Prey-Predator-Parasite: an Ecosystem Model With Fragile Persistence

by

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A Dissertation Presented in Partial Fulfillment
of the Requirements for the Degree
Doctor of Philosophy

Approved March 2017 by the
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May 2017

ABSTRACT

Using a simple SI infection model, I uncover the overall dynamics of the system and how they depend on the incidence function. I consider both an epidemic and endemic perspective of the model, but in both cases, three classes of incidence functions are identified.

In the epidemic form, power incidences, where the infective portion I^p has $p \in (0, 1)$, cause unconditional host extinction, homogeneous incidences have host extinction for certain parameter constellations and host survival for others, and upper density-dependent incidences never cause host extinction. The case of non-extinction in upper density-dependent incidences extends to the case where a latent period is included. Using data from experiments with rhanavirus and salamanders, maximum likelihood estimates are applied to the data. With these estimates, I generate the corrected Akaike information criteria, which reward a low likelihood and punish the use of more parameters. This generates the Akaike weight, which is used to fit parameters to the data, and determine which incidence functions fit the data the best.

From an endemic perspective, I observe that power incidences cause initial condition dependent host extinction for some parameter constellations and global stability for others, homogeneous incidences have host extinction for certain parameter constellations and host survival for others, and upper density-dependent incidences never cause host extinction. The dynamics when the incidence function is homogeneous are deeply explored.

I expand the endemic considerations in the homogeneous case by adding a predator into the model. Using persistence theory, I show the conditions for the persistence of each of the predator, prey, and parasite species. Potential dynamics of the system include parasite mediated persistence of the predator, survival of the ecosystem

at high initial predator levels and ecosystem collapse at low initial predator levels, persistence of all three species, and much more.

ACKNOWLEDGMENTS

First and foremost, I want to express my deep gratitude to my advisor, Dr. Horst R. Thieme. His love and knowledge of mathematics is an inspiration to me, allowing me to see a level of excellence I continually aspire to and hope one day to achieve. Without his support and guidance, my research and dissertation would not have been possible. I also want to thank my wife Cherry Farrell, for her love and understanding during the lengthy and difficult Ph.D. process.

I was honored by the valuable insight and assistance provided to me by Dr. Hal Smith and Dr. Yang Kuang during the creation of this dissertation. I also extend thanks to Dr. Wenbo Tang and Dr. James Collins for their contribution to my successful graduate career. I share the credit of the more sophisticated statistical approach to the work done here with Dr. Jesse Taylor.

Finally, I would like to give a special thanks to Dr. Erica Rutter and Philip Walker, my peers, friends, and sounding boards for research and personal discussions alike.

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Chapter 1

OVERVIEW

Amphibian decline and disappearance (Collins (2010); Rachowicz *et al.* (2005)) have rekindled interest in the role of infectious disease agents in the extinction of their host species. Theoretical work has highlighted the importance of how disease incidence, the rate of new infections, denoted by $\sigma f(S, I)$, depends on the amounts of susceptible individuals, S, and infective individuals, I. Here f is called the incidence function and σ the transmission coefficient.

It is long known that, in deterministic mathematical models, density dependent (or mass action) incidence $f(S, I) = SI$ does not lead to host extinction (Kermack and McKendrick (1927); Hwang and Kuang (2003, 2005)) unless the population is subject to an Allee effect (Friedman and Yakubu (2012); Hilker (2010); Thieme *et al.* (2009)) or there is a reservoir for the disease (Han and Pugliese (2009); Hethcote *et al.* (2005); Holt and Pickering (1985)). Frequency dependent incidence has the form $f(S, I) = \frac{SI}{N}$, where N is the total population size, and $f(S, I) = \frac{SI}{S+I}$ if the disease is of SI type, i.e., the population only consists of susceptible and infectives individuals. Infectious disease models with frequency-dependent incidence, in different parameter regions, are known to show both decline of the host from a disease-free to an endemic equilibrium and extinction of the host (Busenberg *et al.* (1991); Busenberg and van den Driessche (1990); Getz and Pickering (1983); Greenhalgh and Das (1995); May *et al.* (1989); Thieme (1992); Zhou and Hethcote (1994); Hwang and Kuang (2003, 2005); Gao and Hethcote (1992)), but empirical evidence of a population or species eliminated by frequency-dependent parasites seems elusive (de Castro and Bolker (2005)). Consequently, there has been quite some debate whether density-

dependent or frequency-dependent incidence is more appropriate for specific diseases (see Greer *et al.* (2008), (Hethcote, 2000, Sec.2.1), Hethcote *et al.* (2005) and the references therein). The preconception may even have risen that extinction occurs under frequency-dependent incidence only. It is one of the aims of this dissertation to challenge this preconception.

Both density and frequency incidence have bad negative negative likelihood (NLL) fits in laboratory experiments involving tiger salamander larvae and a rhanavirus (Greer *et al.* (2008)). This motivates us to consider alternative incidence functions (of which plenty exist in the literature) and study their potential for host extinction and their fit to the data.

Part 1 of this dissertation will concentrate on epidemic models while Part 2 will focus on endemic models. Endemic models incorporate the demography of the host population while epidemic models restrict themselves on epidemic outbreaks which happen so fast that the natural turnover of the population can be ignored. In Parts 1 and 2, we explore the behavior of three classes of incidence functions in for the following model:

$$S' = Sg(S) - \sigma f(S, I), \qquad I' = \sigma f(S, I) - \mu I. \tag{8.1.1 preview}$$

$g(S)$ is the per capita growth rate of susceptible individuals, and μ is the per capita death rate of infective individuals. The classes of incidence functions considered are

- Homogeneous: $f(\alpha S, \alpha I) = \alpha f(S, I)$.
- Power law: $f(S, I) = \theta(S)I^p$, with $p \in (0, 1), \theta(0) = 0$, and θ strictly increasing.
- Upper density-dependent: for any $N > 0$, there is some c_N such that $f(S, I) \leq c_N SI$ for $0 \leq S, I \leq N$.

In Part 1, we consider an epidemic model, where the disease moves so rapidly through a population that we disregard the growth rate of susceptibles, i.e. $g(S) \equiv 0$. Here, we use the data from Greer *et al.* (2008) to test additional incidence functions for their best fit and their optimal parameters. Additionally, we analyze the three classes of incidence function to determine the following global behaviors of the system:

- Homogeneous: Parameter dependent results including:
    1. Initial condition independent host extinction,
    2. Initial condition independent host survival,
    3. Initial condition dependent host extinction/survival.
- Power law: Host extinction regardless of initial conditions or parameters.
- Upper density-dependent: Host survival regardless of initial conditions or parameters.

Further, in the case of upper density-dependent incidence, we also consider a latent period, where susceptibles are infected, but not yet infective. Although we only make this consideration for a single class of incidence functions, the experiments conducted in Greer *et al.* (2008) assume that there is a latent period which is greater than 24 hours.

In Part 2, we consider the model (8.1.1) with the biologically plausible assumptions that $g(S)$ is strictly decreasing, continuous, $g(0) > 0$, and there is a carrying capacity $K > 0$ with $g(K) = 0$. Since g is strictly decreasing, K is uniquely determined. Biologically we expect $g(S) + \mu \geq 0$ *should* hold for all $S \geq 0$, although we do not enforce this assumption in any analysis. However, it does motivate us to consider a general class of growth rates instead of logistic growth. We reuse the same three

classifications of incidence function of homogeneous, power law, and upper density-dependent. In Part 2, however, the incidence function class behaviors are:

- Homogeneous: Parameter dependent results including:
  1. Initial condition independent host extinction
  2. Initial condition independent host survival
  3. Initial condition dependent host extinction/survival
- Power law: Only incidence functions of the form $f(S,I) = S^q I^p$, are considered. If $p \in (0,1]$ and $q \in (0,1)$, then there is initial condition dependent host extinction for all σ, and an endemic equilibrium if $p+q-1>0$. In this case, numerical evidence suggests for large sigma that all (non-equilibrium) initial conditions lead to host extinction. If $p \in (0,1)$ and $1 \leq q$, there is always an interior equilibrium, which is global stable.
- Upper density-dependent: Host survival regardless of initial conditions or parameters.

For Part 3, we expand upon Equation (8.1.1) by adding a predator into the population. This system of equations is:

$$\begin{aligned} S' =& Sg(S) - \sigma f(S,I) - \kappa_1 SP, \\ I' =& \sigma f(S,I) - \kappa_2 IP - \mu I, \\ P' =& \gamma_1 SP + \gamma_2 IP - \nu P. \end{aligned} \qquad \text{(15.1.1 preview)}$$

Here, $\kappa_1 \geq 0$ is the rate at which one unit of susceptible prey is killed by one unit of predator, and $\kappa_2 > 0$ is the rate at which one unit of infected prey is killed by one unit of predator. $\gamma_1 \geq 0$ is the rate of per unit predator biomass increase by killing

(and eating) one unit of susceptible prey, while $\gamma_2 \in \mathbb{R}$ is the analog for infected prey. We assume $\kappa_2 > \kappa_1$, i.e., infective prey are more easily caught than susceptible prey by the predator. Additionally, we assume $\kappa_1 + \kappa_2 > 0$, and $\kappa_1 = 0$ if and only if $\gamma_1 = 0$. Additionally, we assume if $\gamma_1 = 0$, then $\gamma_2 > 0$, so the predator has food available that provides it sustenance. Note that all constants are non-negative with the possible exception of γ_2 which is positive if eating infected prey has a positive effect on a predator and negative if eating infected prey has a negative effect, i.e. poisoning the predator. Even with this possibility, we assume that the predator cannot be infected by eating an infective prey.

There is a large literature base on predator-prey-parasite models using density-dependent incidence (Arino *et al.* (2004); Bairagi *et al.* (2007); Chattopadhyay *et al.* (2003); Mukherjee (2016); Khan *et al.* (2016); Venturino (1994); Xiao and Chen (2001a, 2002, 2001b); Yongzhen *et al.* (2011), to name a few). The only cases of using a homogeneous incidence we are aware of are in Haque *et al.* (2009); Han *et al.* (2001); Hethcote *et al.* (2004); Ghosh and Li (2016), which use frequency dependent incidence. Chen and Wen (2016) consider a predator-prey-parasite model using a saturation disease incidence. A model similar to ours is analyzed in Ruan and Freedman (1991), using general functions for predation and infection. In our model, we restrict our considerations of incidence functions to homogeneous incidence functions. We do this because homogeneous incidences can cause extinction in the SI subsystem and because of the relative ease in analysis when compared to power laws. This decision is (in part) what motivated us to explore homogeneous incidence functions so heavily in Part 2. When a specific incidence function is needed (e.g. numerical simulations), we choose asymmetric frequency dependent incidence, due to its rich dynamics and the poor fit that (symmetric) frequency dependence had on the data from Greer *et al.* (2008). Our model uses simple, density dependent, predator-prey dynamics for preda-

tion on both infectives and susceptibles. More complex predator-prey interactions (in a predator-prey-parasite model) are seen in Arino *et al.* (2004); Bairagi *et al.* (2007); Chen and Wen (2016); Ghosh and Li (2016); Mukherjee (2016); Khan *et al.* (2016); Xiao and Chen (2001a, 2002); Yongzhen *et al.* (2011), where, in some cases, this is in exchange for simplified host-parasite dynamics. While the addition of a predator to the model seems small, it provides a wealth of possible dynamics:

(i) Predator-mediated extinction of the parasite and survival of the prey and predator

(ii) Parasite-mediated extinction of the predator and survival of the prey and parasite

(iii) Parasite-mediated persistence of the predator

(iv) Predator-mediated survival of all three species at high initial predator levels and parasite-mediated extinction of all three species at low initial predator levels

(v) Predator-mediated extinction of the parasite and survival of the prey and predator at high initial predator levels and parasite-mediated extinction of all three species at low initial predator levels

(vi) Parasite-mediated extinction of all three species at all initial predator levels

(vii) Persistence of all three species.

Analytically, we provide support or proof for each of the above possibilities. I highlight the case of a predator consuming infective prey only, as the analysis of the interior equilibrium in this case is quite thorough and interesting.

PART 1 -

SI MODEL: EPIDEMIC PERSPECTIVE

Chapter 2

INTRODUCTION

As said in Chapter 1, both density and frequency incidence have bad negative negative logarithmic likelihoods (NLL) fits in the laboratory experiments conducted in Greer *et al.* (2008). We begin our exploration of alternative incidence functions with multiplicative incidence functions.

2.1 Multiplicative Incidence

Sometimes, the incidence function is assumed to factor into a term that depends on the susceptibles and a term that depends on the infectives.

$$f(S, I) = f_1(S) f_2(I). \tag{2.1.1}$$

Often, the choice for f_1 is simply $f_1(S) = S$. The classical mass action (density-dependent) incidence is given by $f(S, I) = SI$. Other choices are

$f_2(I) = \dfrac{\kappa I^\nu}{1 + \alpha I^\nu}$, $\nu > 1$ (Regoes *et al.* (2002); Ruan and Wang (2003)),

$f_2(I) = \dfrac{I}{1 + (I/k)}$ (Capasso and Serio (1978), analog of a Holling type II/Michaelis-Menten/Monod functional response by a consumer to a food source),

$f_2(I) = k \ln(1 + (I/k))$ (adopted from Briggs and Godfray (1995) where I is the amount of virus particles released after the death of an infected insect, called negative binomial incidence in Greer *et al.* (2008)),

$f_2(I) = k(1 - e^{-I/k})$ (Ivlev (1955); Skellam (1951), analog of an Ivlev/Skellam functional response to a food source by a consumer),

$f_2(I) = \dfrac{I}{1+(I/k)+\sqrt{1+2(I/k)}}$ (adopted from Heesterbeek and Metz (1993) where it is used to model contact rates),

$f_2(I) = \dfrac{aI}{(a^\gamma + I^\gamma)^{1/\gamma}}$ (adopted from Kribs-Zaleta (2009) where it is used for predation) with the limit case (as $\gamma \to \infty$)

$f_2(I) = \min\{a, I\}$, (Blackman (1905), analog of a Blackman functional response to a food source by a consumer).

There are few empirical or semi-empirical studies that give some information about the form of f. Brown and Hasibuan (Brown and Hasibuan (1995)) fit $f(S,I) = \kappa(SI)^p$ for a fungal disease in spider mites and found a best fit with $p = 0.4 \pm 0.04$. It should be mentioned that the disease is transmitted to susceptible mites from the cadavers of infected mites. Actually, power laws have been suggested early on in the history of mathematical epidemiology ((Bailey, 1975, p.128), (Capasso, 1993, Ch.3) Liu *et al.* (1987, 1986); Wilson and Worcester (1945))

From a theoretical point of view, a power law I^p with $0 < p < 1$ is very problematic because, in epidemic models, it leads to outbreaks ex nihilo (with no initial infectives, see Remark 4.1.3) and, in endemic models, it makes the disease-free equilibrium always unstable (Liu *et al.* (1987)).

A possibility to avoid this and still have some power law involved is $f_2(I) = \dfrac{I}{1-\epsilon+\epsilon I^{1-p}}$ with $0 \le \epsilon \le 1$ which interpolates between I and I^p. A parameter sparser version chooses $\epsilon = 1/2$ which provides $f_2(I) = \dfrac{I}{1+I^{1-p}}$ (where the factor 2 has been absorbed into σ).

2.2 Homogeneous Incidence

Frequency-dependent incidence $f(S,I) = SI/(S+I)$ is a very special case of homogeneous incidence where $f(\alpha S, \alpha I) = \alpha f(S,I)$ for all $S, I, \alpha \geq 0$. A very general class is

$$f(S,I) = \frac{SI}{[qI^\beta + pS^\beta]^{1/\beta}}$$

with $p, q \geq 0$, $p+q=1$, $\beta > 0$ which has the limiting cases $S^q I^p$ and $\min\{S,I\}$ (Hadeler (1989, 1992, 1993, 2012); Hadeler *et al.* (1988)). For $\beta = 1$, we obtain the asymmetric frequency-dependent incidence

$$f(S,I) = \frac{SI}{qI+pS}. \tag{2.2.1}$$

The constant risk incidence $f(S,I) = S$ is a special case ($q=1$ and $p=0$).

A variation of the minimum function is

$$f(S,I) = \min\{S, kI\}. \tag{2.2.2}$$

Actually, if $h : \mathbb{R}_+ \to \mathbb{R}_+$ is an arbitrary increasing function with $h(0) = 0$, the definitions

$$f(S,I) = Sh(I/S), \qquad \text{and} \qquad f(S,I) = Sh\big(I/(S+I)\big)$$

provide homogeneous incidence functions (Hadeler (2012)). In the salamander/ranavirous fits, they cannot quite compete with the power functions and the negative binomial function but their fits are not too bad, either.

2.3 Non-Multiplicative Incidence

Some of these incidences have been fitted to laboratory experiments involving tiger salamander larvae and a rhanavirus Greer *et al.* (2008). Both density and frequency

dependent incidence gave bad negative logarithm likelihoods (NLL) fits and were even outperformed by the constant risk incidence $f(S, I) = S$. The best (NLL) fits were obtained by the double power law $f(S, I) = S^q I^p$ with $0 < p < q < 1$, the mixed law $f(S, I) = SI^p$ with $0 < p < 1$ and by $f(S, I) = Sk \ln(1 + I/k)$. (We have rewritten some of the incidence functions in order to bring them to a similar form).

It is one of the purposes of this dissertation to emphasize that these fits (and some more we will do) leave the question completely open as to whether ranavirus drives salamander larval populations into extinction. We will also show that general homogeneous incidence functions yield better fits than their frequency-dependent special case, though not as good fits as powers in I or the negative binomial incidence function. We will also discuss whether the fitting procedure that is used is appropriate for homogeneous incidence functions. To alleviate any potential issues with the fitting procedure, we also apply the alternative fitting method of continuous time Markov chains.

In this part, we will concentrate on epidemic models which ignore the natural turnover of the population (reproduction and deaths from other causes of the disease) because they occur on a much slower time-scale. In Part 2, we use endemic models which take the natural turnover into account.

2.4 Preview of Results

As for the dynamics of the susceptible part of the population, the differential equation

$$-S' = \sigma f(S, I) \tag{2.4.1}$$

is used. This one equation usually does not form a closed system; the form of the other equations depends on the model assumptions: whether or not a latency period is ignored, whether the disease is fatal or whether there are recoveries from the disease.

Still, a lot of information can be teased out of this one equation, in particular that the disease incidence tends to 0 as time tends to infinity.

Theorem 2.4.1. *Let $f : \mathbb{R}_+^2 \to \mathbb{R}_+$ be continuous, $f(0, I) = 0$ for all $I \in \mathbb{R}_+$. Assume that I is a non-negative bounded uniformly continuous function on $\mathbb{R}_+$ and S a non-negative solution of (2.4.1) on $\mathbb{R}_+$. Then $S_\infty = \lim_{t\to\infty} S(t)$ exists and $f(S(t), I(t)) \to 0$ as $t \to \infty$.*

The proof can be found in Appendix A.

For the constant risk incidence $f(S, I) = S$, which performs relatively well in the fits, (2.4.1) forms a closed model,

$$S' = -\sigma S, \tag{2.4.2}$$

with the solution $S(t) = S_0 e^{-\sigma t}$. This means, that epidemics operating under the constant risk incidence inevitably eradicate the host population.

For all other incidences, we need more equations to obtain a closed system. In the simplest case, we assume that the population consists of susceptible and infective individuals only and add the differential equation

$$I' = \sigma f(S, I) - \mu I \tag{2.4.3}$$

for the infective part of the population. Here $\mu > 0$ is the per capita rate of dying from the disease. This model has the consequence that the infective part of the host population inevitably dies out.

Theorem 2.4.2. *Let $f : \mathbb{R}_+^2 \to \mathbb{R}_+$ be continuous, $f(0, I) = 0$ for all $I \in \mathbb{R}_+$, and S, I be non negative solutions of (2.4.1) and (2.4.3). Then $S_\infty = \lim_{t\to\infty} S(t)$ exists and $I(t) \to 0$ as $t \to \infty$.*

The proof can be found in Appendix A.

