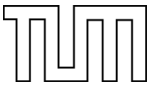

Technische Universität München

Department of Mathematics

Bachelor's Thesis

Dynamics of social learning in vaccination

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Munich,

Zusammenfassung

In den letzten Jahren ist sinkende Impfrate zu einer tickenden Zeitbombe geworden, die durch die steigende Anzahl von Impfskeptikern verursacht wird. Dadurch sind Ausbrüche von Epidemien nur noch eine Frage der Zeit. Das Ziel der Thesis ist die Analyse dieser Impfdynamiken. Der erste Abschnitt stellt die Schwierigkeiten in diesem Gebiet sowie aktuelle Ergebnisse in der Forschung dar. Später werden die beiden Behaviour-Incidence Modelle von Bauch et al. [1] analysiert, worin die Impfskepsis durch soziales Lernen modelliert wird. Das Langzeitverhalten des Standardmodells weist durch die Zeitskalenanalyse keine Oszillationen auf. Das Delay-Modell hingegen deutet durch die Existenz von Hopfpunkten auf mögliche Oszillationen hin. Beide Systeme zeigen eine Schwäche in der Modellierung des Impfverhaltens. Im letzten Abschnitt wird deshalb eine Verbesserung präsentiert, welches auf die Dynamiken der Impfer und Nicht-Impfer basiert.

Abstract

In recent years, the decline in vaccination has grown into ticking time bombs, causing outbreaks of preventable infectious diseases. This problem is caused by the rising numbers of vaccine hesitant people. Our goal in this work is to examine the dynamics of vaccinations with hesitancy. The first chapter introduces difficulties and recent results on this topic to outline its importance in society. We then analyze both behaviour incidence models of Bauch et al. [1], where social learning describes the vaccine scepticism. The long-term characteristics of the standard model don't have any oscillations by the time scale analysis. However, the scepticism turns into hesitancy when the system includes a fixed delay in the incidence term. The existence of Hopf points in this delay model shows evidence for oscillations. In the last section, we illustrate the problems regarding the behaviour dynamics of Bauch's model [1] and finish with a possible improvement.

Contents

1 Introduction

The prevention of disease outbreaks in modern society has turned into a serious matter, especially in times of declining vaccination rates. In addition to that, there aren't any optimal vaccination strategies. This dilemma is mainly caused by a difference in the priorities of community and individual. The first one aims for eradication or at least a prevention of diseases in the population resulting in "herd immunity", while individuals weigh their infection risk with the vaccine risk to stay healthy. This phenomenon leads to the possibility of individuals rejecting vaccination, even if the coverage is below the "herd immunity level" as stated in the paper of Clarkson et al. [3].

In recent years, there have been growing numbers of vaccine hesitant people, despite the relative safeness of the vaccinations. The paper of MacDonald et al. [7] describes this hesitancy as the personal uncertainty in accepting or refusing shots due to convenience, complacency and confidence. Convenience represents social and geographical barriers of vaccination such as affordability, accessibility and quality of service. Vaccine complacency happens when perceived infection risk is below a threshold where shots are deemed unnecessary. Lastly, confidence describes the public trust in vaccination.

The works of Larson et al. [5] suggest that this trust is affected by public concerns about the vaccine industry, the government policies, the reliability of the information source and the safeness of vaccination. It decreases with growing concerns, which results in hesitancy or distrust. Additionally, rumors and opinions of vaccine enemies support the decline of trust. To handle these problems, the paper of Fahlquist [10] recommends that the information regarding immunizations should be appealing, transparent and enlightening with interactive communication. It also suggests that the government should be responsible for individuals harmed by the side effects of shots to promote public trust.

In conclusion, the following table (Figure 1) provides a possible categorization of the determinants for vaccine hesitancy. Some of these are communications, social norms or social network structures, etc.

Table 1
Working Group on Vaccine Hesitancy Determinants Matrix.

Contextual influences Influences arising due to historic, socio-cultural, environmental, health system/institutional, economic or political factors	a. Communication and media environment b. Influential leaders, immunization programme gatekeepers and anti- or pro-vaccination lobbies c. Historical influences d. Religion/culture/gender/socio-economic e. Politics/policies f. Geographic barriers g. Perception of the pharmaceutical industry
Individual and group influences Influences arising from personal perception of the vaccine or influences of the social/peer environment	a. Personal, family and/or community members' experience with vaccination, including pain b. Beliefs, attitudes about health and prevention c. Knowledge/awareness d. Health system and providers – trust and personal experience e. Risk/benefit (perceived, heuristic) f. Immunization as a social norm vs. not needed/harmful
Vaccine/vaccination – specific issues Directly related to vaccine or vaccination	a. Risk/benefit (epidemiological and scientific evidence) b. Introduction of a new vaccine or new formulation or a new recommendation for an existing vaccine c. Mode of administration d. Design of vaccination programme/Mode of delivery (e.g., routine programme or mass vaccination campaign) e. Reliability and/or source of supply of vaccine and/or vaccination equipment f. Vaccination schedule g. Costs h. The strength of the recommendation and/or knowledge base and/or attitude of healthcare professionals

Figure 1: Table of the vaccine hesitancy determinants [7]

In terms of communication, the impacts of unfavorable events for vaccination and infection are discussed in the paper of Bauch et al. [2]. By their findings, vaccination lowers the infection peak to a similar level with or without unfavorable events. These events mainly play a role in the tail of the outbreaks, as shown in Figure 2. Interestingly, the case of immunizations with both adverse events possesses a long infection tail. This phenomenon is caused by the infection of the remaining unvaccinated individuals. The impact of these events varies with different social network structures since their information influences each individual or community differently.

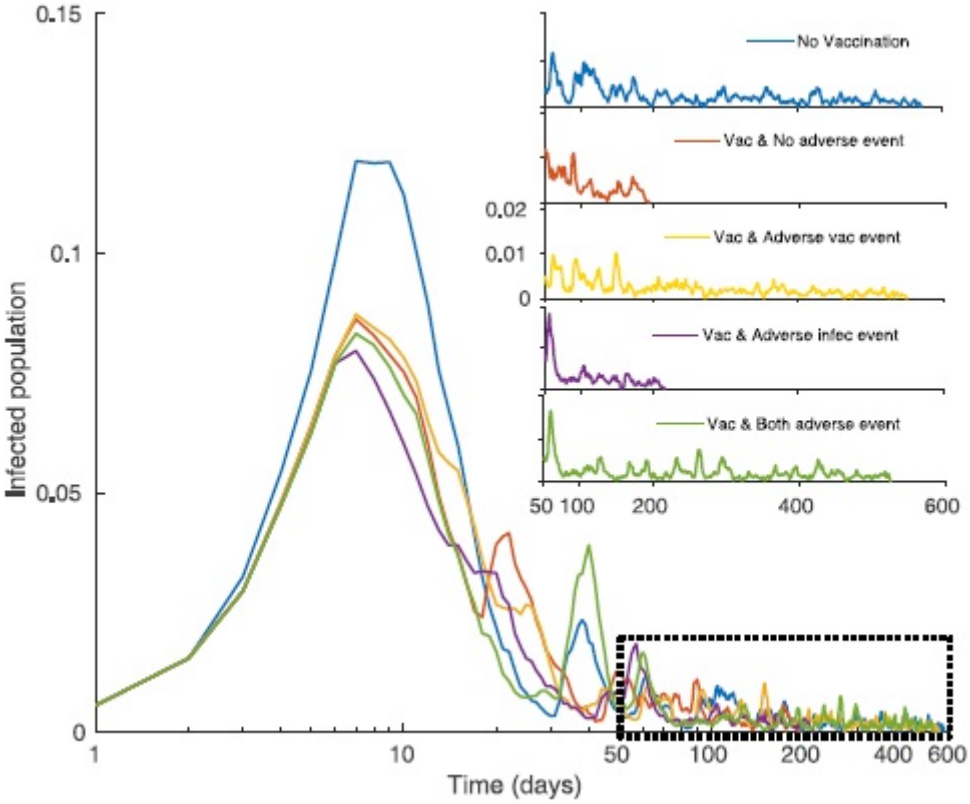


Figure 2: A simulation of relative infected population over time [2]

In this sense, the paper of Oraby et al. [11] suggests that the effects of social norms are also important. They can either promote vaccine coverage or drop it to suboptimal levels, which results in a system with bistable behaviour that may oscillate. Regarding oscillations, the norms weaken their strength and occurrence to stabilize the system. These properties are modelled by improving Bauch's behaviour incidence model [1], where the vaccination dynamics are described by social learning.

This learning allows individuals to improve their vaccine strategy after comparing its payoff with that from a randomly chosen person. The payoff represents the difference between the perceived infection risk and vaccine risk, which is positive if the shot is necessary and negative vice versa. However, the explanatory power of Bauch's system is sometimes limited since it only uses modelled data for the risks and disease incidence. Lastly, it can not determine the occurrence of hesitancy, which is dependent on random events [1].

