

Transmission of Infectious Diseases and of Catchy Ideas

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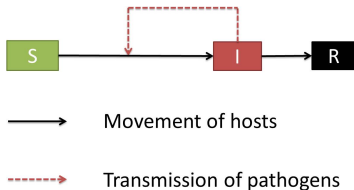


Figure: Schematic representation of the classical *SIR*-model.

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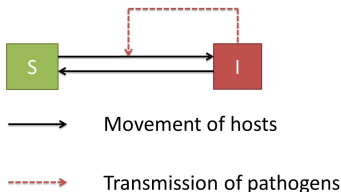
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In many cases though the goal is to **maximize** the spread of an idea. Most religions want believers to spread the faith, Ford Motor Company¹ wants satisfied customers to brag about their shiny new toys, I want you to tell your colleagues and students back home about my talk. And parents try to inoculate their teenagers against drugs by putting other ideas into their heads.

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- **Will the behavioral response be sufficient to prevent future flare-ups?**
- **Which policies will enhance effectiveness of awareness?**

Spread of the disease is assumed to be of type *SIS*

We assume that recovery from infection does not confer immunity. Here we work with an ODE-based *SIS*- model without demographics. Variables s and i represent *fractions* of hosts in the **S**- and **I**-compartments.

$$\begin{aligned}\frac{ds}{dt} &= -\beta i s + \delta i \\ \frac{di}{dt} &= \beta i s - \delta i.\end{aligned}\tag{1}$$

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$$\begin{aligned}\frac{ds}{dt} &= -\beta i s + \delta i \\ \frac{di}{dt} &= \beta i s - \delta i.\end{aligned}\tag{1}$$

We get $R_0 = \frac{\beta}{\delta}$ and:

- The **disease-free equilibrium** $DFE = (s^*, i^*) = (1, 0)$ is globally asymptotically stable iff $R_0 \leq 1$ and unstable if $R_0 > 1$.
- The **endemic equilibrium** $EE = (s^{**}, i^{**}) = (\frac{\delta}{\beta}, 1 - \frac{\delta}{\beta})$ does not exist when $R_0 \leq 1$ and is asymptotically stable when $R_0 > 1$.

Adding a behavioral response: a basic *S**A**S*-model

We incorporate a behavioral response by adding an **A**-compartment of *aware* hosts to the basic *SIS* model. Aware hosts are susceptible but contract the disease at a lower rate $\beta_a < \beta$.

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$$\begin{aligned}\frac{da}{dt} &= \alpha_i(i) s i + \alpha_a(i) s a - \beta_a a i, \\ \frac{di}{dt} &= (\beta s + \beta_a a - \delta) i; \quad s + a + i = 1.\end{aligned}\tag{2}$$

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The term $\alpha_i(i) s i$ models gain of awareness due to **direct information about prevalence**; the term $\alpha_a(i) s a$ models creation of awareness due to **indirect information obtained from aware hosts**.

Theorem

*(Sahneh, Chowdhury and Scoglio 2012) For suitable choices of parameters, we may get an endemic equilibrium $EE = (s^{**}, a^{**}, i^{**})$ with $i^{**} < 1 - \frac{\delta}{\beta}$ or no endemic equilibrium at all, even when $R_0 = \frac{\beta}{\delta} > 1$.*

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How reliable are these predictions?

Two drawbacks of the basic *SAIS*-model

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- The model assumes that hosts never lose awareness. This is unrealistic. Attention is limited and it is very likely that the level of awareness will decay over time, especially when the proportion of infectious hosts has been driven to very low endemic levels.
- Hosts will most likely differ in their willingness to spread awareness of the disease.

An SAIS-model with awareness decay

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*(Juher, Kiss, and Saldaña 2014) For suitable choices of parameters, we may get an endemic equilibrium $EE = (s^{**}, a^{**}, i^{**})$ with $i^{**} \ll 1 - \frac{\delta}{\beta}$. However, when $R_0 = \frac{\beta}{\delta} > 1$, the disease will **not** be driven to extinction in this model.*

Would awareness protect against future flare-ups?

Assume parameters have been chosen so that the model

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How robust is this prediction if we incorporate more realistic features into our model?

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Similarly to aware hosts, unwilling hosts will contract the infection at a reduced rate $\beta_u < \beta$ due to behavior modification.