In Chapter 4, we will show that any incidence of the form $f(S,I) = \theta(S)I^p$ with $0 < p < 1$ and a strictly increasing function θ with $\theta(0) = 0$ leads to unconditional extinction in epidemic models of the form (2.4.1) and (2.4.3). This includes $f(S,I) = S^qI^p$ and $f(S,I) = SI^p$ which provide the best NLL fits.

In Chapter 5, we will show that any epidemic with homogeneous incidence leads to initial-value independent host extinction if σ/μ is large enough. For intermediate values of σ/μ, there may be initial-value dependent host extinction which occurs if the ratio I_0/S_0 is large enough and does not occur otherwise. For small enough σ/μ, there is host survival for all initial conditions.

In Chapter 6, we will show that any incidence which satisfies $f(S,I) \leq cSI$ with a constant $c > 0$ independent of S and I never leads to extinction. This includes the so-called negative binomial incidence $f(S,I) = kS\ln(1+(I/k))$ and the interpolating incidence $f(S,I) = SI/(1+I^{1-p})$ which is like a density-dependent incidence for small $I > 0$ and like a power p in I for large I. They yield the third and fourth best NLL fits which are only slightly worse than those for the two front-runners mentioned above. Actually, it is enough to have $f(S,I) \leq cSI$ for all $S, I \geq 0$ with $S + I \leq S_0 + I_0$, where S_0 and I_0 are the initial values; so $f(S,I) = SI^\nu/(1+\alpha I^\nu)$, $\nu \geq 1$, is also covered. We call such incidences *upper density-dependent*.

Non-extinction even holds for a more realistic epidemic model which takes into account that infected individuals do not become infective immediately after infection and do not die from the disease at a constant per capita rate. This result, which is only proven for upper density-dependent incidence functions, is proven in Section 6.2. Fitting the survivorship curves in Greer *et al.* (2008) by delayed exponential, gamma, and lognormal distributions, we estimate the average time from infection to disease death in the experiments as 13.1 days approximately. Previous laboratory

and field studies showed that most infected salamander larvae usually die within 8-14 days (Greer *et al.* (2008)).

Chapter 3

FITTING IF INCIDENCES REVISITED

3.1 Negative Binomial Approach

We revisit the infection experiments in Greer *et al.* (2008). In these experiments, a group of susceptible salamander larvae was exposed to a group of infective salamander larvae for the period of one day. The experiments took advantage of one feature of ranavirus infection which is not incorporated in model (2.4.1) and (2.4.3), namely that infected larvae do not immediately become infective. So we can assume that the number of infective larvae remains equal to its initial value throughout the experiments.

In the notation of (2.4.1), the infection experiments can be described by the differential equation

$$S' = -\sigma f(S, I_0), \qquad S(0) = S_0. \tag{3.1.1}$$

If $f(S, I) = S\phi(I)$, this equation is solved by

$$S(t) = S_0 e^{-\sigma\phi(I_0)t}.$$

If f is not of this form, (3.1.1) is replaced by

$$S' = \sigma S \frac{f(S_0, I_0)}{S_0}, \qquad S(0) = S_0, \tag{3.1.2}$$

which is solved by

$$S(t) = S_0 e^{-\sigma\phi_0 t}, \qquad \phi_0 = \frac{f(S_0, I_0)}{S_0}. \tag{3.1.3}$$

If f is homogeneous, we have $\phi_0 = f(1, I_0/S_0)$. As an example, if we consider the frequency-dependent transmission function, we will use $\phi_0 = \dfrac{1}{1 + (S_0/I_0)}$. For some

of the infection experiments, (3.1.2) is a really rough approximation of (3.1.1) because all susceptible individuals had been infected when the exposure ended after one day (see Table 3.2). So this replacement may lead to some considerable error. See the discussion at the end of this section.

Using the data and analysis approach from Greer *et al.* (2008), we compute table 3.1, which is a re-computation of table 2 in Greer *et al.* (2008) with more incidence functions, notably a few new homogeneous functions.

Let n be the number of experiments, τ the common length of the experiments, and $S_i(t)$ the number of susceptibles in experiment number i at time t. Then the probability that, at the end of experiment i, there are $S_i(\tau)$ susceptibles left from $S_i(0)$ susceptibles at the beginning is given by

$$p_i(\tau) = \binom{S_i(0)}{S_i(\tau)} P_i^{S_i(\tau)} (1 - P_i)^{S_i(0) - S_i(\tau)}. \tag{3.1.4}$$

Here

$$P_i = \exp(-\sigma \phi_i \tau), \qquad \phi_i = \frac{f(S_i(0), I_i(0))}{S_i(0)}, \tag{3.1.5}$$

with $I_i(0)$ denoting the number of infectives at the beginning of the i^{th} experiment. In the experiments, the salamander larvae were together for 1 day, so $\tau = 1$ is used in the following calculations.

To get the likelihood, we take the product of the probabilities of all the experiments, so if n is the total number of experiments, (in our case $n = 24$) we have

$$\text{likelihood} = \prod_{i=1}^{n} p_i(1).$$

Using a Matlab (Mathworks 2005) simplex search method called fminsearch, we minimized the negative log likelihood for the data to determine optimal parameters.

Thus, we computed

$$\min\left\{\sum_{i=1}^{n} -\ln\binom{S_i(0)}{S_i(1)} - S_i(1)\ln P_i - (S_i(0)-S_i(1))\ln(1-P_i)\right\}, \tag{3.1.6}$$

where the minimum is taken over the parameters of the chosen function.

For an explanation why minimizing the negative log-likelihood function provides a parameter fit, see (Diekmann *et al.*, 2013, p.318, p.487).

After obtaining the optimal parameters and maximal likelihood, we use the corrected Akaike information criterion ($\mathrm{AIC_c}$) values and Akaike weights (which is interpreted as the probability of the transmission function being the best fit) to rank the a particular transmission function as being the best model for the data. The $\mathrm{AIC_c}$ is calculated by the formula

$$\mathrm{AIC_c} = -2\ln(\text{max likelihood}) + 2K\frac{n}{n-K-1},$$

where K is the number of parameters in the model. The Akaike weight for the ith experiment is given by the formula

$$\text{Akaike weight} = \frac{\exp\{-\Delta_i/2\}}{\sum \exp\{-\Delta_j/2\}},$$

where Δ_j is the difference of the minimal $\mathrm{AIC_c}$ across all functions tested and the $\mathrm{AIC_c}$ of the current function, and the summation is over all functions.

See (Martcheva, 2015, Sec.6.4) for a use of the Akaike information criterion in connection with least square fitting of several epidemic models to data from an influenza epidemic in an English boarding school.

3.2 Our Observations and Reservations

We return to the question whether the use of (3.1.5) for incidences which are not of the form $f(S,I) = S\phi(I)$ may lead to bad fits. We have considered two examples

Types of transmission	Function	σ (units)	Additional parameters	NLL	AIC_c value	ΔAIC_c	Akaike weight
Power (in I only)	σSI^p	1.379 ($H^{-p}day^{-1}$)	$p = 0.255$ (†)	20.20	44.98	0.00	0.2308
Negative Binomial (in I)	$\sigma kS\ln(1 + I/k)$	4.459 ($H^{-1}day^{-1}$)	$k = 0.130$ (H)	20.66	45.90	0.92	0.1457
Interpolating	$\sigma SI/(1 + I^{1-p})$	1.978 ($H^{-1}day^{-1}$)	$p = 0.165$ (†)	20.95	46.46	1.48	0.1099
Power (in S and I)	$\sigma S^q I^p$	1.628 ($H^{1-p-q}day^{-1}$)	$p = 0.263$ (†)	19.98	47.16	2.18	0.07768
			$q = 0.953$ (†)				
Contact Rates	$\sigma SI/(1 + kI + \sqrt{1 + 2kI})$	3.661 ($H^{-1}day^{-1}$)	$k = 0.898$ (H^{-1})	21.32	47.21	2.23	0.07566
Predation	$\sigma SI/(a^\gamma + I^\gamma)^{1/\gamma}$	92.595 ($H^{-1}day^{-1}$)	$a = 1.91 \times 10^{-5}$ (H)	20.29	47.79	2.81	0.0566
			$\gamma = 0.081$ (†)				
Neg. Binomial Frequency	$\sigma S\ln(1 + I/(S + I))$	1.129 (day^{-1})		22.98	48.15	3.17	0.04725
Homogeneous Power law	$\sigma S^q I^p$	3.032 (day^{-1})	$p = 0.121$ (†)	21.95	48.47	3.49	0.04025
Frequency with Exponent	$S(I/(S + I))^p$	3.400 (day^{-1})	$p = 0.163$ (†)	22.06	48.69	3.72	0.03601
N. Bin. Asymmetric Frequency	$\sigma S\ln(1 + I/(pS + qI))$	4.379 (day^{-1})	$p = 0.0497$ (†)	22.12	48.81	3.83	0.034
Asymmetric Frequency	$\sigma SI/(pS + qI)$	3.035 (day^{-1})	$p = 0.035$ (†)	22.12	48.81	3.83	0.03399
Michaelis-Menten	$\sigma SI/(1 + I/k)$	1.078 ($H^{-1}day^{-1}$)	$k = 3.162$ (H)	22.15	48.87	3.90	0.0329
Weighted Minimum	$\sigma\min\{S, \alpha I\}$	3.033 (day^{-1})	$\alpha = 7.593$ (†)	22.15	48.87	3.90	0.0329
Constant Risk	σS	2.541 (day^{-1})		24.07	50.32	5.35	0.01593
General Homogeneous	$\sigma SI/(pS^\beta + qI^\beta)^{(1/\beta)}$	3.032 (day^{-1})	$p = 0.121$ (†)	21.74	50.68	5.70	0.01336
			$\beta = 1.08 \times 10^{-14}$ (†)				
Ivlev/Skellam Law	$\sigma kS(1 - e^{-I/k})$	0.522 ($H^{-1}day^{-1}$)	$k = 6.528$ (H)	23.56	51.69	6.72	0.008029
Blackwell	$\sigma S\min\{k, I\}$	1.946 ($H^{-1}day^{-1}$)	$k = 1.31$ (H)	23.91	52.38	7.40	0.005694
Very General	$\sigma SI/((pS)^\alpha + (qI)^\beta).^\gamma$	1.862 (day^{-1})	$p = 0.000113$ (†)	20.04	53.41	8.43	0.003405
			$\beta = 0.407$ (†)				
			$\alpha = 0.275$ (†)				
	$\sigma SI\left(1 + \frac{S+I}{k} + \sqrt{1 + 2\frac{S+I}{k}}\right)^{-1}$		$\gamma = 1.95$ (†)				
Sexual Transmission		0.772 ($H^{-1}day^{-1}$)	$k = 120$ (H)	42.64	89.85	44.87	4.166×10^{-11}
Density-Dependent	σSI	0.247 ($H^{-1}day^{-1}$)		43.86	89.90	44.92	4.061×10^{-11}
Beddington/DeAngelis/Dietz	$\sigma SI/(1 + (S + I)/k)$	0.369 ($H^{-1}day^{-1}$)	$k = 154$ (H)	42.75	90.06	45.08	3.744×10^{-11}
Power (in S only)	$\sigma S^q I$	0.348 ($H^{-q}day^{-1}$)	$q = 0.913$ (†)	43.26	91.10	46.12	2.233×10^{-11}
Frequency Dependent	$\sigma SI/(S + I)$	15.763 ($H^{-p}day^{-1}$)		68.45	139.08	94.10	8.516×10^{-22}
Minimum	$\sigma\min\{S, I\}$	11.241 (day^{-1})		113.86	229.89	184.91	1.62×10^{-41}

Table 3.1: Fits of various incidence types to the infection experiments in Greer *et al.* (2008) using the binomial distribution. † means that the parameter is dimensionless. H stands for host. If p and q appear and only the value for p is given, then $q = 1 - p$.

were the differential equation $S' = -\sigma f(S, I_0)$, $S(0) = S_0$, has solutions in closed form.

Consider $f(S, I) = S^q I^p$ with $0 < q < 1$ and $p \geq 0$. In this case, (3.1.1) can be explicitly solved using

$$\frac{1}{1-q}\frac{d}{dt}S^{1-q} = S^{-q}S' = -\sigma I_0^p,$$

which yields

$$S(t) = S_0\Big[1 - (1-q)\sigma I_0^p S_0^{q-1} t\Big]_+^{\frac{1}{1-q}}. \tag{3.2.1}$$

Here $[r]_+ = r$ if $r \geq 0$ and $[r]_+ = 0$ if $r \leq 0$. In comparison, (3.1.3) provides

$$S(t) = S_0 e^{-\sigma I_0^p S_0^{q-1} t}. \tag{3.2.2}$$

This suggests to use (3.1.6) with

$$P_i = \Big[1 - (1-q)\sigma I_i(0)^p S_i(0)^{q-1}\tau\Big]_+^{\frac{1}{1-p}}.$$

We could not find much difference in the fits because for both choices q turns out to be very close to 1; notice that the limit of (3.2.1) and (3.2.2) are the same as $q \to 1$.

We also consider $f(S, I) = \min\{S, kI\}$ and solve

$$S' = -\sigma \min\{S, a\}, \qquad a = kI_0, \quad S(0) = S_0.$$

We solve by cases (recall $a = kI_0$),

$$S(1) = \begin{cases} S_0 e^{-\sigma}, & S_0/a \leq 1 \\ a e^{(S_0/a)-(\sigma+1)}, & 1 < S_0/a < \sigma + 1 \\ S_0 - \sigma a, & S_0/a \geq \sigma + 1. \end{cases} \tag{3.2.3}$$

Unfortunately, the cases depend on the parameters we want to estimate. But going through the cases, one sees that

$$S(1) = \min\Big\{S_0 e^{[(S_0/a)-1]_+ - \sigma}, a e^{[(S_0/a)-1]_+ - \sigma}, [S_0 - a(\sigma+1)]_+ + a\Big\}. \tag{3.2.4}$$

With $b = kI_0/S_0$,

$$\frac{S(1)}{S_0} = \min\Big\{ \min\{1,b\}e^{[\,(1/b)-1]_+ - \sigma}, \big[1-b(\sigma+1)\big]_+ + b\Big\}. \tag{3.2.5}$$

If we use (3.1.3), we obtain

$$\frac{S(1)}{S_0} = e^{-\sigma \min\{1,b\}}, \qquad b = kI_0/S_0.$$

This agrees with (3.2.3) if $S_0/a \le 1$, but not otherwise. This suggests to use (3.1.6) with

$$P_i = \min\Big\{ \min\{1,b_i\}e^{[\,(1/b_i)-1]_+ - \sigma}, \big[1-b_i(\sigma+1)\big]_+ + b_i\Big\}, \tag{3.2.6}$$

rather than

$$P_i = e^{-\sigma \min\{1,b_i\}}, \tag{3.2.7}$$

where $b_i = kI_i(0)/S_i(0)$.

With (3.2.7), we obtain the fits $\sigma = 2,5406$ per day and $k = 12.914$ per unit of host with NLL 24.07 and $\mathrm{AIC_c}$ 52.71; with (3.2.6), $\sigma = 3.0325$ per day and $k = 3.654$ per unit of host with NLL 22.15 and $\mathrm{AIC_c}$ 48.87. While the goodness of fits are not so different, the different estimates of k are somewhat worrisome.

For reassurance, we numerically solve the equation

$$S' = -\sigma f(S, I_0), \qquad S(0) = S_0 \tag{3.2.8}$$

with the estimated σ and possibly other parameters, and post them together with the outcome of the experiments. See Tables 3.2 and 3.3: Table 3.2 shows the design of the experiments and the outcome for every one of the replicates. In Table 3.3, the horizontal lines separate experiments done with the same initial numbers of susceptible larvae, S_0, and same number of infected larvae, I_0, they were exposed to (columns 2 and 3). Column 1 repeats the experiment labels from Table 3.2. Column 4 shows how many of the initially susceptible larvae had not been infected after one day of

Experiment label	A							B				C			D							E		
No. of replicates	7							4				3			7							3		
Initial Susceptibles	1							8				40			1							80		
Exposure Infectives	1							8				40			8							8		
Final Susceptibles	1	0	0	0	0	0	0	1	1	0	2	1	0	2	0	0	0	0	0	0	0	8	4	12
Final Susc. (mean)	0.143							1				1			0							8		

Table 3.2: The experiments varied the number of exposed susceptibles and the number of infectives they were exposed to for 24 hours. Each experimental constellation was replicated several times. The "final" susceptibles are those initial susceptibles that were not infected during the 24 hours of exposure.

Experimental results				$S(1)$ for differential equation solutions with respective types of incidence									
Exp. Lab.	Init. Sus.	Init. Inf.	Fin. S. mean	Power in S, I	Negat. Binom.	Homog. Powers	Ivlev	Power in S	Con-tact	Pred-ation	Inter-polat.	min $\{k, I\}$	Asym. Freq.
A	1	1	0.143	0.184	0.286	0.023	0.616	0.702	0.359	0.260	0.372	0.573	0.045
B	8	8	1	0.529	0.732	0.183	0.720	0.597	0.712	0.766	0.747	1.127	0.356
C	40	40	1	0.768	1.456	0.913	1.332	0.000	1.597	1.175	1.236	1.155	1.782
D	1	8	0	0.049	0.092	0.005	0.090	0.041	0.089	0.096	0.093	0.159	0.043
E	80	8	8	7.130	7.324	5.441	7.196	10.014	7.119	7.658	7.473	8.371	4.860

Table 3.3: Comparison of final susceptibles from the experiments to $S(1)$ from the corresponding differential equation solutions with the respective incidences. See text for more explanations.

exposure (they were still found susceptible at the end of the experiment), averaged over the replicates of the experiment. The other columns show $S(1)$ for the solution of (3.2.8) where f is the respective incidence function.

3.3 Another Approach: A Markov Chain

Following this procedure, we decided to attempt a more sophisticated statistical approach. Dr. Jesse Taylor (Arizona State) suggested the alternate technique of using a continuous time death Markov chain. $p_s(t)$ is the probability that s larvae are susceptible at time $t \in [0,1]$, with $s = 0, 1, 2, \ldots$. For each experiment we form the linear autonomous ODE system on the time interval $[0,1]$,

$$\begin{aligned} p_s' = &\quad -p_s \sigma f(s, I_0), && s = S_0, \\ p_s' = &\quad p_{s+1}\sigma f(s+1, I_0) - p_s \sigma f(s, I_0), && s = 1, \ldots, S_0 - 1, \\ p_0' = &\quad p_1 \sigma f(1, I_0). && s = 0, \end{aligned}$$

with

$$p_s(0) = \begin{cases} 1, & s = S_0, \\ 0, & s \neq S_0. \end{cases}$$

This has the matrix exponential solution, call it A, which we compute via Matlab. A is an $(S(0)+1) \times (S(0)+1)$ matrix, for the possible states of 0, 1, 2, ... $S(0)$. The elements of A have the form $a_{i,j} = p_{j-1}(1)$, given $S(0) = i - 1$. Equivalently, $a_{i,j}$ is the probability of beginning with $i-1$ individuals and ending with $j-1$ individuals. Thus for experiment k, we define

$$P_k = a_{S_k(0)+1, s+1} = p_s^k(1),$$

with s being the number of susceptibles at the end of the k^{th} experiment and $p_s^k(1)$ the probability of of s susceptible larvae are susceptible at the end of the k^{th} experiment.

Using these probabilities, we define the as likelihood

$$P = P_1 \cdots P_n$$

Given this likelihood, we follow the same procedures as above to determine the Akaike weight, and thus generate Table 3.4. In the case where the incidence $f(S, I) = S\phi(I)$,

this method will simplify to the previous, thus incidences with those forms will have the same fit and optimal parameters.

