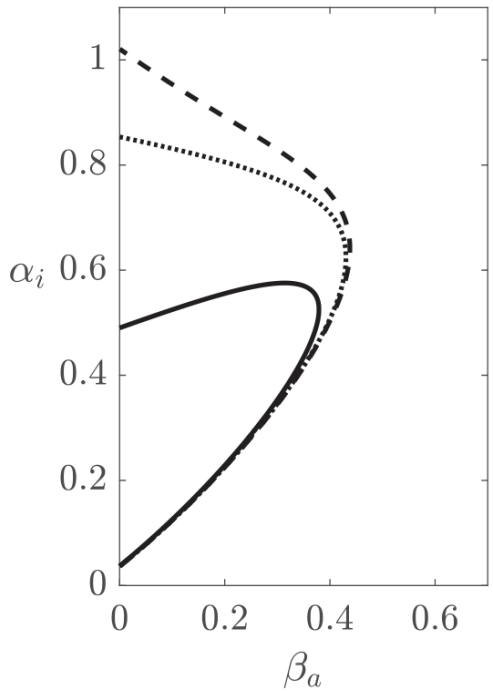


FIG. 1. Hopf-bifurcation curves in the (β_a, α_i) parameter space for $\varepsilon = 0$ (dashed line), $\varepsilon = 10^{-5}$ (dotted line) and $\varepsilon = 10^{-4}$ (solid line). Parameters: $\delta = 1$, $\delta_a = 0.01$, $\delta_u = 0.05$, $\beta = 3$, $\beta_u = 0.5$, $\alpha_a = 0.01$, $\nu_a = 1$.

$$S_m = \max\{\mathrm{Re}(\lambda) \mid \lambda \in \sigma(J(P_3))\}$$

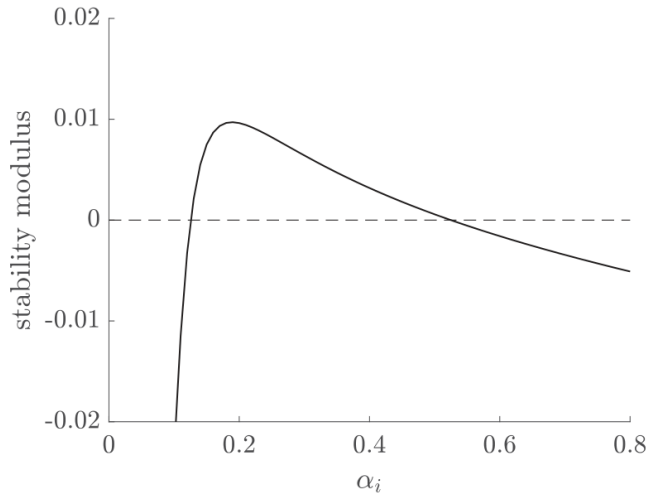


FIG. 2. Stability modulus of the Jacobian matrix at the endemic equilibrium of Eq. (2) as a function of α_i. Parameters: $\delta = 1$, $\delta_a = 0.01$, $\delta_u = 0.05$, $\beta = 3$, $\beta_a = 0.1$, $\beta_u = 0.5$, $\alpha_a = 0.01$, $\nu_a = 1$, $\varepsilon = 10^{-4}$.

Hopf diagram using the FGA[2] on regular random networks of size 10.000:

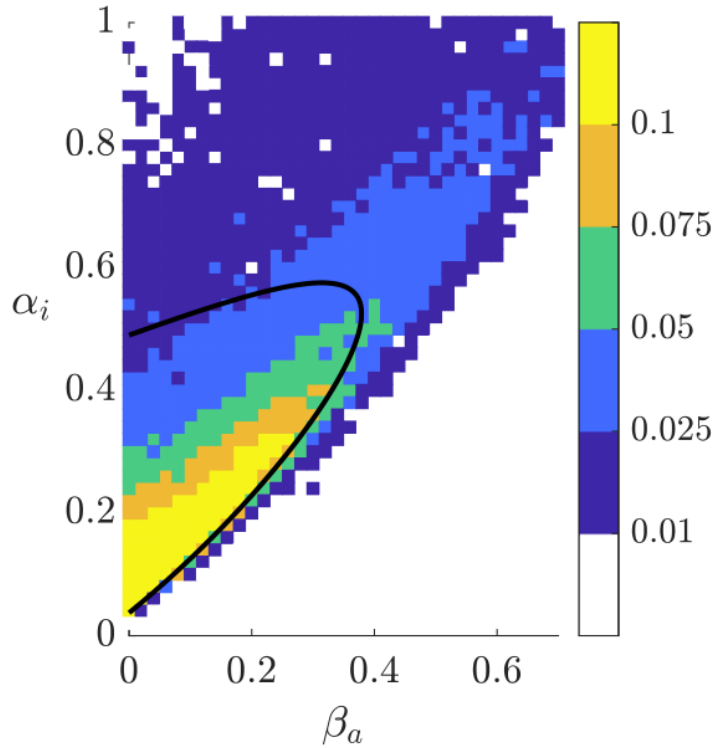


FIG. 9. Hopf diagram obtained according to the description in Sec. IV D of the FGA with $N = 10\,000$ nodes and time $T = 3000$. The gradient of colors evidences the amplitude of the averaged signal of the fraction of infected nodes. The black line is the algebraic Hopf-bifurcation curve for $\varepsilon = 10^{-4}$.

Linear noise approx: The standard deviation of random fluctuations about the mean fraction $\sim \frac{1}{\sqrt{N}} = 0.01$

[2] Fast Gillespie Algorithm. The nodal degree is not a parameter of the algorithm.