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The term $-\delta_u(i) u$ represents **decay of the behavioral response over time** for hosts in the **U**-compartment.

The SAUIS-model permits periodic flare-ups

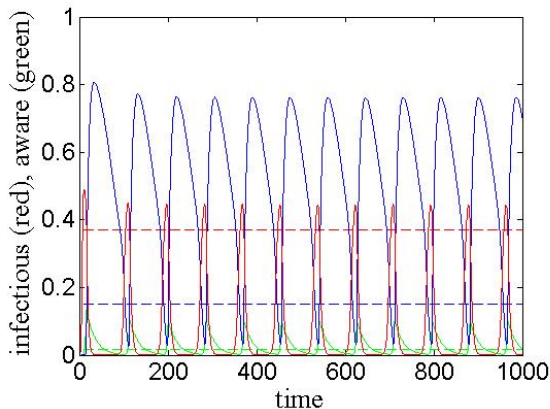


Figure: For certain choices of the parameters $\alpha_i(i), \alpha_a(i), \beta_a, \delta_a(i), \alpha_u(i), \delta_u, \beta, \beta_a, \beta_u, \delta$ we get sustained oscillations of large magnitude.

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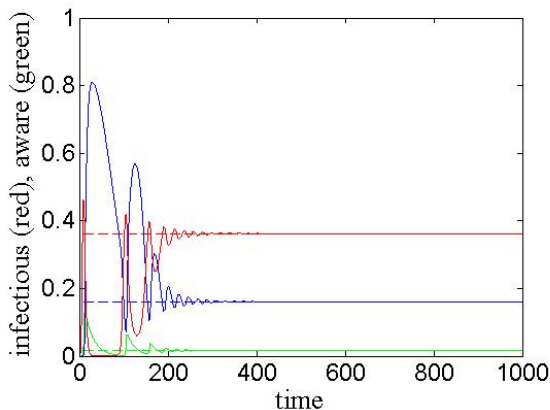


Figure: When we increase $\alpha_i(i)$ (gain of awareness by direct observation) by a factor of 10 but keep all other parameters as in the previous simulation, the oscillations disappear.

Potential implications for public health policy

One can interpret $\alpha_i(i)$ as the rate at which trained professionals who have access to reliable data become aware of the outbreak. Such professionals will then alert the public. Our results indicate that in this interpretation flare-ups may be prevented by increasing the monitoring effort.

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To what extent do these assumptions distort the predictions?

Host-level models

Examples of host-level models:

- Stochastic **network-based models** assumed that hosts can make contact **only** along the edges of a given graph that represents the underlying **contact network**.

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- Awareness usually will spread on a different network than the disease itself. **Multilayer** or **multiplex networks** provide a useful framework for modeling this phenomenon. This approach was in fact taken in (F. D. Sahneh, C. M. Scoglio and Van Mieghem, P. 2013).

Do catchy ideas really spread like pathogens?

Existing single- or multilayer network models assume that transmission of information or pathogens along edges represent independent events. In our context, these models would correspond to ODE-based *SIS*-, *SAIS*-, or *SAUIS*-models with constant rate coefficients.

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However, our results on existence of oscillations in *SAUIS*-models are based on nonlinear functional responses $\alpha_j(i)$, $\alpha_a(i)$, $\alpha_u(i)$. This nonlinearity reflects a threshold like property that can be observed in the spread of fads: Most people will adopt it only if they observe it in a certain minimum number > 1 of other people in their social neighborhood.

A direction of future research

One can try to incorporate the dependencies into (multilayer) network-based models by assuming a sigmoid-shaped distribution of the infection probability for a susceptible host, depending on the proportion of infectious neighbors.

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As far as I know, such models have not yet been investigated.

- [1] F. D. Sahneh, F. N. Chowdhury and C. M. Scoglio (2012). On the existence of a threshold for preventive behavioral responses to suppress epidemic spreading. *Scientific reports* **2**, 632.
- [2] D. Juher, I. Z. Kiss and J. Saldaña (2014). Analysis of an epidemic model with awareness decay on regular random networks. *Journal of Theoretical Biology* to appear.
- [3] F. D. Sahneh, C. M. Scoglio and Van Mieghem, P. (2013). Generalized Epidemic Mean-Field Model for Spreading Processes over Multilayer Complex Networks. *IEEE/ACM Transactions on Networking* **21**(5), 1609–1620.