Types of transmission	Function	σ (units)	Additional Parameters parameters	NLL	AICc Value	Δ AICc	Akaike weight
Power (in I only)	σSI^p	1.379 ($H^{-p}day^{-1}$)	$p = 0.255$ (†)	20.20	44.98	0.00	0.2107
Negative Binomial (in I)	$\sigma kS\ln(1+I/k)$	4.459 ($H^{-1}day^{-1}$)	$k = 0.130$ (H)	20.66	45.90	0.92	0.133
Interpolating	$\sigma SI/(1+I^{1-p})$	1.978 ($H^{-1}day^{-1}$)	$p = 0.165$ (†)	20.95	46.46	1.48	0.1003
Contact Rates	$\sigma SI/(1+\kappa I+\sqrt{1+2\kappa I})$	3.661 ($H^{-1}day^{-1}$)	$\kappa = 0.898$ (H^{-1})	21.32	47.21	2.23	0.06907
Power (in S and I)	$\sigma S^q I^p$	1.579 ($H^{1-p-q}day^{-1}$)	$p = 0.246$ (†)	20.05	47.31	2.33	0.06575
			$q = 0.958$ (†)				
Homogeneous Power law	$\sigma S^q I^p$	2.644 (day^{-1})	$p = 0.126$ (†)	21.38	47.34	2.36	0.06484
Negative Binomial and Frequency	$\sigma S\ln(1+I/(S+I))$	1.196 (day^{-1})		22.66	47.51	2.53	0.05938
Predation	$\sigma SI/(a^\gamma+I^\gamma)^{1/\gamma}$	871261.415 ($H^{-1}day^{-1}$)	$a = 2.68\times10^{-20}$ (H)	20.23	47.66	2.68	0.05524
			$\gamma = 0.023$ (†)				
Frequency with Exponent	$\sigma S(I/(S+I))^p$	3.327 (day^{-1})	$p = 0.248$ (†)	21.70	47.96	2.99	0.04736
N. Binomial and Asymmetric Frequency	$\sigma S\ln(1+I/(pS+qI))$	4.184 (day^{-1})	$p = 0.125$ (†)	21.83	48.24	3.26	0.04134
Asymmetric Frequency	$\sigma SI/(pS+qI)$	2.902 (day^{-1})	$p = 0.089$ (†)	21.83	48.24	3.26	0.04124
Weighted Minimum	$\sigma\min\{S,\alpha I\}$	3.039 (day^{-1})	$\alpha = 3.698$ (†)	22.04	48.66	3.68	0.03349
Michaelis-Menten	$\sigma SI/(1+I/k)$	1.078 ($H^{-1}day^{-1}$)	$k = 3.162$ (H)	22.15	48.87	3.90	0.03003
General Homogeneous	$\sigma SI/(pS^\beta+qI^\beta)^{(1/\beta)}$	2.644 (day^{-1})	$p = 0.126$ (†)	21.34	49.88	4.90	0.01814
			$\beta = 3.63\times10^{-14}$ (†)				
Constant Risk	σS	2.541 (day^{-1})		24.07	50.32	5.35	0.01454
Ivlev/Skellam Law	$\sigma kS(1-e^{-I/k})$	0.522 ($H^{-1}day^{-1}$)	$k = 6.528$ (H)	23.56	51.69	6.72	0.00733
Blackwell	$\sigma S\min\{k,I\}$	1.946 ($H^{-1}day^{-1}$)	$k = 1.31$ (H)	23.91	52.38	7.40	0.005198
Very General	$\sigma SI/((pS)^\alpha+(qI)^\beta)^\gamma$	2.877 (day^{-1})	$p = 1.53\times10^{-22}$ (†)	20.05	53.44	8.46	0.003066
			$\beta = 0.0429$ (†)				
			$\alpha = 0.0676$ (†)				
			$\gamma = 18.2$ (†)				
Sexual Transmission	$\sigma\frac{SI}{1+\frac{S+I}{k}+\sqrt{1+2\frac{S+I}{k}}}$	1.348 ($H^{-1}day^{-1}$)	$k = 17.1$ (H)	39.31	83.19	38.21	1.062×10^{-9}
Beddington/DeAngelis/Dietz	$\sigma SI/(1+(S+I)/k)$	0.584 ($H^{-1}day^{-1}$)	$k = 27.5$ (H)	39.64	83.85	38.87	7.64×10^{-10}
Power (in S only)	$\sigma S^q I$	0.152 ($H^{-q}day^{-1}$)	$q = 1.186$ (†)	40.50	85.58	40.60	3.214×10^{-10}
Density-Dependent	σSI	0.247 ($H^{-1}day^{-1}$)		43.86	89.90	44.92	3.707×10^{-11}
Frequency Dependent	$\sigma SI/(S+I)$	8.439 ($H^{-p}day^{-1}$)		50.63	103.44	58.46	4.261×10^{-14}
Minimum	$\sigma\min\{S,I\}$	6.824 (day^{-1})		53.75	109.68	64.70	1.877×10^{-15}

Table 3.4: Fits of various incidence types to the infection experiments in Greer *et al.* (2008) using the stochastic method. † means that the parameter is dimensionless. H stands for host. If p and q appear and only the value for p is given, then $q = 1 - p$.

Chapter 4

INCIDENCE WITH POWERS < 1 OF THE INFECTIVES: UNCONDITIONAL EXTINCTION

We now consider an incidence function with power $p \in (0, 1)$ for the infectives and a general functional dependence of the incidence on the susceptibles,

$$S' = -\theta(S)I^p, \qquad I' = \theta(S)I^p - \mu I, \tag{4.1.1}$$

under the initial conditions

$$S(0) = S_0 > 0, \quad I(0) = I_0 > 0. \tag{4.1.2}$$

Here $\theta : \mathbb{R}_+ \to \mathbb{R}_+$ is a strictly increasing continuous function with $\theta(0) = 0$, and μ is the per capita disease death rate.

We rewrite the differential equation for the infectives as

$$\frac{d}{dt}\frac{1}{1-p}I^{1-p} = I^{-p}I' = \theta(S) - \mu I^{1-p}.$$

By the variation of constants formula,

$$I^{1-p}(t) = I^{1-p}(0)e^{-(1-p)\mu t} + \int_0^t (1-p)\theta(S(t-r))e^{-(1-p)\mu r}dr. \tag{4.1.3}$$

Notice that, if $I_0 > 0$, we have that $I(t) > 0$ for all $t \geq 0$.

As for the differential equation for the susceptibles, we notice that, if a solution existing on $[0, r]$ for some $r > 0$ satisfies $S(r) = 0$, then the extension $S(t) = 0$ for all $t > r$ is a solution and the only biologically meaningful one. By Theorem 2.4.2, the host population goes extinct.

So we can concentrate on the case that $S(t) > 0$ for all $t \geq 0$. We separate the variables and integrate the S' portion of (4.1.1),

$$\int_{S(t)}^{S_0} \frac{ds}{\theta(s)} = \int_0^t I^p(s)ds. \tag{4.1.4}$$

We notice that S is a decreasing non-negative function of $t \geq 0$. By (4.1.3), since θ is increasing,

$$I^{1-p}(t) \geq \int_0^t (1-p)\theta(S(t))e^{-(1-p)\mu(t-r)}dr = \theta(S(t))\frac{1-e^{-(1-p)\mu t}}{\mu}. \tag{4.1.5}$$

Further,

$$\begin{aligned}\int_0^t I^p(s)ds \geq& \int_0^t \Big(\theta(S(s))\frac{1-e^{-(1-p)\mu s}}{\mu}\Big)^{p/(1-p)}ds \\ \geq& \theta(S(t))^{p/(1-p)}\int_0^t \Big(\frac{1-e^{-(1-p)\mu s}}{\mu}\Big)^{p/(1-p)}ds.\end{aligned}$$

We combine this inequality with (4.1.4),

$$\frac{1}{\theta(S(t))^{p/(1-p)}}\int_{S(t)}^{S_0}\frac{ds}{\theta(s)} \geq \int_0^t \Big(\frac{1-e^{-(1-p)\mu s}}{\mu}\Big)^{p/(1-p)}ds. \tag{4.1.6}$$

Since the integral on the right hand side diverges to infinity as $t \to \infty$, $S(t) \to 0$ as $t \to \infty$. By Theorem 2.4.2, the host population goes extinct.

Theorem 4.1.1. *For any initial value $S_0, I_0 > 0$, the epidemic modeled by (4.1.1) drives the host population into extinction.*

Remark 4.1.2. It is sufficient for unconditional extinction that the incidence satisfies $f(S,I) \geq \theta(S)I^p$ for all $S, I \geq 0$. Then (4.1.4) and (4.1.5) hold as inequalities with $\geq$ replacing $=$. This again results in the inequality (4.1.6) which implies $S(t) \to 0$ as $t \to \infty$.

We are not aware of the observations in Theorem 4.1.1 being made before, but it could well be as they follow from standard considerations. However, powers in I are not often considered by the more mathematically oriented part of the epidemiological

community because I^p is not a Lipschitz continuous function of I at $I = 0$ for $p \in (0, 1)$, creating problem with uniqueness of solutions:

Remark 4.1.3 (*epidemic outbreaks ex nihilo)***.** If $I_0 = 0$, then $I(t) = 0$ and $S(t) = S_0$ for all $t \geq 0$ is a solution of (4.1.1). But (4.1.3), with $I_0 = 0$, provides a second solution. Since we have not used $I_0 > 0$ in our previous analysis (except for excluding the solution $I \equiv 0$), in this second solution, which describes an epidemic outbreak out of nothing, the host population is driven into extinction.

For more details see Appendix B.

Chapter 5

HOMOGENEOUS INCIDENCE: PARAMETER-DEPENDENT EXTINCTION

We now consider homogeneous incidence functions,

$$f(\alpha S, \alpha I) = \alpha f(S, I) \text{ for all } S, I, \alpha \geq 0.$$

We also assume that f is continuous and that $f(S, 1)$ is an an increasing function of S, $f(0, 1) = 0$ and $f(1, 1) > 0$. See Section 2.2 for examples.

We introduce the ratio of infectives to susceptibles, $r = I/S$. With the *hazard function*

$$h(r) = f(1, r), \qquad r \geq 0, \tag{5.1.1}$$

the model takes the form

$$\begin{aligned} S' =& -\sigma S h(r), \\ r' =& \sigma h(r)(1 + r) - \mu r. \end{aligned} \tag{5.1.2}$$

We rephrase,

$$\begin{aligned} S' =& -\sigma S h(r), \\ r' =& r[\sigma \xi(r) - \mu], \end{aligned} \tag{5.1.3}$$

with the *per unit ratio growth rate*

$$\xi(r) = \frac{(1 + r)h(r)}{r}, \quad r > 0. \tag{5.1.4}$$

Since f is homogeneous,

$$\xi(r) = f(1 + r^{-1}, 1 + r), \quad r > 0. \tag{5.1.5}$$

For all homogeneous incidence functions we consider, except the constant risk function, we have $h(0) = 0$. Since we already know that the constant risk function always leads to host extinction, we assume $h(0) = 0$. We always have $h(r) > 0$ for $r > 0$. Notice that

$$h(r)/r = f(1/r, 1).$$

Since $f(S, I)$ is an increasing function of $S \geq 0$,

$$h'(0) = \lim_{r \to 0} h(r)/r = \lim_{S \to \infty} f(S, 1) := f(\infty, 1)$$

exists in $(0, \infty]$ with $h'(0) = \infty$ being possible. By (5.1.4),

$$h'(0) = \lim_{r \to 0} \xi(r), \tag{5.1.6}$$

and so we define

$$\xi(0) = h'(0) = f(\infty, 1). \tag{5.1.7}$$

If $\sigma h'(0) < \mu$, 0 is a locally asymptotically stable equilibrium of the ODE for r.

We have the following threshold result for host extinction. Define the *basic reproduction number of the disease* by

$$\mathcal{R}_0 = \frac{\sigma}{\mu} \lim_{S \to \infty} f(S, 1),$$

$\mathcal{R}_0$ is the mean number of secondary infections caused by one average infective host that is introduced into a population of infinitely many susceptible hosts.

Theorem 5.1.1. *(a) If $\mathcal{R}_0 > 1$, the host is extinguished by the epidemic.*

(b) If $(\sigma/\mu)\xi(r) < 1$ for all $r \geq 0$, the host survives the epidemic.

(c) Let $\mathcal{R}_0 < 1$ and let there exist some $r > 0$ such that $(\sigma/\mu)\xi(r) \geq 1$. Then there exists some unique $r^\sharp > 0$ such that $(\sigma/\mu)\xi(r^\sharp) = 1$ and $(\sigma/\mu)\xi(r) < 1$ for all $r \in (0, r^\sharp)$. The host survives the epidemic if $r(0) \in [0, r^\sharp)$, and is extinguished by the epidemic if $r(0) \geq r^\sharp$.

Proof. (a) If $\mathcal{R}_0 > 1$, then $\sigma h'(0) > \mu$ and any solution r with $r(0) > 0$ is bounded away from 0 and so is $h(r(t))$ as a function of time $t \geq 0$. This implies that S decreases exponentially and the host dies out.

(b) and (c). In the case of (b), let $r^\sharp = \infty$.

If $r(0) \geq r^\sharp$, $r(t) \geq r^\sharp$ for all $t \geq 0$ and the host dies out by the same arguments as in part (a).

If $r(0) < r^\sharp$, $r(t) \to 0$ as $t \to \infty$. Since $\sigma\xi(0) < \mu$, there are $\epsilon > 0$ and $M \geq 1$ such that $r(t) \leq Me^{-\epsilon t}$ for all $t \geq 0$ and all solutions with $r(0) < r^\sharp$. There also is some $c > 0$ such that $h(r) \leq cr$ for all $r \in [0, M]$ and so

$$S(t) \geq S_0 \exp\Big(- \sigma c \int_0^t r(s)ds\Big) \geq S_0 \exp\Big(- \sigma c \int_0^t Me^{-\epsilon s}ds\Big) \geq S_0 e^{-\sigma c M/\epsilon}.$$

□

If the ratio growth rate function ξ is decreasing, $\sup_{r\geq 0}(\sigma/\mu)\xi(r) = \xi(0)$, so case c cannot occur and there is no initial-value dependent outcome for the host.

Corollary 5.1.2. *Let ξ be decreasing. Then, independently of the initial conditions, the host is extinguished by the epidemic if $\mathcal{R}_0 > 1$, and survives the epidemic if $\mathcal{R}_0 < 1$.*

Examples of incidence functions which give rise to decreasing ξ are the asymmetric frequency-dependent incidence function $f(S, I) = \frac{SI}{pS+qI}$ with $0 < p \leq q$, $1 = p + q$, and the negative binomial frequency-dependent incidence function $f(S, I) = S \ln\big(1 + \frac{I}{S+I}\big)$. In the first case, $\mathcal{R}_0 = \sigma/(p\mu)$ and in the second $\mathcal{R}_0 = \sigma/\mu$.

Chapter 6

UPPER DENSITY-DEPENDENT INCIDENCE: NO EXTINCTION

6.1 Overview

An incidence function $f : \mathbb{R}_+^2 \to \mathbb{R}_+$ is called *upper density-dependent* if, for any $N > 0$, there exists some $c_N > 0$ such that

$$f(S, I) \leq c_N SI, \qquad 0 \leq S, I \leq N. \tag{6.1.1}$$

This inequality holds for the incidence functions $f(S, I) = Sk\ln(1+(I/k))$, $f(S, I) = \frac{SI}{1+I^{1-p}}$, $f(S, I) = \frac{SI}{1+(1/k)(S+I)}$, $f(S, I) = SI^2$, and many more.

We will show that epidemic outbreaks of diseases governed by upper density-dependent incidences cannot drive the host population into extinction. This even holds for models which take more features of the tiger salamander/ranavirus system into account than the simple ordinary differential systems we have considered so far.

6.2 Infection Age

The infection experiments in Greer *et al.* (2008) are based on the assumptions that freshly infected salamander larvae will not be infective for at least one day and that they will be infective after five days. Further the survival curves in Figure 2 of Greer *et al.* (2008) show that infected salamander larvae do not die at a constant per capita rate μ. In fact, the per capita death rate is zero for at least 7 days after infection. This suggests to work with an infection-age model which can more or less

directly use the survival curves in Figure 2 whenever the mathematical means allow to do this.

Let $j(t,\cdot)$ be the infection-age distribution of infected hosts at time t; the total number of infected hosts is given by

$$J(t) = \int_0^\infty j(t,a)da, \qquad t \geq 0 \tag{6.2.1}$$

The input into the infected stage is the disease incidence, the rate of new cases, which in an epidemic model equals $-S'$, the negative of the rate of change of the susceptible part of the population. This provides the equation ((Thieme, 2003, Sec.13.1)),

$$j(t,a) = -S'(t-a)\mathcal{F}(a), \qquad t > a \geq 0, \tag{6.2.2}$$

$\mathcal{F}(a)$ is the probability of not having died from the infection at infection age a. $\mathcal{F}$ is a decreasing function and $\mathcal{F}(0) = 1$. There are two cases: Either $\mathcal{F}(a) > 0$ for all $a \in [0,\infty)$, or there is a unique $a^\diamond \in (0,\infty)$ such that $\mathcal{F}(a) > 0$ for all $a \in [0,a^\diamond)$ and $\mathcal{F}(a) = 0$ for all $a \in (a^\diamond,\infty)$. In the first case, we define $a^\diamond = \infty$. Let $j_0(a)$ denote the hosts that are already infected at time 0 and have the infection age a. For consistency, we assume that $j_0(a) = 0$ if $a > a^\diamond$. The quotient $\frac{\mathcal{F}(a)}{\mathcal{F}(a-t)}$ is the probability of not having died from the infection between age $a-t$ and age a if $t < a < a^\diamond$. Then

$$j(t,a) = \begin{cases} \mathcal{F}(a)\frac{j_0(a-t)}{\mathcal{F}(a-t)}, & t < a < a^\diamond \\ 0, & a > a^\diamond. \end{cases} \tag{6.2.3}$$

It is convenient to define $j_0(a)/\mathcal{F}(a) = 0$ if $\mathcal{F}(a) = 0$. Then we can write

$$j(t,a) = \mathcal{F}(a)\frac{j_0(a-t)}{\mathcal{F}(a-t)}, \qquad t < a < \infty. \tag{6.2.4}$$

By (6.2.3) and (6.2.2),

$$J(t) = \int_0^t (-S'(t-a))\mathcal{F}(a)da + \int_t^\infty j_0(a-t)\frac{\mathcal{F}(a)}{\mathcal{F}(a-t)}da.$$

Since $\mathcal{F}$ is decreasing,

$$J(t) \leq \int_0^t (-S'(t-a))\mathcal{F}(a)da + \int_0^\infty j_0(a)da.$$

By the fundamental theorem of calculus.

$$S(t) + J(t) \leq S(0) + J(0), \qquad t \geq 0.$$

The infective force is given by

$$I(t) = \int_0^\infty \xi(a)j(t,a)da, \tag{6.2.5}$$

where $\xi(a)$ is the infection-age dependent per capita infectivity.