Now, recent results of the paper from Li et al. [6] have shown further improvements of Bauch's behaviour incidence model [1]. It is unrealistic that an individual only compares its strategy with that of a randomly chosen person. Therefore, the improvement allows them to consider the weighted average payoff gain of people with the same strategy instead. Furthermore, it includes the influence of social network structures on an individual's learning behaviour. Lastly, there is a motivating effect for unvaccinated individuals and those with failed immunizations. This idea works with a promoting strategy, which boosts the payoff of infected individuals with unsuccessful vaccination to a higher level than those without it. Further modifications of this model are also possible, which will not be discussed in this work.

2 Mathematical tools

In this section, we define some necessary tools for analyzing the long-term behaviour of continuous ODEs. For convenience reasons, we do not state their proofs in this work. These can be found in the book "Methods and models in mathematical biology" [9]. So let us consider $f(x) \in C^1(\mathbb{R}^n, \mathbb{R}^n)$ and $\dot{x} = f(x)$. For $f(\bar{x}) = 0$ with $\bar{x} \in \mathbb{R}^n$ let $A = f'(\bar{x}) = \frac{\partial f_i}{\partial x_k}(\bar{x})$ define the Jacobian matrix of the linearized system $\dot{z} = Az$, then the following holds [9]:

Definition 2.1 ***(Asymptotic stability of a stationary point)*** *[9]*
A stationary point $\bar{x}$ is called asymptotically stable if it is stable and if there is $\delta_0 > 0$ such that for all x_0 satisfying $|x_0 - \bar{x}| < \delta_0$ it is

$$\lim_{t \to \infty} |x(t) - \bar{x}| = 0$$

Definition 2.2 ***(Hyperbolic)*** *[9]*
$\bar{x}$ is called hyperbolic if $0 \notin Re\ \sigma(f'(\bar{x}))$, where $\sigma(f'(x))$ denotes the spectrum of $f'(x)$.

Theorem 2.3 ***(Hartmann and Grobmann)*** *[9]*
Let $\bar{x}$ be hyperbolic. Then, there is a neighbourhood U of $\bar{x}$ and a homeomorphism $H : U \to \mathbb{R}^n$ with $H(\bar{x}) = 0$, which maps the trajectories of $\dot{x} = f(x)$ one-to-one into trajectories of the linearized system w.r.t. the time course.

Definition 2.4 ***(ω-limit set)*** *[9]*
Consider a trajectory $x(t)$ with initial value x_0. The ω limit set of x_0 is given by:

$$\omega(x_0) = \{y \,|\, \exists (t_n),\ t_n \nearrow \infty,\ y = \lim_{n \to \infty} x(t_n)\}$$

Definition 2.5 ***(Periodic orbit, homoclinic orbit, heteroclinic orbit)*** *[9]*
Consider a ODE $\dot{x} = f(x, , t)$, $x \in \mathbb{R}^n$. A solution $x(t)$ with initial condition x_0 is called trajectory of x_0. A non-constant trajectory with $x(t + T) = x(t)$ for some $T > 0$ is called a ***periodic orbit****. A non-constant trajectory $x(t)$ that ends at a stationary point x_1 and starts at a stationary point x_2 is called* ***heteroclinic orbit*** *if $x_1 \neq x_2$ and* ***homoclinic orbit*** *else. A heteroclinic cycle is a finite number of heteroclinic orbits, connecting n stationary points in a cyclic manner.*

Theorem 2.6 ***(Poincaré-Bendixson)*** *[9]*
Consider a trajectory $x(t) \in \mathbb{R}^2$ (or $x(t) \in D$, where $D \subset \mathbb{R}^2$ is compact and connected, positively invariant) of the ODE $\dot{x} = f(x)$, f smooth, with only finitely many roots. If $x(t)$ is bounded, then the ω-limit set is one of the following objects:

- *A stationary point*
- *A periodic orbit*
- *A homoclinic orbit or heteroclinic cycle*

Theorem 2.7 ***(Negative criterion of Bendixson)*** *[9]*
Let $D \subset \mathbb{R}^2$ be a simply connected region and $(f, g) \in C^1(D, \mathbb{R})$ with $div(f, g) = \frac{\partial f}{x} + \frac{\partial g}{y} \neq 0$ and without change of sign in D then the system:

$$\begin{cases} \dot{x} = f(x, y) \\ \dot{y} = g(x, y) \end{cases}$$

has no closed orbits lying entirely in D.

Theorem 2.8 *(Stability criteria for continuous ODE's) [9]*
Consider the linear case $\dot{x} = Ax$ with stationary point $\bar{x}$ and $A \in \mathbb{C}^{n\times n}$. Let $\sigma(A)$ be the spectrum of A.

1. *$\bar{x}$ is asymptotically stable $\Leftrightarrow$ Re $\sigma(A) < 0$*
2. *$\bar{x}$ is stable $\Leftrightarrow$ Re $\sigma(A) \leq 0$ and all eigenvalues with $Re(\lambda) = 0$ are semi-simple (i.e., geometric and algebraic multiplicity are the same)*
3. *If there is a $\lambda \in \sigma(A)$ with $Re(\lambda) > 0$, then $\bar{x}$ is unstable. (The reversed direction is wrong!)*

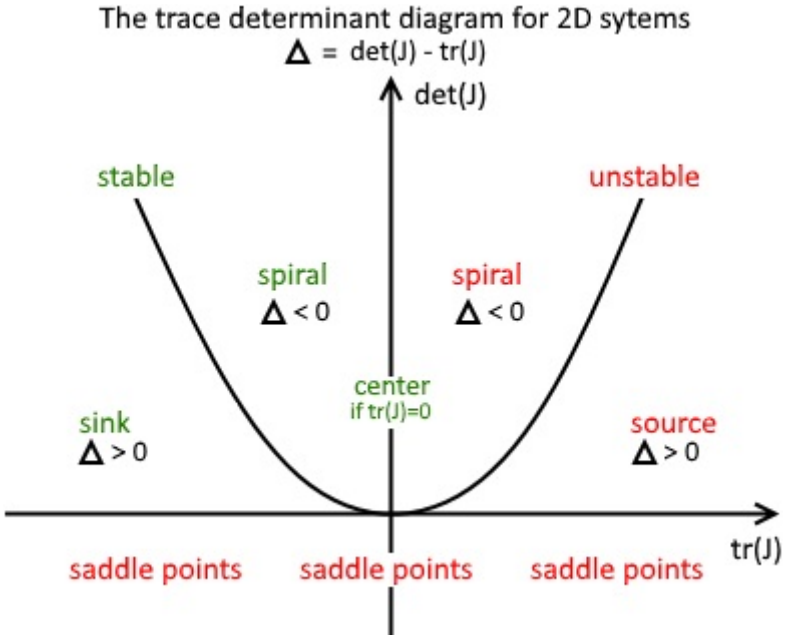


Figure 3: Illustration of the Trace-Determinant Diagram

Sometimes eigenvalues are hard to determine, then the Trace-Determinant Diagram or Criteria (Figure 3) is used for the stability of stationary states. However, this is only applicable to 2D systems. For higher dimensional systems, we need the Hurwitz Criteria (Thm 2.9) instead. In the following, we specify this criterion for 3D-systems:

Theorem 2.9 ***(Routh-Hurwitz Criteria for k=3)*** *[9]*
Let us consider the characteristic polynomial of a three dimensional Jacobian matrix, which is $\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3$. Then all eigenvalues have negative real parts if and only if the determinants of the Hurwitzmatrix H_j are positive i.e if $\det H_j > 0$ for $j = 1, ..., 3$, which is equivalent to $a_1 > 0$; $a_3 > 0$; $a_1a_2 > a_3$.

In the literature, the transcritical bifurcation is defined by the normal form (for more details see [4]). For convenience reasons, we introduce our definition of this bifurcation.

Definition 2.10 ***(Transcritical Bifurcation)*** *[9]*
A bifurcation is called transcritical bifurcation if the two stationary points of the system exchange their stability, exactly at the parameter value, where both points meet.

Theorem 2.11 ***(Hopf point)*** *[9]*
Consider the system $\dot{x} = f_\mu(x)$, $x \in \mathbb{R}$ and assume that f_{μ_0} has an equilibrium at (x_0, μ_0) with the following properties:
The Jacobian $f'_\mu(x_0)$ has exactly one pair of purely imaginary complex eigenvalues (no other eigenvalues with real parts = 0).
Then there is a (locally) smooth curve of equilibria $(x(\mu), \mu)$ with $x(\mu_0) = x_0$. The eigenvalues $\lambda(\mu), \bar{\lambda}(\mu)$ of the Jacobian $f'_\mu(x_0)$ which are purely imaginary at $\mu = \mu_0$, depend smoothly on μ. If additionally the condition:

$$\frac{d}{d\mu}(Re\lambda(\mu)|_{\mu=\mu_0} \coloneqq d \neq 0$$

is satisfied, then there exists a Hopf point.

