HELSINGIN YLIOPISTO — HELSINGFORS UNIVERSITET — UNIVERSITY OF HELSINKI

Tiedekunta/Osasto — Fakultet/Sektion — Faculty	Laitos — Institution — Department
Matemaattis-luonnontieteellinen	Matematiikan ja tilastotieteen laitos

Tekijä — Författare — Author
Patrick Blom

Työn nimi — Arbetets titel — Title

Spread of Disease Awareness During an Epidemic

Oppiaine — Läroämne — Subject		
Soveltava matematiikka		
Työn laji — Arbetets art — Level	Aika — Datum — Month and year	Sivumäärä — Sidoantal — Number of pages
Pro gradu -tutkielma	11/2020	44

Tiivistelmä — Referat — Abstract

Epidemian aikana harvat elävät kuten aikaisemmin ja sen sijaan muuttavat käytöstään uuden tilanteen valossa. Yleensä käytöksen muutos ymmärretään ennaltaehkäisevinä tapoina pienentää tartuntakontakin mahdollisuutta taudille alttiiden ja jo tartunnan saaneiden välillä. Tämä tutkielma pohtii toisenlaista tilannetta, jossa alttiit, tullessaan tietoisiksi taudista, alkavat harkitsemaan rokotusta tautia vastaan.

Perinteistä SIR-mallia muunnellaan siten, että siinä otetaan huomioon tietoisuuden leviäminen epidemiasta tai sen mahdollisuudesta, joka sitten innostaa yksilöitä hakemaan rokotusta. Tietoisuus leviää joko tasaisesti populaation ulkopuolisesta lähteestä tai populaation sisällä tapahtuvista kontakteista tiedostamattomien ja joko tartunnan saaneiden tai taudin jo tiedostavien välillä.

Näiden malleille lasketaan tasapainopisteet sekä ehdot niiden stabiilisuudelle. Kaikki kolme mallia pienentävät huomattavasti epidemian lopullista kokoa. Jos tietoisuuden leviäminen riippuu tartunnan saaneen populaation koosta, joissain tapauksissa rokotukset voivat epästabilisoida endeemisen tasapainopisteen ja luoda oskillaatioita. Ulkopuolisesta lähteestä sekä muista tietoisista riippuvalla mallilla on myös tautivapaat tasapainopisteet, jotka voivat olla stabiileja ja jotka tällöin voivat estää laajemman epidemian syntymisen, jos tietoisuuden leviäminen on tarpeeksi voimakasta.

Ulkopuolista lähteestä ja tietoista riippuvista tietoisuusmalleista luodaan myös vaihtoehtomallit, joissa yksilöt ovat kontaktissa rajattuun määrään muita yksilöitä, ja määritellään näiden tasapainopisteet. Myös tässä tapauksessa näiden kahden mallin tautivapaat tasapainopisteet voidaan stabilisoida tehokkaalla tietoisuuden leviämisellä.

Avainsanat — Nyckelord — Keywords
Biomatematiikka, epidemiologia, SIR-malli

Säilytyspaikka — Förvaringsställe — Where deposited
E-Thesis / Helda

Muita tietoja — Övriga uppgifter — Additional information

HELSINGIN YLIOPISTO — HELSINGFORS UNIVERSITET — UNIVERSITY OF HELSINKI

Tiedekunta/Osasto — Fakultet/Sektion — Faculty	Laitos — Institution — Department
Faculty of Science	Department of Mathematics and Statistics

Tekijä — Författare — Author
Patrick Blom

Työn nimi — Arbetets titel — Title

Spread of Disease Awareness During an Epidemic

Oppiaine — Läroämne — Subject
Applied Mathematics

Työn laji — Arbetets art — Level	Aika — Datum — Month and year	Sivumäärä — Sidoantal — Number of pages
Master's Thesis	11/2020	44

Tiivistelmä — Referat — Abstract

In times of epidemics, few people continue living as usual and instead often change their behaviour in light of the new situation. Usually the change in behaviour is taken to be preventive measures aimed the reduce the probability of infectious contact between susceptibles and infected. This thesis considers a different scenario, where susceptibles, once gaining awareness of the disease, begin to consider vaccination against the disease.

The basic SIR-model is modified to include awareness of the epidemic or the possibility thereof, that inspires individuals to vaccinate themselves. The awareness is spread either from a constant source outside the population or through contacts between unaware individuals and either the infected or individuals already aware of the disease.

The equilibria of these models and the conditions for their stability are established. All three models significantly reductions to the final size of the epidemic. In case of awareness spread being dependent on the size of the infected population, under some conditions the introducing vaccinations can destabilize the endemic equilibrium and lead to oscillations. Constant and aware-dependent awareness models also have disease-free equilibria, which can be stable and prevent a major epidemic from happening if the spread of awareness is strong enough.

Lattice-model analogues of the constant and aware-dependent awareness models, where individuals are connected to a limited number of other individuals, are also established along with their equilibria. Also in this case, the disease-free equilibria of the two models can be stabilised with an effective spread of awareness.

Avainsanat — Nyckelord — Keywords
Biomathematics, epidemiology, SIR model

Säilytyspaikka — Förvaringsställe — Where deposited
E-Thesis / Helda

Muita tietoja — Övriga uppgifter — Additional information

University of Helsinki
Faculty of Science
Department of Mathematics and Statistics

Master's Thesis

Spread of Disease Awareness During an Epidemic

Patrick Blom

Applied Mathematics - Biomathematics

Instructor: Eva Kisdi

November 14, 2020

I would like to express my gratitude to my instructor Eva Kisdi, as well as my parents and friends, who have given me guidance, support, and who have shown incredible patience during the writing of this thesis.

Contents

1 Introduction

Information has never been as accessible in human society as now, when the rise of social media has created a new 'horizontal' axis of information among peers next to the older, 'vertical' axis based on traditional mass media. The role of information is critical in epidemics, when people adjust their behaviour based on the perceived threat the disease imposes on the individual. This behaviour change is usually something aimed to reduce the rate of infection, such as limiting contacts with others or improving hygiene. This kind of behaviour change has been studied before, for example in [1], [2] and [3]. The awareness to the epidemic can also create a demand for a vaccination, if such is available. This work will consider how to model the effect of disease awareness resulting in a willingness to vaccinate.

Chapter 2 is a short introduction to basis of our analysis, the classic SIR-model with demographic changes included in it. The SIR model of epidemics, first presented by Kermack and McKendrick in 1927, is one of the basic compartmental models of epidemiology. Describing diseases that spread through human-to-human contact and against which the individual gains a lifelong immunity after recovering, the simplicity of the SIR model allows for easy analysis. We'll go through the basic terms of the model and conclusions, such as the basic reproduction number R_0 and the existence and stability of a endemic equilibrium. We also consider the final size of a rapid epidemic.

In chapter 3 we modify the SIR-model to introduce three possible sources of awareness: sources outside the population such as mass media, infected individual, and other individuals already aware of the epidemic and the vaccine. We then study the effects awareness will have on disease spread, especially on the final size of the epidemic. We also look at pre-epidemic spread of awareness and how strong it should be to prevent an outbreak of a major epidemic. We finish the chapter with simplifying the model by reducing the time becoming epidemic aware and becoming vaccinated to zero, which gives us more explicit results.

Another assumption the SIR-model makes is that the population is well-mixed and that an infected individual poses an equal threat to all susceptibles. In chapter 4 we switch from the global model to a more localized lattice model, where each individual is in contact with only a limited number of other individuals. After introducing the disease model and the method of pair approximation needed to close the system, we modify that model to incorporate vaccination/awareness. We conclude by studying the stability of the disease-free equilibrium and see that with a high enough rate of vaccination awareness, the outbreak of a major epidemic can be prevented.

2 SIR-model

The population is divided into three groups; the susceptible S, the infected I and the recovered R. The sizes of the populations S, I and R are required to be positive in order for them to be biologically admissible in the situation being modelled. The infected spread the non-lethal disease through contact with susceptible at a rate β and recover at a rate α, gaining permanent immunity from further infections. It is also assumed that the timescale of the epidemic is long enough to justify the inclusion of population dynamics in the model. The size of the population $N = S + I + R$ is fixed, with births happening at a rate μ among the entire population and balancing the deaths. Unless mentioned otherwise, we'll assume from here on that these parameters are strictly positive. The dynamics of this model are described by differential equations

$$\dot{S} = \mu N - \beta SI - \mu S \tag{1a}$$

$$\dot{I} = \beta SI - \alpha I - \mu I \tag{1b}$$

$$\dot{R} = \alpha I - \mu R. \tag{1c}$$

However, since the recovered population R does not affect the dynamics of the two other groups in any way, we can reduce the system to the first two equations (1a)-(1b).

2.1 Basic Reproduction Number R_0

It is not certain that introducing an infected individual into a susceptible population creates a major epidemic. The key to determining the possibility of a major outbreak is the *basic reproduction number* R_0, the expected number of secondary cases per primary case in a 'virgin' population. [4] In case of the SIR-model, the probability $P(t)$ that an infected individual is still infectious after time t since catching the disease is exponentially distributed, with

$$\dot{P}(t) = -(\alpha + \mu) P(t).$$

The expected time of the infectious period, before either recovery or death, is therefore $(\alpha + \mu)^{-1}$. Since the number of infectious contacts the infectious individual has per unit of time in a wholly susceptible population is βN, the basic reproduction number is

$$R_0 = \frac{\beta N}{\alpha + \mu}. \tag{2}$$

The initial growth rate of the epidemic in an entirely susceptible population

$$\dot{I}(0) = (\beta N - \alpha - \mu) I(0)$$

needs to be positive in order for a major outbreak to happen. Since $I(0) > 0$, this can happen only when R_0 is more than 1.

2.2 Equilibria

At an equilibrium $\left(\bar{S}, \bar{I}\right)$, the differential equations (1a)-(1b) are all zero and the system is static. The system has a trivial equilibrium $(N, 0)$, which is of course the population unaffected by any epidemic. In case of an *endemic equilibrium*, where $\bar{I} > 0$, equation (1b) can only be zero if

$$\bar{S} = \frac{\alpha + \mu}{\beta} = \frac{N}{R_0}. \tag{3}$$

Inserting this to the equation (1a) and solving $\dot{S} = 0$ for $\bar{I}$ gives us

$$\bar{I} = \frac{\mu}{\beta}\left(R_0 - 1\right). \tag{4}$$

Since all the parameters are strictly positive, an endemic equilibrium can thus exist only when the basic reproduction number R_0 is greater than one.

2.3 Stability of the Equilibrium

The Jacobian matrix for the system (1a)-(1b) at the equilibrium $\left(\bar{S}, \bar{I}\right)$ is

$$A = \begin{bmatrix} -\left(\beta\bar{I} + \mu\right) & -\beta\bar{S} \\ \beta\bar{I} & \beta\bar{S} - (\alpha + \mu) \end{bmatrix}. \tag{5}$$

For the non-trivial equilibrium calculated above, the Jacobian matrix becomes

$$A = \begin{bmatrix} -\mu R_0 & -(\alpha + \mu) \\ \mu\left(R_0 - 1\right) & 0 \end{bmatrix}. \tag{6}$$

The eigenvalues for the Jacobian matrix A are the roots λ of the characteristic equation

$$\det\left(A - \lambda I\right) = \lambda^2 + \mu R_0 \lambda + \mu\left(R_0 - 1\right)(\alpha + \mu) = 0. \tag{7}$$

This quadratic equation is easy to solve and we get the eigenvalues

$$\lambda = \frac{-\mu R_0 \pm \sqrt{\left(\mu R_0\right)^2 - 4\mu\left(R_0 - 1\right)(\alpha + \mu)}}{2}. \tag{8}$$

As $4\mu\left(R_0 - 1\right)(\alpha + \mu) > 0$, both roots will have a negative real part, proving the non-trivial equilibrium to be asymptotically stable. However, it is possible that $\left(\mu R_0\right)^2 - 4\mu\left(R_0 - 1\right)(\alpha + \mu) < 0$ and the roots are complex conjugates. In this case, the equilibrium would be a focus and the system would approach the equilibrium oscillatorilly. Otherwise both roots are negative and real, making the equilibrium a stable node.

2.4 Final Size of the Epidemic

If the epidemic spreads quickly in the population, the demographic changes, births and deaths, will have only a negligible effect in the overall dynamics during the epidemic. In this case we can assume that $\mu \approx 0$ and reduce the system to

$$\dot{S} = -\beta SI \tag{9a}$$

$$\dot{I} = \beta SI - \alpha I \tag{9b}$$

$$\dot{R} = \alpha I. \tag{9c}$$

The basic reproduction number will now be $R_0 = \frac{\beta N}{\alpha}$.

Since $S(t)$ is a decreasing function when $S(t)$ and $I(t)$ are positive, there is a point in time t_0 so that $\dot{I}(t) = (\beta S(t) - \alpha) I(t) < 0$ for all $t > t_0$. From this we can see that the number of infected $I(t)$ will approach zero when $t \to \infty$. Since the epidemic is now guaranteed to die out, we can consider the question of the final size of the epidemic. What portion of the population will be infected at any point during the epidemic? We can solve this by figuring the part of the population that stays susceptible at any point in time. Starting from the equation

$$\frac{dI}{dS} = \frac{\beta SI - \alpha I}{-\beta SI} = -1 + \frac{\alpha}{\beta}\frac{1}{S},$$

separation of variables gives us the identity

$$\frac{\alpha}{\beta} \ln S(t) - S(t) - I(t) = C$$

for all values of time t. Specifically, we can write the equation

$$\frac{\alpha}{\beta} \ln S(\infty) - S(\infty) - I(\infty) = \frac{\alpha}{\beta} \ln S(-\infty) - S(-\infty) - I(-\infty).$$

In an uninfected population $S(-\infty) = N$ and $I(-\infty) = 0$. Additionally, as shown above, $I(\infty) = 0$. These identities will reduce the above equation to

$$\ln s(\infty) = -R_0 (1 - s(\infty)), \tag{10}$$

where the only unknown is $s(\infty) = \frac{S(\infty)}{N}$, the portion of population N still susceptible after the epidemic. This can be solved numerically. In figure 1 the fraction $1 - s(\infty)$ of the population who have been infected at any point during the epidemic has been presented as a function of the basic reproduction number R_0.

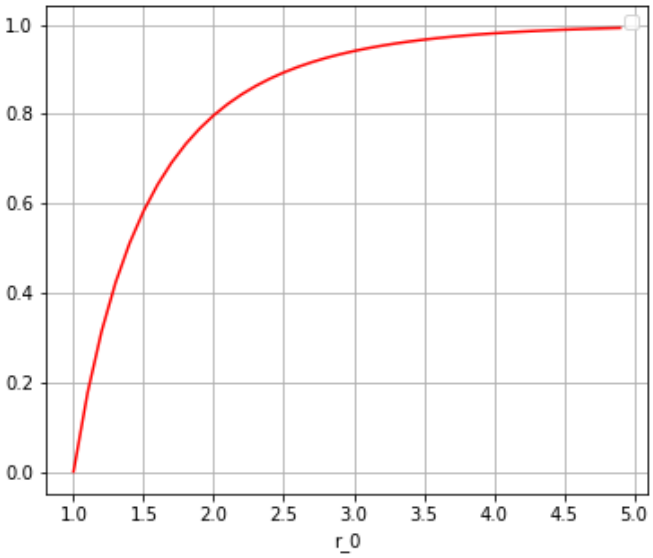


Figure 1: Fraction $1 - s(\infty)$ of the population infected in the epidemic.

3 Model with Spread of Awareness

We now modify this model to include the spread of awareness of the disease and available vaccination. The susceptible S can be separated into two sub-groups, the unaware S_U and the aware S_A. The unaware can be described as those susceptibles who are either ignorant of the disease or do not perceive the epidemic as "their problem" and therefore do not change their behaviour. Susceptibles become aware in a process described by a yet undefined function $\gamma_S(S_U, I, S_A, R_A) \geq 0$ and start to consider taking vaccination. They do so at a rate $\nu > 0$, but they can also get infected before the vaccination in the same way as the unaware. Those vaccinated will gain total immunity to the disease and will, along with the recovered, constitute the immune population, which we will label as R to point out the similarity between this model and the basic SIR-model. Upon vaccination the aware susceptibles will first move to the population of aware immune R_A, who, depending on the chosen model, might still be active in spreading awareness among the rest of the population. If the epidemic is a low-burning one, both the aware susceptibles and immune could also "forget" the epidemic, becoming unaware at a rate u. The unaware immune R_U can be reminded that the epidemic is still happening. That process will be described by the function $\gamma_R(R_U, I, S_A, R_A) \geq 0$. We'll assume that the disease is severe enough that the infected cannot ignore it and thus we'll consider tracking the awareness among the infected unnecessary.