By (6.2.5) and (6.2.2),

$$I(r) = -\int_0^r \xi(a)S'(r-a)\mathcal{F}(a)da + \int_r^\infty \xi(a)j_0(a-r)\frac{\mathcal{F}(a)}{\mathcal{F}(a-r)}da. \tag{6.2.6}$$

We now proceed in a similar way as in Diekmann (1978); Thieme (1977). We define the cumulative infective force by

$$C(t) = \int_0^t I(r)dr. \tag{6.2.7}$$

After a substitution in the second integral of (6.2.6),

$$\begin{aligned} C(t) &= -\int_0^t \int_0^r \xi(a)S'(r-a)\mathcal{F}(a)dadr + C_0(t), \\ C_0(t) &= \int_0^t \int_0^\infty \xi(a+r)j_0(a)\frac{\mathcal{F}(a+r)}{\mathcal{F}(a)}dadr. \end{aligned}$$

We change the order of integration,

$$\begin{aligned} C(t) &= -\int_0^t \Big(\int_a^t S'(r-a)dr\Big)\xi(a)\mathcal{F}(a)da + C_0(t), \\ C_0(t) &= \int_0^t \int_0^\infty \xi(a+r)j_0(a)\frac{\mathcal{F}(a+r)}{\mathcal{F}(a)}dadr. \end{aligned}$$

By the fundamental theorem of calculus,

$$C(t) = \int_0^t \Big(S_0 - S(t-a)\Big)\xi(a)\mathcal{F}(a)da + C_0(t), \tag{6.2.8}$$

We have that $C_0(t) \to C_0^\infty$ with

$$C_0^\infty = \int_0^\infty \int_0^\infty \xi(a+r) j_0(a) \frac{\mathcal{F}(a+r)}{\mathcal{F}(a)} da dr, \tag{6.2.9}$$

provided that this integral exists. To find conditions for the latter we introduce the expectation of remaining infected life,

$$D(a) = \int_0^\infty \frac{\mathcal{F}(r+a)}{\mathcal{F}(a)} dr, \qquad a \geq 0, \tag{6.2.10}$$

i.e., the average time from infection age a to the time of disease death (Thieme, 2003, Sec. 12.4).

Proposition 6.2.1. *(a) Assume that ξ is bounded and let $\tilde{\xi}$ be its supremum. Then*

$$C_0^\infty \leq \tilde{\xi} \int_0^\infty j_0(a) D(a) da. \tag{6.2.11}$$

(i) If D is bounded and $\tilde{D}$ its supremum, then

$$C_0^\infty \leq \tilde{\xi} \tilde{D} J(0).$$

(ii) If $a^\sharp > 0$ and $j_0(a) = 0$ for $a > a^\sharp$ and D is bounded on $[0, a^\sharp]$, then

$$C_0^\infty \leq \tilde{\xi} J(0) \sup D([0, a^\sharp]).$$

(b) Assume that $j_0/\mathcal{F}$ is bounded. Then

$$C_0^\infty \leq \sup(j_0/\mathcal{F}) \int_0^\infty a \xi(a) \mathcal{F}(a) da.$$

For all practical purposes, there is a finite number of initially infected hosts which thus have a finite maximum infection age. So case (a)(ii) applies and the cumulative initial infected force is bounded.

C is increasing by (6.2.7). Assume that C_0^∞ exists. Then C_∞ exists. Further, in an epidemic model (without population turnover), the number of susceptibles $S(t)$ is

decreasing and converges to $S_\infty \geq 0$ as $t \to \infty$. We apply Beppo Levi's theorem of monotone convergence to (6.2.8) and obtain

$$\begin{aligned} C_\infty &= \big(S_0 - S_\infty\big)\kappa_1 + C_0^\infty, \\ \kappa_1 &= \int_0^\infty \xi(a)\mathcal{F}(a)da. \end{aligned} \tag{6.2.12}$$

6.3 No Extinction

The considerations in the previous sections hold for arbitrary incidence functions.

We now assume that the dynamics of the susceptible part of the host population obeys the differential equation

$$S' = -\sigma f(S, I) \leq 0, \tag{6.3.1}$$

where f is an upper density-dependent incidence function and I is the infective force (6.2.5).

Notice that $S(t) \leq S(0)$ and, by (6.2.5), $I(t) \leq \tilde{\sigma} J(t) \leq \tilde{\sigma}(J(0) + S(0))$ with $\tilde{\sigma} = \sigma\tilde{\xi}$, $\tilde{\xi} = \sup \xi$. By (6.1.1), there is some $c > 0$ (which depends on the initial data of the solution) such that S satisfies the differential inequality

$$S' \geq -cSI.$$

This differential inequality is solved by

$$S(t) \geq S_0 e^{-cC(t)}, \tag{6.3.2}$$

with the cumulative infective force $C(t)$ in (6.2.7), and

$$S_\infty \geq S_0 e^{-cC_\infty} > 0. \tag{6.3.3}$$

If we want some more information, we can substitute this inequality into (6.2.12),

$$C_\infty \le S_0\big(1 - e^{-cC_\infty}\big)\kappa_1 + C_0^\infty. \tag{6.3.4}$$

This can be rewritten as

$$1 \le S_0 \frac{1 - e^{-cC_\infty}}{C_\infty}\kappa_1 + \frac{C_0^\infty}{C_\infty}. \tag{6.3.5}$$

Notice that the right hand side of this equation is a strictly decreasing function of C_∞. So

$$C_\infty \le C^\sharp,$$

where $C^\sharp > 0$ is the unique solution of

$$1 = S_0 \frac{1 - e^{-cC^\sharp}}{C^\sharp}\kappa_1 + \frac{C_0^\infty}{C^\sharp}, \tag{6.3.6}$$

and

$$S_\infty \ge S_0 e^{-cC^\sharp} > 0. \tag{6.3.7}$$

Theorem 6.3.1. *We assume that for any $N > 0$ there exists some $c_N > 0$ such that*

$$f(S, I) \le c_N SI, \qquad 0 \le S, I \le N. \tag{6.3.8}$$

Further we assume that the initial cumulative infective force is bounded. Then an epidemic outbreak does not drive the host population into extinction.

That $S_\infty > 0$ was already noticed in Capasso and Serio (1978) for the system $0 \ge S' \ge -cSI$, $I' = -S' - \mu I$, but without the more precise information in (6.3.6) and (6.3.7).

For completeness, we also prove the following result.

Theorem 6.3.2. *We assume that for any $N > 0$ there exists some $\epsilon_N > 0$ such that*

$$f(S, I) \ge \epsilon_N SI, \qquad 0 \le S, I \le N. \tag{6.3.9}$$

Further, we assume that the initial cumulative infective force is unbounded. Then the epidemic outbreak drives the host population into extinction.

Proof. If C_0 is unbounded, then $C(t) \to \infty$ as $t \to \infty$ by (6.2.8). Similarly as above, for any solution there is some $\epsilon > 0$ such that $S' \leq -\epsilon SI$ and

$$S(t) \leq S_0 e^{-\epsilon C(t)} \to 0, \qquad t \to \infty.$$

□

6.4 Density-Dependent Incidence

Under density dependence incidence, $S' = -SI$, we obtain the equations,

$$S_\infty = S_0 e^{-C_\infty} > 0 \tag{6.4.1}$$

and

$$1 = S_0 \frac{1 - e^{-C_\infty}}{C_\infty} \kappa_1 + \frac{C_0^\infty}{C_\infty}$$

From the second equation, we see that, as $S_0 \to \infty$, also $C_\infty \to \infty$ and so $S_\infty \to 0$ by the first equation. While, in our model, host populations do not go completely extinct under density-dependent incidence, very large host population can be brought down to near-extinction by an epidemic outbreak and then go extinct by stochastic effects (which are not included in our model).

6.5 Fitting the Survival Function

Sizing up the curves in (Greer *et al.*, 2008, Fig.2), we first felt that the survival function $\mathcal{F}$ roughly looks like

$$\mathcal{F}(a) = \begin{cases} 1, & 0 \le a \le a_3, \\ e^{\mu(a_3 - a)}, & a > a_3. \end{cases} \tag{6.5.1}$$

with $a_3 \ge 6$ [days]. We call a survival function of this form a *delayed exponential survival function*.

It is difficult to obtain information about ξ. In the experiments, it was assumed that $\xi(a) = 0$ for $0 \le a \le a_1$ where a_1 is some number between 1 and 5 days: All infective larvae had an infection age between 5 and 6 days because 5 days are enough for infection to develop and the exposure lasted for one day.

By (6.2.10) and (6.5.1), the expected remaining infected life at infection age a is given by

$$D(a) = [a_3 - a]_+ + \frac{1}{\mu}$$

and provides a bounded (actually, eventually constant) function of a. The average time from the moment of infection till disease death is given by

$$D(0) = a_3 + \frac{1}{\mu}.$$

The variance of the time from infection to disease death is given by ((Thieme, 2003, Sec.12.3))

$$V = 2\int_0^\infty a\mathcal{F}(a)da - D(0)^2 = \mu^{-2}.$$

A least square fit of $\mathcal{F}$ to the survival date in Greer *et al.* (2008) provides estimates for a_3 and μ, which allow us to estimate $D(0)$, $a_3 = 11.4491$, $\mu = 0.5151$, and $D(0) = 11.4491 + 1/0.5151 \approx 13.390$. With this estimate of μ, we calculate

the variance $V = \mu^{-2} = 0.5151^{-2} \approx 3.7689$. Previous laboratory and field studies showed that most infected salamander larvae usually die within 8-14 days (Greer *et al.* (2008)). Notice that our figures show the time from infection to disease death of susceptible salamander larvae that were actually infected while (Greer *et al.*, 2008, Fig.2) considers all susceptible salamander larvae.

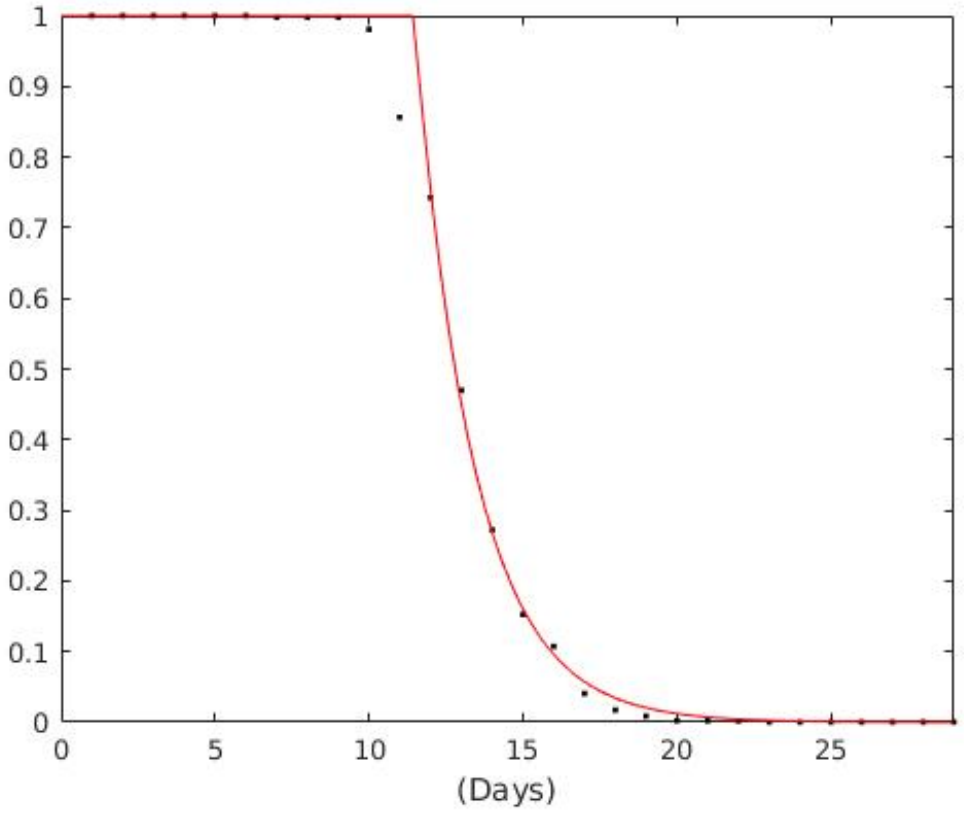

Figure 6.5.1: Each point is the proportion of remaining infected salamander larvae on that day. The curve is the fitted delayed exponential survival function.

Motivated by the work by Sartwell (Sartwell (1950, 1966)) who fitted lognormal distributions to the incubation periods of various infectious diseases, we also fitted a lognormal (Figure 6.5.3) and a gamma distribution (Figure 6.5.2) to the time from infection to disease death, both of which turn out to yield better fits than the delayed

exponential. Using the errors obtained from the least squares fits, we can generate Akaike weights via the least squares fit, where we determine the $\mathrm{AIC_c}$ value by

$$\mathrm{AIC}_C = n \log(\mathrm{SSE}) + 2K,$$

where n is the number of experiments, K is the number of parameters, and the SSE is the least square error. Once the $\mathrm{AIC_c}$ is obtained, we calculate the Akaike weights as we did in Section 3. The results can be seen in Table 6.1. According to (Burnham and Anderson, 2002, Chapter 2) (see also (Martcheva, 2015, Sec.6.4.1)), there is substantial support in the data if $\Delta AIC_\mathrm{c} \leq 2$, considerably less support if $4 \leq \Delta AIC_\mathrm{c} \leq 7$, and essentially no support if $\Delta AIC_\mathrm{c} \geq 10$.

Function	SSE	$\mathrm{AIC_c}$	$\Delta\mathrm{AIC_c}$	Akaike weight
Lognormal	4.5×10^{-3}	-249.52	0.00	.9315
Gamma	5.4×10^{-3}	-244.30	5.2203	.0685
Delayed Exponential	2.26×10^{-2}	-202.94	46.5783	7.158×10^{-11}

Table 6.1: Akaike weights are determined using the optimal least squares fit of the proposed function to the survival curve.

The gamma distribution, the density of which is $a^{\kappa-1}e^{-\theta a}$ (the normalizing constants have been dropped) has shape parameter $\kappa = 49.28$ and scale parameter $\theta = .2652$. The calculated mean and variance from this curve are 13.069 and 3.47 respectively. The expected remaining infected life satisfies $D(a) \to 1/\theta$ as $a \to \infty$ (Remark C.1.2).

If the time from infection to disease death is lognormally distributed, the survival function $\mathcal{F}$ has the form ((Thieme, 2003, (12.14)))

$$\mathcal{F}(a) = (2\pi)^{-1/2} \int_{b(a)}^{\infty} e^{-y^2/2} dy, \quad b(a) = (1/\zeta) \ln(a/m), \qquad a \geq 0. \tag{6.5.2}$$

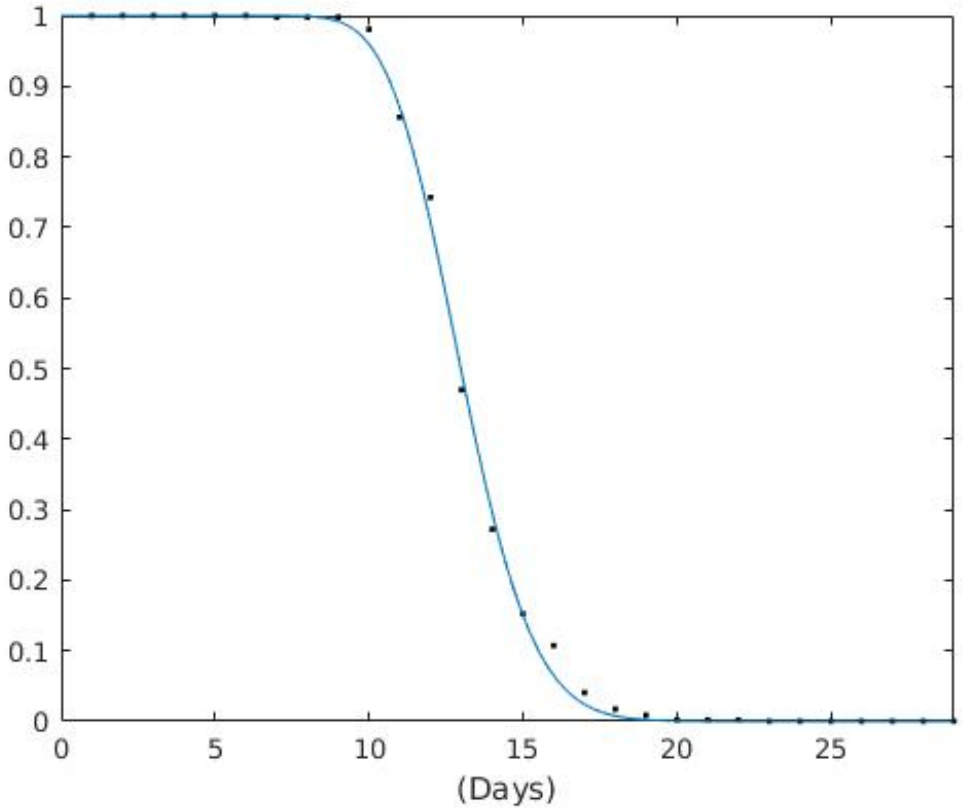

Figure 6.5.2: Each point is the proportion of remaining infected salamander larvae on that day. The curve is the fitted survival function if the time from infection to disease death is gamma distributed.

The best fit of the lognormal distribution has the mean of the associated normal distribution as $\ln m = 2.5619$, and the standard deviation of the associated normal distribution as $\zeta = 0.1429$. With these, we calculated the mean and variance of the lognormal distribution and found $D(0) = 13.0935$ days and 3.5371 respectively. If determined directly from the data, the mean is 13.6481 days and the variance is 4.0092.

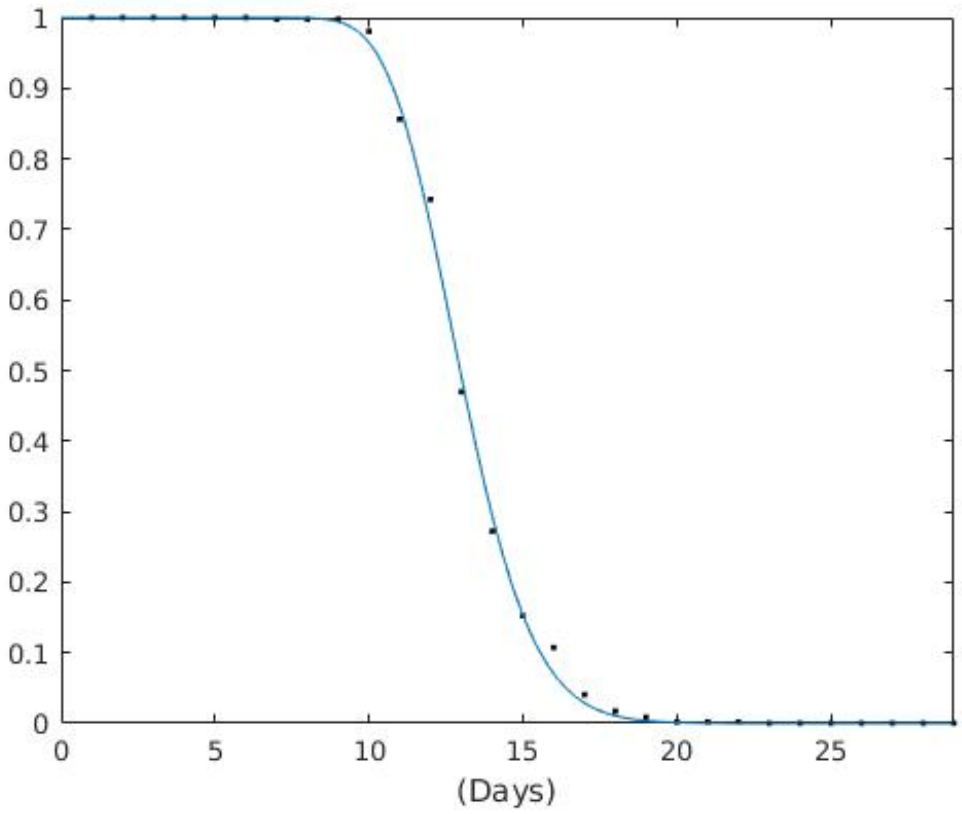

Figure 6.5.3: Each point is the proportion of remaining infected salamander larvae on that day. The curve is the fitted survival function if the time from infection to disease death is lognormally distributed.

If the time between between infection and disease death is lognormally distributed, the expected remaining infected life at infection age a, $D(a)$, converges to infinity as $a \to \infty$.