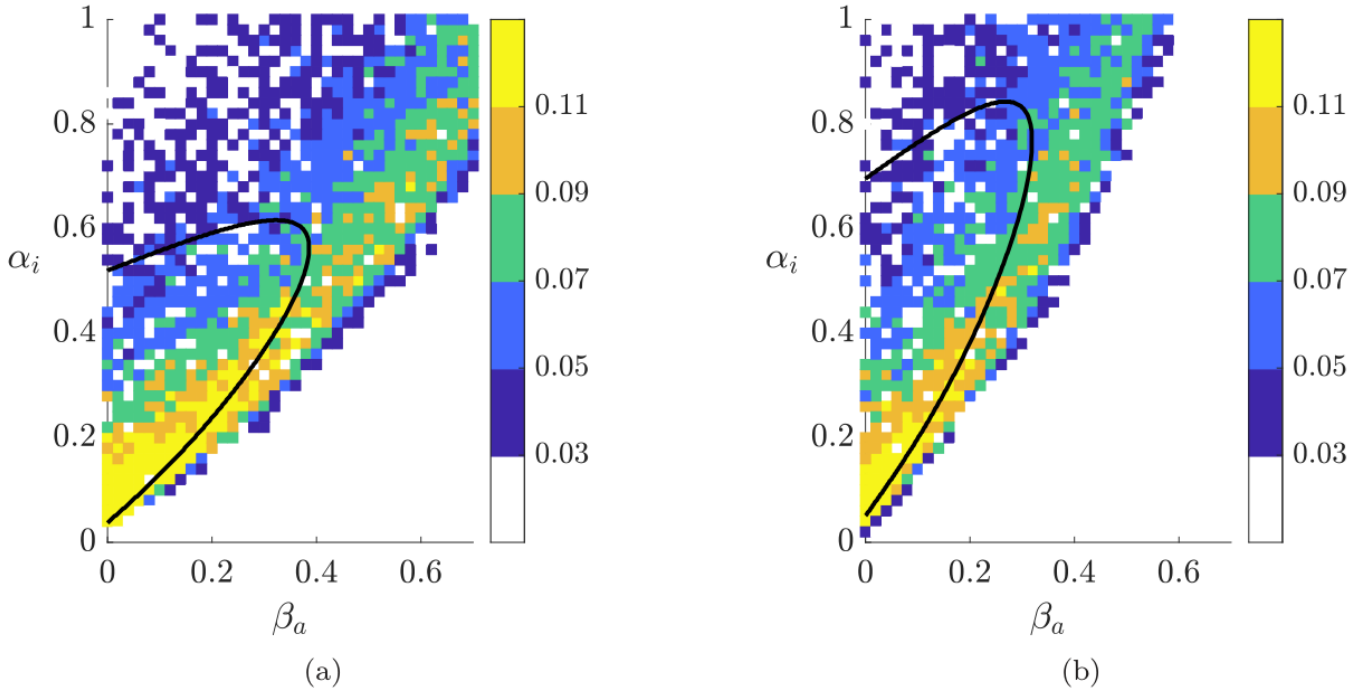


FIG. 10. Hopf diagram of SAUIS-ε obtained according to the description in Sec. IV D with $N = 1000$ nodes and time $T = 3000$, in two different network architectures of mean degree 50: (a) Poisson and (b) Exponential. Parameters: $\delta = 1$, $\delta_a = 0.01$, $\delta_u = 0.05$, $\beta = 3$, $\beta_u = 0.5$, $\alpha_a = 0.01$, $\nu_a = 1$, $\varepsilon = 10^{-4}$, $i(0) = 0.1$, $a(0) = u(0) = 0.2$, 50 experiments for each pair (β_a, α_i). The gradient of colors evidences the amplitude of the signal corresponding to the fraction of infected nodes. The black line is the theoretical Hopf-bifurcation curve.

Linear noise approx: The standard deviation of random fluctuations about the mean fraction $\sim \frac{1}{\sqrt{N}} = 0.0316$

Juher, Rojas & J.S. (2022): SAUIS-ε model with non-constant rates

The model:

$$\begin{aligned}
\frac{da}{dt} &= \alpha_i s\, i + \alpha_a s\, a - \beta_a\, a\, i - \boxed{\delta_a \sigma_m(i)\, a} - \varepsilon a, \\
\frac{du}{dt} &= \boxed{\delta_a \sigma_m(i)\, a} + \boxed{\alpha_u \sigma_m(i)\, s\, a} - \beta_u\, u\, i - \delta_u\, u - \varepsilon u, \\
\frac{di}{dt} &= (\beta\, s + \beta_a a + \beta_u u - \delta)\, i + (1 - i)\varepsilon,
\end{aligned}$$

with $s + a + u + i = 1$, $\varepsilon \geqslant 0$.

With a high disease prevalence, the awareness decay rate δ_a and the alerting rate α_u will be lower than when $i \approx 0$.

$\hookrightarrow$ Both rates are modulated by the function $\sigma_m(i)$.