Modifying the SIR-model to include the changes described above we arrive to a system

$$\begin{aligned}
\dot{S}_U &= \mu N - \beta S_U I - \gamma_S(S_U, I, S_A, R_A) + uS_A - \mu S_U \\
\dot{S}_A &= \gamma_S(S_U, I, S_A, R_A) - \beta S_A I - \nu S_A - uS_A - \mu S_A \\
\dot{I} &= \beta(S_U + S_A)I - \alpha I - \mu I \\
\dot{R}_A &= \alpha I + \nu S_A + \gamma_R(R_U, I, S_A, R_A) - uR_A - \mu R_A \\
\dot{R}_U &= uR_A - \gamma_R(R_U, I, S_A, R_A) - \mu R_U.
\end{aligned}$$

3.1 Three simple choices for the functions γ_S and γ_R

We'll assume that the processes of informing the susceptible and the immune are similar and thus both can be represented with a function $\gamma_X(X_U, I, S_A, R_A)$, where X_U marks the targeted unaware population. We want the *awareness function* $\gamma_X(X_U, I, S_A, R_A)$ to model the spreading of information about the epidemic and its risks among the unaware population $U = S_U + R_U$. First we will note the somewhat self-evident observation that the function γ_X is at least non-negative everywhere. Otherwise we could have a situation where people willfully ignore the epidemic in the face of evidence. While a large enough population will contain some contrarians, it can be safely assumed that they will stay as a fringe phenomenon. We must also make sure that $\gamma_X(0, I, S_A, R_A) = 0$ for any I, S_A or R_A to preserve positivity.

There are several possible ways awareness can be spread. The first possibility, and the easiest to control, is that information about the epidemic and available vaccination is spread in mass media. We'll assume that this coverage is based on the potential threat

posed by the epidemic and is thus not dependent on the size of the outbreak currently. If the coverage is sufficiently omnipresent, we can model this simply by defining a rate a_C that a given susceptible is won over at a particular moment. This would give us the function $\gamma_{X,C}(X_U, I, S_A, R_A) = \gamma_C X_U = a_C X_U$.

The second route is personal experience with the epidemic via family, friends and acquaintances falling victim. We can model this by giving (non-contracting) contact between susceptible and infected the possibility to create enough concern to make the susceptible interested in protecting themselves from the epidemic a rate a_I. The resulting candidate for the awareness function is $\gamma_{X,I}(X_U, I, S_A, R_A) = \gamma_I(I) X_U = a_I I X_U$.

A third possible source are other aware individuals who will spread what they've learned to others. Of course this requires an initial group of susceptibles or already recovered who have learned from the epidemic from somewhere else, so we have to assume that the aware population $A = S_A + R_A$ is nonzero at the start of the epidemic. Modelling this means including a process where at a rate a_A aware and unaware individuals meet and the aware convince the unaware. This would give us the term $\gamma_{X,A}(X_U, I, S_A, R_A) = \gamma_A(S_A, R_A) X_U = a_A A X_U$.

The three awareness functions $\gamma_X(X_U, I, S_A, R_A)$ chosen here are all in the form of $\gamma(I, S_A, R_A) X_U$, where the *gamma function* $\gamma(I, S_A, R_A)$ describes the information pressure an (unaware) individual faces. From this point on, the systems we will focus on will be of the form

$$\dot{S}_U = \mu N - \beta S_U I - \gamma(I, S_A, R_A) S_U + u S_A - \mu S_U \tag{11a}$$

$$\dot{S}_A = \gamma(I, S_A, R_A) S_U - \beta S_A I - \nu S_A - u S_A - \mu S_A \tag{11b}$$

$$\dot{I} = \beta(S_U + S_A) I - \alpha I - \mu I \tag{11c}$$

$$\dot{R}_A = \alpha I + \nu S_A + \gamma(I, S_A, R_A) R_U - u R_A - \mu R_A \tag{11d}$$

$$\dot{R}_U = u R_A - \gamma(I, S_A, R_A) R_U - \mu R_U. \tag{11e}$$

3.2 Pre-Epidemic Equilibrium

In case of two of the above gamma functions, γ_C and γ_A, the spread of the awareness does not rely on the presence of the infected among the population. For these there exist pre-epidemic equilibria of awareness. This leads to vaccinations that can hinder or even completely stop the inital spread of the disease. If the total number of susceptibles at the start of the epidemic $S_0 = S_U + S_A$ is less than the total population N, then the basic reproduction number will be

$$R_0 = \frac{\beta S_0}{\alpha + \mu}$$

after the spread of awareness preceding the epidemic. We aim to create a situation where the total number of susceptibles S_0 at the start of the epidemic is low enough to make the reproduction number $R_0 < 1$ and prevent a major outbreak of the disease.

When $I = 0$, the model describes the spread of awareness with the equations

$$\dot{S}_U = \mu N - \gamma\left(S_A, R_A\right) S_U + u S_A - \mu S_U \tag{12a}$$
$$\dot{S}_A = \gamma\left(S_A, R_A\right) S_U - \nu S_A - u S_A - \mu S_A \tag{12b}$$
$$\dot{R}_A = \nu S_A + \gamma\left(S_A, R_A\right) R_U - u R_A - \mu R_A \tag{12c}$$
$$\dot{R}_U = u R_A - \gamma\left(S_A, R_A\right) R_U - \mu R_U. \tag{12d}$$

3.2.1 Constant Awareness Spread

Starting first with the function $\gamma_C = a_C$, system (12) becomes

$$\dot{S}_U = \mu N - a_C S_U + u S_A - \mu S_U \tag{13a}$$
$$\dot{S}_A = a_C S_U - \nu S_A - u S_A - \mu S_A \tag{13b}$$
$$\dot{R}_A = \nu S_A + a_C R_U - u R_A - \mu R_A \tag{13c}$$
$$\dot{R}_U = u R_A - a_C R_U - \mu R_U. \tag{13d}$$

The pre-epidemic equilibrium is the point $\left(\bar{S}_U, \bar{S}_A, \bar{R}_A, \bar{R}_U\right)$ where all equations of (13) are zero. Equation (13b) requires that in an equilibrium the population of aware susceptible S_A can be expressed as

$$\bar{S}_A = \frac{a_C}{\nu + u + \mu} \bar{S}_U.$$

Using this expression for $\bar{S}_A$ in equation (13a), we get the equation

$$\begin{aligned} \mu N &= \frac{(a_C + \mu)(\nu + u + \mu) - u a_C}{\nu + u + \mu} \bar{S}_U \\ &= \frac{\mu(a_C + u + \mu) + \nu(a_C + \mu)}{\nu + u + \mu} \bar{S}_U. \end{aligned}$$

This gives the population of the unaware susceptibles S_U as

$$\bar{S}_U = \frac{\mu(\nu + u + \mu)}{\mu(a_C + u + \nu + \mu) + a_C \nu} N$$

and consequently the equilibrium for the all susceptibles S is now

$$\bar{S} = \bar{S}_U + \bar{S}_A = \frac{\mu(a_C + u + \nu + \mu)}{\mu(a_C + u + \nu + \mu) + a_C \nu} N \tag{14}$$

and the equilibrium of the total immune population R is

$$\bar{R} = N - \bar{S} = \frac{a_C \nu}{\mu(a_C + u + \nu + \mu) + a_C \nu} N.$$

The ratio between the aware and unaware in the immune population R can be solved with equation (13d) by writing R_A as $R - R_U$ and solving the resulting equation

$$u(\bar{R} - \bar{R}_U) = (a_C + \mu)\bar{R}_U.$$

This gives the equilibrium of unaware immune R_U as

$$\bar{R}_U = \frac{u}{a_C + \mu + u}\bar{R},$$

making the aware immune population as

$$\bar{R}_A = \frac{a_C + \mu}{a_C + \mu + u}\bar{R}.$$

Together, the equilibrium for system (13) is

$$\begin{aligned}\left(\bar{S}_U, \bar{S}_A, \bar{R}_A, \bar{R}_U\right) = \Bigg(& \frac{\mu(\nu + u + \mu)}{\mu(a_C + u + \nu + \mu) + a_C\nu}N, \frac{a_C\mu}{\mu\left(a_C + u + \nu + \mu\right) + a_C\nu}N, \\ & \frac{a_C\nu\left(a_C + \mu\right)}{\left(a_C + \mu + u\right)\left(\mu(a_C + u + \nu + \mu) + a_C\nu\right)}N, \\ & \frac{a_C\nu u}{\left(a_C + \mu + u\right)\left(\mu(a_C + u + \nu + \mu) + a_C\nu\right)}N\Bigg). \end{aligned} \tag{15}$$

The Jacobian matrix of model (13) is the 4x4 matrix

$$\begin{bmatrix} -\left(a_C + \mu\right) & u & 0 & 0 \\ a_C & -\left(\nu + u + \mu\right) & 0 & 0 \\ 0 & \nu & -\left(u + \mu\right) & a_C \\ 0 & 0 & u & -\left(a_C + \mu\right) \end{bmatrix}.$$

It can be seen that this is a block lower triangular matrix. This means that the characteristic equation of this matrix is the multiplication of the characteristic equations of the diagonal 2x2 matrices. Further, the lower right matrix is merely the upper left matrix in the special case of $\nu = 0$ after trivial permutations of rows and columns. Thus, the analysis of the equilibrium's stability can be reduced to the two-dimensional system

$$\begin{bmatrix} -\left(a_C + \mu\right) & u \\ a_C & -\left(\nu + u + \mu\right) \end{bmatrix}. \tag{16}$$

Here and further on we will use *the Routh-Hurwitz criterion*, which is a necessary and sufficient requirement for the stability of a linear system. For a second order system, the criterion states that the characteristic equation has no roots with positive real parts when its coefficients share the same sign. [5] The characteristic equation for (16) is

$$\Phi_C\left(\lambda\right) = \det\begin{bmatrix} -\left(a_C + \mu + \lambda\right) & u \\ a_C & -\left(\nu + u + \mu + \lambda\right) \end{bmatrix} \tag{17}$$

$$= \lambda^2 + \left(a_C + \nu + u + 2\mu\right)\lambda + \left(a_C + \mu\right)\left(\nu + \mu\right) + u\mu. \tag{18}$$

Since all the parameters are positive, the coefficients are all positive as well and according to the Routh-Hurwitz criterion all the eigenvalues of matrix (16) and by extention of the Jacobian of system (13) have negative real parts. The disease-free equilibrium (15) is therefore asymptotically stable.

3.2.2 Aware-Dependent Awareness Spread

The other gamma function with a pre-epidemic equilibrium is $\gamma_A = a_A A$. Here awareness essentially spreads as a "disease" in contacts between aware and unaware individuals. Examining the dynamics of the total unaware population $U = S_U + R_U$ on one hand and of the total aware population $A = S_A + R_A$ on the other, we get the SIS system

$$\dot{U} = \mu N - a_A A U + u A - \mu U \tag{19a}$$

$$\dot{A} = a_A A U - (u + \mu) A. \tag{19b}$$

In this model the spread of awareness has a basic reproduction number,

$$R_0^A = \frac{a_A N}{u + \mu},$$

that needs to be more than 1 for an "awareness epidemic" to happen.

From equation (19b) we can see that apart from the trivial equilibrium $\left(\bar{U}, \bar{A}\right) = (N, 0)$, we have the equilibrium

$$\begin{aligned}\left(\bar{U}, \bar{A}\right) &= \left(\frac{u+\mu}{a_A}, N - \frac{u+\mu}{a_A}\right) \\ &= \left(\frac{1}{R_0^A} N, \frac{R_0^A - 1}{R_0^A} N\right)\end{aligned}$$

Coming back to the system

$$\dot{S}_U = \mu N - a_A A S_U + u S_A - \mu S_U \tag{20a}$$

$$\dot{S}_A = a_A A S_U - \nu S_A - u S_A - \mu S_A \tag{20b}$$

$$\dot{R}_A = \nu S_A + a_A A R_U - u R_A - \mu R_A \tag{20c}$$

$$\dot{R}_U = u R_A - a_A A R_U - \mu R_U, \tag{20d}$$

the second equation gives us the equilibrium of aware susceptibles as

$$\begin{aligned}\bar{S}_A &= \frac{a_A \bar{A}}{\nu + u + \mu} \bar{S}_U \\ &= \left(\frac{a_A N}{\nu + u + \mu} - \frac{u + \mu}{\nu + u + \mu}\right) \bar{S}_U\end{aligned} \tag{21}$$

Inserting this expression of $\bar{S}_A$ into the first equation allows us to write the equilibrium population of unaware susceptibles as

$$\bar{S}_U = \frac{\mu N}{\mu + a_A \bar{A} \left(1 - \frac{u}{\nu + u + \mu}\right)}.$$

The denominator in this can be prettified to

$$\frac{\left(a_A N - (u + \mu)\right)(\nu + \mu) + \mu\left(\nu + u + \mu\right)}{\nu + u + \mu},$$

which gives the the equilbrium population of unaware susceptibles the form

$$\bar{S}_U = \frac{\mu\left(\nu + u + \mu\right)}{\left(a_A N - (u+\mu)\right)\left(\nu+\mu\right) + \mu\left(\nu + u + \mu\right)} N. \tag{22}$$

From (21) and the fact that $a_A\bar{A} = a_A N - (u+\mu)$ we can express the equilibrium of the total susceptible population as

$$\bar{S} = \bar{S}_U + \frac{a_A\bar{A}}{\nu+u+\mu}\bar{S}_U = \frac{a_A N + \nu}{\nu+u+\mu}\bar{S}_U,$$

and plugging in (22) gives the expression

$$\bar{S} = \frac{\mu\left(a_A N + \nu\right)}{\left(a_A N - (u+\mu)\right)\left(\nu+\mu\right) + \mu\left(\nu + u + \mu\right)} N. \tag{23}$$

Combining the equations (20c) and (20d) we get the differential equation describing the evolution of the population of all immune, which painlessly gives us the equilibrium for the immune population as

$$\bar{R} = \frac{\nu}{\mu}\bar{S}_A.$$

By expressing the population of aware immune as $R - R_U$ like before, we can find out from equation (20d) that

$$\bar{R}_U = \frac{u}{u + a_A\bar{A} + \mu}\bar{R},$$

and consequently,

$$\bar{R}_A = \frac{a_A\bar{A} + \mu}{u + a_A\bar{A} + \mu}\bar{R}.$$

We have thus expressed $\bar{R}_A$ and $\bar{R}_U$ in terms of $\bar{A}$ and $\bar{S}_A$, $\bar{S}_A$ in terms of $\bar{S}_U$ and $\bar{S}_U$ in terms of $\bar{A}$. This chain gives us the equilibrium

$$\begin{aligned}\left(\bar{S}_U, \bar{S}_A, \bar{R}_A, \bar{R}_U\right) = \Bigg(&\frac{\mu\left(\nu + u + \mu\right)}{\left(a_A N - (u+\mu)\right)\left(\nu+\mu\right) + \mu\left(\nu + u + \mu\right)} N,\\ &\frac{a_A\mu\bar{A}}{\left(a_A N - (u+\mu)\right)\left(\nu+\mu\right) + \mu\left(\nu + u + \mu\right)} N,\\ &\frac{a_A\bar{A} + \mu}{u + a_A\bar{A} + \mu}\frac{a_A\nu\bar{A}}{\left(a_A N - (u+\mu)\right)\left(\nu+\mu\right) + \mu\left(\nu + u + \mu\right)} N,\\ &\frac{u}{u + a_A\bar{A} + \mu}\frac{a_A\nu\bar{A}}{\left(a_A N - (u+\mu)\right)\left(\nu+\mu\right) + \mu\left(\nu + u + \mu\right)} N\Bigg).\end{aligned} \tag{24}$$

in terms of $\bar{A} = N - \frac{u+\mu}{a_A}$.