Theorem 6.5.1. *If the time from infection to disease death is lognormally distributed, then* $D(a) \to \infty$ *as* $a \to \infty$*,*

$$D(a) > \frac{\zeta^2 a}{\ln(a/m) + \zeta^2}, \qquad a/m \geq e^{1-\zeta^2}.$$

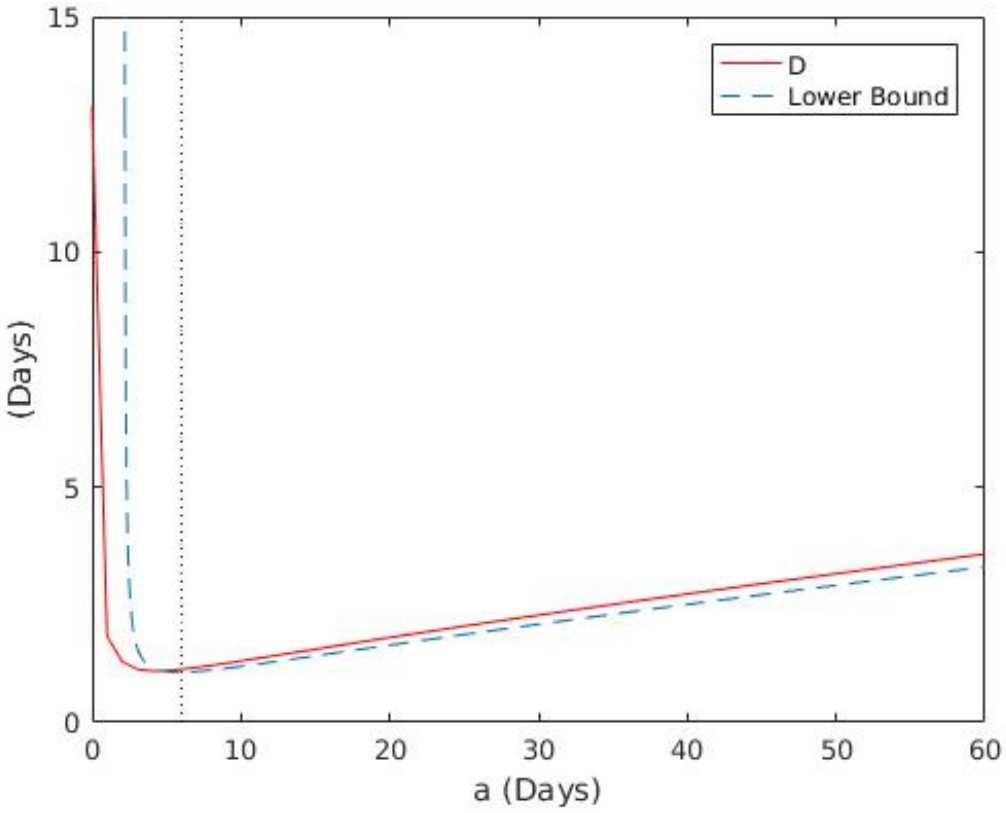

Figure 6.5.4: The solid curve shows the expected remaining infected life at infection age a and the dashed curve the lower estimate from Thm.6.5.1 if the time between infection and disease death is fitted by a lognormal distribution. The dotted vertical line indicates $a/m = e^{1-\zeta^2}$, which is the smallest point at which Thm.6.5.1 guarantees the lower bound estimate is valid.

See Appendix C for the proof and Figure 6.5.4 for an illustration. The unboundedness of D can make the cumulative initial infective force and thus the final size of the susceptible population very sensitive to the infection-age distribution of the initially infected hosts. This is illustrated by the subsequent remark which is also proved in Appendix C.

Remark 6.5.2. Assume that the time from infection to disease death is lognormally distributed and that there are $c > 0$ and $\xi_0 > 0$ such that $\xi(a) \geq \xi_0$ for all $a \geq c$. Then there exists some integrable $j_0 : \mathbb{R}_+ \to \mathbb{R}_+$ such that $\int_0^\infty j_0(a)da < \infty$ and $C_0^\infty = \infty$.

Chapter 7

DISCUSSION

Theoretical studies have shown that the potential of an infectious disease to drive a population into extinction by itself appears to be linked to the form of the disease incidence function, i.e., to the functional relationship between the rate of new cases on the one hand, $\sigma f(S, I)$, and to the amount of susceptibles S, infectives I and possible other classes of the host population on the other hand. Conventional knowledge seems to be restricted to the insight, though, that under density-dependent (aka mass action) incidence the host population cannot be driven into extinction (Kermack and McKendrick (1927)) while under frequency-dependent (formerly know as 'standard') incidence it can for appropriate parameter values (Busenberg *et al.* (1991); Busenberg and van den Driessche (1990); Getz and Pickering (1983); Greenhalgh and Das (1995); May *et al.* (1989); Thieme (1992); Zhou and Hethcote (1994)).

Many other types of incidence functions than density and frequency dependent incidences have been considered in the literature (see Chapters 2 and 2), but the respective studies have been more interested in so called "complicated behavior" of the disease dynamics (Derrick and van den Driessche (2003); Glendinning and Perry (1997)) than in the more fundamental question of host extinction versus mere host decline.

Our mathematical considerations identified three classes of incidence functions that give different results for this fundamental problem.

(i) Power incidence functions, in which the functional dependence of the incidence on the number of infective hosts is give by a term I^p with $0 \leq p < 1$, are associated

with unconditional (irrespective of parameter and initial values) host extinction in epidemic outbreaks.

(ii) Homogeneous incidence functions, in which the risk of an average susceptible host to be infected depends on the ratio of infectives to susceptibles, are mostly associated with parameter-dependent host extinction: In certain parameter regions of the transmission coefficient σ and the per capita disease death rate μ, epidemic outbreaks lead to host extinction while in others they lead to more host decline. Homogeneous incidence functions contain the frequency-dependent incidence as a special case but are a much wider class than that. The homogeneous power incidence function $f(S, I) = S^q I^p$ with $q + p = 1$, $0 \leq q, p \leq 1$, which contains the constant risk incidence as the special case $q = 1, p = 0$ is associated with unconditional host extinction.

(iii) Upper density-dependent incidences, for which for any $N > 0$ there exists some $c_N > 0$ such that $f(S, N) \leq c_N SI$ for $0 \leq S, I \leq N$, are never associated with host extinction but only with (sometimes very substantial) host decline. They include density-dependent, negative binomial and interpolation incidences.

This dissertation has been partially motivated by the infection experiments in Greer *et al.* (2008) involving tiger salamander larvae and ranavirus. Using a maximum likelihood fit and the Akaike information criterion, the attempt was made to determine which incidence functions may be appropriate to describe this infection process. We repeat this attempt considering some more incidence functions. The incidence functions that fare best are powers $\sigma S^q I^p$ and $\sigma S I^p$ with $p, q \in (0, 1)$, the negative binomial $\sigma S k \ln(1 + I/k)$ and interpolations $\sigma \frac{SI}{1+I^{1-p}}$ between density and power incidence (Notice that we flipped p and q compared to Greer *et al.* (2008) and rescaled the negative binomial to make it look more like the three others.) Both density- and frequency-dependent incidence fare poorly, even much worse than the constant risk in-

cidence σS. The negative binomial and asymmetric frequency-dependent incidences, as the best fitting homogeneous incidence functions with $\Delta\mathrm{AIC_c}$ values of 3.17 and 3.83, are in some kind of grey zone as, according to (Burnham and Anderson, 2002, Chapter 2) (see also (Martcheva, 2015, Sec.6.4.1)), there is substantial support in the data if $\Delta AIC_c \leq 2$ and considerably less support if $4 \leq \Delta AIC_c \leq 7$.

According to our analysis of epidemic models of simple SI type, this leaves the question completely open as to whether ranavirus has the potential to drive salamander populations into extinction. On the one hand, we show that under any incidence containing I^p with $0 < p < 1$ the host population goes extinct, for any values of the transmission coefficient σ and all initial values of the susceptible and infective part of the populations. On the other hand, we show that, under the negative binomial incidence and the interpolation incidence, the host population never goes extinct. More generally, under any incidence function satisfying $f(S, I) \leq cSI$, with a constant $c > 0$, extinction never occurs in an epidemic model. This also holds for models that take into account that infected salamander larvae do not immediately become infective. In Table 7.1, we modify Table 3.4 to show only the type, NLL, and Akaike weight in order to give a clear picture as to why the question of host extinction is still completely open.

Notice that σSI^p and $\sigma \frac{SI}{1+I^{1-p}}$ behave very similarly for large values of I; so it is the behavior of the incidence function at low values of I that matters in the question of host extinction.

The constant risk incidence σS fits the experiments in Greer *et al.* (2008) not too badly, counter-intuitively so at first sight. (It should be needless to state that constant risk frequency always leads to host extinction.) The exposure tanks, in which the experiments were conducted, measured $55 \times 38 \times 30$ cm and contained 55 l of aged tap water. The size of a typical salamander larva used in the experiments is not

Types of transmission	Function	Type	NLL	Δ AICc
Power (in I only)	σSI^p	P	20.20	0.00
Negative Binomial (in I)	$\sigma kS\ln(1+I/k)$	UD	20.66	0.92
Interpolating	$\sigma SI/(1+I^{1-p})$	UD	20.95	1.48
Contact Rates	$\sigma SI/(1+\kappa I+\sqrt{1+2\kappa I})$	UD	21.32	2.23
Power (in S and I)	$\sigma S^q I^p$	P	20.05	2.33
Homogeneous Power law	$\sigma S^q I^p$	H,P	21.38	2.36
Negative Binomial and Frequency	$\sigma S\ln(1+I/(S+I))$	UD	22.66	2.53
Predation	$\sigma SI/(a^\gamma+I^\gamma)^{1/\gamma}$	UD	20.23	2.68
Frequency with Exponent	$\sigma S(I/(S+I))^p$	H	21.70	2.99
N. Binomial and Asymetric Frequency	$\sigma S\ln(1+I/(pS+qI))$	H	21.83	3.26
Asymetric Frequency	$\sigma SI/(pS+qI)$	H	21.83	3.26
Weighted Minimum	$\sigma\min\{S,\alpha I\}$	H	22.04	3.68
Michaelis-Menten	$\sigma SI/(1+I/k)$	H	22.15	3.90
General Homogeneous	$\sigma SI/(pS^\beta+qI^\beta)^{(1/\beta)}$	H	21.34	4.90
Constant Risk	σS	H,P	24.07	5.35
Ivlev/Skellam Law	$\sigma kS(1-e^{-I/k})$	UD	23.56	6.72
Blackwell	$\sigma S\min\{k,I\}$	N/A	23.91	7.40
Very General	$\sigma SI/((pS)^\alpha+(qI)^\beta)^\gamma$	N/A	20.05	8.46
Sexual Transmission	$\sigma\dfrac{SI}{1+\frac{S+I}{k}+\sqrt{1+2\frac{S+I}{k}}}$	UD	39.31	38.21
Beddington/DeAngelis/Dietz	$\sigma SI/(1+(S+I)/k)$	UD	39.64	38.87
Power (in S only)	$\sigma S^q I$	N/A*	40.50	40.60
Density-Dependent	σSI	UD	43.86	44.92
Frequency Dependent	$\sigma SI/(S+I)$	H	50.63	58.46
Minimum	$\sigma\min\{S,I\}$	H	53.75	64.70

Table 7.1: Fits of various incidence types to the infection experiments in Greer *et al.* (2008) using the stochastic method. The incidence type is listed here for ease. P is power law, UD is upper density-dependent, and H is homogeneous. N/A means that this function does not fall under any of the three categorizations. For power in S only, the general function $\sigma S^q I$ is not of any of the three types, however, if $q>1$ (as was found for the best fit) it is upper density-dependent, hence the classification of N/A*.

reported in Greer *et al.* (2008), but the typical length at maturity was chosen as 40 mm in the model in Bolker *et al.* (2008). Further, as reported in Greer *et al.* (2008), "when two larvae were placed in a tank they tended to spend time in close proximity together." Finally, the minimum initial ratio of infective to susceptible salamander larvae in all infection experiments has been 0.1; at the onset of an epidemic in the field one may expect much lower ratios. The way they have been designed, the experiments may not have caught the shape of the incidence function for low values of I, which decides about the disease's potential of host extinction. This may be why the power incidence function fares much better than homogeneous incidence functions in the fits. From a theoretical point, power incidence is hardly acceptable because it allows infectious outbreaks ex nihilo (see Remark 4.1.3).

Before our considerations in Section 3.3, we thought that another reason homogeneous incidence functions do not fit so well might be that the theoretical underpinning of the fitting procedure in Section 3.1 is only rigorous for incidence functions that are linear in S, i.e. $f(S, I) = S\phi(I)$. However, the continuous time Markov chain calculations do not provide a substantially better fit than the ODE approximation, which disproves that conjecture.

For upper density-dependent incidences, it is possible to include in the model that infected salamander larvae are not immediately infective and that the time from infection to disease death is not exponentially distributed. From among delayed exponential, gamma and lognormal distributions, the lognormal distribution yields the best fit. For lognormal distributions the expected remaining infected life is an unbounded function of infection age, the time since infection. This makes the final size of an epidemic quite sensitive to the infection age distribution of the initial infectives. So even a small number of initial infectives can cause a severe decline (though not

extinction) of the host population if a large proportion of the initial infectives has a high infection age.

Although the question 'can diseases by themselves can eradicate the host species?' is still open, this may be in part to the limited data we have access to. Perhaps with more data, a trend would emerge, allowing us to make a more educated statement concerning which incidence functions are the most appropriate.

PART 2 -

SI MODEL: ENDEMIC PERSPECTIVE

Chapter 8

INTRODUCTION

We continue our mathematical exploration into the role of a the disease incidence function, now using an endemic model. One of the purposes of this part is to show that frequency-dependent incidence is not the only type of incidence that can cause host extinction, but that incidences that are quite close to those found to be good fits in Greer *et al.* (2008) can do that as well.

To do that we choose as simple a model as possible, namely of SI type, with two ordinary differential equations for the density of susceptibles, S and the density of infectives, I,

$$S' = Sg(S) - \sigma f(S, I), \qquad I' = \sigma f(S, I) - \mu I. \tag{8.1.1}$$

Here, to keep our analysis simple, we have assumed that infective individuals do not reproduce and to not compete for vital resources. The reason may be that infected individuals are too weak to sample food and care for their offspring or that they lose interest in doing so. The term $g(S)$ is the per capita growth rate of the susceptible part of the population at density S, i.e., the difference of the per capita birth rate and of the per capita death rate of susceptible individuals. Biologically, we expect the relation $g(S) + \mu \geq 0$ to hold for all $S \geq 0$, i.e. that susceptible individuals do not die at a faster rate than infective individuals. Although we do not enforce this in our analysis, it leads us to consider a general growth function instead of using logistic growth, which violates this condition. In Kuang and Beretta (1998) a similar model is analyzed as a predator-prey model, using logistic growth and asymmetric frequency dependent predation. Since Kuang and Beretta (1998) are viewing this model as a

predator-prey model, the choice of logistic growth is not concerning, as it is in our case. Due to the closeness of these two models, we will compare them throughout this part. We make the plausible assumption that g is differentiable and $g'(S) < 0$ for $S > 0$ and $g'(0) \leq 0$. This implies g is a decreasing and continuous function of the size of the susceptible population due to resource competition. We also assume that $g(0) > 0$ and that there is a carrying capacity $K > 0$ for the host population where $g(K) = 0$. Since g is strictly decreasing, K is uniquely determined.

$\sigma f(S, I)$ denotes the *disease incidence*, i.e., the number of new infections per unit of time. f is called the *incidence function* and σ the *incidence coefficient*. It is difficult to give a generally valid interpretation of σ as it depends on the incidence function. Even the dimension of σ may change, see (Greer *et al.*, 2008, Table 2), and Tables 3.1, and 3.4.

μ denotes the death rate of infective individuals. Since susceptible individuals should never suffer a higher per capita death rate than infective individuals, the consistency relation $g(S) + \mu \geq 0$ should hold for all $S \geq 0$. We do not make this assumption, but it motivates us to consider a general class of growth rates instead of the usual logistic one, $g(S) = \gamma - \nu S$, often written as $\gamma(1 - (S/K))$, with positive constants γ, ν, K.

In the context of system (8.1.1), the incidence is frequency-dependent if the incidence function f is of the form

$$f(S, I) = 2\frac{SI}{S + I}, \qquad S, I \geq 0. \tag{8.1.2}$$

This f is (positively) homogeneous (of degree one), i.e.,

$$f(\alpha S, \alpha I) = \alpha f(S, I), \qquad \alpha, I, S \geq 0. \tag{8.1.3}$$

In order to compare the destructive potential of homogeneous incidence functions, we introduce the normalization,

$$f(1,1) = 1. \tag{8.1.4}$$

This explains the factor 2 in (8.1.2) which may appear strange at first sight.

Model (8.1.1) can be rewritten to be the same as the model in Kuang and Beretta (1998) (which is viewed as a predator-prey system), using $g(S) = a\Big(1 - \frac{S}{K}\Big)$ and $f(S,I) = \frac{SI}{\frac{1}{m+1}S + \frac{m}{m+1}I}$ (normalized so $f(1,1) = 1$). Many of the parameters transfer fairly clearly between our work and that of Kuang and Beretta (1998), however we point out that the normalization causes a slight change in the infection coefficient, namely that $\sigma = \frac{f}{1+m}$. Additionally, the requirement of $f = c$ in Kuang and Beretta (1998) is needed to account for an infection, as opposed to predation.

We will show that, for any homogeneous incidence function f, the disease will drive the host species into extinction for certain parameter constellations (σ sufficiently large) and that the host and the parasite coexist for other parameter constellations. This means that the same parasite can drive a host into extinction at one location and can coexist with the same host at another location. We also observe that in the case of a homogeneous incidence function, we have that the *incidence coefficient*, σ, has the units of day^{-1}. In this case, σ has the interpretation of proportion of infections per day.

Here are some examples for homogeneous incidence functions,
the constant risk incidence

$$f(S,I) = S, \tag{8.1.5}$$

homogeneous power laws,

$$f(S,I) = S^q I^p; \qquad q,p > 0, \quad p+q = 1, \tag{8.1.6}$$

and asymmetric versions of frequency-dependent incidence,

$$f(S,I) = \frac{SI}{pS + qI}; \qquad q, p > 0, \quad p + q = 1. \tag{8.1.7}$$

The numbers p and q in (8.1.6) and (8.1.7) are related to the contact activity of susceptibles and infectives. If $p > q$, susceptibles are more active than infectives. The standard, symmetric, incidence function is contained in (8.1.7) as the special case $p = q = 1/2$. More general classes of homogeneous incidence functions are presented in Section 12.2 and 12.3.2.

The constant risk incidence (8.1.5) (which is the limiting case of both (8.1.6) and (8.1.7) for $p = 0$ and $q = 1$) looks like an unlikely candidate because it does not depend on the density of infectives at all; still it fits the experiments in Greer *et al.* (2008) relatively well. The exposure tanks, in which the experiments were conducted, measured $55 \times 38 \times 30$ cm and contained 55 l of aged tap water. The size of a typical salamander larva used in the experiments is not reported in Greer *et al.* (2008), but the typical length at maturity was chosen as 40 mm in the model in Bolker *et al.* (2008). Further, as reported in Greer *et al.* (2008), "when two larvae were placed in a tank they tended to spend time in close proximity together." Finally, the minimum initial ratio of infective to susceptible salamander larvae in all infection experiments has been 0.1; at the onset of an epidemic in the field one may expect much lower ratios.

For a field model, one may like to modify the constant risk incidence function to another homogeneous one,

$$f(S,I) = \sigma \min\{S, \gamma I\}, \tag{8.1.8}$$

where γ should be chosen sufficiently larger than 1. In the infection experiments in Greer *et al.* (2008), there would be no difference between the constant risk incidence

and the minimum incidence if γ were chose greater than or equal to 10. Notice that also (8.1.6) and (8.1.7) come close to (8.1.5) if $p > 0$ is chosen almost zero.