$$\sigma_m(i) = \frac{1}{1 + (i/\eta)^m}, \quad m \geqslant 1.$$

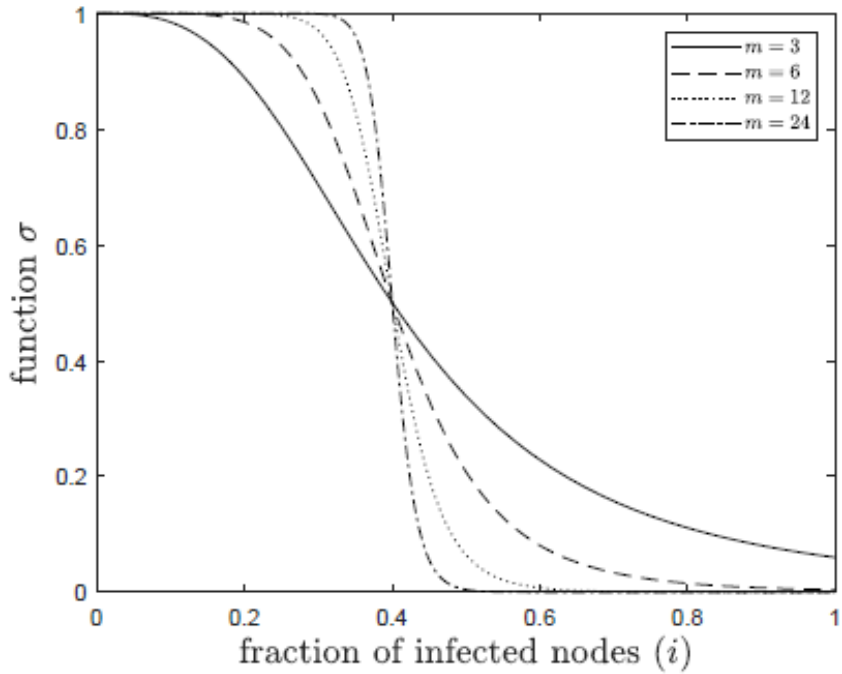


Figure 1: Shape of function $\sigma_m(i)$ with $\eta = 0.4$ for different values of m.

The sharpness of the reduction of the rates is controlled by m.

Lemma: This system with $\varepsilon = 0$ has at least one endemic equilibrium if $0 \leqslant \beta_a < \beta_u < \delta < \beta$ and $R_0^a < \frac{R_0 - R_0^d}{1 - R_0^d}$.

The proof is based, first, on the fact that any endemic equilibrium lies inside the plane

$$i = 1 - \frac{\delta}{\beta} - \left(1 - \frac{\beta_a}{\beta}\right) a - \left(1 - \frac{\beta_u}{\beta}\right) u,$$

which reduces the problem to finding the common zeros of two functions $f_1(a, u)$ and $f_2(a, u)$, and, second, on a version of the Poincaré-Miranda theorem in a triangular domain which guarantees the existence of at least one common zero.

For ε small enough, the system with $\varepsilon > 0$ also has an endemic equilib.

From now on we will restrict ourselves to parameter values that satisfy these two conditions. In particular, they imply $R_0^d < 1 < R_0$.

The tuning parameter is m which is related to the abrupt change in the awareness decay.