To help us in analysing the stability of this equilibrium, we will replace the resistant populations R_A and R_U with the total aware and unaware populations A and U, since

$R_A = A - S_A$ and $R_U = U - S_U$. The Jacobian matrix of the resulting system, (19a)-(19b) and (20a)-(20b), is then

$$M = \begin{bmatrix} -\left(a_A\bar{A} + \mu\right) & u - a_A\bar{U} & 0 & 0 \\ a_A\bar{A} & a_A\bar{U} - (u + \mu) & 0 & 0 \\ 0 & a_A\bar{S}_U & -(\nu + u + \mu) & a_A\bar{A} \\ 0 & -a_A\bar{A} & u & -\left(a_A\bar{A} + \mu\right) \end{bmatrix},$$

a lower block triangular matrix. This again means that we can divide the stability analysis into the study of two two-dimensional systems, the "awareness" system (19a)-(19b) and the system of susceptibles (20a)-(20b).

The Jacobian matrix of the system of unawares and awares (19a)-(19b) at the equilibrium $\left(\bar{U}, \bar{A}\right)$ is then

$$M_1 = \begin{bmatrix} -\left(a_A\bar{A} + \mu\right) & u - a_A\bar{U} \\ a_A\bar{A} & a_A\bar{U} - (u + \mu) \end{bmatrix} = \begin{bmatrix} -\left(a_A\bar{A} + \mu\right) & -\mu \\ a_A\bar{A} & 0 \end{bmatrix},$$

which gives the characteristic function

$$\Phi_1(\lambda) = \det(M_1 - \lambda I) = \lambda^2 + \left(a_A\bar{A} + \mu\right)\lambda + a_A\bar{A}\mu.$$

It is easy to explicitly find the eigenvalues to be $(\lambda_1, \lambda_2) = \left(-\mu, -a_A\bar{A}\right)$.

Continuing to the system (20a)-(20b), the Jacobian matrix at the equilibrium $\left(\bar{U}, \bar{A}, \bar{S}_U, \bar{S}_A\right)$ is now

$$M_2 = \begin{bmatrix} -(\nu + u + \mu) & a_A\bar{A} \\ u & -\left(a_A\bar{A} + \mu\right) \end{bmatrix}.$$

This gives the characteristic equation

$$\Phi_2(\lambda) = \det(M_2 - \lambda I) = \lambda^2 + \left(\nu + u + \mu + a_A\bar{A} + \mu\right)\lambda + (\nu + u + \mu)\left(a_A\bar{A} + \mu\right) + \mu u.$$

As in the stability analysis of the constant awareness spread model, using the Routh-Hurwitz criterion shows that the roots of the second-order polynomial $\Phi_2(\lambda)$ have negative real parts. Since for the lower triangular matrix M we get the characteristic equation

$$\Phi_A(\lambda) = \det(M - \lambda I) = \det(M_1 - \lambda I)\det(M_2 - \lambda I),$$

which has for its roots the roots of the previous characteristic equations $\Phi_1(\lambda)$ and $\Phi_2(\lambda)$. The disease-free equilibrium is then proven to be asymptotically stable in this system as well.

3.2.3 Preventing a Major Outbreak

As mentioned before, if the spread of awareness (and vaccinations) before the epidemic are effective enough, it is possible to reduce the total number of all susceptibles at the start of the epidemic S_0 so that the new basic reproduction number after the awareness spread has reached its equilibrium satisfies the condition

$$\widetilde{R}_0 = \frac{\beta S_0}{\alpha + \mu} < 1,$$

meaning that no major outbreak would happen. We need to find the values of the awareness parameters a_C and a_A for which the total susceptible population

$$S_0 < \frac{\alpha + \mu}{\beta}. \tag{25}$$

It should be noted that while a_C is a dimensionless parameter, in the aware-dependent awareness model the parameter a_A has the dimension N^{-1}, since $\gamma_A(t) = a_A A(t) = a_A N a(t)$, where $a(t)$ is the portion of the population that are aware at time t. To make the two models compararble with each other, we will now contrast a_C with the dimensionless $a_A N$.

An important detail to keep in mind before examining the awareness is that we need to make sure that our vaccination goal is actually achievable. No amount of awareness is going to help if the aware susceptibles do not then take action to vaccinate themselves. In both models, if the awareness parameter a_C or a_A is far larger than the other parameters, the equilibria of the susceptible population S as calculated in (14) and (23) approach the limit

$$\frac{a_C \mu}{a_C(\nu + \mu)} N = \frac{a_A N \mu}{a_A N(\nu + \mu)} N = \frac{\mu}{\nu + \mu} N.$$

From now we'll assume that this limit is below the goal $(\alpha + \mu)/\beta$, which gives us the condition

$$\nu > \mu \left(\frac{\beta N}{\alpha + \mu} - 1 \right) = \mu(R_0 - 1). \tag{26}$$

Starting with the model with constant awareness spread, we insert the equilibrium for the susceptible population calculated in (14) and solve the parameter a_C needed for the inequality

$$\frac{a_C \mu + \mu(u + \nu + \mu)}{a_C(\nu + \mu) + \mu(u + \nu + \mu)} N < \frac{\alpha + \mu}{\beta} \tag{27}$$

to be true. This inequality can be rewritten as

$$a_C(\beta \mu N - (\alpha + \mu)(\nu + \mu)) < \mu((\alpha + \mu) - \beta N)(u + \nu + \mu).$$

From (26) and assuming that the basic reproduction number in (2) is greater than one, i.e. the disease is something that needs preventing, we see that both $\beta \mu N - (\alpha + \mu)(\nu + \mu)$ and $(\alpha + \mu) - \beta N$ are now negative. Therefore, inequality (25) is true when a_C is larger than treshold

$$\bar{a}_C = \frac{\mu(\beta N - (\alpha + \mu))(u + \nu + \mu)}{((\alpha + \mu)(\nu + \mu) - \beta \mu N)} = \frac{\mu(R_0 - 1)(u + \nu + \mu)}{\nu - \mu(R_0 - 1)} > 0 \tag{28}$$

Similarly we can find out the awareness parameter a_A needed to prevent a major outbreak in the aware-dependent awareness model. Getting the total susceptible population (23) under the goal (25) requires that

$$\begin{aligned} a_A N(\beta \mu N - (\alpha + \mu)(\nu + \mu)) < & \mu(\alpha + \mu)(u + \nu + \mu) \\ & - \beta \nu \mu N - (u + \mu)(\nu + \mu)(\alpha + \mu). \end{aligned}$$

However, since $(u+\mu)(\nu+\mu)(\alpha+\mu) = \mu(\alpha+\mu)(u+\nu+\mu)+u\nu(\alpha+\mu)$, this inequality becomes

$$a_A N(\beta\mu N - (\alpha+\mu)(\nu+\mu)) > -\nu(\beta\mu N + u(\alpha+\mu)).$$

Assuming again that (26) is true, from this we get the inequality

$$\bar{a}_A N = \frac{\nu(\beta\mu N + u(\alpha+\mu))}{((\alpha+\mu)(\nu+\mu) - \beta\mu N)} = \frac{\nu(\mu R_0 + u)}{\nu - \mu(R_0 - 1)} > 0 \tag{29}$$

as a requirement for an aware-dependent spread of awareness to prevent a major outbreak.

Comparing the prevention thresholds in (28) and (29) shows that $\bar{a}_A N$ will be higher than $\bar{a}_C$ when

$$\nu\mu R_0 + u\nu > \mu(R_0 - 1)(u+\mu) + \nu\mu R_0 - \mu\nu.$$

This leads to the inequality

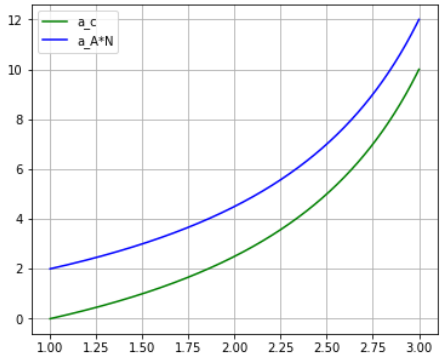
$$\nu(u+\mu) > \mu(R_0 - 1)(u+\mu),$$

which is always true when the required vaccination level in (26) is kept.

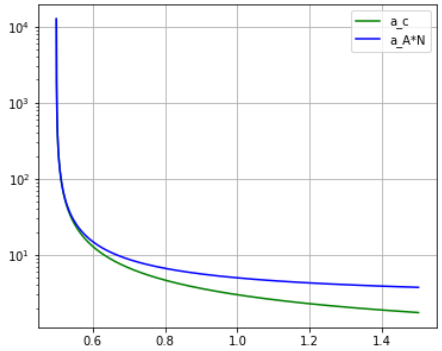
The two thresholds show similar behaviour when the parameters are varied. The figure 2 shows thresholds (28) and (29) when the basic reproduction number, rate of demographic change or rate of vaccination changes. The key factor is the difference between the vaccination rate ν and its theoretical lower limit $\mu(R_0 - 1)$ in the denominator. The higher the vaccination rate is, the more relaxed the spread of awareness can be while still creating the necessary number of vaccinated individuals before the introduction of the disease. Both thresholds grow without limit as the rate of infection β approaches the limit

$$\frac{(\nu+\mu)(\alpha+\mu)}{\mu N}.$$

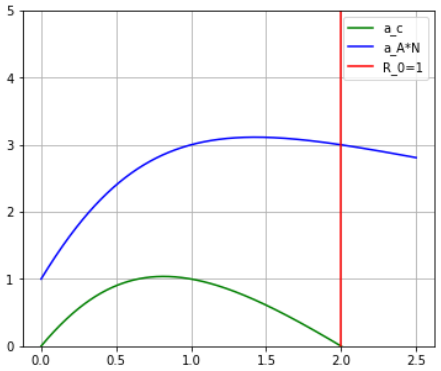
Meanwhile with increasing ν the thresholds decrease and approach their asymptotes, $\mu(R_0 - 1)$ for a_C and $\mu(R_0 + u)$ for $a_A N$. The effect of μ on the new basic reproduction number $\widetilde{R}_0 = \beta S_0/(\alpha+\mu)$ is more complicated. With low values of μ the number of susceptibles in disease-free equilibria (22) and (23), which are the initial susceptibles S_0 at the beginning of the epidemic, are small and give the disease limited room to spread. On the other hand, increasing μ lowers the expected time $(\alpha+\mu)^{-1}$ an individual stays infectious. These two effects lead to the non-monotonic behaviour as seen in figure 2c.



(a) As a function of the basic reproduction number R_0



(b) As a function of the rate of vaccination ν



(c) As a function of the rate of demographic change μ

Figure 2: Threshold awareness to prevent epidemic. Unless varied, the parameters are: $N = 1, \beta = 2, \mu = \alpha = 0.5, u = 1, \nu = 2$.

3.3 Basic Reproduction number and the Endemic Equilibrium of the Disease

We will now examine the existence and stability of the equilibrium of system (11) in a situation where the awareness starts to spread only after the outbreak of the actual epidemic. The basic reproduction number R_0 does not change from the ordinary SIR-model. At the start of the epidemic the entire population N consists of unaware susceptibles S_U bar an infinitesimal fraction of infected. The growth rate of the epidemic during the initial stage is thus

$$\dot{I} = (\beta N - \alpha - \mu)\, I.$$

This is positive if and only if the reproduction number

$$R_0 = \frac{\beta N}{\alpha + \mu} > 1.$$

The equilibrium for the total susceptible population, both aware and unaware, will also not change. For some non-zero infected population $\bar{I}$, the stationarity of the system

requires that the equations equal to zero. Specifically, it must be that

$$\dot{I} = \left[\beta(\bar{S}_U + \bar{S}_A) - (\alpha + \mu)\right] \bar{I} = 0.$$

This requires that the terms inside the brackets equal zero, thus giving us the equilibrium for the total susceptible population

$$\bar{S} = \bar{S}_U + \bar{S}_A = \frac{\alpha + \mu}{\beta} = \frac{N}{R_0}. \tag{30}$$

Since $S_U = S - S_A$, we can express, with the help of aware susceptibles S_A, the dynamics of the unaware susceptibles S_U with those of the total susceptible population S. The dynamics of S is described by the sum of equations (11a) and (11b), giving us the equation

$$\dot{S} = \mu N - \beta SI - \mu S - \nu S_A.$$

At the equilibrium $\left(\bar{S}, \bar{S}_A, \bar{I}, \bar{R}_A, \bar{R}_U\right)$, this equation must also be zero. Using the value of $\bar{S}$ we got in (30), we now have

$$\beta \frac{N}{R_0} \bar{I} = \mu N \left(1 - \frac{1}{R_0}\right) - \nu \bar{S}_A.$$

Multiplying both sides with R_0/N, we see that the equilibrium for the infected population

$$\bar{I} = \bar{I}_0 - b\bar{S}_A, \tag{31}$$

where $\bar{I}_0 = (\mu/\beta)(R_0 - 1)$ and $b = \nu/(\alpha + \mu)$.

Once $\bar{S}_A$ is solved, we can continue to solve the equilibrium for the recovered populations. The dynamics of the total recovered population R is described by the differential equation

$$\dot{R} = \alpha I + \nu S_A - \mu R$$

and the total recovered population in the endemic equilibrium is accordingly

$$\bar{R} = \frac{\alpha \bar{I} + \nu \bar{S}_A}{\mu}.$$

From $\dot{R}_U = 0$ we can express the equilibrium of the unaware recovered population R_U relative to that of the aware recovered R_A as

$$\bar{R}_U = \frac{u}{\bar{\gamma} + \mu} \bar{R}_A.$$

where $\bar{\gamma} = \gamma\left(\bar{I}, \bar{S}_A, \bar{R}_A\right)$ is the value of the gamma function in the equilibrium. Then, since now

$$\bar{R} = \bar{R}_U + \bar{R}_A = \left(1 + \frac{u}{\bar{\gamma} + \mu}\right) \bar{R}_A = \frac{u + \bar{\gamma} + \mu}{\bar{\gamma} + \mu} \bar{R}_A,$$

we can then figure out that

$$\bar{R}_A = \frac{\bar{\gamma}+\mu}{u+\bar{\gamma}+\mu}\frac{\alpha\bar{I}+\nu\bar{S}_A}{\mu},$$
$$\bar{R}_U = \frac{u}{u+\bar{\gamma}+\mu}\frac{\alpha\bar{I}+\nu\bar{S}_A}{\mu}$$

are the populations of aware and unaware recovered in the endemic equilibrium.