The homogeneous power law (8.1.6) has not been tested in Greer *et al.* (2008), but it is close to the power laws in Table 2 in Greer *et al.* (2008) where $f(S, I) = SI^p$ with $p = 0.255$ or $f(S, I) = S^q I^p$ with $q = 0.953$ and $p = 0.263$. (Notice that p and q have been switched.) Both these power laws provided relatively good fits. The results in Table 3.1, where we use the same fitting procedure used in Greer *et al.* (2008), found $p = 0.121$ and $q = .0889$ for the homogeneous power law, with a worse fit as compared to the power in S and I (Δ AIC_Cs of 3.94 and 2.18). In Table 3.4, we use the continuous time Markov Chain and obtain $p = 0.126$ and $q = .0884$ for the homogeneous power law, and a fit just slightly worse than power in S and I (Δ AIC_Cs of 2.33 and 2.36). The parameters q and p can be related to the relative contact activity of susceptibles and infectives, respectively.

In Section 10.2 we show that a power law incidence function $f(S, I) = S^q I^q$ has initial-data dependent host extinction for any $q \in (0, 1)$ and any $p > 0$, while in Section 10.3 we show power law incidences with $0 < p < 1 \leq q$ have global stability of an interior equilibrium.

In Section 12.1 we prove that the constant risk incidence function $f(S, I) = S$ has the potential of global host extinction and if one compares the potential for unconditional (i.e., for initial conditions independent) parasite-mediated host extinction of the constant risk incidence and the symmetric frequency dependent incidence, one finds that $\sigma \geq g(0)$ is sufficient and necessary for the constant risk incidence while $\sigma \geq \frac{1}{2}(g(0) + \mu)$ is sufficient and necessary for the symmetric frequency dependent incidence.

All incidence functions $f(S, I)$ in Greer *et al.* (2008) and in this dissertation have the following properties:

(i) $f(S, I)$ is an increasing and concave function of both $S \geq 0$ and $I \geq 0$;

(ii) f is continuous;

(iii) $f(0, I) = 0$ for all $I \geq 0$.

All incidence functions mentioned so far and in Greer *et al.* (2008), except the constant risk function, also have the following plausible property.

(iv) $f(S, 0) = 0$ for all $S \geq 0$.

In Section 12.7, we will give conditions for general homogeneous incidence functions to have global and initial-data dependent disease-mediated host extinction. In order to explore whether our conditions are sharp, we give conditions for uniform host persistence in Section 12.8 and for disease extinction in Section 12.9.

Without making further assumptions on the incidence function, it is difficult to give a complete, succinct, non-overlapping, and gapless description of the scenarios that can happen. Here is a very rough one. Recall that f is an increasing function of both variables and that f is homogeneous.

Theorem 8.1.1 (Preview of scenarios)**.**

$f(\infty, 1) < \frac{\mu}{\sigma}$	*The equilibrium with no disease and the host at carrying capacity is locally asymptotically stable.*
$f\left(\frac{\mu}{g(0)}, 1\right) <$ $\frac{\mu}{\sigma} < f(\infty, 1)$	*The disease invades the host population and persists, and there exists a coexistence equilibrium, where both the host and the infectious agent are present.*
$\frac{\mu}{\sigma} < f\left(\frac{\mu}{g(0)}, 1\right)$	*The disease drives its host into extinction: If* $I(0)/S(0) > 0$*, then* $S(t) \to 0$ *as* $t \to \infty$*.*

In general, nothing can be said about the stability of the coexistence equilibrium in the second scenario. The last scenario comes with the condition that $f(S, I)$ is strictly increasing in $I > 0$ (actually f partially differentiable in I and $\partial_I f(S, I) > 0$) which is satisfied for all homogeneous incidence functions except the minimum incidence and the constant risk incidence (which are analyzed in Section 12.10 and Section 12.1, respectively). The condition for host extinction is ambiguous as to whether increasing disease mortality facilities or impedes host extinction. Because f is homogeneous, this condition can be rewritten as $1 < f(\frac{\sigma}{g(0)}, \frac{\sigma}{\mu})$. Since f is increasing in both arguments, increasing disease mortality impedes host extinction because infectious hosts have less time available to transmit the disease.

The scenarios in Theorem 8.1.1 do not capture the bistable cases in which there is a coexistence equilibrium but there is also initial-condition-dependent disease-mediated host extinction. The most extreme cases occur for power laws with $p \in (0, 1]$ and $q \in (0, 1)$, (even if it is inhomogeneous) where this happens for any parameter constellation (Theorem 10.2.4) and the host goes extinct in finite time. Unconditional host extinction happens for power incidence functions only when they are homogeneous (Section 12.11). In Section 10.2 we have numerical support for what we call 'practical host extinction,' where all initial conditions excluding the unstable interior equilibrium will tend toward host extinction, when σ is large enough. Initial-condition-dependent disease-mediated host extinction does not occur for the generalized frequency-dependent incidence with $0 < p \leq 1/2 \leq q < 1$ and for the constant risk incidence.

Conditions for initial-condition-dependent and unconditional host extinction will be exemplified for the minimum incidence function in Section 12.10. In Section 12.12 and 12.13, we will make a detailed bifurcation analysis for asymmetric frequency-

dependent incidence with sharp conditions for both global and initial-data-dependent host extinction.

As a second purpose of this part, we will show that this simple model, with the incidence function (8.1.7) and $p > q$, can have rich dynamics including periodic solutions, heteroclinic orbits, and bistability. It appears to be the smallest epidemic model found so far to have such rich dynamics. In Kuang and Beretta (1998), it was proven that a similar predator-prey model could not have non-trivial periodic orbits if the interior equilibrium is locally asymptotically stable. There is numerical evidence that our simple model can support a locally asymptotically stable interior equilibrium surrounded by an unstable periodic orbit. The reason for this is that the logistic growth function in Kuang and Beretta (1998) is replaced by Ricker type growth rate to satisfy the specific consistency rules of an epidemic model rather than a predator-prey model.

Chapter 9

SOME GENERAL RESULTS

As said before, we make the plausible assumption that the per capita growth rate $g(S)$ of the susceptible part of the population is a strictly decreasing and continuous function of the size S of that part. Further we assume that $g(0) > 0$ and $g(S) < 0$ for some $S > 0$. Then there is a unique $K \in (0, \infty)$ such that $g(K) = 0$. K is called the *carrying capacity* of the host population. We use the notation

$$S^\infty = \limsup_{t\to\infty} S(t) \qquad \text{and} \qquad S_\infty = \liminf_{t\to\infty} S(t).$$

Theorem 9.1.1. *For all non-negative solutions S and I, we have $S(t) \le \max\{K, S(0)\}$ for all $t \ge 0$ and $S^\infty \le K$. For the total host population size $H(t) = S(t) + I(t)$,*

$$H(t) \le \max\{K, S(0)\}\frac{g(0)+\mu}{\mu} + H(0)e^{-\mu t}$$

and

$$H^\infty \le \sup\{S(g(S)+\mu)/\mu; S \in [0, S^\infty]\} \le S^\infty\frac{g(0)+\mu}{\mu} \le K\frac{g(0)+\mu}{\mu}.$$

Proof. Notice that for any solution, $S'(t) < 0$ as long as $S(t) > K$. This implies that S is bounded and $S^\infty \le K$. If $S(t) < K$ for some $t > 0$. Then $S(s) \le K$ for all $s \ge t$. We add the differential equations for S and I,

$$H' = Sg(S) - \mu I = S(g(S)+\mu) - \mu H.$$

Since S is bounded, so is H. The first inequality for H follows from using an integrating factor. By the fluctuation method, there exists a sequence (t_n) with $t_n \to \infty$,

$H(t_n) \to H^\infty$, and $H'(t_n) \to 0$. We substitute these relations into the differential equation for H,

$$0 = \lim_{n\to\infty} [S(t_n)(g(S(t_n)) + \mu) - \mu H(t_n)].$$

By the limit rules,

$$H^\infty = \frac{1}{\mu} \lim_{n\to\infty} [S(t_n)(g(S(t_n)) + \mu)].$$

After choosing a subsequence, $S(t_n) \to S^\diamond$ for some $S^\diamond \in [0, S^\infty]$ and, since g is continuous,

$$H^\infty = \frac{1}{\mu}[S^\diamond(g(S^\diamond) + \mu)].$$

This implies the formulas for H^∞. □

Corollary 9.1.2. *If $S(t) \to 0$ as $t \to \infty$, then $H(t) = S(t) + I(t) \to 0$.*

With our assumptions on f, we only have local Lipschitz continuity on $(0, \infty)$, thus only solutions starting and staying in $(0, \infty)^2$ are subject to the standard uniqueness proof. In particular, we can have spontaneous outbreaks of the disease although there are no infectives initially present. However, this is not possible for the susceptible population.

Theorem 9.1.3. *If $S(r) = 0$ for any $r \geq 0$, then $S(t) = 0$ for $t \geq r$.*

Proof. We perform a time shift so we can assume that $S(0) = 0$. We only wish to consider non-negative solutions for S and I, which allows us to create the differential inequality $S' \leq Sg(S)$. We integrate the differential inequality:

$$\begin{aligned} S' &\leq Sg(S) \\ e^{-\int_0^t g(S(r))dr}(S' - Sg(S)) &\leq 0 \\ \frac{d}{dt}\left[e^{-\int_0^t g(S(r))dr} S\right] &\leq 0 \\ e^{-\int_0^t g(S(r))dr} S(t) &\leq 0. \end{aligned}$$

We see that the only solution is $S \equiv 0$. $\square$

With this, we can make an improvement to our uniqueness considerations.

Theorem 9.1.4. *Solutions with $S(0) \geq 0$ and $I(0) > 0$ are unique in forward time.*

Proof. Suppose $x(t) = (x_1(t), x_2(t))$ and $y(t) = (y_1(t), y_2(t))$ are solutions to (8.1.1) with $x(0) = y(0)$. Since our assumptions imply that the vector field is locally Lipschitz on $(0,\infty)^2$, if $0 < x_1(t), y_1(t)$ for all $t \geq 0$, solutions are unique. If $0 < x_1(t), y_1(t)$ does not hold, call t_0 the first time where x or y solution enters $\{0\} \times (0, \infty)$. Without loss of generality, we choose $x_1(t_0) = 0$. Suppose, toward contradiction, that $y_1(t_0) > 0$. However, this clearly cannot happen, since $x(t) = y(t)$ when $x \in (0, \infty)^2$ or $y \in (0, \infty)^2$, by uniqueness of solutions in $(0, \infty)^2$. In a similar fashion, we can see $x_2(t_0) = y_2(t_0)$.

Therefore, we have $x(t_0) = y(t_0)$. Using Theorem 9.1.3, we have $S(t) = 0$ for $t \geq t_0$, which is equivalent to $x_1(t) = y_1(t) = 0$ for $t \geq t_0$. Then, for $t \geq t_0$, system (8.1.1) reduces to

$$S(t) \equiv 0, \qquad I' = -\mu I,$$

which has the unique solution for $t \geq t_0$ of $I(t) = I(t_0)e^{-\mu(t-t_0)}$. Thus $x(t) = y(t)$ for $t \in [0, t_0)$, and $t \in [t_0, \infty)$. $\square$

Since we may not have unique solutions, we are motivated to obtain a persistence result which does not require uniqueness of solutions. We do so in a very general manner.

Let D be an nonempty subset of $\mathbb{R}^N$, $N \in \mathbb{N}$, and $f : D \to \mathbb{R}^N$ be continuous.

Assume that, for any $x^\circ \in D$, there exists a solution $x : \mathbb{R}_+ \to D$ of $x' = f(x)$ on $\mathbb{R}_+$, $x(0) = x^\circ$, which does not need to be unique.

Let $\rho : D \to \mathbb{R}_+$ be continuous. The vector field f is called *uniformly ρ-persistent* if there exists some $\epsilon > 0$ such that $\liminf_{t\to\infty} \rho(x(t)) \geq \epsilon$ for all solutions $x : \mathbb{R}_+ \to D$ of $x' = f(x)$ on $\mathbb{R}_+$ with $\rho(x(0)) > 0$.

The vector field f is called *uniformly weakly ρ-persistent* if the same statement holds with the limit inferior being replaced by the limit superior.

Obviously, uniform persistence implies uniform weak persistence. We explore conditions under which the converse holds. Since we do not assume that solutions to $x' = f(x)$ are uniquely determined by their initial conditions, the results in Smith and Thieme (2011); Zhao (2003) cannot be directly applied though the proofs can be adapted.

(CA) There exists a closed subset B of D such that the following hold:

(i) For any $0 < \epsilon_2 < \infty$, the set $\{x \in B; \rho(x) \leq \epsilon_2\}$ is compact.

(ii) For any solution $x : \mathbb{R}_+ \to D$ to $x' = f(x)$ on $\mathbb{R}_+$ with $\rho(x(0)) > 0$, there exists some $r > 0$ such that $x(t) \in B$ for all $t \geq r$.

(iii) There exists no $t \in (0, \infty)$, $s \in (0, t)$ and no solution $x : [0, t] \to B$ of $x' = f(x)$ on $[0, t]$ such that $\rho(x(0)) > 0$, $\rho(x(s)) = 0$ and $\rho(x(t)) > 0$.

Theorem 9.1.5. *Suppose that f maps bounded subsets of D into bounded subsets of $\mathbb{R}^N$ and that the assumption (CA) is satisfied. Then f is uniformly ρ-persistent if it is weakly uniformly ρ-persistent.*

Proof. Suppose that f is weakly uniformly ρ-persistent but not uniformly ρ-persistent. Then there exists some $\epsilon_0 > 0$ such that

$$\limsup_{t\to\infty} \rho(x(t)) > \epsilon_0 \tag{9.1.1}$$

for all solutions $x : \mathbb{R}_+ \to D$ to $x' = f(x)$ on $\mathbb{R}_+$ with $\rho(x(0)) > 0$.

Further, there exists a sequence (ϵ_n) in $(0, \epsilon_0/2)$ and a sequence (x_n) of solutions $x_n : \mathbb{R}_+ \to D$ of $x_n' = f(x_n)$ on $\mathbb{R}_+$ such that, for all $n \in N$, $\rho(x_n(0)) > 0$ and $\limsup_{t\to\infty} \rho(x_n(t)) > \epsilon_0$ and $\liminf_{t\to\infty} \rho(x_n(t)) < \epsilon_n$.

Then there exist sequences (r_n), (s_n), $t_n)$, (u_n) in $(0, \infty)$ such that, for all $n \in \mathbb{N}$,

$$
\begin{aligned}
&r_n > n,\\
&\rho(x_n(r_n)) = \epsilon_0, \qquad \rho(x_n(r_n + s_n)) = \epsilon_n, \qquad \rho(x_n(r_n + s_n + t_n)) = \epsilon_0/2,\\
&\rho(x_n(r_n + s_n + t_n + u_n)) = \epsilon_0,\\
&\rho(x_n(s)) < \epsilon_0, \quad s \in (r_n, r_n + s_n + t_n + u_n),\\
&x_n(t) \in B, \quad t \geq r_n.
\end{aligned}
$$

Set $y_n : \mathbb{R}_+ \to D$ by $y_n(t) = x_n(r_n + t)$ for $t \geq 0$. Then $y_n' = f(y_n))$ on $\mathbb{R}_+$ and

$$
\begin{aligned}
&\rho(y_n(0)) = \epsilon_0, \qquad \rho(y_n(s_n)) = \epsilon_n, \qquad \rho(y_n(s_n + t_n)) = \epsilon_0/2,\\
&\rho(y_n(s_n + t_n + u_n)) = \epsilon_0,\\
&\rho(y_n(s)) < \epsilon_0, \quad s \in (0, s_n + t_n + u_n),\\
&y_n(t) \in B, \quad t \geq 0.
\end{aligned}
$$

Since the set $B_0 = \{x \in B; \rho(x) \leq \epsilon_0\}$ is compact by (CA) (i), there is some $c > 0$ such that $\|y_n(t)\| \leq c$ for all $n \in \mathbb{N}$ and $t \in [0, s_n+t_n+u_n]$. Since f maps bounded subsets of D into bounded subsets of $\mathbb{R}^N$, there is some $\tilde{c} > 0$ such that $\|y_n'(t)\| = \|f(y_n(t))\| \leq \tilde{c}$ for all $n \in \mathbb{N}$ and $t \in [0, s_n + t_n + u_n]$. Since ρ is uniformly continuous on the compact set B_0, this implies that the sequence (u_n) is bounded away from 0.

We claim that $s_n + t_n + u_n \to \infty$ as $n \to \infty$.

If not, after choosing subsequences, $s_n \to s$, $t_n \to t$ and $u_n \to u$ with $s, t, u \in (0, \infty)$, $u > 0$. By the Arzela-Ascoli theorem, $y_n \to y$ uniformly on $[0, s + t]$ with some continuous function $y : [0, s + t] \to B$ which solves $y' = f(y)$ on $[0, s + t]$ and satisfies $\rho(y(0)) > 0$, $\rho(y(s)) = 0$, and $\rho(y(s + t)) > 0$.

This contradicts (CA).

So $s_n + t_n + u_n \to \infty$ as $n \to \infty$. By the Arzela-Ascoli theorem, $y_n \to y$ uniformly on all bounded subsets of $\mathbb{R}_+$ with some continuous function $y : \mathbb{R}_+ \to B$ which solves $y' = f(y)$ on $\mathbb{R}_+$ and satisfies $\rho(y(0)) > 0$ and $\rho(y(t)) \leq \epsilon_0$ for all $t \geq 0$. This is a contradiction to (9.1.1). □

Theorem 9.1.6. *If $S(t) \to K$ as $t \to \infty$ then $I \to 0$ as $t \to \infty$*

Proof. We wish to apply Barbalat's Lemma, which requires differentiability of the vector field and uniform continuity of $S'(t)$. Since both S and I are bounded by Theorem 9.1.1, S' is uniformly continuous on $\mathbb{R}_+$, and thus the vector field meets the requirements.

Therefore, we have that $\lim_{t\to\infty} S'(t) = 0$, which provides us with

$$0 = Kg(K) - \lim_{t\to\infty} f(K, I(t)),$$

which implies $\lim_{t\to\infty} f(K, I(t)) = 0$. By our assumptions on f, this equality can only be achieved if $\lim_{t\to\infty} I(t) = 0$. □

Corollary 9.1.7. *If $S(t) \geq K$ for all $t \geq 0$, then $I \to 0$ as $t \to \infty$.*

Chapter 10

POWER LAW INCIDENCES

10.1 General Results

Power laws have been suggested early on in the history of mathematical epidemiology ((Bailey, 1975, p.128), (Capasso, 1993, Ch.3) Liu *et al.* (1987, 1986); Wilson and Worcester (1945)) but have not become popular, in particular for a power of I between 0 and 1. Let us explain why. Consider the differential equation

$$I' = \sigma S^q I^p - \mu I, \qquad I(0) = 0,$$

where $q \geq 0$ and $p \in (0, 1)$. This equation has one trivial solution, namely $I \equiv 0$. We claim that there also is a solution with $I(t) > 0$ for all $t > 0$. We divide by I^p,

$$\frac{1}{1-p}\frac{d}{dt} I^{1-p} = I' I^{-p} = \sigma S^q - \mu I^{1-p}.$$

We use the variation of constants formula,

$$\frac{1}{1-p} I^{1-p}(t) = \sigma \int_0^t S^q(s) e^{-\mu(1-p)(t-s)} ds$$

which yields

$$I(t) = \left((1-p)\sigma \int_0^t S^q(s) e^{-\mu(1-p)(t-s)} ds\right)^{\frac{1}{1-p}}.$$

While the problem of multiple solutions to the same initial data does not arise if one assumes $I(0) > 0$, the existence of solutions where infective individuals spontaneously appear ex nihilo is certainly worrisome. Mathematically, it is linked to the fact that I^q is not Lipschitz continuous around 0 which results in nonuniqueness of solutions starting at 0. See the Appendix B for proof.