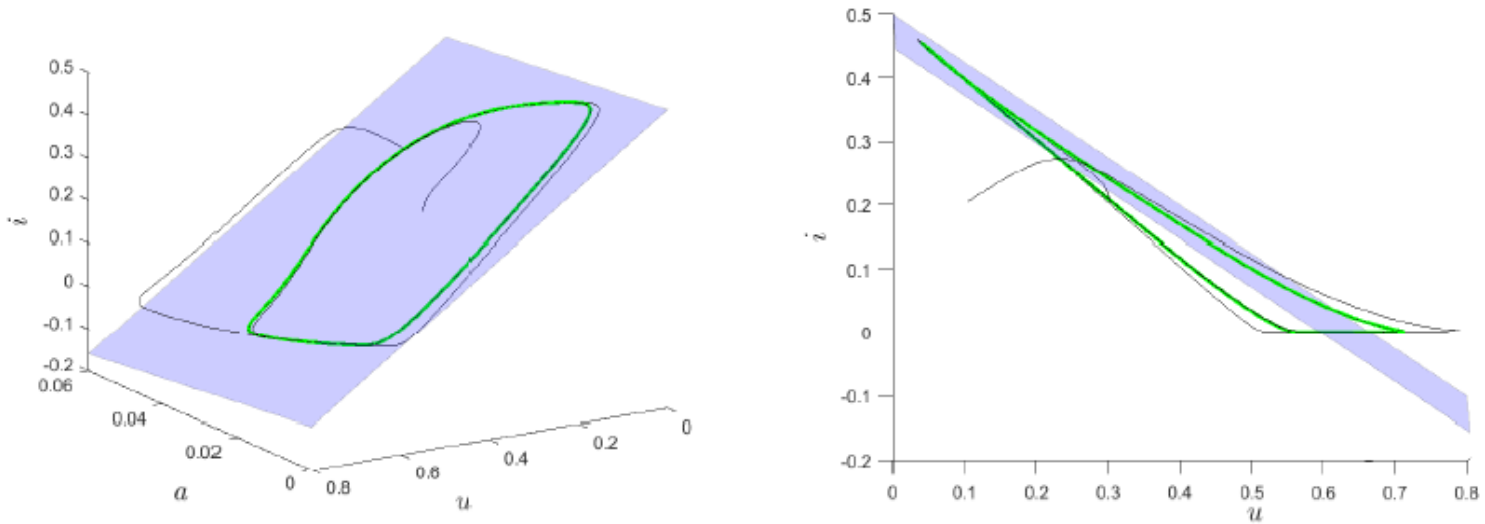
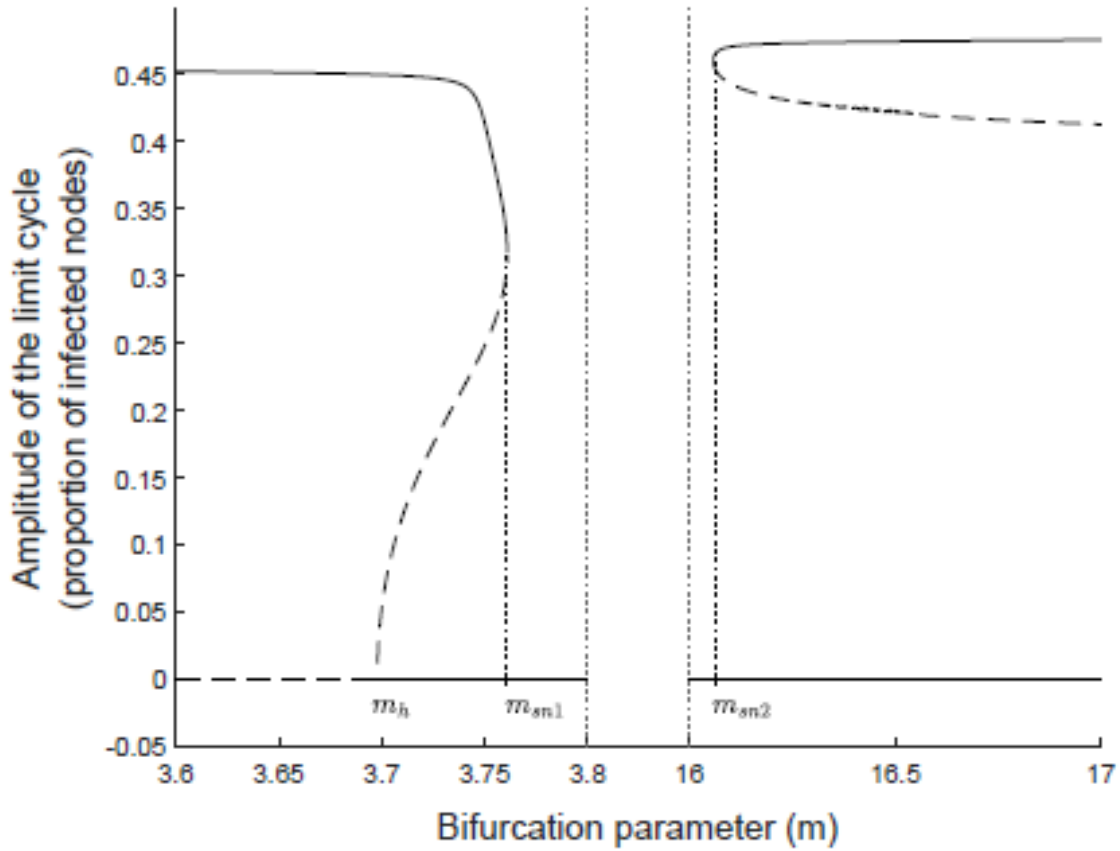


Figure 2: Two solutions of system (1) for $m = 3.5$ tending to the limit cycle (in green) whose dynamics is close to the plane (2) (in light blue).

β	β_a	β_u	α_i	α_a	ν_a	δ	δ_a	δ_u	ε	η
2	0	0.5	0.001	0.015	3	1	0.01	0.03	10^{-5}	0.4

Table 1: Standing values of some parameters

Numerical computation of the limit cycles and their stability using the Poincaré map.



Amplitude = difference between the largest and the smallest fractions of infected individuals along the periodic orbit.