Additionally, a Hopf bifurcation occurs if the corresponding generic conditions are fulfilled (for more details see [9]). For simplicity's sake, we do not consider them in this work. In the next step, we want to find a necessary and sufficient condition for the existence of Hopf points using the Routh-Hurwitz Criteria (Thm 2.9), so let us consider the system:

$$\frac{dz}{dt} = f_\alpha(z), z \in \mathbb{R}^n \alpha \in \mathbb{R} \tag{1}$$

with an equilibrium (z^*, α^*) and $f \in C^\infty$. A Hopf point exists if the following holds:

Theorem 2.12 ***(Liu's Theorem)*** *[8]*
Assume there is a smooth curve of equilibria $(z(\alpha), \alpha)$ with $z(\alpha^*) = z^*$ for the system (1). Let $p(\lambda; \alpha)$ be the corresponding characteristic polynomial, then there is a Hopf point if the following conditions hold:

1. $p_0(\alpha^*) > 0, \hat{H}_1(\alpha^*) > 0, \hat{H}_2(\alpha^*) > 0, ..., \hat{H}_{n-2}(\alpha^*) > 0, \hat{H}_{n-1}(\alpha^*) = 0$
2. $\frac{d}{d\alpha}(\hat{H}_{n-1}(\alpha^*)) \neq 0$

where $\hat{H}_i$ are the Hurwitz determinants.

Proof. For convenience reasons, we do not state the proof in this work. It can be found in the paper of Junichi Minagawa [8]. □

3 The behaviour incidence model

In this section, we work with the behaviour incidence model of Bauch et al. [1], where vaccine scepticism is described by social learning. Here, individuals improve their vaccine strategy by comparing its payoff with that from a randomly chosen person. This payoff represents the difference between infection risk and vaccine risk, which is positive if the shot should be taken and negative the other way round.

3.1 Bifurcation analysis

We are interested in the effects and long term characteristics of Bauch's SIR-behaviour incidence model [1] with the simplification $\tau = 0$:

$$\begin{aligned} \dot{s} &= \mu(1-\varepsilon x) - \mu s - \beta s i \\ \dot{i} &= -\mu i - \gamma i + \beta s i \\ \dot{x} &= kx(1-x)(i-\omega) \end{aligned} \tag{2}$$

where $x(t) \in [0,1]$ denotes the proportion of vaccinators in the population at time t and ω being the risk evolution curve, which describes the time evolution of the vaccine penalty. Here, s is the relative number of susceptibles, i the relative number of infectives and r the relative number of recovered w.r.t the total population. We assume that $s+i+r=1$ and all parameters are positive, where μ is the birth rate or death rate per capita, ε is the vaccine efficacy, β is the transmission rate, γ is the recovery rate and k is a scale factor.

3.1.1 Steady states

Let us begin with the analysis by determining the stationary states of the system (2):

Theorem 3.1 ***(disease-free steady states)***
The disease-free stationary states of the system (2) are:

- $(1,0,0)$
- $(1-\varepsilon,0,1)$

Proof. We assume that $i=0$ in the absence of infection. Hence, the disease-free stationary states are the solutions of the following equations

$$\begin{cases} 0 = (1-\varepsilon x) - s \\ 0 = -kx\omega(1-x) \end{cases}$$

which yield our claim. □

Theorem 3.2 ***(endemic steady states)***
The endemic stationary states of the system (2) are:

- $(\frac{\mu+\gamma}{\beta}, \frac{\mu}{\mu+\gamma} - \frac{\mu}{\beta}, 0)$
- $(\frac{\mu+\gamma}{\beta}, \frac{\mu(1-\varepsilon)}{\mu+\gamma} - \frac{\mu}{\beta}, 1)$
- $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ *where* $x_0 = \frac{1}{\varepsilon}(1 - \frac{\mu+\gamma}{\beta} - \frac{\mu+\gamma}{\mu}\omega)$ *with* $\beta \in (\frac{\mu(\mu+\gamma)}{\mu-(\mu+\gamma)\omega}, \frac{\mu(\mu+\gamma)}{\mu(1-\varepsilon)-(\mu+\gamma)\omega})$

Proof. Analogously the endemic steady states are the solutions of the equations

$$\begin{cases} 0 = \mu(1-\varepsilon x) - \mu s - \beta s i \\ 0 = -\mu i - \gamma i + \beta s i \\ 0 = kx(1-x)(i-\omega) \end{cases}$$

which finish our proof. □

3.1.2 Stability analysis

Now we consider the stability of these states. So, let us start with the general Jacobian matrix J:

$$J(s,i,x) = \begin{pmatrix} -\mu - \beta i & -\beta s & -\mu\varepsilon \\ \beta i & -\mu - \gamma + \beta s & 0 \\ 0 & kx(1-x) & k(1-2x)(i-\omega) \end{pmatrix} \tag{3}$$

We state that the Hartmann & Grobmann theorem (Thm 2.3) holds for our stationary points since the condition $0 \notin \operatorname{Re}\sigma(f'(\bar{x}))$ will always be fulfiled. Therefore, the stability criteria (Thm 2.8) is applicable, which we need afterward.

Theorem 3.3
The disease-free stationary state $(1,0,0)$ *is* $\begin{cases} \textit{stable} & \textit{if } \beta < \mu + \gamma \\ \textit{unstable} & \textit{if } \beta > \mu + \gamma \end{cases}$

Proof. For (1,0,0) the Jacobian matrix reads

$$J(1,0,0) = \begin{pmatrix} -\mu & -\beta & -\mu\varepsilon \\ 0 & -\mu - \gamma + \beta & 0 \\ 0 & 0 & k\omega \end{pmatrix}$$

with the eigenvalues $\lambda_1 = -\mu$, $\lambda_2 = -k\omega$, $\lambda_3 = -\mu - \gamma + \beta$. The claim follows by the stability criteria (Thm 2.8) since all parameters are positive by assumption. □

Theorem 3.4
The disease-free stationary state $(1-\varepsilon, 0, 1)$ *is unstable.*

Proof. For $(1-\varepsilon,0,1)$ the Jacobian matrix reads:

$$J(1-\varepsilon,0,1) = \begin{pmatrix} -\mu & -\beta(1-\varepsilon) & -\mu\varepsilon \\ 0 & -\mu - \gamma + \beta(1-\varepsilon) & 0 \\ 0 & 0 & k\omega \end{pmatrix}$$

with the eigenvalues $\lambda_1 = -\mu$, $\lambda_2 = k\omega$, $\lambda_3 = -\mu - \gamma + \beta(1-\varepsilon)$. We conclude by the stability criteria (Thm 2.8) with $\lambda_2 > 0$ that $(1-\varepsilon,0,1)$ is unstable. □

Theorem 3.5
The endemic steady state $(\frac{\mu+\gamma}{\beta}, \frac{\mu}{\mu+\gamma} - \frac{\mu}{\beta}, 0)$ *is* $\begin{cases} \textit{stable} & \textit{if } \beta \in (\mu+\gamma, \frac{\mu(\mu+\gamma)}{\mu-\omega(\mu+\gamma)}) \\ \textit{unstable} & \textit{else} \end{cases}$

Proof. For $(\frac{\mu+\gamma}{\beta}, \frac{\mu}{\mu+\gamma} - \frac{\mu}{\beta}, 0)$ the Jacobian matrix reads

$$J(\tfrac{\mu+\gamma}{\beta}, \tfrac{\mu}{\mu+\gamma} - \tfrac{\mu}{\beta}, 0) = \begin{pmatrix} -\frac{\beta\mu}{\mu+\gamma} & -(\mu+\gamma) & -\mu\varepsilon \\ \frac{\beta\mu}{\mu+\gamma} - \mu & 0 & 0 \\ 0 & 0 & k(\frac{\mu}{\mu+\gamma} - \frac{\mu}{\beta} - \omega) \end{pmatrix}$$

with the characteristic polynomial

$$\chi_J(\lambda) = (\lambda - k\mu(\frac{1}{\mu+\gamma} - \frac{1}{\beta} - \frac{\omega}{\mu}))(\lambda^2 + \frac{\beta\mu}{\mu+\gamma}\lambda + (\mu+\gamma)(\frac{\beta\mu}{\mu+\gamma} - \mu))$$

which yields the eigenvalues with the following properties

- $\lambda_1 = k\mu(\frac{1}{\mu+\gamma} - \frac{1}{\beta} - \frac{\omega}{\mu}) = \begin{cases} < 0 \text{ if } \beta < \frac{\mu(\mu+\gamma)}{\mu-\omega(\mu+\gamma)} \\ > 0 \text{ if } \beta > \frac{\mu(\mu+\gamma)}{\mu-\omega(\mu+\gamma)} \end{cases}$
- $\lambda_2 = -\frac{\beta\mu}{2(\mu+\gamma)} - \frac{1}{2}\sqrt{(\frac{\beta\mu}{\mu+\gamma})^2 - 4(\mu+\gamma)(\frac{\beta\mu}{\mu+\gamma} - \mu)} < 0$
- $\lambda_3 = -\frac{\beta\mu}{2(\mu+\gamma)} + \frac{1}{2}\sqrt{(\frac{\beta\mu}{\mu+\gamma})^2 - 4(\mu+\gamma)(\frac{\beta\mu}{\mu+\gamma} - \mu)} = \begin{cases} < 0 \text{ if } \beta > \mu+\gamma \\ > 0 \text{ if } \beta < \mu+\gamma \end{cases}$

then this steady state is stable by the stability criteria (Thm 2.8) if $\beta \in (\mu+\gamma, \frac{\mu(\mu+\gamma)}{\mu-\omega(\mu+\gamma)})$ and the claim follows. □