We cannot analyze power laws in the endemic case as generally as we could in the epidemic case. First, we only consider the case of $\theta(S) = S^q$. Second, we must consider a few ranges of p and q separately. Our system is

$$\begin{aligned} S' =& Sg(S) - \sigma S^q I^p, \\ I' =& \sigma S^q I^p - \mu I. \end{aligned} \tag{10.1.1}$$

We note that in Table 3.1 $p, q \in (0,1)$, with $p + q > 1$ are found, and so we begin with $p, q \in (0,1)$.

10.2 $0 < p, q < 1$

We can get a powerful result here with little effort. Note that the following two results work for more than just $0 < p, q < 1$.

Proposition 10.2.1. *Consider any solution to system (10.1.1) with $p \in (0,1), q > 0$, and initial data $S(0), I(0) > 0$. Then the (uniform) persistence of S implies the (uniform) persistence of I.*

Proof. Suppose that S persists, i.e. $S_\infty > 0$. From the I Equation from System (10.1.1), we can form the following equation:

$$\frac{1}{1-p}\frac{d}{dt} I^{1-p} = I' I^{-p} = \sigma S^q - \mu I^{1-p}.$$

Applying the method of fluctuations, we have a sequence t_k, with $t_k \to \infty$ as $k \to \infty$ such that $\frac{d}{dt} I^{1-p} = 0$, and $\lim_{k\to\infty} I(t_k) = I_\infty$. We have

$$0 = \sigma \lim_{k\to\infty} S^q(t_k) - \mu I_\infty^{1-p} \geq \sigma S_\infty^q - \mu I_\infty^{1-p},$$

which implies $I_\infty \geq \left(\frac{\sigma S_\infty^q}{\mu}\right)^{\frac{1}{1-p}} > 0$.

Similarly, if S persists uniformly, then there is some $\varepsilon > 0$ such that $S_\infty > \varepsilon$, which implies $I_\infty \geq \left(\frac{\sigma S_\infty^q}{\mu}\right)^{\frac{1}{1-p}} \geq \left(\frac{\sigma \varepsilon^q}{\mu}\right)^{\frac{1}{1-p}} > 0$. □

Corollary 10.2.2. *Any solution to system (10.1.1) with $p \in (0,1), q > 0$, and initial data $S(0), I(0) > 0$ cannot converge to $(K, 0)$.*

Thus, if there are any infectives initially, we cannot have eradication of the disease, if $p \in (0,1)$ and $0 < q$.

We mention that for $\frac{p+q-1}{1-p} > 0$ (as in Table 2 in Greer *et al.* (2008)), there is always a coexistence equilibrium. For a coexistence equilibrium, we have

$$I = (\sigma/\mu)^{\frac{1}{1-p}} S^{\frac{q}{1-p}}, \qquad g(S) = \sigma S^{q-1} I^p.$$

We substitute the first equation into the second

$$g(S) = \sigma^{\frac{1}{1-p}} \mu^{\frac{p}{p-1}} S^{\frac{p+q-1}{1-p}}. \tag{10.2.1}$$

If the power of S is positive, this equation has a unique solution $S > 0$ by the intermediate value theorem. We also note that S is a strictly decreasing function of σ.

The Jacobian matrix of the interior equilibrium, which we denote (S^*, I^*), is

$$\begin{pmatrix} S^* g'(S^*) + (1-q) g(S^*) & -p\mu \\ q g(S^*) & (p-1)\mu \end{pmatrix},$$

which has determinant

$$\det J(S^*, I^*) = (p-1) S^* g'(S^*) \mu + (p+q-1) g(S^*) \mu. \tag{10.2.2}$$

If $p < 1 < p+q$ (as in Table 2 in Greer *et al.* (2008)), then the determinant is positive, and we satisfy $\frac{p+q-1}{1-p} > 0$. Thus we can expect the interior equilibrium to be a stable or unstable node, but never a saddle point.

The trace T, is

$$T = S^* g'(S^*) + (p-1)\mu + (1-q) g(S^*) \tag{10.2.3}$$

which can be either positive or negative. Using Equation 10.2.1, we can determine the limiting behavior of the S^* with respect to σ, we have

$$\lim_{\sigma\to 0^+} S^* = K, \qquad \lim_{\sigma\to\infty} S^* = 0.$$

Then, using $I^* = (\sigma/\mu)^{\frac{1}{1-p}}(S^*)^{\frac{q}{1-p}}$ we have

$$\lim_{\sigma\to 0^+} I^* = 0, \qquad \lim_{\sigma\to\infty} I^* = 0.$$

With the limiting behavior of S^*, we consider the trace as a function of σ and see the following limiting behavior

$$\begin{aligned} \lim_{\sigma\to 0^+} T(\sigma) =& S^* g'(K) + (p-1)\mu < 0, \\ \lim_{\sigma\to\infty} T(\sigma) =& (p-1)\mu + (1-q)g(0). \end{aligned} \tag{10.2.4}$$

From this we see if $g(0) > \dfrac{1-p}{1-q}\mu$, there is some $\sigma \in (0,\infty)$ such that $T(\sigma) = 0$, by the intermediate value theorem. Since the determinant is always positive, we will have a locally asymptotically stable equilibrium for sufficiently small σ and an unstable equilibrium for sufficiently large σ. This motivates us to consider what conditions will ensure a Hopf bifurcation.

Theorem 10.2.3. *If $g \in C^2(0,\infty)$, $f(S,I) = S^q I^p$, with $q \in (0,1)$, $\quad p < 1 < p+q$ and $g(0) > \dfrac{1-p}{1-q}\mu$, will give a generic global Hopf bifurcation of periodic solutions.*

The proof, which is based on Sard's lemma (Deimling (1985), lemma 1.4) and Chow and Mallet-Paret (1978), is similar to the one of Theorem 12.13.1, which is the technically more demanding one. Therefore, the proof of this theorem is omitted and the proof of Theorem 12.13.1 will be presented in detail. Also, the properties of a global Hopf bifurcation in comparison to a mere Hopf bifurcation will be explained at that occasion.

Since we have an unstable equilibrium for very large σ, we wonder if we have 'practical host extinction,' where all initial conditions, besides the interior equilibrium, will lead to host extinction. Using Matlab (Mathworks 2005), we perform some numerical simulations to lend support to the idea, and came up with this figure, using $g(S) = \frac{\kappa}{b+S^{\alpha}} - \theta, \kappa = 101, b = 1, \alpha = 2, \theta = 1, \mu = 5, p = 0.263, q = 0.953$, and $\sigma = 700$. This has the p and q values found in Table 3.1, $g(0) = \frac{\kappa}{b} - \theta > \frac{1-p}{1-q}\mu$, and preserves the biologically motivated inequality $g(S)+\mu \geq 0$ for all S. Meeting all these criteria contributed to the exceedingly high σ required to make the interior equilibrium have a positive trace of 0.1442, and thus be unstable. As Figure 10.2.1 shows, there seem to be no periodic orbits. Many other parameters and initial conditions very close to the equilibrium were tested and all showed the practical extinction of all initial conditions within $(0,\infty)^2 \setminus (S^*, I^*)$.

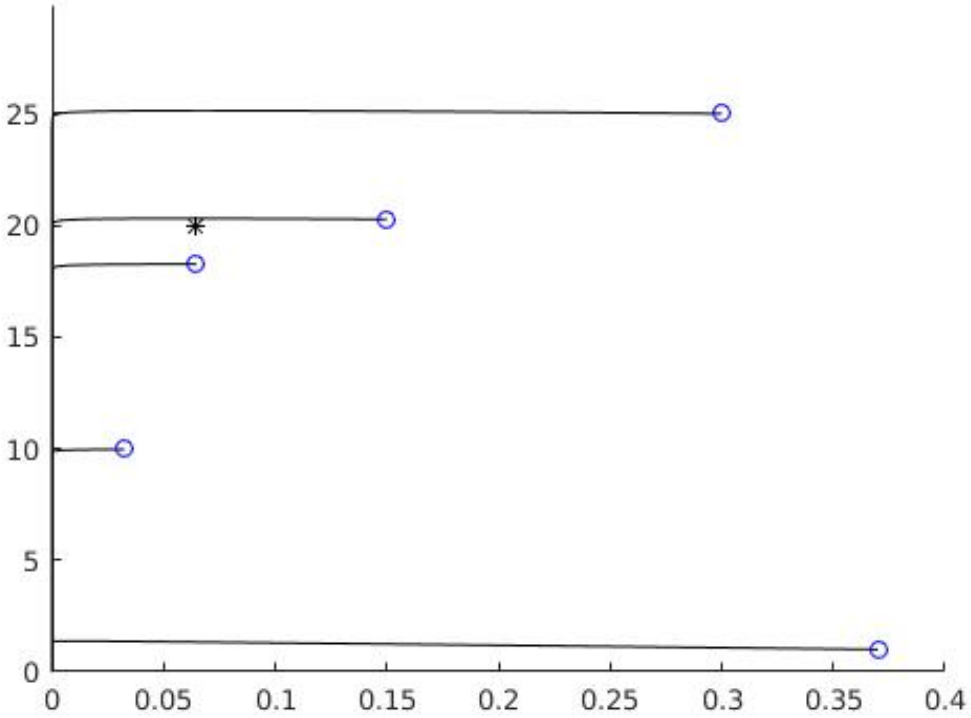

Figure 10.2.1: This is generated using $\sigma = 700$. Each blue circle represents an initial condition, and the black lines are the trajectory from said initial condition. The black asterisk is the equilibrium (S^*, r^*), which is unstable. The trace is 0.1442. There appears to be no periodic orbit.

Even if we have a stable interior equilibrium, we always obtain initial-data dependent host extinction for $0 < q < 1$ and $p \geq 0$.

Theorem 10.2.4. *If $p > 0, q \in (0,1)$ and the initial data $S_0 > 0$ and $I_0 > 0$ satisfy*

$$S_0^{1-q} I_0^{-p} < \frac{(1-q)\sigma}{(1-q)g(0) + p\mu},$$

$S(t)$ becomes zero at finite time and stays zero thereafter and $I(t) \to 0$ as $t \to \infty$.

Notice that the range of initial data which lead to host extinction increases if σ increases, or either $g(0)$ or μ decrease.

Proof. Notice that all solutions with $S(0) = S_0 \geq 0$ and $I(0) = I_0 > 0$, satisfy $S(t) \geq 0$ and $I(t) \geq 0$ for all $t \geq 0$.

Since $I' \geq -\mu I$, $I(t) \geq I_0 e^{-\mu t}$. As long as S is positive,

$$S' S^{-q} = S^{1-q} g(S) - \sigma I^p \leq S^{1-q} g(S) - \sigma I_0^p e^{-p\mu t}.$$

Notice that the left hand side is the derivative of $\frac{1}{1-q} S^{1-q}$,

$$(d/dt) S^{1-q}(t) \leq (1-q) S^{1-q}(t) g(0) - (1-q)\sigma I_0^p e^{-p\mu t}.$$

We multiply by an integrating factor,

$$(d/dt)\Big(S^{1-q}(t) e^{-(1-q)g(0)t} \Big) \leq -(1-q)\sigma I_0^p e^{-p\mu t} e^{-(1-q)g(0)t}.$$

We integrate the differential inequality,

$$e^{-(1-q)g(0)t} S^{1-q}(t) \leq S_0^{1-q} - \frac{(1-q)\sigma I_0^p}{(1-q)g(0) + p\mu}(1 - e^{-[(1-q)g(0)+p\mu]t}).$$

This implies that $S(t)$ becomes zero at finite time if

$$S_0^{1-q} I_0^{-p} < \frac{(1-q)\sigma}{(1-q)g(0) + p\mu}.$$

Once $S(t_0) = 0$ for some $t_0 > 0$, then $S(t) = 0$ for all $t \geq t_0$ is *a* solution. However, using Theorem 9.1.4, we know that this extension is in fact the unique solution. Applying Corollary 9.1.2 implies that $I(t) \to 0$ as $t \to \infty$. □

10.3 $0 < p < 1 \leq q$

We recall that both Proposition 10.2.1 and Corollary 10.2.2 still apply in this range of p and q.

We revisit Equation (10.2.3) with $q = 1$ and see

$$T = S^* g'(S^*) + (p-1)\mu + (1-q) g(S^*) = S^* g'(S^*) + (p-1)\mu < 0,$$

since g is strictly decreasing and $q = 1$. Thus the interior equilibrium is always locally asymptotically stable. This finding motivates the following Theorem.

Theorem 10.3.1. *For all σ, system (10.1.1) with $0 < p < 1 \leq q$ has an interior equilibrium which is globally asymptotically stable for solutions in $(0,\infty)^2$.*

Proof. Suppose, toward contradiction, that $S^\infty < \gamma$ for all $0 < \gamma$. Let $\varepsilon = K/2$ and choose $\delta > 0$ such that $g(\varepsilon) - \sigma\varepsilon^{q-1}\delta^p > 0$. Then, if necessary, shrink ε until $\sigma\varepsilon^q < \mu\delta^{1-p}$. Note that $g(\varepsilon) - \sigma\varepsilon^{q-1}\delta^p > 0$ still holds for a smaller ε. Perform a time shift such that $S(t) < \varepsilon$ for all $t \geq 0$. Then

$$\frac{I'}{I^p} = \sigma S^q - \mu I^{1-p} < \sigma\varepsilon^q - \mu I^{1-p} < \mu\delta^{1-p} - \mu I^{1-p},$$

which is negative so long as $\delta < I$, which implies $I^\infty \leq \delta$.

Perform another time shift until $I(t) \leq \delta$ for all $t \geq 0$. Then we have

$$\frac{S'}{S} = g(S) - \sigma S^{q-1} I^p > g(\varepsilon) - \sigma\varepsilon^{q-1}\delta^p > 0,$$

implying the exponential growth of S to infinity, a contradiction to $S^\infty < \gamma$ for all $\gamma > 0$. Thus $S^\infty > \gamma > 0$ for some $\gamma > 0$. We apply Theorem 9.1.5 in order we can upgrade our uniform weak persistence to uniform persistence, i.e. $S_\infty > \varepsilon > 0$ for some $\varepsilon > 0$. Proposition 10.2.1 then implies the uniform persistence of I.

Next, we show that there are no periodic orbits, by using Dulac's criterion, with $\rho = \dfrac{1}{SI^p}$. Then, if we convert our system of differential equations into a vector field with $f_1(S,I) = S'$ and $f_2(S,I) = I'$, then

$$\nabla(\rho f) = \frac{g'(S)}{I^p} - (q-1)\sigma S^{q-2} - \frac{\mu(1-p)}{SI^p} < 0.$$

Thus there are no periodic orbits in $(0,\infty)^2$. We have bounded solutions by Theorem 9.1.1. Combined with uniform persistence of both S and I, there is some $\varepsilon > 0$ such that all solutions will enter $[\varepsilon, H^\infty]^2$, and never leave. Thus, since the interior equilibrium is the only equilibrium in the set, all solutions in $(0,\infty)^2$ must converge to it by Poincaré-Bendixson. □

This is a very interesting result, as it radically changes the behavior between the epidemic case and the endemic case. Recall that in the epidemic case, the host was driven to extinction regardless of initial conditions or parameters.

Chapter 11

UPPER DENSITY-DEPENDENT INCIDENCES

An incidence function $f : \mathbb{R}_+^2 \to \mathbb{R}_+$ is called *upper density-dependent* if, for any $N > 0$, there exists some $c_N > 0$ such that

$$f(S, I) \leq c_N SI, \qquad 0 \leq S, I \leq N. \tag{6.1.1 revisited}$$

This inequality holds for the incidence functions $f(S, I) = Sk\ln(1 + (I/k))$, $f(S, I) = \frac{SI}{1+I^{1-p}}$, $f(S, I) = \frac{SI}{1+(1/k)(S+I)}$ and many more and also for $f(S, I) = SI^2$.

Theorem 11.1.1. *Suppose that $f(S, I)$ is upper density-dependent. Then for $S(0) > 0$, and $I(0) \geq 0$, the host persists uniformly, i.e. there is some $\varepsilon > 0$ such that $S_\infty > \varepsilon$.*

Proof. Choose $N = 2H^\infty$, with H^∞ as defined in Theorem 9.1.1. Theorem 9.1.1 also tells us that eventually all trajectories with $S(0), I(0) \geq 0$ will enter $[0, N]^2$ at some point, thus we perform a time shift (if needed) such that $0 \leq S(t), I(t) \leq N$ for all $t \geq 0$. Since f is upper density-dependent, we obtain our c_N, as per Equation 6.1.1. Suppose toward contradiction that $S^\infty < \dfrac{\mu}{4\sigma c_N}$. Perform a time shift such that $\sigma c_N S(t) < \dfrac{\mu}{2}$ for all $t \geq 0$. Considering the infective differential equation, we have

$$I' = \sigma f(S, I) - I\mu \leq \sigma c_N SI - I\mu < I\Big(\frac{\mu}{2} - \mu\Big) = -\frac{I\mu}{2},$$

which implies $I(t) \leq I(0)e^{-t(\mu/2)}$, so $I(t) \to 0$ as $t \to \infty$. Choose $\varepsilon > 0$ such that $g\Big(\dfrac{\mu}{2\sigma c_N}\Big) > \sigma c_N \varepsilon$, and perform another time shift until $I(t) < \varepsilon$ for all $t \geq 0$. Then the S' equation becomes

$$S' = Sg(S) - \sigma f(S, I) \geq Sg(S) - \sigma c_N SI > S\Big(g\Big(\frac{\mu}{2\sigma c_N}\Big) - \sigma c_N \varepsilon\Big),$$

which implies the exponential growth of S, a contradiction. Thus we have $S^\infty > \frac{\mu}{4\sigma c_N}$.

We wish to invoke Theorem 4.2 from Smith and Thieme (2011), so we check the assumptions, using the persistence function $\rho(S, I) = S$. Recall that we have unique solutions, which gives $S(t) = 0$ if and only if $S(t) \equiv 0$. Then we see

$$\rho(S, I) = 0 \iff S = 0 \iff S \equiv 0 \iff \rho(S, I) \equiv 0,$$

fulfilling a requirement of the theorem. For another requirement, we choose $B = B_k = [0, H^\infty]^2$. Finally, in the language of Smith and Thieme (2011), we have this system is uniformly weakly ρ persistent. All this together implies uniform persistence, which is equivalent to $S_\infty > \varepsilon$ for some $\varepsilon > 0$. □

Chapter 12

HOMOGENEOUS INCIDENCE

12.1 Constant Risk Incidence

The constant risk is linear and lacks the property $f(S,0)=0$. We use non-linearity and $f(S,0)=0$ in our analysis of general homogeneous functions, which is why we consider the constant risk by itself. With the constant risk incidence, the equation for the susceptibles in (8.1.1) decouples,

$$S' = S(g(S) - \sigma). \tag{12.1.1}$$

We obtain global host extinction if the infection coefficient σ is sufficiently large.

Theorem 12.1.1. *If $g(0) \leq \sigma$, $S(t) \to 0$ and $I(t) \to 0$ as $t \to \infty$.*

Proof. We can assume that $S(0) > 0$, otherwise $S(t) = 0$ for all $t \geq 0$. Since $g(0) \leq \sigma$, $g(S) - \sigma < 0$ for all $S > 0$ and $S(t)$ is strictly decreasing and converges towards a limit $S_\infty \geq 0$. If $S_\infty > 0$, the derivative S' is negative and bounded away from zero and S decreases to $-\infty$, a contradiction. Further

$$I' = \sigma S(t) - \mu I.$$

Since $S(t) \to 0$, also $I(t) \to 0$. □

This result is sharp because the strict decrease of g implies the following result.

Theorem 12.1.2. *Let $g(0) > \sigma > 0$ and S^* be the unique density with $g(S^*) = \sigma$. Then $S(t) \to S^*$ and $I(t) \to \sigma S^*/\mu$.*

In particular, survival or extinction of the host do not depend on the initial condition.