The population equilibria are now all solved in terms of $\bar{S}_A$. We can now insert the value of $\bar{I}$ in (31) to equation (11b), which in equilibrium is

$$\begin{aligned}\dot{S}_A &= \bar{\gamma}\bar{S}_U - \beta\bar{S}_A\left(\bar{I}_0 - b\bar{S}_A\right) - (\mu+u+\nu)\,\bar{S}_A \\ &= b\beta\bar{S}_A^2 - \left(\bar{I}_0\beta+\mu+u+\nu\right)\bar{S}_A + \bar{\gamma}\left(\bar{S}-\bar{S}_A\right) \\ &= \frac{R_0\nu}{N}\bar{S}_A^2 - (\mu R_0+u+\nu+\bar{\gamma})\,\bar{S}_A + \bar{\gamma}\bar{S} = 0. \end{aligned} \tag{32}$$

This is the furthest one could go without defining the gamma function describing the spread of awareness. Once we'll solve the equilibrium for S_A for the specific model, we can then express the endemic equilibrium as

$$\begin{aligned}&\left(\bar{S},\bar{S}_A,\bar{I},\bar{R}_A,\bar{R}_U\right) \\ &= \left(\frac{\alpha+\mu}{\beta},\bar{S}_A,\frac{\mu}{\beta}(R_0-1) - \frac{\nu}{\alpha+\mu}\bar{S}_A,\frac{\bar{\gamma}+\mu}{u+\bar{\gamma}+\mu}\frac{\alpha\bar{I}+\nu\bar{S}_A}{\mu},\frac{u}{u+\bar{\gamma}+\mu}\frac{\alpha\bar{I}+\nu\bar{S}_A}{\mu}\right).\end{aligned} \tag{33}$$

3.3.1 Constant Awareness Spread

Picking first the function $\gamma_C = a_C$, system (11) becomes

$$\dot{S} = \mu N - \beta SI - \nu S_A - \mu S \tag{34a}$$
$$\dot{S}_A = a_C(S-S_A) - (\beta I+\nu+u+\mu)\,S_A \tag{34b}$$
$$\dot{I} = \beta SI - \alpha I - \mu I \tag{34c}$$
$$\dot{R}_A = \alpha I + \nu S_A + a_C R_U - uR_A - \mu R_A \tag{34d}$$
$$\dot{R}_U = uR_A - a_C R_U - \mu R_U. \tag{34e}$$

With $\bar{\gamma}_C = a_C$, equation (32) of S_A becomes

$$\frac{R_0\nu}{N}\bar{S}_A^2 - (\mu R_0+u+\nu+a_C)\,\bar{S}_A + a_C\frac{N}{R_0} = 0.$$

The quadratic formula gives the equilibrium population of aware susceptible S_A

$$\bar{S}_A = \frac{(\mu R_0+u+\nu+a_C) \pm \sqrt{(\mu R_0+u+\nu+a_C)^2 - 4\nu a_C}}{2\nu}\bar{S}.$$

The two roots are real and positive. Expanding and rearranging the term under the square root shows that

$$\begin{aligned}(\mu R_0 + u + \nu + a_C)^2 - 4\nu a_C &= ((\mu R_0 + u) + (\nu + a_C))^2 - 4\nu a_C \\ &= (\mu R_0 + u)^2 + (\mu R_0 + u)(\nu + a_C) + (\nu + a_C)^2 - 4\nu a_C \\ &= (\mu R_0 + u)^2 + (\mu R_0 + u)(\nu + a_C) + (\nu - a_C)^2 \geq 0.\end{aligned} \tag{35}$$

Furthermore,

$$\begin{aligned}(\mu R_0 + u + \nu + a_C) &= \sqrt{(\mu R_0 + u + \nu + a_C)^2} \\ &> \sqrt{(\mu R_0 + u + \nu + a_C)^2 - 4\nu a_C}.\end{aligned}$$

The greater root, while valid from a purely mathematical point of view, would give a population of aware susceptibles that is larger than the population of susceptibles in total; remembering (35), we can evaluate

$$\begin{aligned}\bar{S}_A &= \frac{(\mu R_0 + u + \nu + a_C) + \sqrt{(\mu R_0 + u + \nu + a_C)^2 - 4\nu a_C}}{2\nu}\bar{S} \\ &> \frac{(\nu + a_C) + \sqrt{(\nu + a_C)^2 - 4\nu a_C}}{2\nu}\bar{S} = \frac{\nu + a_C + \sqrt{(\nu - a_C)^2}}{2\nu}\bar{S} \\ &= \frac{\nu + a_C + |\nu - a_C|}{2\nu}\bar{S}.\end{aligned}$$

The numerator is now either $2a_C$, if $a_C > \nu$, or 2ν otherwise. In either case $\bar{S}_A$ would be larger than $\bar{S}$. Since we require that all populations, including S_U, have to be positive, this $\bar{S}_A$ is not admissible in our model and therefore the smaller root

$$\bar{S}_A = \frac{(\mu R_0 + u + \nu + a_C) - \sqrt{(\mu R_0 + u + \nu + a_C)^2 - 4\nu a_C}}{2\nu}\bar{S} \tag{36}$$

is the only possible equilibrium for population S_A in the endemic case. This, along with (33), provides the the endemic equilibrium.

We can now calculate how large the constant a_C needs to be in order for the equilibrium of the infected population $\bar{I}$ as calculated in (31) to become zero. That is equal to

$$(\mu R_0 + u + \nu + \bar{a}_C) - 2\mu(R_0 - 1) = \sqrt{(\mu R_0 + u + \nu + \bar{a}_C)^2 - 4\nu\bar{a}_C}.$$

Raising both sides to the power of two and arranging the terms of a_C to the left hand side and the rest to the right gives the equation

$$\bar{a}_C(\nu - \mu(R_0 - 1)) = \mu(R_0 - 1)(\mu + u + \nu).$$

From this we can see that the constant $\bar{a}_C$ needed to remove the disease from the population is the same as threshold (28) to make the disease-free equilibrium stable. When $\nu > \mu(R_0 - 1)$, equilibrium $\bar{I}$ is strictly positive when $a_C < \bar{a}_C$. On the other hand, if $\nu < \mu(R_0 - 1)$, threshold $\bar{a}_C$ is negative and $\bar{I}$ is strictly positive for all positive values of a_C. As can be seen from (33), the positivity of both $\bar{R}_A$ and $\bar{R}_U$ follow from the positivity of $\bar{S}_A$ and $\bar{I}$.

Stability of the Equilibria The Jacobian of system (34) in the endemic equilibrium is then

$$\begin{bmatrix} -\left(\beta\bar{I}+\mu\right) & -\nu & -\beta\bar{S} & 0 & 0 \\ a_C & -\left(\beta\bar{I}+\mu+u+\nu+a_C\right) & -\beta\bar{S}_A & 0 & 0 \\ \beta\bar{I} & 0 & \beta S-(\alpha+\mu) & 0 & 0 \\ 0 & \nu & \alpha & -(u+\mu) & a_C \\ 0 & 0 & 0 & u & -(a_C+\mu) \end{bmatrix}$$

This is again a lower block triangular matrix, and we have already proven in (18) that the characteristic equation of the lower right matrix has negative real roots. The stability of the system now depends on the upper left matrix

$$M_C = \begin{bmatrix} -\left(\beta\bar{I}+\mu\right) & -\nu & -(\alpha+\mu) \\ a_C & -\left(\beta\bar{I}+\mu+u+\nu+a_C\right) & -\beta\bar{S}_A \\ \beta I & 0 & 0 \end{bmatrix}$$

which has the characteristic function

$$\begin{aligned} \Phi_C(\lambda) &= \det(M_C - \lambda I) \\ &= -\lambda^3 - (2C_1 + C_2)\lambda^2 - \left(C_1^2 + C_1C_2 + C_3 + C_5\right)\lambda - (C_1C_3 + C_2C_3 - C_4), \end{aligned}$$

where

$$\begin{aligned} C_1 &= \beta\bar{I} + \mu, & C_4 &= \nu\left(\beta\bar{S}_A\right)\left(\beta\bar{I}\right), \\ C_2 &= u + \nu + a_C, & C_5 &= a_C\nu. \\ C_3 &= \beta\bar{I}(\alpha+\mu), \end{aligned}$$

Due to the positivity of the parameters, as well as the equilibria $\bar{S}_A$ and $\bar{I}$, the coefficients $C_1, \ldots, C_5$ are also positive.

To assess the stability of the equilibrium we study the roots of the characteristic function $\Phi_C(\lambda)$, which are identical to the roots of function $\Phi_{C-}(\lambda) = -\Phi_C(\lambda)$. We can apply here the Routh-Hurwitz criterion for third order polynomials. The polynomial $P(\lambda) = \lambda^3 + c_2\lambda^2 + c_1\lambda + c_0$ has all its roots in the open left half side of the complex plane if and only if c_2 and c_0 are positive and $c_2c_1 > c_0$. [6] We can prove now that in the case of the function $\Phi_{C-}(\lambda)$ these are true for all positive parameters. The coefficients of the polynomial $\Phi_{C-}(\lambda)$ are all positive, as are all the terms $C_1, \ldots, C_5$ and

$$C_2C_3 = \nu\beta\bar{S}\beta\bar{I} + (u + a_C)\beta\bar{S}\beta\bar{I} > \nu\beta\bar{S}\beta\bar{I} > \nu\beta\bar{S}_A\beta\bar{I} = C_4, \tag{37}$$

since $\bar{S} > \bar{S}_A$. The last criterion, the inequality

$$(2C_1 + C_2)\left(C_1^2 + C_1C_2 + C_3 + C_5\right) > C_1C_3 + C_2C_3 - C_4.$$

can be written to be

$$(2C_1 + C_2)\left(C_1^2 + C_1C_2 + C_5\right) + C_1C_3 + C_4 > 0,$$

which holds true always. Therefore the function $\Phi_{C-}(\lambda)$, and the characteristic function $\Phi_C(\lambda)$, only has roots with negative real parts, making the endemic equilibrium locally asymptotically stable.

To collect the results we have gained both here and in (3.2.1), system (34) with constant awareness spread has two possible equilibria, an endemic one (33) with $\bar{S}_A$ as defined in (36) and a disease-free one in (15). An example of the effect of the constant a_C on the endemic equilibrium is shown in figure 3. When a_C is below threshold (28), both equilibria are biologically viable, but only the endemic equilibrium is asymptotically stable. Once the disease is introduced into the population, the epidemic stays endemic, though the fraction of the population infected in the equilibrium is reduced compared to a system with no spread of awareness or vaccination. Above the threshold the endemic equilibrium ceases to be biologically viable, and only the disease-free equilibrium, which is now asymptotically stable, exists.

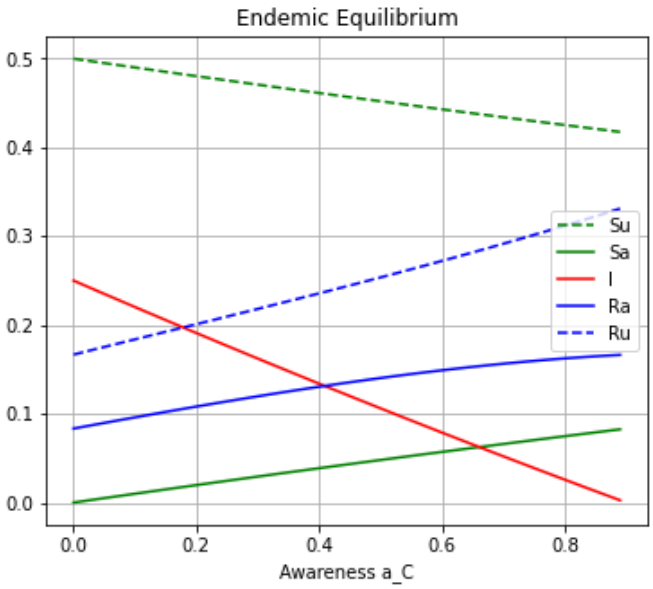


Figure 3: Endemic equilibrium $\left(\bar{S}, \bar{S}_A, \bar{I}, \bar{R}_A, \bar{R}_U\right)$ as a function of a_C. Parameters are: $N = 1, \beta = 2, \mu = \alpha = 0.5, u = 1, \nu = 2$.

3.3.2 Infected-Dependent Awareness Spread

The other two possiblities unfold in a similar fashion. With the function $\gamma_I = a_I I$ and equilibria $\bar{I}$ as presented in (31) we have

$$\begin{aligned}\bar{\gamma}_I S_U = a_I \bar{I} \bar{S}_U &= a_I \left(\bar{I}_0 - \frac{\nu}{\alpha+\mu}\bar{S}_A\right)\left(\bar{S} - \bar{S}_A\right) \\ &= a_I \frac{\nu}{\alpha+\mu}\bar{S}_A^2 - a_I\left(\bar{I}_0 + \frac{\nu}{\alpha+\mu}\bar{S}\right)\bar{S}_A + a_I \bar{I}_0 \bar{S},\end{aligned}$$

which can now be inserted to equation (32) to give the quadratic equation for $\bar{S}_A$

$$\begin{aligned}\dot{S}_A &= \left(\frac{R_0\nu}{N} + a_I\frac{\nu}{\alpha+\mu}\right)\bar{S}_A^2 - \left(\mu R_0 + u + \nu + a_I\left(\bar{I}_0 + \frac{\nu}{\alpha+\mu}\bar{S}\right)\right)\bar{S}_A + a_I\bar{I}_0\bar{S} \\ &= \frac{R_0}{N}\left(\nu + a_I\frac{\nu}{\beta}\right)\bar{S}_A^2 - \left(\mu R_0 + u + \nu + a_I\left(\bar{I}_0 + \frac{\nu}{\beta}\right)\right)\bar{S}_A + a_I\bar{I}_0\bar{S} = 0. \qquad (38)\end{aligned}$$

The constant and second-order terms of this quadratic equation are always positive, while the first-order term is always negative. This means that according to the Routh-Hurwitz criterion, both roots have positive real parts. However, only the smaller root is biologically viable. Treating $\dot{S}_A$ in (38) as a function of $\bar{S}_A$, we see that as a parabola opening up, it is negative between the two roots and positive elsewhere. When $\bar{S}_A = \bar{S}$, this function has the value

$$\begin{aligned}&\bar{S}\left(\frac{R_0}{N}\left(\nu + a_I\frac{\nu}{\beta}\right)\bar{S} - \mu R_0 - u - \nu - a_I\left(\bar{I}_0 + \frac{\nu}{\beta}\right) + a_I\bar{I}_0\right) \\ =&\bar{S}\left(\nu + a_I\frac{\nu}{\beta} - \mu R_0 - u - \nu - a_I\bar{I}_0 - a_I\frac{\nu}{\beta} + a_I\bar{I}_0\right) = \bar{S}\left(-\mu R_0 - u\right) < 0.\end{aligned}$$

The negativity means that $\bar{S}$ is between the two roots. Since S_A is supposed to be a subgroup of S, this disqualifies the larger of the roots as a possible equilibrium for S_A. We also require that $\bar{I} = \bar{I}_0 - \nu/\left(\alpha+\mu\right)\bar{S}_A$ is positive as well. If $\bar{S}_A = I_0\left(\alpha+\mu\right)/\nu$, the function in (38) becomes

$$\begin{aligned}&\frac{\alpha+\mu}{\nu}\bar{I}_0\left(\frac{R_0}{N}\left(\nu + a_I\frac{\nu}{\beta}\right)\frac{\alpha+\mu}{\nu}\bar{I}_0 - \mu R_0 - u - \nu - a_I\bar{I}_0 - a_I\frac{\nu}{\beta} + a_I\frac{\nu}{\beta}\right) \\ &= \frac{\alpha+\mu}{\nu}\bar{I}_0\left(\mu\left(R_0 - 1\right) + a_I\bar{I}_0 - \mu R_0 - u - \nu - a_I\bar{I}_0\right) = \frac{\alpha+\mu}{\nu}\bar{I}_0\left(-\mu - u - \nu\right) < 0.\end{aligned}$$

Again, the negativity shows that only the smaller root provides a biologically viable equilibrium for the infected population I. This leaves the smaller root of (38)

$$\begin{aligned}\bar{S}_A =& \frac{\left(\mu R_0 + u + \nu + a_I\bar{I}_0 + \frac{a_I\nu}{\beta}\right)}{2\left(\nu + a_I\frac{\nu}{\beta}\right)}\bar{S} \\ &- \frac{\sqrt{\left(\mu R_0 + u + \nu + a_I\bar{I}_0 + \frac{a_I\nu}{\beta}\right)^2 - 4\left(\nu + a_I\frac{\nu}{\beta}\right)a_I\bar{I}_0}}{2\left(\nu + a_I\frac{\nu}{\beta}\right)}\bar{S}. \qquad (39)\end{aligned}$$

as the only biologically possible equilibrium for aware susceptible population S_A.

Finally, $\bar{S}_A$ in (39) could have complex values, if the discriminant is negative. Presenting the discriminant as a function of a_I, we have

$$\begin{aligned}&\left(\bar{I}_0^2 - 2\frac{\nu}{\beta}\bar{I}_0 + \frac{\nu^2}{\beta^2}\right) a_I^2 \\ &+2\left[\left(\bar{I}_0 + \frac{\nu}{\beta}\right)(\mu R_0 + u + \nu) - 2\nu\bar{I}_0\right] a_I \\ &+ (\mu R_0 + u + \nu)^2 < 0. \end{aligned} \tag{40}$$

The constant coefficient is positive, as well as the second-order coefficient $\left(\bar{I}_0 - \nu/\beta\right)^2$. We can also figure out that the first order coefficient is always positive, since

$$\begin{aligned}&\frac{\nu^2}{\beta} + \bar{I}_0(\mu R_0 + u) + \frac{\mu R_0 + u}{\beta}\nu + \nu\bar{I}_0 - 2\nu\bar{I}_0 \\ =&\frac{\nu^2}{\beta} + \bar{I}_0(\mu R_0 + u) + \frac{\mu R_0 + u}{\beta}\nu - \nu\frac{\mu}{\beta}(R_0 - 1) \\ =&\frac{\nu^2}{\beta} + \bar{I}_0(\mu R_0 + u) + \frac{\mu + u}{\beta}\nu > 0\end{aligned}$$

when ν is positive. The discriminant is thus always positive when a_I is positive and and $\bar{S}_A$ is always real-valued.