Theorem 3.6

The endemic steady state $(\frac{\mu+\gamma}{\beta}, \frac{\mu(1-\varepsilon)}{\mu+\gamma} - \frac{\mu}{\beta}, 1)$ *is* $\begin{cases} \textit{stable} & \textit{if } \beta > \frac{\mu(\mu+\gamma)}{\mu(1-\epsilon)-\omega(\mu+\gamma)} \\ \textit{unstable} & \textit{if } \beta < \frac{\mu(\mu+\gamma)}{\mu(1-\epsilon)-\omega(\mu+\gamma)} \end{cases}$

Proof. For $(\frac{\mu+\gamma}{\beta}, \frac{\mu(1-\varepsilon)}{\mu+\gamma} - \frac{\mu}{\beta}, 1)$ the Jacobian matrix reads

$$J(\tfrac{\mu+\gamma}{\beta}, \tfrac{\mu(1-\varepsilon)}{\mu+\gamma} - \tfrac{\mu}{\beta}, 1) = \begin{pmatrix} -\frac{\beta\mu(1-\varepsilon)}{\mu+\gamma} & -(\mu+\gamma) & -\mu\varepsilon \\ \frac{\beta\mu(1-\varepsilon)}{\mu+\gamma} - \mu & 0 & 0 \\ 0 & 0 & -k(\frac{\mu(1-\varepsilon)}{\mu+\gamma} - \frac{\mu}{\beta} - \omega) \end{pmatrix}$$

with the characteristic polynomial

$$\chi_J(\lambda) = (\lambda + k\mu(\frac{1-\varepsilon}{\mu+\gamma} - \frac{1}{\beta} - \frac{\omega}{\mu}))(\lambda^2 + \frac{\beta\mu(1-\varepsilon)}{\mu+\gamma}\lambda + (\mu+\gamma)(\frac{\beta\mu(1-\varepsilon)}{\mu+\gamma} - \mu))$$

by further calculations we get the eigenvalues with the following properties

- $\lambda_1 = -k\mu(\frac{1-\varepsilon}{\mu+\gamma} - \frac{1}{\beta} - \frac{\omega}{\mu}) = \begin{cases} < 0 \text{ if } \beta > \frac{\mu(\mu+\gamma)}{\mu(1-\epsilon)-\omega(\mu+\gamma)} \\ > 0 \text{ if } \beta < \frac{\mu(\mu+\gamma)}{\mu(1-\epsilon)-\omega(\mu+\gamma)} \end{cases}$
- $\lambda_2 = -\frac{\beta\mu(1-\varepsilon)}{2(\mu+\gamma)} - \frac{1}{2}\sqrt{(\frac{\beta\mu(1-\varepsilon)}{\mu+\gamma})^2 - 4(\mu+\gamma)(\frac{\beta\mu(1-\varepsilon)}{\mu+\gamma} - \mu)} < 0$
- $\lambda_3 = -\frac{\beta\mu(1-\varepsilon)}{2(\mu+\gamma)} + \frac{1}{2}\sqrt{(\frac{\beta\mu(1-\varepsilon)}{\mu+\gamma})^2 - 4(\mu+\gamma)(\frac{\beta\mu(1-\varepsilon)}{\mu+\gamma} - \mu)} = \begin{cases} < 0 \text{ if } \beta > \frac{\mu+\gamma}{1-\varepsilon} \\ > 0 \text{ if } \beta < \frac{\mu+\gamma}{1-\varepsilon} \end{cases}$

then at least one of the condition $\beta > \frac{\mu+\gamma}{1-\varepsilon}$ or $\beta > \frac{\mu(\mu+\gamma)}{\mu(1-\epsilon)-\omega(\mu+\gamma)}$ is required for the stability of this stationary state.

Now assume the steady state is stable and $\beta > \frac{\mu+\gamma}{1-\varepsilon}$ holds. Then $-k\mu(\frac{1-\varepsilon}{\mu+\gamma} - \frac{1}{\beta} - \frac{\omega}{\mu}) > 0$ implies $\lambda_1 > 0$, which results in instability that contradicts the assumption. Checking the other condition yields that the eigenvalues fulfil the stability criteria (Thm 2.8) and the claim follows. □

Theorem 3.7

The endemic steady state $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ *is* $\begin{cases} \textit{stable} & \textit{if } \beta > \frac{\mu}{\omega}(\frac{\varepsilon k x_0(1-x_0)}{\mu+\gamma} - 1) \\ \textit{unstable} & \textit{if } \beta < \frac{\mu}{\omega}(\frac{\varepsilon k x_0(1-x_0)}{\mu+\gamma} - 1) \end{cases}$

Proof. For $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ the Jacobian matrix reads

$$J(\tfrac{\mu+\gamma}{\beta}, \omega, x_0) = \begin{pmatrix} -\mu - \omega\beta & -(\mu+\gamma) & -\mu\varepsilon \\ \beta\omega & 0 & 0 \\ 0 & kx_0(1-x_0) & 0 \end{pmatrix}$$

with the characteristic polynomial

$$\chi_J(\lambda) = \lambda^3 + (\mu + \omega\beta)\lambda^2 + \beta\omega(\mu+\gamma)\lambda + \mu\varepsilon\beta\omega k x_0(1-x_0)$$

checking the Hurwitz Criteria (Thm 2.9) yields

- $a_1 = \mu + \omega\beta > 0$
- $a_3 = \mu\varepsilon\beta\omega k x_0(1-x_0) > 0$
- $a_1 a_2 > a_3 \Leftrightarrow \beta\omega(\mu+\omega\beta)(\mu+\gamma) > \mu\varepsilon\beta\omega k x_0(1-x_0)$

therefore, the condition $\beta > \frac{\mu}{\omega}(\frac{\varepsilon k x_0(1-x_0)}{\mu+\gamma} - 1)$ needs to hold for the stability of the endemic state $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ and the claim follows. □

3.1.3 Bifurcation

In this part, we determine possible bifurcations in our system (2). So, let us consider the stationary states with their stability conditions:

a) $\begin{cases} (1,0,0) & \text{is } \begin{cases} \text{stable} & \text{if } \beta < \mu+\gamma \\ \text{unstable} & \text{if } \beta > \mu+\gamma \end{cases} \\ (\frac{\mu+\gamma}{\beta}, \frac{\mu}{\mu+\gamma} - \frac{\mu}{\beta}, 0) & \text{is } \begin{cases} \text{stable} & \text{if } \beta \in (\mu+\gamma, \frac{\mu(\mu+\gamma)}{\mu-\omega(\mu+\gamma)}) \\ \text{unstable} & \text{else} \end{cases} \end{cases}$

b) $\begin{cases} (1-\varepsilon, 0, 1) & \text{is unstable} \\ (\frac{\mu+\gamma}{\beta}, \frac{\mu(1-\varepsilon)}{\mu+\gamma} - \frac{\mu}{\beta}, 1) & \text{is } \begin{cases} \text{stable} & \text{if } \beta > \frac{\mu(\mu+\gamma)}{\mu(1-\epsilon)-\omega(\mu+\gamma)} \\ \text{unstable} & \text{if } \beta < \frac{\mu(\mu+\gamma)}{\mu(1-\epsilon)-\omega(\mu+\gamma)} \end{cases} \end{cases}$

c) $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ is $\begin{cases} \text{stable} & \text{if } \beta > \frac{\mu}{\omega}(\frac{\varepsilon k x_0(1-x_0)}{\mu+\gamma} - 1),\ \beta \in (\frac{\mu(\mu+\gamma)}{\mu-(\mu+\gamma)\omega}, \frac{\mu(\mu+\gamma)}{\mu(1-\varepsilon)-(\mu+\gamma)\omega}) \\ \text{unstable} & \text{if } \beta < \frac{\mu}{\omega}(\frac{\varepsilon k x_0(1-x_0)}{\mu+\gamma} - 1),\ \beta \in (\frac{\mu(\mu+\gamma)}{\mu-(\mu+\gamma)\omega}, \frac{\mu(\mu+\gamma)}{\mu(1-\varepsilon)-(\mu+\gamma)\omega}) \end{cases}$

Corollary 3.8 *By comparing the stability conditions of the stationary states, we conclude by definition 2.10 that there is a transcritical bifurcation in case (a) and (b). The last one* $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ *acts as a transfer between them as illustrated below (Figure 4).*

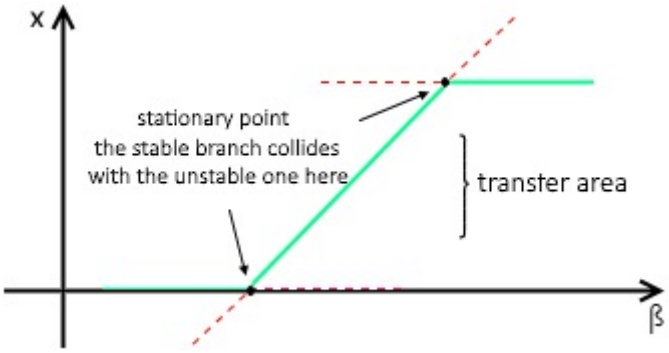


Figure 4: Illustration of the transfer in Corollary 3.8. The stable branch (green) meets the unstable one (red) at two stationary points, where they exchange their stability.