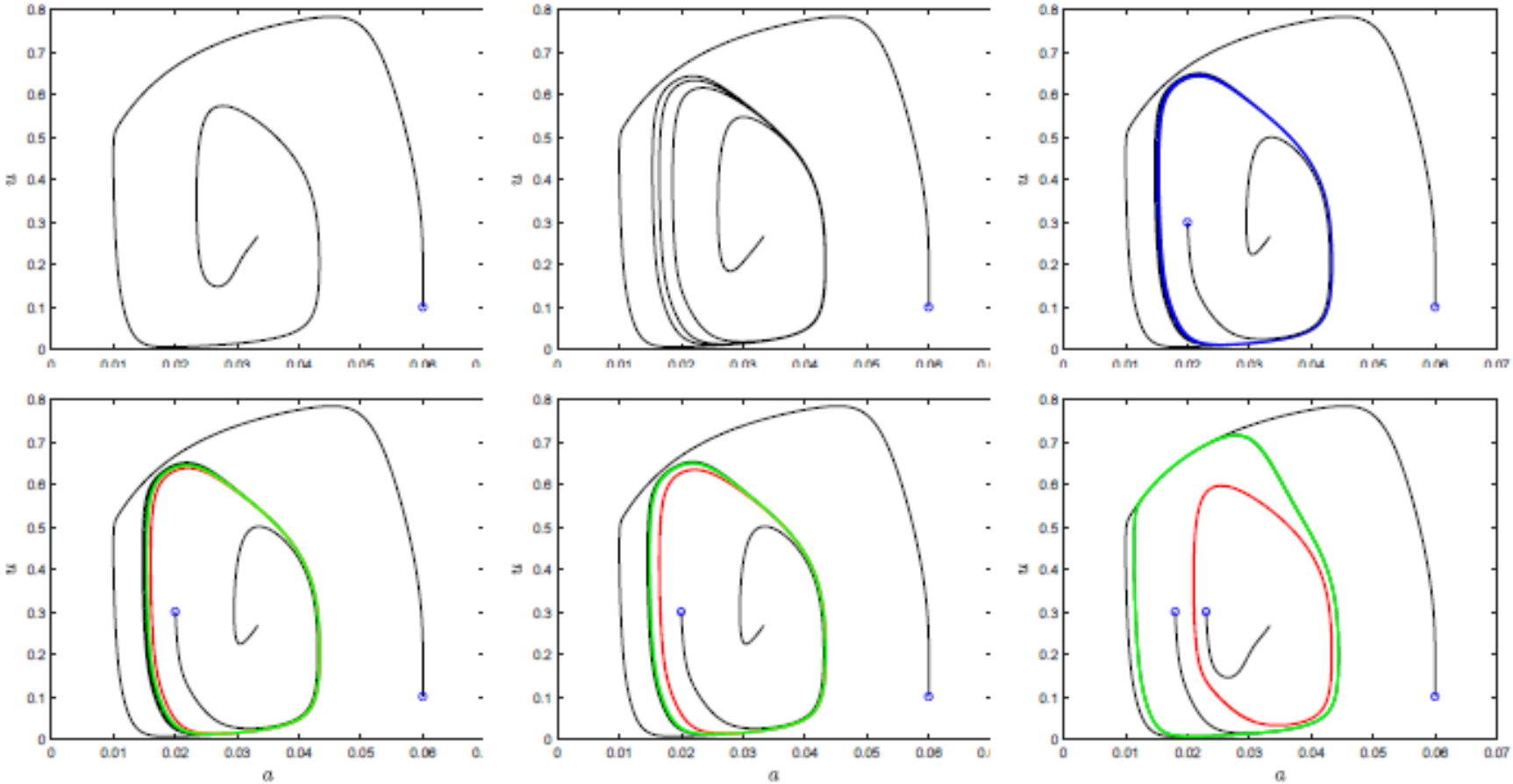
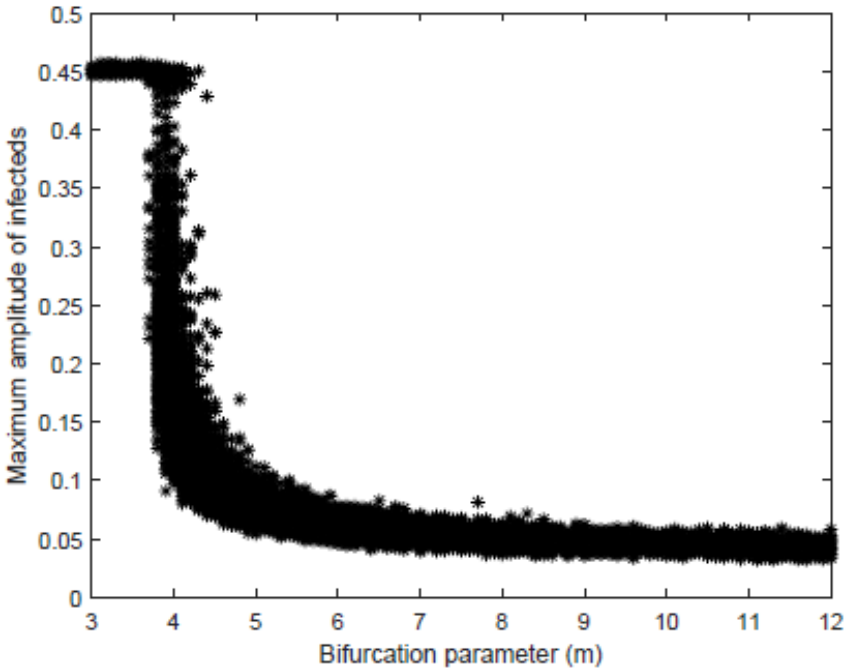
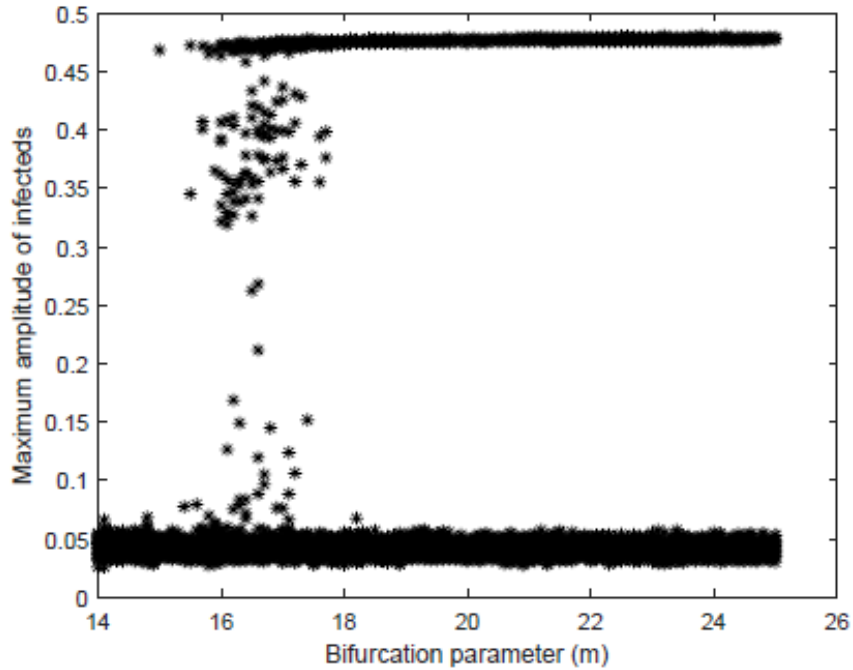


Figure 4: Projections on the (a, u)-plane of some solutions of system (1) for $m = 16$ (top left), $m = 16.05$ (top middle), $m = 16.0575$ (top right), $m = 16.05755$ (bottom left), $m = 16.06$ (bottom middle) and $m = 17$ (bottom right). The orbit in green corresponds to the hyperbolic attractive limit cycle, the orbit in red to the hyperbolic repulsive limit cycle and the orbit in blue to the non-hyperbolic semistable limit cycle. Blue circles mark the initial conditions.

Stochastic detection of bistability:





$n = 1000$ stochastic realizations of an epidemic with $N = 10^5$.

https://sites.google.com/view/epimod-girona2023/

Thank you for your attention!