Stability of the Equilibria Using the same arguments as in the stability analysis in section 3.3.1, the stability of this system depends on the stability of the reduced system

$$\dot{S} = \mu N - \beta SI - \nu S_A - \mu S, \tag{41a}$$

$$\dot{S}_A = a_I(S - S_A)I - (\beta I + \mu + u + \nu)S_A. \tag{41b}$$

$$\dot{I} = \beta SI - (\alpha + \mu)I. \tag{41c}$$

The Jacobian matrix of this system at the equilibrium $\left(\bar{S}, \bar{S}_A, \bar{I}\right)$ is

$$M_I = \begin{bmatrix} -\left(\beta\bar{I} + \mu\right) & -\nu & -(\alpha + \mu) \\ a_I\bar{I} & -\left(\beta\bar{I} + \mu + u + \nu + a_I\bar{I}\right) & a_I\bar{S} - (a_I + \beta)\bar{S}_A \\ \beta\bar{I} & 0 & 0 \end{bmatrix},$$

the characteristic function of which is

$$\begin{aligned}\Phi_I(\lambda) &= \det(M_I - \lambda I) \\ &= -\lambda^3 - (2C_1 + C_2)\lambda^2 - \left(C_1^2 + C_1C_2 + C_3 + C_5\right)\lambda - (C_1C_3 + C_2C_3 + C_4),\end{aligned}$$

where

$$\begin{aligned}C_1 &= \beta\bar{I} + \mu, & C_4 &= \nu\left(\beta\bar{I}\right)\left(a_I\bar{S} - (a_I + \beta)\bar{S}_A\right), \\ C_2 &= u + \nu + a_I\bar{I}, & C_5 &= a_I\nu\bar{I}. \\ C_3 &= \beta\bar{I}(\alpha + \mu),\end{aligned}$$

Since at equilibrium $\dot{S}_A = 0$, we can evaluate

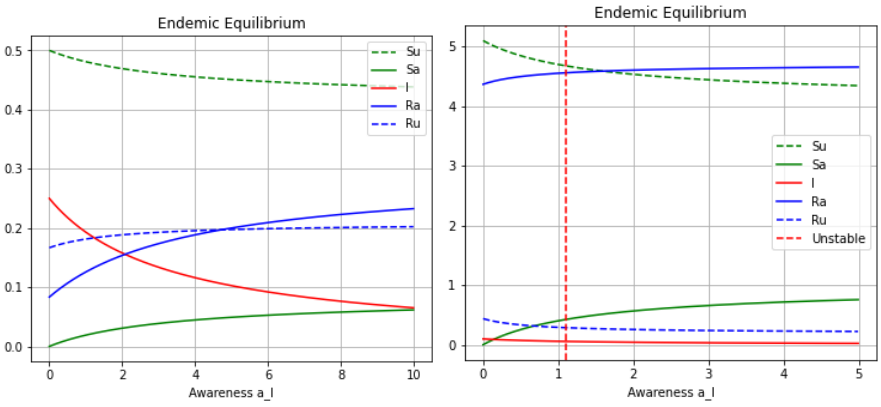
$$a_I \bar{S} = (a_I + \beta)\, \bar{S}_A + \frac{u + \nu + \mu}{\bar{I}} \bar{S}_A > (a_I + \beta)\, \bar{S}_A.$$

Therefore all the defined terms $C_1, \ldots, C_5$ are positive. This leaves the inequality

$$(2C_1 + C_2)\left(C_1^2 + C_1 C_2 + C_5\right) + C_1 C_3 > C_4 \tag{42}$$

as the only criterion for asymptotic stability. However, depending on the parameters chosen, this can be untrue and the endemic equilibrium can be unstable, as we will see below.

Figure 4 features the endemic equilibria for two systems with infected-dependent awareness spread and similar basic reproduction numbers for the disease. In both cases the epidemic will stay endemic, since maintaining a non-zero population of aware individuals requires an infected population to spread concern. High values of a_I and ν will however reduce the size of the infected population $\bar{I}$ needed to maintain the equilibrium. With the parameters of figure 4b, criterion (42) is true for any value of a_I and the endemic equilibrium is always asymptotically stable. Figure 4b is an example of a system with a possible unstable equilibrium. Here criterion (42) is not true when a_I is larger than $\tilde{a}_I \approx 1.1031$. In this system increasing the rate of awareness spread without increasing the rate of vaccination will make the endemic equilibrium unstable.

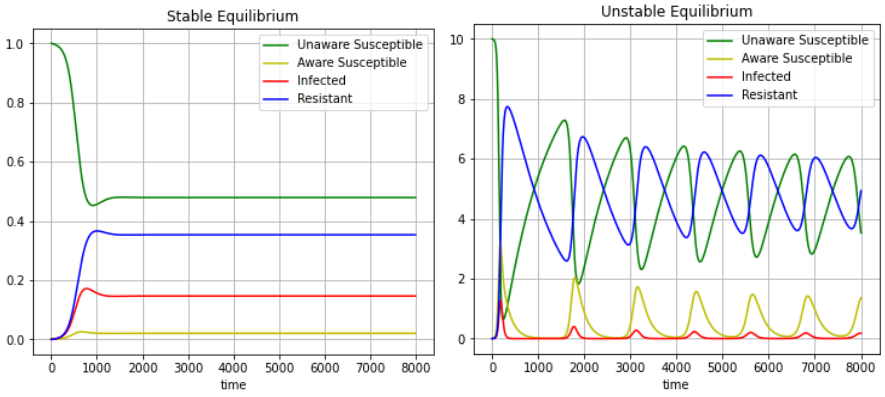


(a) (b)

Figure 4: Endemic equilibria for two systems with infected-dependent awareness spread. Parameters used on the left are $N = 1, \beta = 2, \mu = \alpha = 0.5, u = 1, \nu = 2$, and on the right $N = 10$, $\beta = 1$, $\mu = 0.1$, $\alpha = 5$, $u = 0.01$ and $\nu = 0.5$.

The population dynamics of these two systems when $a_I = 2$ are shown in figure 5. In 5a, a reduced, but endemic presence of infected creates a constant pressure for vaccinations, keeping the equilibrium stable. Meanwhile, in the system depicted in 5b, the populations oscillate around the unstable equilibrium. The vaccination ends up

being 'too effective'. The epidemic is virtually over, but with it the population also loses its motivator for the vaccinations. As the population turnover brings new, unvaccinated individuals and takes away those with acquired resistance, the population of unaware susceptibles S_U grows large enough that the epidemic can start to grow again.



(a) (b)

Figure 5: Population dynamics of two systems with infected-dependent awareness spread. Parameters used on the left and right are same as in figure 4, with $a_I = 2$.

3.3.3 Aware-Dependent Awareness Spread

Since the aware immune population R_A now also contributes to the spread of awareness and is therefore unignorable, we must now take into account the total aware population A as well as the population of aware susceptibles S_A when examining the stability of this equilibrium. This leads us to the system

$$\dot{S} = \mu N - \beta SI - \nu S_A - \mu S, \tag{43a}$$
$$\dot{S}_A = a_A \left(S - S_A\right) A - \left(\beta I + \mu + u + \nu\right) S_A \tag{43b}$$
$$\dot{I} = \beta SI - \left(\alpha + \mu\right) I, \tag{43c}$$
$$\dot{A} = a_A A \left(N - A - I\right) + \left(\alpha - \beta S_A\right) I - \left(u + \mu\right) A, \tag{43d}$$

With the third function $\gamma_A = a_A A$ equation (32) becomes

$$\frac{R_0 \nu}{N} \bar{S}_A^2 - \left(\mu R_0 + u + \nu + a_A \bar{A}\right) \bar{S}_A + a_A \bar{A} \bar{S} = 0 \tag{44}$$

and to solve it we first need an expression for $\bar{A}$. By adding equations (11b) and (20c) we get

$$\dot{A} = a_A U A + \left(\alpha - \beta S_A\right) I - \left(u + \mu\right) A.$$

By using the expressions $U = N - A - I$ and $\bar{I} = \bar{I}_0 - b\bar{S}_A$ and evaluating the above equation at zero, from the quadratic equation

$$-a_A\bar{A}^2 + \left(a_A N - a_A\bar{I}_0 - u - \mu\right)\bar{A} + \left(\alpha\bar{I}_0 - \left(b\bar{S}_A + \beta\bar{I}_0\right)\bar{S}_A + b\beta\bar{S}_A^2\right)$$

we can solve the equilibrium population of all aware $\bar{A}$ as a function of $\bar{S}_A$

$$\begin{aligned}\bar{A}_\pm =& \frac{a_A\left(N - \bar{I}_0\right) + a_A b\bar{S}_A - u - \mu}{2a_A} \\ &\pm \frac{\sqrt{\left(a_A\left(N - \bar{I}_0\right) + a_A b\bar{S}_A - u - \mu\right)^2 + 4a_A\left(\alpha\bar{I}_0 - \left(b\bar{S}_A + \beta\bar{I}_0\right)\bar{S}_A + b\beta\bar{S}_A^2\right)}}{2a_A}\end{aligned}$$

and insert it to equation (44) to solve $\bar{S}_A$.

The Jacobian of system (43) in the endemic equilibrium is now

$$M_A = \begin{bmatrix} -\left(\beta\bar{I} + \mu\right) & -\nu & -\left(\alpha + \mu\right) & 0 \\ a_A\bar{A} & -\left(a\bar{A} + \beta\bar{I} + \mu + \nu + u\right) & -\beta\bar{S}_A & a\left(\bar{S} - \bar{S}_A\right) \\ \beta\bar{I} & 0 & 0 & 0 \\ 0 & -\beta\bar{I} & -a_A\bar{A} + \alpha - \beta\bar{S}_A & a_A\left(N - \bar{I} - 2\bar{A}\right) - (u + \mu) \end{bmatrix}.$$

The characteristic polynomial will be a fourth-order polynomial

$$\begin{aligned}\Phi_A(\lambda) &= \det\left(M_A - \lambda I\right) \\ &= \lambda^4 + C_3\lambda^3 + C_2\lambda^2 + C_1\lambda + C_0.\end{aligned}$$

According to the Routh-Hurwitz criterion, this equilibrium is stable if and only if all coefficients C_0–C_3, as well as the terms $(C_3C_2 - C_1)$ and $(C_3C_2 - C_1)C_1 - C_3^2C_4$, are positive.

To get an idea of the behaviour of this system, we will fix the parameters to those used in figure (6). In this case, only $\bar{A}_-$ crosses with the S_A-nullcline

$$\bar{A} = \frac{\left(\beta\bar{I} + \mu + u + \nu\right)\bar{S}_A}{a_A\left(\bar{S} - \bar{S}_A\right)}$$

in the biologically admissable $[0, N] \times [0, N]$-space and it is therefore the equilibrium of population A we want. Figure (6) shows the endemic equilibria as parameter a_A is grown. When the constant a_A is below threshold (29), the endemic equilibrium is asymptotically stable, as it fulfills the Routh-Hurwitz criterion given above, with a decreased infected population $\bar{I}$ in the equilibrium. Above this threshold, the infected population $\bar{I}$ is negative and the only biologically viable equilibrium is the asymptotically stable disease-free equilibrium as calculated in (24).

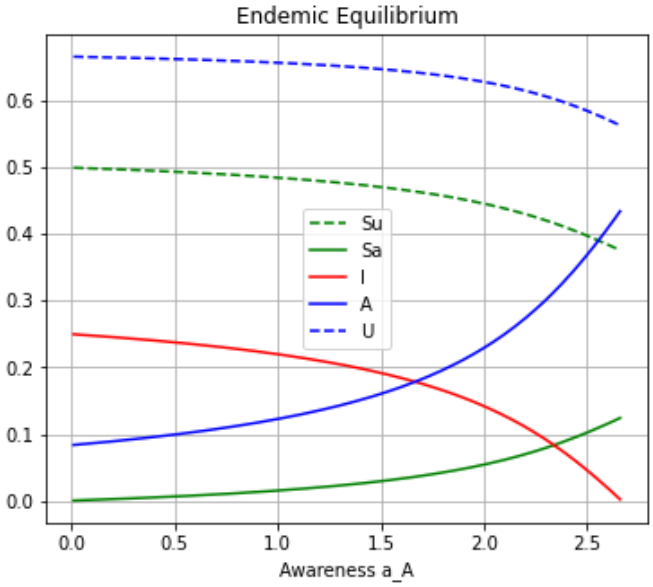


Figure 6: Endemic equilibrium for a system with aware-dependent awareness spread. The parameters are $N = 1, \beta = 2, \mu = \alpha = 0.5, u = 1, \nu = 2$.

3.4 Effect of Awareness on Final Size of Epidemic

We can again consider the special case $\mu = 0$ and study how the spread of awareness can influence the final size of the epidemic. Unfortunately, unlike with the ordinary SIR-model, it does not seem to be possible to find any explicit answer for the final size of the unaffected population, and so we will move to numerical evalutations and example cases.

We'll choose suitable parameters for an example case. We'll choose N=1 and hence deal with relative proportions of total population rather than with absolute population numbers. Measuring time in weeks, let's assume that the expected time an individual spends infected is two weeks. This requires that the parameter $\alpha = 0.5$. We declare the reproduction number of the disease to be three, which requires the parameter β to be 1.5. The expected time a susceptible individual aware of the epidemic around him gets vaccinated is half a week and the expected time they will forget the epidemic will be a week. This means that $\nu = 2$ and $u = 1$.

In figuring out the portion of the population who are unaffected by the epidemic, we must consider not just those who manage to stay susceptible, but also those who become aware and get vaccinated. This means adding to the system $\left(\dot{S}, \dot{S}_A, \dot{I}, \dot{A}\right)$ the differential equation

$$\dot{V} = \nu S_A$$

to describe the growth of the vaccinated population V. Then, for a given value of a, we can simply use Euler's method with timestep $h = 0.01$ and initial values $(S, S_A, I, A, V) =$

$(0.98, 0, 0.01, 0.01, 0)$ to evaluate the size of each group. By step 1000, the epidemic will have died down and the susceptible and the vaccinated population will not change in any meaningful way. The figure 7 below shows the portion of the population that is neither susceptible nor vaccinated at this late stage of the epidemic for the given value of awareness parameter a_X.

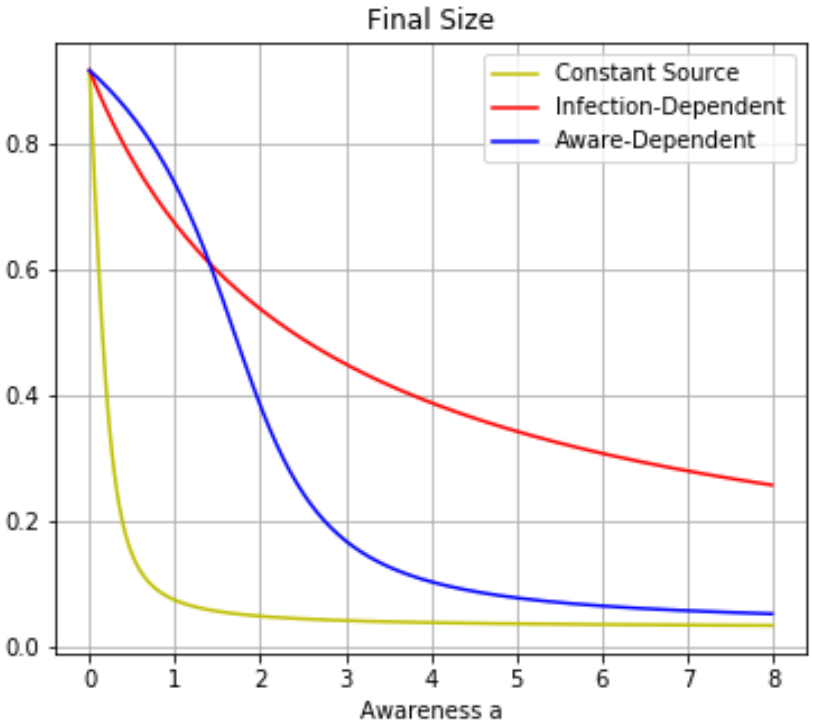


Figure 7: The final size of the epidemic affected by the spreading of awareness. Parameters are $N = 1, \beta = 1.5, \alpha = 0.5, u = 1, \nu = 2$.

It is important to understand that these graphs are not comparable, as the different awareness parameters a describe entirely different processes and achieving the same values involve different things in different models.

3.5 The Limiting Model

As we have seen in the previous chapter, model (11) could be at best reduced down to a system of three equations, the analysing of which was still a considerable effort. One way to simplify this model even further is to add an assumption that the aware susceptibles will not waste time with the vaccination, viz. that ν is very large. This effectively removes the aware susceptibles from the equations by keeping S_A close to zero, as any aware individual quickly vaccinates himself and becomes immune. If we also assume that γ is a function of the susceptible population S and the infected population I, the

equations for the simplified model are

$$\begin{aligned}\dot{S} &= \mu N - \beta SI - \gamma(S,I) - \mu S \\ \dot{I} &= \beta SI - \alpha I - \mu I \\ \dot{R} &= \alpha I + \gamma(S,I) - \mu R.\end{aligned}$$

Since the equation for $\dot{I}$ remains unchanged, the results we derived from it, the value for R_0 and the equilibrium of susceptible population S in (30), are also valid now.