3.2 Time scale analysis

In fact, a population doesn't immediately vaccinate after a disease outbreak, although the numbers of susceptible and infected change drastically. This leads to three processes that act on two different time scales. For simplicity sake, assume that $\dot{x}$ is on the slow time scale $\hat{\tau}$ and that the other two are on the fast time scale $\widetilde{\tau}$. In the following, we study the resulting slow and fast system with the same notations (Subsection 3.1) to determine if oscillations occur in the long term.

3.2.1 Fast System

Let us begin with the fast system:

$$\begin{aligned} \dot{s} &= \mu(1-\varepsilon x) - \mu s - \beta s i \\ \dot{i} &= -\mu i - \gamma i + \beta s i \\ \dot{x} &= \widetilde{\delta} k x (1-x)(i-\omega) \end{aligned} \tag{4}$$

with the quasi steady state assumption $\widetilde{\delta} \to 0$, which is equivalent to $\dot{x}$ being constant. Then the system (4) reduces to

$$\begin{aligned} \dot{s} &= \mu(1-\varepsilon x) - \mu s - \beta s i \\ \dot{i} &= -\mu i - \gamma i + \beta s i \end{aligned} \tag{5}$$

Stationary states

Theorem 3.9 ***(disease-free steady state)***
The disease-free stationary state of our system (5) is given by $(\widetilde{s}_1, \widetilde{i}_1) = (1-\varepsilon x, 0)$

Proof. Analogously to the proofs from the previous Subsection 3.1.1. □

Theorem 3.10 ***(endemic steady state)***
The endemic stationary state of our system (5) is given by $(\widetilde{s}_2, \widetilde{i}_2) = (\frac{\mu+\gamma}{\beta}, \frac{\mu(1-\varepsilon x)}{\mu+\gamma} - \frac{\mu}{\beta})$

Proof. Analogously to the proofs from the previous Subsection 3.1.1. □

Stability analysis

For stability analysis, we need the general Jacobian matrix $\widetilde{J}$ of the fast system (5) given by:

$$\widetilde{J}(s,i) = \begin{pmatrix} -(\mu+\beta i) & -\beta s \\ \beta i & -(\mu+\gamma)+\beta s \end{pmatrix}$$

Here, we note that the Hartmann & Grobmann theorem (Thm 2.3) holds for our stationary states in the fast system, since $0 \notin \text{Re } \sigma(f'(\bar{x}))$ will always be fulfiled. This allows us to apply the stability criteria (Thm 2.8) afterward.

Theorem 3.11

The disease-free stationary state $(\widetilde{s}_1, \widetilde{i}_1)$ *is a* $\begin{cases} \textit{unstable saddle} & \textit{if } \beta > \frac{\mu+\gamma}{(1-\varepsilon x)} \\ \textit{stable node} & \textit{if } \beta < \frac{\mu+\gamma}{(1-\varepsilon x)} \end{cases}$

Proof. The Jacobian matrix of disease-free steady state $(\widetilde{s}_1, \widetilde{i}_1)$ reads

$$\widetilde{J}(\widetilde{s}_1, \widetilde{i}_1) = \begin{pmatrix} -\mu & -\beta(1-\varepsilon x) \\ 0 & -(\mu+\gamma)+\beta(1-\varepsilon x) \end{pmatrix}$$

with the following eigenvalues

- $\widetilde{\lambda}_1 = -\mu < 0$
- $\widetilde{\lambda}_2 = -(\mu+\gamma)+\beta(1-\varepsilon x) \begin{cases} > 0 & \text{if } \beta > \frac{\mu+\gamma}{(1-\varepsilon x)} \\ < 0 & \text{if } \beta < \frac{\mu+\gamma}{(1-\varepsilon x)} \end{cases}$

which yield the claim. □

Theorem 3.12

The endemic stationary state $(\widetilde{s}_2, \widetilde{i}_2)$ *is a* $\begin{cases} \textit{stable node} & \textit{if } \beta > \frac{\mu+\gamma}{(1-\varepsilon x)} \\ \textit{unstable saddle} & \textit{if } \beta < \frac{\mu+\gamma}{(1-\varepsilon x)} \end{cases}$

Proof. Analogously the Jacobian matrix of the endemic steady state $(\widetilde{s}_2, \widetilde{i}_2)$ reads

$$\widetilde{J}(\widetilde{s}_2, \widetilde{i}_2) = \begin{pmatrix} \frac{\beta\mu(1-\varepsilon x)}{\mu+\gamma} & -(\mu+\gamma) \\ \frac{\beta\mu(1-\varepsilon x)}{\mu+\gamma} - \mu & 0 \end{pmatrix}$$

with

- $tr(\widetilde{J}) = -\frac{\beta\mu(1-\varepsilon x}{\mu+\gamma} < 0$
- $det(\widetilde{J}) = (\mu+\gamma)(\frac{\beta\mu(1-\varepsilon x)}{\mu+\gamma} - \mu)$

applying the Trace-Determinant Criteria (Figure 3) yields our result. □

Periodic Orbits

Let us analyze the behaviour of this fast system. We separate two cases depending on the value of β. If $\beta > \frac{\mu+\gamma}{(1-\varepsilon x)}$, then the endemic steady state is stable and the one without infection is unstable. Therefore, the disease breaks out and we get the following statement:

Theorem 3.13

For $\beta > \frac{\mu+\gamma}{(1-\varepsilon x)}$*, the solutions of the fast system (5) tend to a stationary point on the slow manifold.*

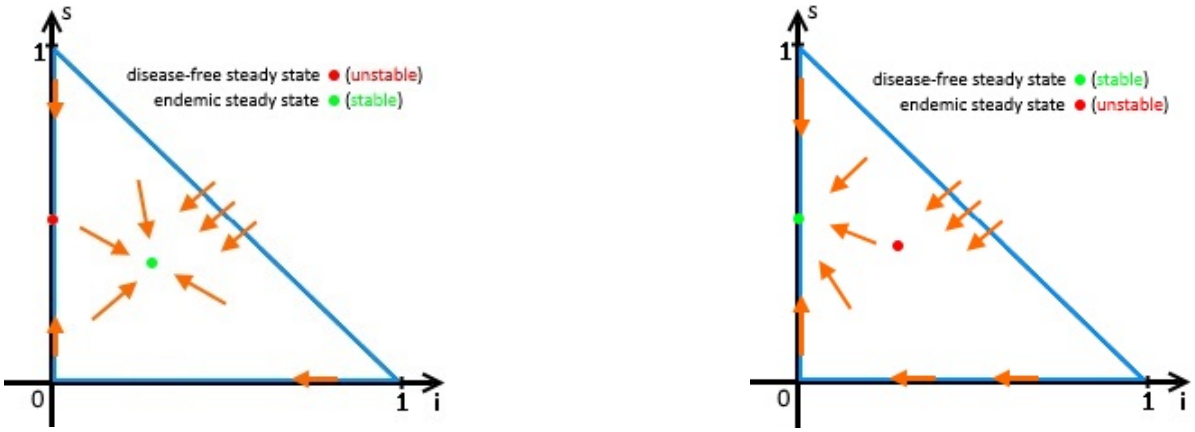
Proof. Assume that $\beta > \frac{\mu+\gamma}{(1-\varepsilon x)}$ and rescale the system (5) with $\frac{1}{i}$, which reads

$$\begin{aligned}\frac{\dot{s}}{i} &= \frac{\mu(1-\varepsilon x)}{i} - \frac{\mu s}{i} - \beta s \\ \frac{\dot{i}}{i} &= -\mu - \gamma + \beta s\end{aligned}$$

then $\frac{\partial}{\partial s}\frac{\dot{s}}{i} = -\frac{\mu}{i} - \beta < 0$ and $\frac{\partial}{\partial i}\frac{\dot{i}}{i} = 0$ holds and we conclude that $\frac{\partial}{\partial s}\frac{\dot{s}}{i} + \frac{\partial}{\partial i}\frac{\dot{i}}{i} < 0$. Then the Negative criterion of Bendixon (Thm 2.7) yields that the system doesn't have any periodic orbits or any heteroclinic and homoclinic cycles (5).
In the next step, we want to apply the Poincare Bendixion theorem (Thm 2.6). For $\beta > \frac{\mu+\gamma}{(1-\varepsilon x)}$, consider an invariant region $D = [(s,i) : s \geq 0, i \geq 0, s+i \leq 1]$. There, the disease-free steady state is unstable (Thm 3.11) and the endemic one is stable (Thm 3.12). This results in Figure 5 a) by considering