Continuing with the equation $\dot{S} = 0$ and using the fact that $\beta\bar{S} = \alpha + \mu$, we get the equation

$$(\alpha + \mu)\,\bar{I} = \mu\left(N - \bar{S}\right) - \gamma\left(\bar{S}, \bar{I}\right).$$

Dividing both sides with $\alpha + \mu$ we get the equilibrium for the infected population

$$\begin{aligned}\bar{I} &= \frac{\mu}{\alpha+\mu}\left(N - \bar{S}\right) - \frac{\gamma\left(\bar{S},\bar{I}\right)}{\alpha+\mu} \\ &= \frac{\mu}{\beta}\left(R_0 - 1\right) - \frac{\gamma\left(\bar{S},\bar{I}\right)}{\alpha+\mu}.\end{aligned} \tag{45}$$

3.5.1 Existence of Equilibria

The spread of information decreases the total number of infected, depending on the function $\gamma(S,I) \geq 0$. We'll again use simple functions of S and I. First, using the γ-function $\gamma_C\left(\bar{S},\bar{I}\right) = a_C\bar{S} = a_C\frac{\alpha+\mu}{\beta}$, the above equation for $\bar{I}$ becomes

$$\bar{I} = \frac{\mu}{\beta}\left(R_0 - 1\right) - \frac{a_C}{\beta},$$

which can be rearranged to the form

$$\bar{I} = \frac{\mu}{\beta}\left(R_0 - \left(1 + \frac{a_C}{\mu}\right)\right),$$

resembling the equilibrium (4) for the infected population in the basic SIR-model.

Next choice is the $\gamma_I\left(\bar{S},\bar{I}\right) = a_I\bar{S}\bar{I} = a_I\frac{\alpha+\mu}{\beta}\bar{I}$, which the spread of awareness through non-infecting contact with infected. Inserting this into equation (45) and gathering the terms containing $\bar{I}$ on the left side gives us

$$\left(1 + \frac{a_I}{\beta}\right)\bar{I} = \frac{\beta + a_I}{\beta}\bar{I} = \frac{\mu}{\beta}\left(R_0 - 1\right).$$

Multiplying both sides with the constant $\frac{\beta+a_I}{\beta}$ now gives the equilibrium

$$\bar{I} = \frac{\mu}{\beta + a_I}\left(R_0 - 1\right).$$

Adapting the aware-dependent awareness spread to the limiting model is more complicated. Even if the model doesn't have aware susceptibles, the aware recovered R_A,

maintaining their awareness after their recovery or vaccination, could still propagate awareness. Since the point of the limiting model is to simplify the model down to just two unknowns, we shall add the additional assumption of no loss of awareness, which allows us to express the recovered population as $R = R_A = N - I - S$. Now, with $\gamma_A\left(\bar{S}, \bar{I}\right) = a_A \bar{S}\bar{R} = a_A\left(N - \bar{S} - \bar{I}\right)\bar{S}$, the requirement $\dot{S} = 0$ becomes

$$(\beta - a_A)\,\bar{I}\bar{S} = \mu N - \mu\bar{S} - a_A\bar{S}\left(N - \bar{S}\right)$$

When $\beta - a_A \neq 0$, the endemic equilibrium for the infected population would be

$$\bar{I} = \frac{\mu}{\beta - a_A}\left(R_0 - 1\right) - \frac{a_A}{\beta - a_A}\left(N - \bar{S}\right). \tag{46}$$

Since this equilibrium needs to also make biological sense, we need further requirements. When $a_A < \beta$, $\bar{I}$ will be negative if

$$a_A > \frac{\mu\left(R_0 - 1\right)}{N - \bar{S}}.$$

When $a_A > \beta$, the endemic equilibrium will be positive, but it will still not be biologically viable, since it will be larger than $N - \bar{S}$. From (46), we have

$$\bar{I} = \frac{a_A}{a_A - \beta}\left(N - \bar{S}\right) - \frac{\beta}{a_A - \beta}\frac{\mu}{\beta}\left(R_0 - 1\right) > N - \bar{S}.$$

Dividing both sides with $N - \bar{S} > 0$ gives us the inequality

$$\frac{a_A}{a_A - \beta} - \frac{\beta}{a_A - \beta}\frac{\bar{I}_0}{N - \bar{S}} = \frac{a_A - \frac{\bar{I}_0}{N - \bar{S}}\beta}{a_A - \beta} > 1.$$

This is always true, since $\bar{I}_0/\left(N - \bar{S}\right)$ is less than one; the population of non-susceptibles $N - \bar{S}$ consists of both the infected and the recovered and therefore

$$N - \bar{S} - \bar{I}_0 = \frac{N}{R_0}\left(R_0 - 1\right) - \frac{\mu}{\beta}\left(R_0 - 1\right) = \left(\frac{\alpha + \mu}{\beta} - \frac{\mu}{\beta}\right)\left(R_0 - 1\right) > 0.$$

The endemic equilibrium for the aware-dependent awareness spread thus exists only when

$$a_A < \frac{\mu\left(R_0 - 1\right)}{N - \bar{S}} = \frac{\bar{I}_0}{N - \bar{S}}\beta < \beta. \tag{47}$$

3.5.2 Stability in Limiting Models

The equilibria for the limiting models with constant and infected-dependent awareness spread are always asymptotically stable, as the Jacobians

$$M_C = \begin{bmatrix} -\beta\bar{I} - (a_C + \mu) & -\beta\bar{S} \\ \beta\bar{I} & \beta\bar{S} - (\alpha + \mu) \end{bmatrix}$$

and

$$M_I = \begin{bmatrix} -(a_I + \beta)\bar{I} - \mu & -(a_I + \beta)\bar{S} \\ \beta\bar{I} & \beta\bar{S} - (\alpha + \mu) \end{bmatrix}$$

give us characteristic equations $\Phi(\lambda) = M - \lambda I$ that are second-order polynomials of λ with all coefficients positive and thus from the Routh-Hurwitz criterion we can determine that the eigenvalues have negative real parts. Notably, the limiting model with infected-dependent awareness spread has an endemic equilibrium that is always asymptotically stable, unlike the model in 3.3.1.

The stability of the equilibrium in the system with aware-dependent awareness spread is, at least at first sight, a slightly more complicated question. In this case the Jacobian

$$M_A = \begin{bmatrix} 2a_R\bar{S} - (\beta - a_A)\bar{I} - \mu & -(\beta - a_A)\bar{S} \\ \beta\bar{I} & \beta\bar{S} - (\alpha + \mu) \end{bmatrix}$$

has the characteristic equation

$$\Phi_A(\lambda) = \lambda^2 + \left(\mu + (\beta - a_A)\bar{I} - 2a_A\bar{S}\right)\lambda + \beta(\beta - a_A)\bar{S}\bar{I}.$$

If $a_A < \beta$, the stability of the equilibria now depends on the sign of the first order coefficient. Expanding it to a function of a_R gives the inequality

$$\mu + \mu(R_0 - 1) - a_A\left(N - \bar{S}\right) - 2a_A\bar{S} = \mu R_0 - a_A\left(N + \bar{S}\right) > 0,$$

the solutions of which are

$$a_A < \frac{\mu R_0}{N + \bar{S}}. \tag{48}$$

Remembering (47), we can show that

$$\frac{\mu(R_0 - 1)}{N - \bar{S}} > \frac{\mu R_0}{N + \bar{S}},$$

meaning that as a_A grows, the endemic equilibrium changes stability before it vanishes. Multiplying both sides with the denominators gives us the inequality

$$\mu(R_0 - 1)\left(N + \bar{S}\right) > \mu R_0\left(N - \bar{S}\right),$$

and rearranging the terms shows that this is equivalent to

$$R_0\left(N + \bar{S} - N + \bar{S}\right) = 2\frac{N}{\bar{S}}\bar{S} = 2N > N + \bar{S},$$

which is always true. The endemic equilibrium will be positive and asymptotically stable, when a_A is under limit (48), becomes unstable when a_A is between (48) and (47) and finallly ceases to be biologically viable after a_A passes (47).

3.5.3 Final Size of the Epidemic

Finally we will determine the final size of an epidemic when $\mu = 0$ and awareness of the epidemic depends on the non-infectious contacts between susceptibles and infected. In addition of the population S left susceptible at the end of the epidemic, we now have to also consider the vaccinated population V, with $\dot{V} = aSI$. We will assume that the pre-epidemic population is entirely unaware and unvaccinated. We can write

$$\begin{aligned}\frac{\mathrm{d}I}{\mathrm{d}S} &= \frac{\beta SI - \alpha I}{-\left(a_I + \beta\right) SI} \\ &= -\frac{\beta}{a_I + \beta} + \frac{\alpha}{a_I + \beta}\frac{1}{S}.\end{aligned}$$

Like in the basic SIR-model, we can now separate the variables and integrate both sides to gain the identity

$$I\left(t\right) = -\frac{\beta}{a_I + \beta} S\left(t\right) + \frac{\alpha}{a_I + \beta} \ln S\left(t\right) + C$$

valid for all values of time t. Specifically, we can see that

$$I\left(-\infty\right) + \frac{\beta}{a_I + \beta} S\left(-\infty\right) - \frac{\alpha}{a_I + \beta} \ln S\left(-\infty\right) = I\left(\infty\right) + \frac{\beta}{a_I + \beta} S\left(\infty\right) - \frac{\alpha}{a_I + \beta} \ln S\left(\infty\right).$$

Since $I\left(-\infty\right) = I\left(\infty\right) = 0$ and $S\left(-\infty\right) = N$, we can write the above as

$$\ln s\left(\infty\right) = -R_0\left(1 - s\left(\infty\right)\right),$$

which is the same formula as in (10) for the basic SIR-model with $s = S/N$. The reduction in the final size of the epidemic is then from the vaccinated population $V\left(\infty\right)$. From the equation

$$\frac{\mathrm{d}S}{\mathrm{d}V} = \frac{-\left(a_I + \beta\right) SI}{a_I SI} = \frac{\beta + a_I}{a_I}$$

we can again separate the variables to give the identity

$$S\left(t\right) = -\frac{\beta + a_I}{a_I} V\left(t\right) + C$$

for all values of time t. This leads us to the equation

$$S\left(-\infty\right) + \frac{\beta + a_I}{a_I} V\left(-\infty\right) = S\left(\infty\right) + \frac{\beta + a_I}{a_I} V\left(\infty\right).$$

The fraction $v\left(\infty\right) = \frac{V(\infty)}{N}$ of the population vaccinated and shielded from the epidemic at the end is thus

$$v\left(\infty\right) = \frac{a_I}{a_I + \beta}\left(1 - s\left(\infty\right)\right).$$

Essentially, in the portion $1 - s\left(\infty\right)$ of the population that has gained immunity to the disease by the end of the epidemic, $a_I / \left(a_I + \beta\right)$ of these gained it through vaccination and avoided infection.

4 Lattice Model

In this section we will now switch to a different perspective. Previously we have assumed that the population is well-mixed and that an infected individual is an equal threat to everyone else in the population. This is of course simplistic, since people have social networks and interact far more with others inside the network than with strangers outside it. To take these connections into account, instead of analysing the densities of subpopulations as before, we will now shift our focus to the densities of particular connections, or pairs, of individuals in the population.

The model consists of an infinite lattice of sites, where each site is connected to a number z of other sites. Here the word 'lattice' does not necessarily have to imply a spatial regularity within this network of sites. The particular structure, or the lack of one, can tell us how we will close the equations [8], as we will see in 4.2. Each has a state σ. In addition to the previously mentioned states S, I and R, we will now also consider uninhabitated sites with state 0. We will also mark sites inhabited by any of the states S, I, R with X. The density ρ_σ is the probability that a randomly chosen site has the state σ.

We will also define the conditional probability $q_{\sigma'/\sigma}$ that a randomly chosen site with state σ is connected to a site with state σ'. Then the density of pairs $[\sigma\sigma']$ can be written as

$$P_{\sigma\sigma'} = \rho_\sigma q_{\sigma'/\sigma} = \rho_{\sigma'} q_{\sigma/\sigma'} = P_{\sigma'\sigma}. \tag{49}$$

For the densities ρ_σ holds the identity

$$\sum_\sigma \rho_\sigma = 1 \tag{50}$$

and for the conditional probablilites

$$\sum_{\sigma'} q_{\sigma'/\sigma} = 1. \tag{51}$$

4.1 Deriving the Disease Model

To look at the the behaviour of the model we will derive the equations for the evolution of the population densities ρ_σ and the density of pairs $[\sigma\sigma']$. We will choose the unit of time such that all individuals, regardless of the epidemic, will die at a rate 1 and give birth to a new susceptible into a neighboring empty site at a rate μ. We will later see that the birth rate needs to exceed a threshold higher than the death rate in order for the population to be viable.

The infection will spread between an infected and a neighboring susceptible site at a rate β. The spread of the information is again defined by a function γ which we will define later.

The important detail to notice is that the cause of the change in the state of the pair can come from outside the pair. Namely, the empty site of a $[X0]$-pair can be filled by a birth from a third site connected to the empty site as well as by the other half

of the pair. Similarly infection enters a pair from an outside connected infected site. Therefore, to examine the changes in the $[\sigma\sigma']$-pairs, we are also forced to also consider triples $[\sigma\sigma'\sigma'']$, where a site with state σ' is connected to a site with state σ and another site with state σ''.

Let's take a random site with a susceptible with a neighbouring empty site. This $[S,0]$-pair was formed either when the susceptible was created from another connected inhabited site or when the neighbouring site was emptied after the death of its former occupant.

The $[S,0]$-pair can be destroyed after (a) the death of the susceptible, (b) the vaccination of the susceptible, (c) the susceptible becomes infected by an infected from another connected site, (d) the susceptible populates the empty site or (e) the empty site is populated from the outside. These lead to a differential equation

$$\dot{P}_{S0} = \mu P_{X00} + P_{SX} - P_{S0} - \gamma_{S0} - \beta P_{IS0} - \mu z^{-1} P_{S0} - \mu P_{S0X}.$$

From (49) we can conclude that $\dot{P}_{S0}$ and $\dot{P}_{0S}$ are interchangeable.

In case of a randomly chosen site with a susceptible neighbouring another susceptible, both sites of the pair face the same processes. The pair was formed when either the chosen susceptible or its neighbour was given birth, either by the other or by a third party. The pair ceases to exist when either susceptible dies, becomes vaccinated, or becomes infected by an infected from another connected site. From these considerations we get the differential equation

$$\begin{aligned}\dot{P}_{SS} &= \mu z^{-1} P_{S0} + \mu z^{-1} P_{0S} + \mu P_{S0X} + \mu P_{X0S} \\ &\quad - P_{SS} - P_{SS} - \gamma_{SS} - \gamma_{SS} - \beta P_{SSI} - \beta P_{ISS} \\ &= 2\mu z^{-1} P_{S0} + 2\mu P_{S0X} - 2P_{SS} - 2\gamma_{SS} - 2\beta P_{SSI}.\end{aligned}$$

Following similar process for the other pairs, we end up with the system of differential equations for the pair densities

$$\dot{P}_{00} = 2P_{X0} - 2\mu P_{X00} \tag{52a}$$
$$\dot{P}_{S0} = \mu P_{X00} + P_{SX} - P_{S0} - \gamma_{S0} - \beta P_{IS0} - \mu z^{-1} P_{S0} - \mu P_{S0X} \tag{52b}$$
$$\dot{P}_{I0} = \beta \bar{z} P_{IS0} + P_{IX} - P_{I0} - \alpha P_{I0} - \mu z^{-1} P_{I0} - \mu P_{I0X} \tag{52c}$$
$$\dot{P}_{R0} = \gamma_{S0} + \alpha P_{I0} + P_{RX} - P_{X0} - \mu z^{-1} P_{R0} - \mu P_{R0X} \tag{52d}$$
$$\dot{P}_{SS} = 2\mu z^{-1} P_{S0} + 2\mu P_{S0X} - 2P_{SS} - 2\gamma_{SS} - 2\beta P_{SSI} \tag{52e}$$
$$\dot{P}_{IS} = \mu z^{-1} P_{I0} + \mu P_{I0X} + \beta P_{ISS} - 2P_{IS} - \gamma_{IS} - \alpha P_{IS} - \beta z^{-1} P_{IS} - \beta P_{ISI} \tag{52f}$$
$$\dot{P}_{RS} = \mu z^{-1} P_{R0} + \mu P_{R0X} + \gamma_{SS} + \alpha P_{IS} - 2P_{SS} - \gamma_{RS} - \beta P_{RSI} \tag{52g}$$
$$\dot{P}_{II} = 2\beta^{-1} P_{IS} + 2\beta P_{ISI} - 2P_{II} - \alpha P_{II} \tag{52h}$$
$$\dot{P}_{IR} = \alpha P_{II} + \gamma_{IS} + \beta P_{ISR} - 2P_{IR} - \alpha P_{IR} \tag{52i}$$
$$\dot{P}_{RR} = 2\gamma_{RS} + 2\alpha P_{IR} - 2P_{RR}. \tag{52j}$$

In addition, from the identity (51), we conclude

$$\sum_{\sigma'} P_{\sigma\sigma'} = \rho_\sigma \sum_{\sigma'} q_{\sigma'/\sigma} = \rho_\sigma, \tag{53}$$

giving us the system of equations for the evolution of densities

$$\dot{\rho}_\sigma = \sum_{\sigma'} \dot{P}_{\sigma\sigma'}. \tag{54}$$

4.2 Pair Approximation

The problem of the above system is clear. It is not closed, as it still depends on the densities of triplet P_{ABC}. Closing this system is in fact not possible, as the number of triples would in return depend on quadruples and so on infinitely. To create a solvable system, we need to approximate the number of triples as an expression of the doubles. One customary way of doing this is called *pair approximation* [3]. This requires us to bring an additional assumption to consideration. We will assume from now on that in a triple ABC, where the sites A and C are connected to site B, they will be independent of each other. This allows us to close the system; if every site is connected to number z of other sites, then the number of connections to a B-site already connected to a A-site is $(z-1)\,[AB]$. If the remaining connections are independent of the A-site, then the fraction of those connections that are to a C-site should be equal to the fraction $[BC]\,/z\,[B]$ of all connections to B-sites that pair them with C-site. Thus the probability of a ABC-triplet can be expressed as

$$P_{ABC} = P_{AB}\,(z-1)\,\frac{P_{BC}}{z\rho_B} = \frac{z-1}{z} q_{C/B} P_{AB}.$$

In the future we will set the constant $\bar{z} = (z-1)/z$. With the above approximation we can write

$$\dot{P}_{00} = 2P_{X0} - 2\mu\bar{z}q_{X/0}P_{00} \tag{55a}$$

$$\dot{P}_{S0} = \mu\bar{z}q_{X/0}P_{00} + P_{SX} - P_{S0} - \gamma_{S0} - \beta\bar{z}q_{I/S}P_{S0} - \left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{S0} \tag{55b}$$

$$\dot{P}_{I0} = \beta\bar{z}q_{I/S}P_{S0} + P_{IX} - P_{I0} - \alpha P_{I0} - \left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{I0} \tag{55c}$$

$$\dot{P}_{R0} = \gamma_{S0} + \alpha P_{I0} + P_{RX} - P_{R0} - \left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{R0} \tag{55d}$$

$$\dot{P}_{SS} = 2\left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{S0} - 2P_{SS} - 2\gamma_{SS} - 2\beta\bar{z}q_{I/S}P_{SS} \tag{55e}$$

$$\dot{P}_{IS} = \left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{I0} + \beta\bar{z}q_{I/S}P_{SS} - 2P_{IS} - \gamma_{IS} - \alpha P_{IS} - \left(\beta z^{-1} + \beta\bar{z}q_{I/S}\right)P_{IS} \tag{55f}$$

$$\dot{P}_{RS} = \left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{R0} + \gamma_{SS} + \alpha P_{IS} - 2P_{RS} - \gamma_{RS} - \beta\bar{z}q_{I/S}P_{RS} \tag{55g}$$

$$\dot{P}_{II} = 2\left(\beta z^{-1} + \beta\bar{z}q_{I/S}\right)P_{IS} - 2P_{II} - 2\alpha P_{II} \tag{55h}$$

$$\dot{P}_{IR} = \alpha P_{II} + \gamma_{IS} + \beta\bar{z}q_{I/S}P_{SR} - 2P_{IR} - \alpha P_{IR} \tag{55i}$$

$$\dot{P}_{RR} = 2\gamma_{SR} + 2\alpha P_{IR} - 2P_{RR}. \tag{55j}$$

4.3 Defining the γ-terms

The only terms left to be defined are the γ-functions. We'll again simplify the model by assuming that once a susceptible becomes aware, they'll quickly seek and recieve vaccination. This allows us to equate the spread of awareness with the spread of the vaccinations. We'll adapt the three approaches to awareness spread we've been using before to the lattice model. The first approach involves the susceptible in a Sσ-pair "spontaneously" becoming aware and vaccinated due to a constant force ν_C acting on the population. This means that

$$\gamma_{S\sigma} = \nu_C P_{S\sigma}. \tag{56}$$

The other possibility for the spread of awareness on which we will concentrate is through the recovered population. In case of a SR-pair, the susceptible half of the pair can be influenced either by the recovered of the pair or by a recovered individual outside the pair but connected to the susceptible individual. Applying pair approximation, this approach gives the γ-function

$$\gamma_{SR} = \nu_R \left(z^{-1} + \bar{z} q_{R/S}\right) P_{SR}. \tag{57}$$

For Sσ-pairs, where state $\sigma \neq R$, awareness can still spread from an outside recovered individual connected to the susceptible. This means that in these cases

$$\gamma_{S\sigma} = \nu_R \bar{z} q_{R/S} P_{S\sigma}. \tag{58}$$

The third possible vector of awareness spread, non-infectious contact with infected, would yield the same γ-functions with I replacing R. However, from here on we will focus on the disease-free equilibria and therefore the study of the infected-dependent awareness spread will be left for another time.

4.4 Equilibria in the Lattice Model

We can now start to study the equilibria of the system (55). Taking the first equation (55a) and setting it to zero gives us

$$q_{0/0} = \frac{1}{\mu \bar{z}},$$

and as a consequence, since $q_{X/0} = 1 - q_{0/0}$, we get

$$q_{X/0} = \frac{\mu \bar{z} - 1}{\mu \bar{z}}.$$

Furthermore, we can now simplify the term $\left(\mu z^{-1} + \mu \bar{z} q_{X/0}\right)$ found in the equations as

$$\frac{\mu}{z} + \mu \bar{z} - 1 = \mu - 1.$$

Finally the existence of the population requires that the conditional probability $q_{0/0}$ is less than 1. Thus we get condition for the fertility rate

$$\mu > \frac{1}{\bar{z}} = \frac{z}{z-1} > 1.$$

All these results above do not depend on the spread of either the disease or the awareness and thus they will carry to all of the model variations we'll be going through.

4.4.1 Constant spread of awareness

We will continue with the equilibrium points for the densities ρ_σ in case of constant spread of awareness with function γ defined as in equation (56). From the equation $\dot{\rho}_0 = \sum_\sigma \dot{P}_{\sigma 0} = 0$, we get

$$\rho_S + \rho_I + \rho_R - \mu\bar{z}\rho_0 q_{0/0} - (\mu - 1)\,\rho_0 q_{X/0} = 1 - \rho_0 - \mu q_{X/0}\rho_0 = 0,$$

where we can solve the density

$$\rho_0 = \frac{1}{1 + \mu q_{X/0}} = \frac{\bar{z}}{\mu\bar{z} + \bar{z} - 1}. \tag{59}$$

Similarly we get the equilibrium for the density of the susceptible population

$$\rho_s = \frac{\mu q_{X/0}}{\beta q_{I/S} + \nu_C + 1}\rho_0 = \frac{\mu\bar{z} - 1}{\bar{z}(\beta q_{I/S} + \nu_C + 1)}\rho_0, \tag{60}$$

the infected population

$$\rho_I = \frac{\beta q_{I/S}}{\alpha + 1}\rho_S, \tag{61}$$

and the recovered population

$$\rho_R = \nu_C\rho_S + \alpha\rho_I. \tag{62}$$

Using the identity (53), we can see from the equation $\dot{P}_{S0} = 0$ that

$$\rho_S + (\mu\bar{z} - 1)\,\rho_0 q_{X/0} = \left(\beta\bar{z}q_{I/S} + \nu_C + \mu + 1\right)\rho_S q_{0/S}.$$

Dividing this with $\rho_S\,(\nu_C + \mu + 1)$ we have the conditional probability

$$q_{0/S} = \frac{\rho_S + \rho_0 q_{X/0}}{\beta\bar{z}q_{I/S} + \nu_C + \mu + 1} = \frac{\beta q_{I/S} + \nu_C + \mu + 1}{\mu\left(\beta\bar{z}q_{I/S} + \nu_C + \mu + 1\right)}. \tag{63}$$

Similarly from the other equations we get the rest of the conditional probabilities

$$q_{0/R} = \frac{1}{\mu+1}\left[\frac{\nu_C\rho_S}{\nu_C\rho_S+\alpha\rho_I}\left(1+q_{0/S}\right)+\frac{\alpha\rho_I}{\nu_C\rho_S+\alpha\rho_I}\left(1+q_{0/I}\right)\right] \tag{64a}$$

$$q_{S/S} = \frac{\mu-1}{\beta\bar{z}q_{I/S}+\nu_C+1}q_{0/S} \tag{64b}$$

$$q_{S/R} = \frac{1}{\nu_C+2}\left[\frac{\nu_C\rho_S}{\nu_C\rho_S+\alpha\rho_I}q_{S/S}+(\mu-1)\,q_{0/R}\right] \tag{64c}$$

$$q_{R/R} = \nu_C q_{S/R}+\alpha q_{I/R} \tag{64d}$$

$$q_{I/0} = \frac{\beta}{\mu+\alpha+1}\frac{\rho_S}{\rho_0}\left(\frac{1}{\alpha+1}+\bar{z}q_{0/S}\right)q_{I/S} \tag{64e}$$

$$q_{I/I} = \frac{\beta\left(z^{-1}+\bar{z}q_{I/S}\right)}{\alpha+1}q_{I/S} \tag{64f}$$

$$q_{I/R} = \frac{1}{\alpha+2}\left[\frac{\alpha\rho_I}{\alpha\rho_I+\nu_C\rho_S}q_{I/I}+\left(\frac{\nu_C\rho_S}{\alpha\rho_I+\nu_C\rho_S}+\beta\bar{z}q_{S/R}\right)q_{I/S}\right]. \tag{64g}$$

4.4.2 Stability of the disease-free equilibrium

From setting the density ρ_I to zero, we can see that there exists a disease-free equilibrium. Equation (61) leads us to conclude that in this case the conditional probability $q_{I/S}=0$, and since in the equilibrium (64) the conditional probabilities $q_{I/\sigma}$ all depend on $q_{I/S}$, they too are zero. These reduce the rest of the conditional probabilities in the equilibrium (64) to

$$q_{0/S} = q_{0/R} = \frac{1}{\mu} \tag{65a}$$

$$q_{S/S} = q_{S/R} = \frac{\mu-1}{\nu_C+1}q_{0/S} = \frac{\mu-1}{\mu\left(\nu_C+1\right)} \tag{65b}$$

$$q_{R/R} = \nu_C q_{S/R} = \frac{\nu_C\left(\mu-1\right)}{\mu\left(\nu_C+1\right)}. \tag{65c}$$

We'll establish now the stability of the disease-free equilibrium by analysing the dynamics in its neighbourhood. Near the disease-free equilibrium, density ρ_I and probabilities $q_{I/\sigma}$ are positive and close to zero. The epidemic will not spread in the neighborhood of the epidemic-free equilibrium if

$$\begin{aligned}\dot{\rho}_I &= \dot{P}_{I0}+\dot{P}_{IS}+\dot{P}_{II}+\dot{P}_{IR}\\ &= \beta\bar{z}q_{I/S}\left(\rho_S-P_{IS}\right)+\left(\beta z^{-1}+\beta\bar{z}q_{I/S}\right)P_{IS}-\left(1+\alpha\right)\rho_I<0,\end{aligned}$$

which is equivalent to

$$\rho_I\left(\beta\bar{z}q_{S/I}+\beta z^{-1}q_{S/I}-\left(1+\alpha\right)\right)<0 \tag{66}$$

This gives the condition

$$q_{S/I} < \frac{\alpha + 1}{\beta} \tag{67}$$

for the prevention of the epidemic.

Next we need an expression for the conditional probability $q_{S/I}$ near the disease-free equilibrium to compare with the requirement (67). The dynamics of $q_{S/I}$ are described by the differential equation

$$\begin{aligned}\dot{q}_{S/I} &= \frac{d}{dt}\left(\frac{P_{SI}}{\rho_I}\right) = \frac{1}{\rho_I}\left(\dot{P}_{SI} - q_{S/I}\dot{\rho}_I\right) \\ &= (\mu - 1)\, q_{0/I} + \left(\beta \bar{z} q_{S/S} - (\nu_C + 1) - \frac{\beta}{z}\right) q_{S/I} - \beta q_{S/I}^2.\end{aligned} \tag{68}$$

To calculate $q_{S/I}$, we need $q_{0/I}$, which similarly has the differential equation

$$\begin{aligned}\dot{q}_{0/I} &= \frac{d}{dt}\left(\frac{P_{I0}}{\rho_I}\right) = \frac{1}{\rho_I}\left(\dot{P}_{I0} - q_{0/I}\dot{\rho}_I\right) \\ &= 1 + \beta \bar{z} q_{0/S} q_{S/I} - q_{0/I}\,(1 + \alpha + \mu) - \beta q_{S/I} q_{0/I} + (\alpha + 1)\, q_{0/I}.\end{aligned} \tag{69}$$

Since near the disease-free equilibrium $\rho_I < \epsilon$ for some very small $\epsilon > 0$, then from (66) we see that $\dot{\rho}_I = \mathcal{O}(\epsilon)$. Meanwhile the equations (68) and (69) are $\mathcal{O}(1)$ and therefore show that $q_{0/I}$ and $q_{S/I}$ are fast processes compared to the density ρ_I and they will quickly reach their quasi-equilibria near the disease-free equilibrium. From setting equation (69) to zero we get the quasi-equilibrium

$$q_{0/I} = \frac{1 + \beta \bar{z} q_{0/S} q_{S/I}}{\mu + \beta q_{S/I}} = \frac{\mu + \beta \bar{z} q_{S/I}}{\mu\left(\mu + \beta q_{S/I}\right)}. \tag{70}$$

Inserting this into equation (68) and setting the equation to zero gives the third-order polynomial

$$-\beta^2 q_{S/I}^3 \tag{71}$$

$$+\beta\left(\frac{\beta\,(z-1)\,(\mu-1) - \mu z\,(\nu_C+1)^2 - \beta\mu\,(\nu_C+1) - \mu^2 z\,(\nu_C+1)}{\mu z\,(\nu_C+1)}\right) q_{S/I}^2 \tag{71a}$$

$$+\left(\frac{\beta\,(z-1)\,(\mu-1)\,(\nu_C+\mu+1) - \mu^2 z\,(\nu_C+1)^2 - \beta\mu^2\,(\nu_C+1)}{\mu z\,(\nu_C+1)}\right) q_{S/I} \tag{71b}$$

$$+\,(\mu - 1) = 0, \tag{71c}$$

the roots of which are the quasi-equilibrium points of the conditional probability $q_{S/I}$ in the neighbourhood of the epidemic-free equilibrium.

The above third-order polynomial might have more than one root and it is not obvious how many biologically viable roots, if any, exist. However, it is possible to show the existence and the uniqueness of the biologically viable root with the $q_{S/I}$-nullcline and

$q_{0/1}$-nullcline, which can both be expressed as functions of $q_{S/I}$. From equation (68) we solve $q_{0/I}$ as a function

$$q_{0/I} = \frac{\beta q_{S/I}^2 - \left(\beta \bar{z} q_{S/S} - (\nu_C + 1) - \frac{\beta}{z}\right) q_{S/I}}{\mu - 1} =: g_1\left(q_{S/I}\right). \tag{72}$$

We will also define the function $g_2\left(q_{S/I}\right)$ as the $q_{0/I}$-nullcline from (70).