- $i = 0 \quad \Rightarrow \dot{s} = \mu(1-\varepsilon x - s) \begin{cases} < 0 \text{ if } s \rightsquigarrow 1 \\ > 0 \text{ if } s \rightsquigarrow 0 \end{cases}$
- $s = 0 \quad \Rightarrow \dot{i} = -i(\mu+\gamma) < 0$
- $s + i = 1 \quad \Rightarrow \dot{s} + \dot{i} = \mu(1-\varepsilon x) - \mu - \gamma i < 0$

applying the Poincare Bendixion theorem yields our claim. □



(a) Case $\beta > \frac{\mu+\gamma}{(1-\varepsilon x)}$ (b) Case $\beta < \frac{\mu+\gamma}{(1-\varepsilon x)}$

Figure 5: Illustration of the fast system's stability behaviour depending on β

For $\beta < \frac{\mu+\gamma}{(1-\varepsilon x)}$, the infection dies out and we obtain the following result analogously:

Corollary 3.14 *For $\beta < \frac{\mu+\gamma}{(1-\varepsilon x)}$, the solutions of the fast system tend to the stable disease-free steady state in the long term as illustrated in Figure 5 b).*

3.2.2 Slow System

Let us turn to the slow system now:

$$\begin{aligned} \hat{\varepsilon}\dot{s} &= \mu(1-\varepsilon x) - \mu s - \beta s i \\ \hat{\varepsilon}\dot{i} &= -\mu i - \gamma i + \beta s i \\ \dot{x} &= \widetilde{\delta} k x (1-x)(i-\omega) \end{aligned} \tag{6}$$

with the quasi steady state assumption $\hat{\varepsilon} \to 0$, such that

$$\begin{cases} i = \frac{\mu(1-\varepsilon x)}{\mu+\gamma} - \frac{\mu}{\beta} \coloneqq \phi(x) \\ s = \frac{\mu+\gamma}{\beta} \end{cases}$$

then the slow system (6) reduces to

$$\dot{x} = kx(1-x)(\frac{\mu}{\mu+\gamma} - \frac{\mu}{\beta} - \omega - \frac{\mu\varepsilon}{\mu+\gamma}x)$$

Our goal is to analyze long term behaviour of the slow system. Therefore, we use the same approach as stated in the fast system to distinguish between the two cases that depend on the value of β. If $\beta < \frac{\mu+\gamma}{(1-\varepsilon x)}$, then the disease-free steady state is stable. Furthermore $(i-\omega) < 0$ and $\dot{x} < 0$, which implies a continuous decrease of x. Hence, we conclude:

Corollary 3.15 *For $\beta < \frac{\mu+\gamma}{(1-\varepsilon x)}$, the slow systems tend to the stationary point $\bar{x} = 0$.*

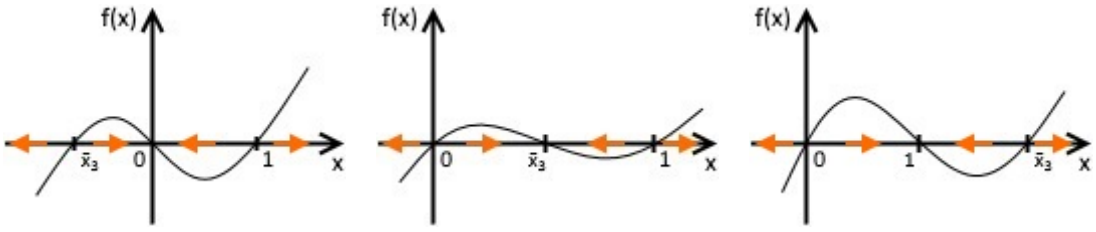
Now we switch to the case $\beta > \frac{\mu+\gamma}{(1-\varepsilon x)}$, where the endemic state is stable. Then, further simplifications of the above equation with $a \coloneqq \frac{\mu}{\mu+\gamma} - \frac{\mu}{\beta} - \omega$ and $b \coloneqq \frac{\mu\varepsilon}{\mu+\gamma}$ leads to the following statement:

Proposition 3.16 *The simplified slow system*

$$\dot{x} = bkx(1-x)(\frac{a}{b} - x) \coloneqq f(x) \tag{7}$$

has the stationary states $\bar{x}_1 = 0$, $\bar{x}_2 = 1$, $\bar{x}_3 = \frac{a}{b}$ with their stability depending on the value of $\frac{a}{b}$.

In the following, we introduce three generic cases for the values of $\frac{a}{b}$ as illustrated in Figure 6. The corresponding stability diagrams result from the signs of $\dot{x} = f(\hat{x})$ with $\hat{x} \in \mathbb{R}\backslash\{\bar{x}_1, \bar{x}_2, \bar{x}_3\}$. A negative sign implies that the value of x decreases and a positive one that it increases, which are indicated by arrows. In conclusion, the stationary points are locally asymptotically stable (Def. 2.1) if we can't leave them by small perturbations, else unstable.



(a) Case $\bar{x}_3 = \frac{a}{b} < 0$ (b) Case $\bar{x}_3 = \frac{a}{b} \in (0,1)$ (c) Case $\bar{x}_3 = \frac{a}{b} > 1$

Figure 6: Illustrations of the stability chart for different values of $\bar{x}_3$

However, we only consider the biologically relevant case 6b since x $\in$ [0,1] by our assumption. The illustration 6b shows that the steady state $\bar{x}_3$ is stable and $\bar{x}_1$ and $\bar{x}_2$ are unstable. Next, we analyze the long term behaviour of this system by defining an invariant region with

$$\dot{x} = kx(1-x)(\phi(x)-\omega) \coloneqq g(\phi(x), x)$$

where $\phi(x) \coloneqq i$, then the invariant region (Figure 7) is given by the observations:

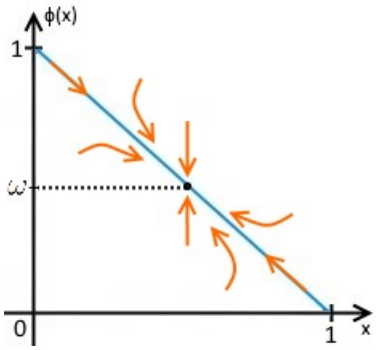


Figure 7: Illustration of invariant region for $\beta > \frac{\mu+\gamma}{(1-\varepsilon x)}$

- if $\phi(x) < \omega$, then $\dot{x} = g(\phi(x), x) < 0 \Rightarrow x$ is decreasing
- if $\phi(x) > \omega$, then $\dot{x} = g(\phi(x), x) > 0 \Rightarrow x$ is increasing

which leads to the following conclusion:

Corollary 3.17 *The long term dynamics on the slow manifold is given by the slow system, which tends to the stationary point* $\bar{x} = \frac{1}{\varepsilon}(1 - \frac{\mu+\gamma}{\beta} - \frac{\mu+\gamma}{\mu}\omega)$ *for* $\beta > \frac{\mu+\gamma}{(1-\varepsilon x)}$.

4 The behaviour incidence model with delay

4.1 Model description

We turn to the modified delay model (8) with the same delay δ and notations (Subsection 3.1) from the paper of Bauch et al. [1]. The delay represents the time an individual requires for a decision about vaccination, which is caused by vaccine hesitancy. Our goal is to find a possible Hopf point in this system. We start with the determination of the stationary states first, followed by their stability analysis. The delay model reads:

$$\begin{aligned} \dot{s} &= \mu(1-\varepsilon x) - \mu s - \beta s i \\ \dot{i} &= -\mu i - \gamma i + \beta s i \\ \dot{x} &= kx(1-x)(i(t-\delta) - \omega) \end{aligned} \tag{8}$$

4.2 Steady states

The stationary states of our behaviour incidence model with delay (8) are identical to those from the one without delay (2) by the following assumptions:

- $\lim\limits_{t\to\infty} s(t) = \lim\limits_{t\to\infty} s(t-\delta) = \hat{s}$
- $\lim\limits_{t\to\infty} i(t) = \lim\limits_{t\to\infty} i(t-\delta) = \hat{i}$
- $\lim\limits_{t\to\infty} x(t) = \lim\limits_{t\to\infty} x(t-\delta) = \hat{x}$

Theorem 4.1
The disease-free stationary states of the delay system (8) are:

- $(1, 0, 0)$
- $(1-\varepsilon x, 0, 1)$

Proof. Analogously to the proof of the system without delay (2). □

Theorem 4.2
The endemic stationary states of the delay system (8) are:

- $(\frac{\mu+\gamma}{\beta}, \frac{\mu}{\mu+\gamma} - \frac{\mu}{\beta}, 0)$
- $(\frac{\mu+\gamma}{\beta}, \frac{\mu(1-\varepsilon)}{\mu+\gamma} - \frac{\mu}{\beta}, 1)$
- $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ *where* $x_0 = \frac{1}{\varepsilon}(1 - \frac{\mu+\gamma}{\beta} - \frac{\mu+\gamma}{\mu}\omega)$ *with* $\beta \in (\frac{\mu(\mu+\gamma)}{\mu-(\mu+\gamma)\omega}, \frac{\mu(\mu+\gamma)}{\mu(1-\varepsilon)-(\mu+\gamma)\omega})$

Proof. Analogously to the proof of the system without delay (2). □

4.3 Stability analysis and model behaviour

Here, we consider the stability of the stationary states. The general Jacobian matrix J is given by:

$$J = J_1 + e^{-\delta} J_2$$

with

$$J_1(s,i,x) = \begin{pmatrix} -\mu-\beta i & -\beta s & -\mu\varepsilon \\ \beta i & -\mu-\gamma+\beta s & 0 \\ 0 & 0 & k(1-2x)(i(t-\delta)-\omega) \end{pmatrix}$$

$$J_2(s(t-\delta), i(t-\delta), x(t-\delta)) = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & k(1-2x) & 0 \end{pmatrix}$$

hence inserting the stationary states $(\hat{s}, \hat{i}, \hat{x})$ yields

$$J(\hat{s}, \hat{i}, \hat{x}) = \begin{pmatrix} -\mu-\beta\hat{i} & -\beta\hat{s} & -\mu\varepsilon \\ \beta\hat{i} & -\mu-\gamma+\beta\hat{s} & 0 \\ 0 & e^{-\delta}k\hat{x}(1-\hat{x}) & k(1-2\hat{x})(\hat{i}-\omega) \end{pmatrix} \tag{9}$$

Theorem 4.3
The stability conditions of the stationary states of the delay model (8) are unaffected by the delay, except for $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$.

Proof. We observe that the difference between the Jacobian matrix (9) of the delay model and the one without delay (3) is the term $e^{-\delta}k\hat{x}(1-\hat{x})$. It is is equal to 0 if $\hat{x}$ takes the value 0 or 1. In conclusion, the stability conditions remain the same for all stationary states except $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$. □

In the following, we want to find out if $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ is a Hopf point by considering its stability:

Theorem 4.4
For large or small values of k, there exists a Hopf point in the system. Furthermore, the spectrum is on the negative half plane with $\delta \in [0, \delta_0)$ *if there is a delay* $\delta_0 \in [\frac{\pi}{2\sqrt{\chi}}, \frac{\pi}{\sqrt{\chi}}]$ *i.e* $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ *is locally stable for* $\delta < \delta_0$ *and unstable for* $\delta > \delta_0$.

Remark 4.5 *We expect that there is a Hopf bifurcation such that the system (8) oscillates.*

Proof. The Jacobian matrix at $(\frac{\mu+\gamma}{\beta}, \omega, x_0)$ reads

$$J((\frac{\mu+\gamma}{\beta}, \omega, x_0) = \begin{pmatrix} -\mu-\omega\beta & -(\mu+\gamma) & -\mu\varepsilon \\ \beta\omega & 0 & 0 \\ 0 & e^{-\delta}kx_0(1-x_0) & 0 \end{pmatrix}$$

with the characteristic polynomial

$$\chi_J(\lambda) = \lambda^3 + (\mu+\omega\beta)\lambda^2 + \beta\omega(\mu+\gamma)\lambda + \mu\varepsilon\beta\omega kx_0(1-x_0)e^{-\delta}$$

Set $a = (\mu+\omega\beta), b = \beta\omega(\mu+\gamma), c = \mu\beta\varepsilon kx_0(1-x_0)$, then the simplification reads

$$\chi_J(\lambda) = \lambda^3 + a\lambda^2 + b\lambda + ce^{-\delta} \tag{10}$$

Using the ansatz $\lambda = 0 + i\hat{\omega}$ (for more details, see [9]) on the charactristic polynomial (10) yields

$$-i\hat{\omega}^3 - a\hat{\omega}^2 + ib\hat{\omega} + ce^{-i\hat{\omega}\delta} := 0$$

resulting in the following real and imaginary part

$$\begin{cases} cos(\hat{\omega}\delta) = \frac{a}{c}\hat{\omega}^2 \\ sin(\hat{\omega}\delta) = \frac{-\hat{\omega}^3+\hat{\omega}b}{c} \end{cases}$$

now apply $sin(\hat{\omega}\delta) = \pm\sqrt{1-\cos(\hat{\omega}\delta)^2}$ to the pair of equations above to obtain

$$\pm\sqrt{1-(\tfrac{a}{c})^2\hat{\omega}^4} = \tfrac{-\hat{\omega}^3+\hat{\omega}b}{c}$$

which exists if and only if $1 > (\frac{a}{c})^2\hat{\omega}^4$ holds since $\hat{\omega} \in \mathbb{R}$, thus by further calculations

$$\hat{\omega}^6 - (2b-a^2)\hat{\omega}^4 + b^2\hat{\omega}^2 - c^2 = 0 \tag{11}$$

The substitution with $\chi = \hat{\omega}^2$ yields

$$\chi^3 - (2b-a^2)\chi^2 + b^2\chi - c^2 = 0 \coloneqq g(\chi) \tag{12}$$

where $1 > \frac{a}{c}\chi^2 \Leftrightarrow 1 > \frac{a}{c}\chi$. Then set $\eta \coloneqq \frac{a}{c}\chi$ and (12) simplifies to

$$\frac{c}{a}^3\eta^3 - (2b-a^2)\frac{c}{a}^2\eta^2 + b^2\frac{c}{a}\eta - c^2 = 0$$

with $1 > \eta$, which is equivalent to

$$\eta^3 - (2b-a^2)\frac{a}{c}\eta^2 + \frac{(ba)^2}{c^2}\eta - \frac{a^3}{c} = 0 \coloneqq f(\eta)$$

Checking the boudaries $\eta = 1$ and $\eta = 0$ gives

$$\begin{cases} f(0) = -\frac{a^3}{c} < 0 \\ f(1) = 1 - \frac{2ba}{c} - \frac{(ba)^2}{c^2} \end{cases}$$

therefore, we conclude that

- $f(0) \longrightarrow 0$ if c or equivalently k is large
- $f(1) \longrightarrow 0$ if k is small

Hence, we can find a solution $\bar{\chi} = \frac{c}{a}\bar{\eta}$ with $g(\chi) = 0$ such that $\bar{\omega} = \pm\sqrt{\bar{\chi}}$ exists, which proofs the existance of a Hopf point.
Inserting $\bar{\omega} = \pm\sqrt{\bar{\chi}}$ into $cos(\hat{\omega}\delta) = \frac{a}{c}\hat{\omega}^2$ yields

$$cos(\pm\sqrt{\bar{\chi}}) = cos(\sqrt{\bar{\chi}}) = \frac{a}{c}\bar{\chi}$$

Set $h(\delta) = \cos(\sqrt{\bar{\chi}}\delta) - \frac{a}{c}\bar{\chi}$, then for $\delta = \frac{\pi}{2\sqrt{\bar{\chi}}}$ and $\delta = \frac{\pi}{\sqrt{\bar{\chi}}}$:

$$h(\frac{\pi}{2\sqrt{\bar{\chi}}}) = cos(\frac{\pi}{2}) - \frac{a}{c}\bar{\chi} = -\frac{a}{c}\bar{\chi} < 0 < 1 - \frac{a}{c}\bar{\chi} = h(\frac{\pi}{\sqrt{\bar{\chi}}})$$

So for $\delta \in [0, \delta_0)$ with $\delta_0 \in [\frac{\pi}{2\sqrt{\bar{\chi}}}, \frac{\pi}{\sqrt{\bar{\chi}}}]$, we conclude that the spectrum is on the negative half plane i.e. the solution is locally stable. If $\delta > \delta_0$, the solution is unstable. □

5 Improvement of the SIR behaviour incidence model

In reality, the stationary points 0 and 1 of the equation $\dot{x}$ don't exist since there will always be vaccination supporters and enemies preventing these values to happen. In addition to that, the fixed delay is unsuitable to represent the hesitancy. Consequently, this equation doesn't fit the vaccination dynamics and we aim to improve it.

So, let us introduce the general concept of the improvement, which we specify for our model afterwards. The total population size N splits into 2 groups: those who have taken the immunization and those who haven't yet. Both group sizes depend on the quantity of infected I. For simplicity's sake, let $\hat{x}_+$ denote the group with immunization and $\hat{x}_-$ the other one with the following dynamics based in the properties of Hill functions:

- $\dot{\hat{x}}_+ = -\frac{\hat{a}\hat{b}^n}{\hat{b}^n+I^n}\hat{x}_+ + \frac{\hat{a}I^n}{\hat{b}^n+I^n}\hat{x}_-$
- $\dot{\hat{x}}_- = -\frac{\hat{a}i^n}{\hat{b}^n+I^n}\hat{x}_- + \frac{\hat{a}\hat{b}^n}{\hat{b}^n+I^n}\hat{x}_+$

where $\hat{a}$ is the maximal influence of $\hat{x}_+$ or $\hat{x}_-$ on the immunization rate and $\hat{b}$ is the half maximal immunization rate. If $\dot{\hat{x}}_+ > 0$ then $\hat{x}_+$ is growing, which implies that people prefer vaccinations and vice versa. The propotion of vaccinators in the population is:

$$\hat{x} = \frac{\hat{x}_+}{\hat{x}_+ + \hat{x}_-} \tag{13}$$

with $\hat{x} \in [0,1]$, where $\hat{N} = \hat{x}_+ + \hat{x}_-$.