In the $\left(q_{S/I}, q_{0/I}\right)$-space, the $q_{0/I}$-nullcline

$$g_2\left(q_{S/I}\right) = \frac{\mu + \beta \bar{z} q_{S/I}}{\mu\left(\mu + \beta q_{S/I}\right)} \tag{73}$$

is always positive, less than one since $\mu > 1$, and a decreasing function, since

$$g_2'\left(q_{S/I}\right) = -\frac{\beta\left(1 - \bar{z}\right)}{\mu + \beta q_{S/I}} < 0 \; \forall q_{S/I} > 0.$$

Meanwhile we can figure that the $q_{S/I}$-nullcline $g_1\left(q_{S/I}\right)$, as a parabola opening to the top and cutting the origin, will be a increasing function above the $q_{S/I}$-axis. This means that the increasing function g_1 and the decreasing function g_2 can cross at most once in the $[0,1] \times [0,1]$-area.

It's easy to see that $g_1(0) - g_2(0) < 0$. On the other hand,

$$\begin{aligned} g_1(1) - g_2(1) &= \frac{\beta\left(1 - \bar{z} q_{S/S}\right) + (\nu_C + 1) + \frac{\beta}{z}}{\mu - 1} - \frac{\mu + \beta \bar{z}}{\mu(\mu + \beta)} \\ &= \frac{\left(\mu^2 + \mu\beta\right)\left[\beta\left(1 - \bar{z} q_{S/S}\right) + \frac{\beta}{z}\right] + \mu + \beta \bar{z}}{\mu(\mu - 1)(\mu + \beta)} \\ &+ \frac{\left(\mu^2 + \mu\beta\right)(\nu_C + 1) - \left(\mu^2 + \mu\beta \bar{z}\right)}{\mu(\mu - 1)(\mu + \beta)}. \end{aligned}$$

Knowing that both $\bar{z}$ and $q_{S/S}$ are less than one leads us to conclude that $g_1(1) - g_2(1) > 0$ and that therefore the nullclines cross when $q_{S/I} \in [0,1]$. We can then see that there exists an unique, biologically viable quasi-equilibrium for $q_{S/I}$ in the neighbourhood of the disease-free equilibrium, which allows us to use (66) to determine whether a minor outbreak dies out or turns into a major outbreak.

We'll finally look at how the pre-epidemic spread of awareness/vaccination can stablize the disease-free equilibrium and prevent an epidemic in a particular case. Let's take a lattice with $z = 8$. We'll set the rate of infection β as 8, the rate of recovery α as 1 and the fertility rate μ as 2. With these parameters inequality (67) requires that that the conditional probability $q_{S/I}$ is below 0.25 in order to prevent a major epidemic. When ν_C is zero and there are no vaccinated before the introduction of the disease, equation (71) gives us the conditional probability $q_{S/I} \approx 0.352$ and the epidemic will spread.

Since (71) is in fact a quadratic function of ν_C, if we set $q_{S/I}$ to 0.25, we can explicitly solve that the treshold value for a major epidemic not happening is $\widetilde{\nu}_C \approx 0.391$. In the

equilibrium preceding the introduction of the disease, using expression (62) for density ρ_R tells us that the density of the total population is

$$\rho_X = \rho_S + \rho_R = (\nu_C + 1)\,\rho_S.$$

From this we can say that when the pre-epidemic equilbrium is stable, the portion of at least

$$\rho_R = \frac{\nu_C}{\nu_C + 1}\rho_X \approx 0.281\rho_X$$

of the total population has been vaccinated.

4.4.3 Immune-dependent spread of awareness

Similarly to section 4.4.1, we can calculate that the disease-free system

$$\begin{aligned}
\dot{P}_{00} &= 2P_{X0} - 2\mu\bar{z}q_{X/0}P_{00} \\
\dot{P}_{S0} &= \mu\bar{z}q_{X/0}P_{00} + P_{SX} - P_{S0} - \nu_R\bar{z}q_{R/S}P_{S0}. - \left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{S0} \\
\dot{P}_{R0} &= \nu_R\bar{z}q_{R/S}P_{S0} + P_{RX} - P_{R0} - \left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{R0} \\
\dot{P}_{SS} &= 2\left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{S0} - 2P_{SS} - 2\nu_R\bar{z}q_{R/S}P_{SS} \\
\dot{P}_{RS} &= \left(\mu z^{-1} + \mu\bar{z}q_{X/0}\right)P_{R0} + \nu_R\bar{z}q_{R/S}P_{SS} - 2P_{RS} - \nu_R\left(z^{-1} + \bar{z}q_{R/S}\right)P_{SR} \\
\dot{P}_{RR} &= 2\nu_R\left(z^{-1} + \bar{z}q_{R/S}\right)P_{SR}. - 2P_{RR},
\end{aligned}$$

where awareness is now spread in contacts between the susceptible and the immune R, has the equilibrium

$$\rho_S = \frac{\mu\bar{z} - 1}{\left(\nu_R q_{R/S} + 1\right)\left(\mu\bar{z} + \bar{z} - 1\right)}, \tag{74}$$

$$\rho_R = \frac{\nu_R q_{R/S}\left(\mu\bar{z} - 1\right)}{\left(\nu_R q_{R/S} + 1\right)\left(\mu\bar{z} + \bar{z} - 1\right)}, \tag{75}$$

$$q_{0/S} = \frac{\nu_R q_{R/S} + \mu + 1}{\mu\left(\bar{z}\nu_R q_{R/S} + \mu + 1\right)}, \tag{76a}$$

$$q_{0/R} = \frac{1}{\mu+1}\left(1 + \frac{\rho_S}{\rho_R}\nu_R\bar{z}q_{R/S}q_{0/S}\right) = \frac{(\mu+1)\,\nu_R\bar{z}q_{R/S} + (\mu+\bar{z})\,(\mu+1)}{\mu\,(\mu+1)\left(\nu_R\bar{z}q_{R/S} + \mu + 1\right)}, \tag{76b}$$

$$q_{S/S} = \frac{\mu - 1}{\nu_R q_{R/S} + 1}q_{0/S} = \frac{(\mu-1)\left(\nu_R q_{R/S} + \mu + 1\right)}{\left(\nu_R q_{R/S} + 1\right)\mu\left(\bar{z}\nu_R q_{R/S} + \mu + 1\right)}, \tag{76c}$$

$$q_{R/R} = \nu_R\left(z^{-1} + \bar{z}q_{R/S}\right)q_{S/R} = z^{-1} + \bar{z}q_{R/S}. \tag{76d}$$

Since the conditional probabilities must fulfill the requirement

$$q_{0/S} + q_{S/S} + q_{R/S} = 1,$$

we can solve the last conditional probability $q_{R/S}$ by inserting expressions of $q_{0/S}$ and $q_{S/S}$ from (76). Multiplying with the denominators gives the equation

$$\begin{aligned}&\left(\nu_R q_{R/S}+1\right)\left(\nu_R q_{R/S}+\mu+1\right)+(\mu-1)\left(\nu_R q_{R/S}+\mu+1\right)\\&+\left(q_{R/S}-1\right)\left(\nu_R q_{R/S}+1\right)\mu\left(\bar{z}\nu_R q_{R/S}+\mu+1\right)=0.\end{aligned}$$

The constant terms cancel each other, leaving us with the equation $q_{R/S}f\left(q_{R/S}\right)=0$, where the function

$$\begin{aligned}f\left(q_{R/S}\right)=&\mu\bar{z}\nu_R^2 q_{R/S}^2+\nu_R\left(\mu\bar{z}+\mu^2+\mu+\nu_R-\mu\bar{z}\nu_R\right)q_{R/S}\\&+\mu(\mu-1)-\left(\left(\mu^2-\mu\right)+(\mu\bar{z}-1)\right)\nu_R.\end{aligned}$$

In the constant awareness model, where the pressure for awareness comes from the 'outside' of the population and is not affected by it, the conditional probability

$$q_{R/S}=\frac{\rho_R}{\rho_S}q_{S/R}=\frac{\nu_C(\mu-1)}{\mu(\nu_C+1)}$$

is always strictly positive for any ν_C larger than zero. On the other hand, here the awareness essentially spreads like an epidemic and thus ν_R has to be sufficiently high to create a strictly positive $q_{R/S}$ and thus an equilibrium with a sustainable population of immune individuals.

We can figure how large ν_R has to be from function $f\left(q_{R/S}\right)$. The second-degree coefficient of $f\left(q_{R/S}\right)$ is always positive. The constant coefficient is positive when

$$\nu_R<\frac{\mu(\mu-1)}{\mu(\mu-1)+(\mu\bar{z}-1)}.$$

When ν_R is under this threshold, the coefficient of the linear term is also positive, as

$$\begin{aligned}&\mu\bar{z}+\mu^2+\mu-(\mu\bar{z}-1)\nu_R\\&>\mu\bar{z}+\mu^2+\mu-\frac{\mu(\mu-1)(\mu\bar{z}-1)}{\mu(\mu-1)+(\mu\bar{z}-1)}\\&=\frac{\mu\left(\mu\bar{z}+\mu^2+\mu\right)(\mu-1)+(\mu\bar{z}+\mu)(\mu\bar{z}-1)}{\mu(\mu-1)+(\mu\bar{z}-1)}+\frac{(\mu-\mu+1)(\mu\bar{z}-1)}{\mu(\mu-1)+(\mu\bar{z}-1)}>0.\end{aligned}$$

This leads us to conclude that the existence of a strictly positive $q_{R/S}$ requires that

$$\nu_R>\frac{\mu(\mu-1)}{\mu(\mu-1)+(\mu\bar{z}-1)},\tag{77}$$

since if ν_R was smaller than this, all the coefficients of the second order polynomial $f\left(q_{R/S}\right)$ would be positive and therefore according to the Routh-Hurwitz criterion, the polynomial would have both of its roots in the open left half plane, leaving them meaningless in this model. On the other hand, if (77) is true, then both $f\left(q_{R/S}\right)$ and $f\left(-q_{R/S}\right)$ have a negative constant term and therefore the polynomial $f\left(q_{R/S}\right)$ must have one positive and one negative real root.

4.4.4 Stability of the Disease-free Equilibrium

Studying the stability of this disease-free equilibrium follows a path very similar to section 4.4.2. The change in the awareness dynamics does not affect the dynamics of the infected and therefore the ODE (66) stays the same in this case, as well as the requirement for the prevention of a major epidemic (67) derived from it. Finding out the quasi-equilibrium of $q_{S/I}$ requires solving the differential equation

$$\begin{aligned}\dot{q}_{S/I} &= \frac{1}{\rho_I}\left(\dot{P}_{SI} - q_{S/I}\dot{\rho}_I\right) \\ &= -\beta q_{S/I}^2 + \left(\beta\bar{z}q_{S/S} - \frac{\beta}{z} - \left(\nu_R\bar{z}q_{R/S} + 1\right)\right) q_{S/I} + (\mu - 1)\, q_{0/I} = 0. \end{aligned} \tag{78}$$

The differential equation $\dot{q}_{0/I} = 0$ stays unchanged in this case as well and (70) applies here in the form

$$q_{0/I} = \frac{1 + \beta\bar{z}q_{0/S}q_{S/I}}{\mu + \beta q_{S/I}}. \tag{79}$$

Inserting this to (78) again gives us the equation

$$-\beta^2 q_{S/I}^3 \tag{80}$$

$$+\beta\left(\frac{\beta\,(z-1)\,q_{S/S} - \nu_R\,(z-1)\,q_{R/S} - (\mu+1)\,z - \beta}{z}\right) q_{S/I}^2 \tag{80a}$$

$$+\frac{\mu\beta\,(z-1)\,q_{S/S} + \beta\,(z-1)\,q_{0/S} - \nu_R\,(z-1)\,q_{R/S} - \beta - z}{z} q_{S/I} \tag{80b}$$

$$+\,(\mu - 1) = 0, \tag{80c}$$

for the quasi-equilibrium of the conditonal probability $q_{S/I}$. There exists exactly one root of this polynomial that is biologically viable in the model. The argument for this is virtually identical to the one for the constant-spread model presented in 4.4.2, with the nullclines

$$g_1\left(q_{S/I}\right) = \frac{\beta q_{S/I}^2 - \left(\beta\bar{z}q_{S/S} - \left(\left(\nu_R\bar{z}q_{R/S} + 1\right) - \frac{\beta}{z}\right) q_{S/I}\right.}{\mu - 1} \tag{81}$$

given by equation (78) and $g_2\left(q_{S/I}\right)$ as $q_{0/I}$ from equation (79).

For the model with the same parameters as those used in 4.4.2, solving equation (80) tells us that the pre-epidemic equilibrium is stable when ν_R is greater than 1.73. This corresponds to an equilibrium where the conditional probability $q_{R/S}$ is 0.22 and using the expression for the density ρ_R in (75) we will get the portion of immune in the pre-epidemic population as

$$\rho_R = \frac{\nu_R q_{R/S}}{\nu_R q_{R/S} + 1}\rho_X \approx 0.276\rho_X.$$

5 Discussion

In chapter 3 we have shown the calculations needed to solve both the pre-epidemic and endemic equilibria and the conditions for the stability of the latter. In a model with awareness spreading through non-infectious contacts between the unaware and the infected, we saw that under certain parameters the endemic equilibrium can become unstable as a result of vaccinations, creating oscillating waves of epidemics. We also calculated the necessary threshold for the awareness to prevent a major epidemic from happening. In chapter 4 we create lattice variants of constant and recovered-dependent awareness spread and determine when the pre-epidemic equilibria are stable. All three approaches see significant reductions in the final size of the epidemic and the size of the endemic population of infected, but only the constant and aware-dependent awareness models are capable of eliminating the disease entirely and permanently. It is however difficult to directly compare the different approaches with each other, due to the "awareness parameter" a representing very different processes in different models. The constant spread of awareness is most effective in frustrating the epidemic per change in parameter a, especially for small a, due to its independence from population dynamics. However, the actual real-life cost of changing or maintaining that parameter is obscured in this model. It might be that recovered-dependent spread of awareness, despite it being a slower process to start, ends up being cheaper, especially if we want to maintain awareness even after the epidemic.

The functions γ describing the spread of awareness are in general very simple and chosen as a compromise of somewhat reasonably modeling the spread while still being calculable. Hidden in them is a very optimistic assumption that more information will always lead to more vaccinations and thus is always good.

There are several ways to question this assumption. On one hand, there is the question of information saturation. If an individual is not convinced after being bombarded every other hour with messages promoting vaccinations, it is unlikely that they'll change their mind if the bombardment intensifies to every hour. In chapter 3 this is taken into account by the existence of aware susceptibles, who do not become even more aware and more likely to vaccinate themselves. However the removal of aware susceptibles as a compartment, needed to make the lattice model somewhat solvable in chapter 4, removed the information saturation from the model. A term γ that would reincorporate saturation into the model, such as

$$\gamma(I) = \frac{aI}{1+bI} \text{ [7]}$$

would be worthy of studying.

On the other hand, health services have limited amount of resources, in which case too much information will only create an unfulfillable demand for vaccinations, overwhelming health care. It would therefore be interesting to consider the maximum number of vaccinations that the health care system is capable of providing and adjusting the desired awareness spread so that the demand stays manageble.

References

[1] Zuzana Chladná, Jana Kopfová, Dmitrii Rachinskii & Samiha C. Rouf, *Global dynamics of SIR model with switched transmission rate*, Journal of Mathematical Biology (2020) 80:1209–1233

[2] Istvan Z. Kiss, Jackie Cassel, Mario Recker & Péter L. Simon. *The impact of information transmission on epidemic outbreaks*, Mathematical Biosciences 225 (2010):1–10

[3] Kazunori Sato, Hirotsugu Matsuda & Akira Sasaki. *Pathogen invasion and host extinction in lattice structured populations*, Journal of Mathematical Biology (1994) 32:251-268

[4] Odo Diekmann, Hans Heesterbeek & Tom Britton. *Mathematical tools for understanding infectious disease dynamics*, Princeton University Press, 2013.

[5] Richard C. Dorf & Robert H. Bishop. *Modern Control Systems*, Pearson Prentice Hall 2008:362-363

[6] Ibid.:363

[7] Anuj Kumar, Prashant K. Srivastava & Yasuhiro Takeuchi. *Modeling the role of information and limited optimal treatment on disease prevalence*, Journal of Theoretical Biology 414 (2017): 103–119

[8] Minus Van Baalen. *Pair Approximations for Different Spatial Geometries*. In: *The Geometry of Ecological Interactions: Simplifying Spatial Complexity*, eds. Dieckmann U, Law R & Metz JAJ, Cambridge University Press 2000:359–387.