Theorem 5.1
The dynamics of vaccinators in the population is given by

$$\dot{\hat{x}} = \hat{a}(\frac{I^n}{I^n + \hat{b}^n}) - \hat{a}\hat{x} \tag{14}$$

with $x \in [0,1]$, where I denotes the number of infected in the population.

Proof. Applying the quotient rule on $\hat{x}$ yields

$$\dot{\hat{x}} = \frac{\dot{\hat{x}}_+}{(\hat{x}_+ + \hat{x}_-)} - \frac{\hat{x}_+(\dot{\hat{x}}_+ + \dot{\hat{x}}_-)}{(\hat{x}_+ + \hat{x}_-)^2}$$

where $(\dot{\hat{x}}_+ + \dot{\hat{x}}_-) = 0$, then further simplification leads to

$$\dot{\hat{x}} = \frac{\dot{x}_+}{\hat{x}_+ + \hat{x}_-}$$

inserting $\dot{\hat{x}}_+$ with $\hat{x}_- = \hat{N} - \hat{x}_+$ yields

$$\dot{\hat{x}} = -\frac{\hat{a}\hat{b}^n\hat{x}_+}{(\hat{b}^n + I^n)(\hat{x}_+ + \hat{x}_-)} + \frac{\hat{a}I^n(\hat{N} - \hat{x}_+)}{(\hat{b}^n + I^n)(\hat{x}_+ + \hat{x}_-)}$$

using the equation (13) with further simplifications yield the claim. □

In our model, we work with relative numbers. Therefore, we arrive with some adjustments at the following statement:

Corollary 5.2 *The improved vaccination dynamics is given by*

$$\dot{x} = a(\frac{i^n}{i^n + b^n}) - ax \tag{15}$$

with $x \in [0,1]$, where i denotes the relative amount of infected in the population.

Further improvements of the new dynamics in (15) are possibile. In the following, we present one of them by introducing the modified SIR-model with the same notations (Subsection 3.1):

$$\begin{aligned} \dot{s} &= \mu(1 - \varepsilon x) - \mu s - \beta s i \\ \dot{i} &= -\mu i - \gamma i + \beta s i \\ \dot{x} &= F(i) - dx + m \end{aligned} \tag{16}$$

with $x \in [0,1]$ and $F(i) = \tilde{a}(\frac{i^n}{\tilde{b}^n + i^n})$, where $\tilde{a}$ is the maximal influence of i on the immunization rate, $\tilde{b}$ is the half maximal immunization rate, m is the basic immunization rate and d is the vaccine risk.

5.1 Bifurcation analysis

We want to find out if there is a Hopf point in the improved model (16), which is necessary for oscillating behaviours. Hence, we introduce the following result:

Theorem 5.3
For large values of $\dot{F}(i^*)$, there is a Hopf point in the system (16).

Remark 5.4 *We expect that there is a Hopf bifurcation in the system (16) such that oscillations occur.*

Proof. Our goal is to prove the existence of the Hopf point with Liu's theorem (2.12). So, let us begin with the simplification of the Jacobian Matrix J from the system (16).

$$J(s^*, i^*, x^*) = \begin{pmatrix} -\mu - \beta i^* & -\beta s^* & -\mu\varepsilon \\ \beta i^* & -\mu - \gamma + \beta s^* & 0 \\ 0 & \dot{F}(i^*) & -d \end{pmatrix}$$

which is

$$J(s^*, i^*, x^*) = \begin{pmatrix} -g & -b & -c \\ a & -f + b & 0 \\ 0 & \dot{F}(i^*) & -d \end{pmatrix}$$

where $f = \mu + \gamma$, $g = \mu + \beta i^*$, $a = \beta i^*$, $b = \beta s^*$, $c = \mu\varepsilon$, with the characteristic polynomial

$$\chi_J(\lambda) = \lambda^3 + (f - b + g + d)\lambda^2 + (\mu(f - b) + af + d(f - b + g))\lambda + d(\mu(f - b) + af) + ac\dot{F}(i^*)$$

Checking the Hurwitz Criteria (Thm 2.9) for $n = 3$ yields

- $a_1 = f + g + d - b > 0$ if $f + g + d > b$
- $a_2 = (\mu(f - b) + af + d(f - b + g)) > 0$ if $\frac{f(\mu+a+d)+dg}{\mu+d} > b$
- $a_1 a_2 > a_3 \Leftrightarrow (f + g - b)(\mu(f - b) + af + d(f - b + g)) + d^2(f + g - b) > ac\dot{F}(i^*)$

We conclude that the last inequality holds if $\dot{F}(i^*)$ is small, then the eigenvalues have negative real parts by Hurwitz Criteria (Thm 2.9) and the corresponding stationary states are stable. For large values of $\dot{F}(i^*)$, $a_1 a_2 < a_3$ holds and there exists an $\dot{F}(\hat{i}^*)$ such that $a_1 a_2 = a_3$. For every given $F(i^*)$ there is a corresponding stationary point (s^*, i^*, x^*). Now we define a family of functions with parameter α by $F_\alpha(i)$, such that

1. $F_\alpha(i^*) = F(i^*)\ \forall\, \alpha$
2. $F_\alpha(i)|_{\alpha=0} = F(i)$
3. $\dot{F}_\alpha(i) = h(\alpha)$ with $h(\alpha) \to \infty$ as $\alpha \to \infty$

where (s^*, i^*, x^*) is a stationary point for all parameter values of α; which corresponds to a smooth curve. By checking the two conditions of Liu's theorem (Thm 2.12) for $n = 3$, we know that (i) holds by the previous result above. The second condition (ii) also holds since

$$\frac{d}{d\alpha}|H_2(\alpha^*)| = \frac{d}{d\alpha} det(H_2(\alpha^*)) = \frac{d}{d\alpha}(a_1 a_2 - F_{\alpha^*}(i)) = -F_{\alpha^*}(i) \neq 0$$

Applying Liu's theorem (Thm 2.12) yields the claim. □

5.2 Disucssion

The importance of immunization has grown in significance during recent years. Researches in this field aim for a better understanding of its dynamics in society, where vaccine hesitancy plays a major role. This hesitancy can lead to low vaccination rates, which are responsible for severe disease outbreaks, such as measles in the UK. However, difficulties arise when implementing and analyzing vaccination models with hesitancy as a factor due to its complexity. Additionally, the modelling of the social processes regarding vaccine hesitancy is challenging since the individual's behaviour depends on many unpredictable aspects such as emotions, experiences, personality, etc. Therefore, single effects and determinants of the scepticism in traditional models are usually insufficient to describe the hesitancy problems in a general context. Nevertheless, these models are essential for a better understanding of the vaccine behaviours with its dynamics in populations.

This work has considered and improved one of these modelling approaches by the analysis of the vaccination dynamics in Bauch's behaviour incidence models [1], where hesitancy is explained by social learning. These specific Bauch's incidence models use the following properties:

Firstly, the long term behaviour of the delay-free system can switch between stationary states with and without vaccinators. They exchange the stability at the corresponding transcritical bifurcation point. Secondly, the time scale analysis shows that the slow system determines the model dynamics, which converges to a stationary point. Therefore, the system does not oscillate. Lastly, oscillations may happen in the behaviour incidence model with the delay by the existence of the Hopf point. However, the generic conditions have yet to be proven for the occurrence of Hopf bifurcations.

The improvement of Bauch's model has been achieved by rebuilding the immunization behaviour based on the dynamics of vaccinated and unvaccinated individuals with the properties of Hill functions and vaccine determinants. These determinants are factors such as communication, social norms and public trust, etc. that cause vaccine hesitancy, as described in the paper of Noni E. MacDonald et al. [7]. A Hopf point exists in the new system under certain situations, where the occurrence of Hopf bifurcation has yet to be shown by the generic conditions. This improvement shares the same limitations as Bauch's models [1], which is caused by factors like modelled incidence data, stochastically driven dynamics, etc. However, the modification is crucial since realistically immunizations supporters and enemies prevent the proportion of vaccinators falling to 0 or rising to 1. In conclusion, the behaviour incidence model with delay and its improved version showed evidence for oscillations by the existence of Hopf points. This might provide a possible explanation for the return of preventable diseases.

Similar adjustments of Bauch's model that focus on the behaviour of individuals have been implemented in the new model of Li et al. [6]. This system is also based on separating the dynamics of vaccinator and non-vaccinators. In contrast to traditional models, the new one includes the effects of social networks on the individual's learning behaviour. Difficulties arise in this area when assigning influence levels for members in socials network. In the context of vaccination, a possible solution allows individuals to compare their strategy payoff with the weighted average gain from all participants with a chosen vaccine strategy. Other articles concerning the dynamics of vaccination focus on single components of hesitancy or interactions between multiple ones.

Further researches may implement the oscillating dynamics of this work into vaccination models to explain the return of certain diseases. Additionally, one can adopt it for models that consider the coexistence of vaccinators and non-vaccinators in populations by bistable behaviour. The oscillations would allow a dynamic switch between them since low immunization rates cause disease outbreaks that make immunizations attractive again. Lastly, this oscillation dynamic can be used to describe hesitancy components like social media, which has a strong influence if the infection is high and vice versa.

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