12.2 A Large Class of Homogeneous Incidence Functions

One important class of homogeneous incidence functions is

$$f(S,I) = (qS^\gamma + pI^\gamma)^{1/\gamma}, \quad S,I > 0, \tag{12.2.1}$$

where $\gamma < 0$ and $p, q > 0$, $p + q = 1$ (Hadeler *et al.* (1988); Hadeler (1989)). Set $\beta = -\gamma$. Then $\beta > 0$ and

$$f(S,I) = \frac{SI}{(qI^\beta + pS^\beta)^{1/\beta}}. \tag{12.2.2}$$

The numbers p and q are related to the contact activity of susceptible and infectives. If $p > q$, susceptibles are more active than infectives. Notice that the denominator causes some saturation in the incidence if S and I are large.

Notice that, for this form of f, $f(s,t) = 0$ if either s or $t = 0$, and

$$\min\{S,I\} \le f(S,I) \le \max\{p^{1/\gamma}, q^{1/\gamma}\} \min\{S,I\}, \qquad S,I \ge 0. \tag{12.2.3}$$

We conclude that $f(S,I) \to 0$ as $S \to 0$ or $I \to 0$ and set

$$f(S,I) = 0, \quad S,I \ge 0, SI = 0. \tag{12.2.4}$$

With this definition, f becomes continuous on $\mathbb{R}^2_+$. One readily checks that f is homogeneous and that f is a concave function of each separate variables.

For all $\gamma < 0$, we recover the constant risk incidence $f(S,I) = S$ by letting $q \to 0$ and $p \to 1$.

Another class is

$$f(S,I) = S^q I^p, \qquad S,I \ge 0, \tag{12.2.5}$$

with $p, q > 0$ and $p + q = 1$. This f is the limit of (12.2.1) as $\gamma \to 0$ (Hadeler *et al.* (1988)). The homogeneous function

$$f(S, I) = \min\{S, I\} \tag{12.2.6}$$

is the limit of (12.2.1) as $\gamma \to -\infty$ by (12.2.3).

Finally, if we set $\beta = 1$, we recover the asymmetric frequency incidence

$$f(S, I) = \frac{SI}{pS + qI}, \tag{12.2.7}$$

and in a further special case of $p = q = 1/2$ we recover (symmetric) frequency dependence

$$f(S, I) = 2\frac{SI}{S + I}. \tag{12.2.8}$$

The incidence function in Kuang and Beretta (1998) is transformed into normalized asymmetric frequency dependence with $p = \dfrac{1}{m+1}$ and $q = \dfrac{m}{m+1}$.

Notice new homogeneous functions can be obtained from known ones by setting

$$\tilde{f}(S, I) = \beta f(\alpha S, \tilde{\alpha} I), \qquad S, I \geq 0 \tag{12.2.9}$$

where $\alpha, \tilde{\alpha}, \beta \geq 0$. Since f is homogeneous, we can restrict this to $0 < \alpha, \tilde{\alpha} < 1$. For $\gamma \in (\infty, 0)$ and for the homogeneous power law, this leads to nothing new because the new parameters can be absorbed into the incidence coefficient σ after possibly changing p and q. In the limiting case $\gamma \to -\infty$, even after absorption, we get

$$f(S, I) = \min\{S, \alpha I\}. \tag{12.2.10}$$

12.3 The Ratio Formulation of the Model

Homogeneous incidence functions cannot be differentiated at the origin (unless they are linear) such that a standard stability analysis at the equilibrium (0,0) is not possible. We assume $f(S, I)$ is continuous, is an increasing and concave function of both $S \geq 0$ and $I \geq 0$, $f(0, I) = 0$ for all $I \geq 0$, and $f(S, 0) = 0$ for all $S \geq 0$.

Therefore, we introduce the ratio of infectives to susceptibles, $r = I/S$. In Kuang and Beretta (1998), they use x for susceptibles, y for infectives, and $u = x/y$ as the ratio term. Note that although a ratio term is employed here, it is the inverse of our considerations, i.e. $u \approx r^{-1}$. With the hazard function $h(r) = f(1, r)$, the model takes the form

$$\begin{aligned} S' =& S(g(S) - \sigma h(r)), \\ r' =& \sigma h(r)(1 + r) - r(g(S) + \mu). \end{aligned} \tag{12.3.1}$$

We rephrase,

$$\begin{aligned} S' =& S(g(S) - \sigma h(r)), \\ r' =& r[\sigma \xi(r) - (g(S) + \mu)], \end{aligned} \tag{12.3.2}$$

with the *per unit ratio growth rate*

$$\xi(r) = \frac{(1 + r)h(r)}{r}, \quad r > 0. \tag{5.1.4 revisited}$$

Since f is homogeneous,

$$\xi(r) = f(1 + r^{-1}, 1 + r), \quad r > 0. \tag{5.1.5 revisited}$$

In some of the analysis performed in Kuang and Beretta (1998) they employ a function $U(u)$, which appears to be an analog of our $h(r)$, and has the form

$$U(u) = \frac{u}{m + u}.$$

Remark 12.3.1. If $h'(0) < \infty$, then h is locally Lipschitz continuous on $[0, \infty)$. Using the standard local Lipschitz argument, we can improve the result of Theorem 9.1.4 to include solutions with $S(0), I(0) \geq 0$. Therefore, we have uniqueness of solutions with $S(0), I(0) \in \mathbb{R}^2$ so long as $h'(0)$ is finite.

12.3.1 Classes of Hazard Functions

For our general homogeneous incidence class we have that

$$h(r) = (q + pr^{\gamma})^{1/\gamma}. \tag{12.3.3}$$

Recall that $\gamma < 0$. For the homogeneous power law,

$$h(r) = r^p \tag{12.3.4}$$

and for the generalized minimum incidence function,

$$h(r) = \min\{1, \alpha r\}. \tag{12.3.5}$$

Creating the hazard function which would be born from the incidence function used in Kuang and Beretta (1998), we see

$$h(r) = \frac{r}{\frac{1}{1+m} + \frac{mr}{m+1}} = \frac{(1+m)r}{1+mr}.$$

We notice that h is increasing (with exception of the minimum function, even strictly). With exception of the homogeneous power incidence,

$$h(\infty) = \lim_{r \to \infty} h(r) < \infty. \tag{12.3.6}$$

Further h is concave and $h(0) = 0$. This implies that $h(r)/r$ is decreasing. If h is strictly concave, then $h(r)/r$ is strictly decreasing as well. Since f is homogeneous,

$$\frac{h(r)}{r} = f(1/r, 1), \qquad \frac{h(r)}{r} \overset{r \to \infty}{\longrightarrow} f(0,1) = 0, \qquad h'(0) = f(\infty, 1), \tag{12.3.7}$$

with the values in the last equality possibly being infinite. With exception of the minimum function, h is differentiable on $(0,\infty)$ and h' strictly decreasing.

For all but the homogeneous power law, h is also differentiable at 0. For the homogeneous power law, $h(r)/r \to \infty$ as $r \to 0$.

For the general homogeneous incidence, equation (5.1.4) has the form

$$\xi(r) = (p + qr^{\gamma})^{1/\gamma}\frac{1+r}{r}, \qquad \gamma < 0.$$

Notice that

$$\xi(0) = q^{1/\gamma}, \qquad \xi(\infty) = p^{1/\gamma}.$$

To find out the monotonicity properties of ξ, we consider

$$\xi(r)^{\gamma} = (pr^{-\gamma} + q)(1+r)^{\gamma}.$$

We take the derivative

$$(d/dr)\xi(r)^{\gamma} = -p\gamma r^{-\gamma-1}(1+r)^{\gamma} + (pr^{-\gamma}+q)\gamma(1+r)^{\gamma-1}.$$

So

$$\gamma\xi(r)^{\gamma-1}\xi'(r) = \gamma(1+r)^{\gamma-1}(pr^{-\gamma} + q - pr^{-\gamma-1}(1+r)).$$

We simplify

$$\xi(r)^{\gamma-1}\xi'(r) = (1+r)^{\gamma-1}(q - pr^{-\gamma-1}).$$

Proposition 12.3.2. *The monotonicity behavior of ξ depends on $\gamma < 0$ in the following way:*

$-1 < \gamma$: ξ is first strictly decreasing and then strictly increasing, the minimum is taken at $r_{\text{ex}} = (p/q)^{1+\gamma}$, and $\xi' < 0$ on $(0, r_{\text{ex}})$ and $\xi' > 0$ on (r_{ex}, ∞).

$\gamma < -1$: ξ is first strictly increasing and then strictly decreasing, the maximum is taken again at $r_{\text{ex}} = (p/q)^{1+\gamma}$ and $\xi' > 0$ on $(0, r_{\text{ex}})$ and $\xi' < 0$ on (r_{ex}, ∞).

$\gamma = -1$ and $p > q$: ξ is strictly decreasing and $\xi' < 0$ on $(0, \infty)$.

$\gamma = -1$ and $p < q$: ξ is strictly increasing and $\xi' > 0$ on $(0, \infty)$.

$\gamma = -1$ and $p = q$ (frequency-dependent incidence): ξ is constant.

We recall that our results will hold for the model considered in Kuang and Beretta (1998) (which is a predator-prey model), and we would be in the case of $\gamma = -1$, $p = \dfrac{1}{m+1}$, and $q = \dfrac{m}{m+1}$. Thus the relation of p to q is the same as the relationship of 1 to m.

We make it an overall assumption that h is concave, increasing and continuous on $\mathbb{R}_+$ and $h(0) = 0$. These assumptions imply that h is Lipschitz continuous on every interval (a, b) with $0 < a < b < \infty$.

12.3.2 Backward Construction of Homogeneous Incidence Function

The function (12.3.5) is known as Blackman functional response in chemostat theory. For $\gamma = -1$, (12.3.3) provides a Michaelis-Menten type functional response. Other functional responses are the Ivlev-functional response (Ivlev (1955))

$$h(r) = 1 - e^{-\alpha r}, \qquad \alpha > 0 \tag{12.3.8}$$

or the logarithmic functional response,

$$h(r) = \ln(1 + \alpha r), \qquad \alpha > 0. \tag{12.3.9}$$

In general, let $h : \mathbb{R}_+ \to \mathbb{R}_+$ be increasing and concave, $h(0) = 0$ and set

$$f(S, I) = Sh(I/S), \qquad S > 0. \tag{12.3.10}$$

Then f has the properties of a homogeneous incidence function. Notice that, if h and $\tilde{h}$ have the above-mentioned properties, so have $h + \tilde{h}$ and $h \circ \tilde{h}$. This allows to construct a zoo of homogeneous incidence functions.

From a modeling point of view, if $\tilde{h}$ is the functional response of choice, it may make more sense to define

$$f(S,I) = S\tilde{h}\Big(\frac{I}{pS+qI}\Big). \tag{12.3.11}$$

The term $\dfrac{I}{pS+qI}$ can be viewed as the probability that a susceptible individual comes into contact with an infected individual, with the denominator weighted by contact rates. Then the associated function $h(r) = f(1,r)$ is

$$h(r) = \tilde{h}\Big(\frac{r}{p+qr}\Big). \tag{12.3.12}$$

If $p \le q$, the associated function ξ is decreasing. More generally, we have the following connection.

Lemma 12.3.3. *Let h_1 and h_2 be increasing and concave, $h_j(0) = 0$ and $h_j(r) > 0$ for $r > 0$, and $h = h_1 \circ h_2$. Then h has the same properties.*

Assume that ξ_2 defined by

$$\xi_2(r) = h_2(r)\frac{1+r}{r}$$

is decreasing. Then ξ defined by $\xi(r) = h(r)^{\frac{1+r}{r}}$ is also decreasing.

Further, if the h_j are differentiable on $(0,\infty)$ and h_1' is decreasing and $\xi_2' \le 0$, then $\xi' \le 0$.

If, in addition, h_1' is strictly decreasing or ξ_2' is strictly negative, then ξ' is strictly negative.

Proof. Recall that h is concave if $h(\beta r + (1-\beta)s) \ge \beta h(r) + (1-\beta)h(s)$ for all $\beta \in (0,1)$ and all $r, s \ge 0$. The concavity of h then readily follows from the concavity of h_1 and h_2 and the increase of h_1. Notice that, for $r > 0$,

$$\xi(r) = \frac{h_1(h_2(r))}{h_2(r)}\xi_2(r).$$

Since h_1 is concave, $\phi(s) = h_1(s)/s$ is a decreasing function of $s > 0$ and the decrease of ξ follows. If the h_j are differentiable, ϕ' inherits the (strict) negativity from the (strict) decrease of h_1' by the mean-value value. Further $\xi(r) = \phi(h_2(r))\xi_2(r)$ and

$$\xi' = (\phi' \circ h_2)\xi_2 + (\phi \circ h_2)\xi_2'.$$

This implies the assertions. □

A decreasing ξ provides very nice dynamics, which are summed up in Section 12.12. Using Lemma 12.3.3, we can produce $f(S, I) = S \ln\left(1 + \frac{I}{S+I}\right)$, which is the homogeneous incidence with the best fit in Table 3.1 and second best in Table 3.4.

Corollary 12.3.4. *The incidence function* $f(S, I) = S \ln\left(1 + \frac{I}{S+I}\right)$ *produces a strictly decreasing* ξ*.*

Proof. We will use Lemma (12.3.3) with $h_1(r) = \ln(1 + r)$ and $h_2(r) = \frac{r}{1+r}$, which gives $\xi_2 = 1$. In addition to the basic properties required for Lemma (12.3.3), h_j are differentiable on $(0, \infty)$, h_1' is strictly decreasing and $\xi_2' \leq 0$, so ξ' is strictly negative.

Our h_1 and h_2 together form $h(r) = \ln\left(1 + \frac{r}{1+r}\right)$. Equation 12.3.10 gives $f(S, I) = Sh(I/S) = S \ln\left(1 + \frac{I/S}{1+I/S}\right) = S \ln\left(1 + \frac{I}{S+I}\right)$. □

Lemma 12.3.5 ((Thieme, 2017, L.3.8))**.** *If* h *is concave, then* $f(S, I) = Sh(I/S)$ *is homogeneous and concave in* (S, I) *for* $S > 0$*,* $I \geq 0$*. If* f *is continuous on* $\mathbb{R}^2_+$*, it is concave there.*

12.4 Equilibria

Equilibria are time-independent solutions. There are two obvious equilibria, $(0, 0)$ and $(K, 0)$, where the host carrying capacity $K > 0$ is uniquely determined by $g(K) =$

0. The Jacobian matrix of our vector field at (0,0) is

$$\begin{pmatrix} g(0) & 0 \\ 0 & \sigma h'(0) - g(0) - \mu \end{pmatrix}.$$

Theorem 12.4.1. *If $\sigma h'(0) < g(0) + \mu$, then (0,0) is a saddle with the $r = 0$ axis being the unstable manifold and the $S = 0$ axis being the unstable manifold.*

If $\sigma h'(0) > g(0) + \mu$, $(0, 0)$ is an unstable node.

The Jacobian matrix at $(K, 0)$ is given by

$$\begin{pmatrix} Kg'(K) & -\sigma K h'(0) \\ 0 & \sigma\xi(0) - \mu \end{pmatrix}.$$

Theorem 12.4.2. *If $\sigma\xi(0) = \sigma h'(0) > \mu$, then (K,0) is a saddle with the $S = 0$ axis being the stable manifold.*

If $\sigma h'(0) < \mu$, $(K, 0)$ is a locally asymptotically stable node.

By (12.3.7), the stability condition is equivalent to $f(\infty, 1) < \frac{\mu}{\sigma}$. See Theorem 8.1.1.

12.4.1 Host-Parasite Coexistence Equilibrium

An interior equilibrium (S^*, r^*) with $S^* > 0$ and $r^* > 0$ satisfies the equations

$$g(S^*) = \sigma h(r^*), \qquad \sigma h(r^*)(1 + r^*) = r^*(g(S^*) + \mu),$$

which simplify to

$$g(S^*) = \sigma h(r^*), \qquad \sigma h(r^*) = r^*\mu. \tag{12.4.1}$$

Since $h(r)/r$ is a decreasing function of $r > 0$, a necessary and sufficient condition for solving the second equation is $\sigma h'(0) > \mu$. $S^* > 0$ can then be found if $g(0) > \sigma h(r^*)$ for a solution $r^* > 0$ for the second equation. Recall that $\sigma h'(0) - \mu$ is the second eigenvalue of the Jacobian matrix at the boundary equilibrium $(K, 0)$.

Except for the minimum incidence function, the decrease of $h(r)/r$ in $r > 0$ is strict and the coexistence equilibrium is unique.

The following has been rewritten.

Theorem 12.4.3. *Let h be increasing and $h(r)/r$ be a (strict) decreasing function of $r > 0$.*

(a) There is no interior equilibrium if $\sigma h'(0) \leq \mu$ or if $g(0) \leq \sigma h(r)$ for any solution $r > 0$ of $\sigma h(r) = \mu r$.

(b) There is an interior equilibrium (which is unique) if $\sigma h'(0) > \mu$ and if $g(0) > \sigma h(r^*)$ for a solution $r^* > 0$ of $\sigma h(r^*) = \mu r^*$. The equilibrium is given by (S^*, r^*) with $g(S^*) = \sigma h(r^*)$.

(c) If a coexistence equilibrium exists, the boundary equilibrium $(K, 0)$ is a saddle.

Corollary 12.4.4. *Assume that h is increasing and $h(r)/r$ a decreasing function of $r > 0$.*

Then a coexistence equilibrium (S^*, r^*) exists (with $r^* \in (0, g(0)/\mu)$) if

$$\frac{\mu}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)}.$$

The coexistence equilibrium is unique if $h(r)/r$ is a strictly decreasing function of $r > 0$.

Proof. We rewrite the condition as

$$\frac{1}{h'(0)} < \sigma/\mu < \frac{g(0)/\mu}{h(g(0)/\mu)}.$$

By the intermediate value theorem, there exists some $r \in (0, h(g(0)/\mu))$ with $\sigma/\mu = r^*/h(r^*)$. Since h is increasing,

$$\sigma h(r^*) \leq \sigma h(g(0)/\mu) < g(0).$$

Now apply Theorem 12.4.3 (b). □

Corollary 12.4.5. *Assume that h is increasing and $h(r)/r$ a decreasing function of $r > 0$.*

Then there is no coexistence equilibrium (S^*, r^*) if $\sigma < \frac{\mu}{h'(0)}$ or $\sigma > \frac{g(0)}{h(g(0)/\mu)}$.

Proof. The first condition has already been dealt with in Theorem 12.4.3 (a). Let $\sigma > \frac{g(0)}{h(g(0)/\mu)}$. To apply Theorem 12.4.3 (a) again, let $r > 0$ be a solution of $\sigma h(r) = \mu r$. Then

$$r/h(r) = \sigma/\mu > \frac{g(0)/\mu}{h(g(0)/\mu)}.$$

Since $h(r)/r$ is a decreasing function of r, $r > \dfrac{g(0)}{\mu}$. Since h is increasing,

$$\sigma h(r) \geq \sigma h(g(0)/\mu) \geq g(0). \qquad \square$$

A similar proof shows the following.

Corollary 12.4.6. *Assume that h is increasing and $h(r)/r$ a decreasing function of $r > 0$ with the decrease being strict on $(0, g(0)/\mu]$.*

Then there is no coexistence equilibrium (S^*, r^*) if $\sigma \leq \frac{\mu}{h'(0)}$ or $\sigma \geq \frac{g(0)}{h(g(0)/\mu)}$.

12.4.1.1 Stability of the Coexistence Equilibrium

Since $\sigma\xi(r^*) = g(S^*) + \mu$ as well at a coexistence equilibrium, we obtain the following Jacobian matrix from System 9.1.7,

$$J(S^*, r^*) = \begin{pmatrix} S^* g'(S^*) & -\sigma S^* h'(r^*) \\ -r^* g'(S^*) & \sigma r^* \xi'(r^*) \end{pmatrix}.$$

The determinant is

$$\det J(S^*, r^*) = \sigma S^* r^* g'(S^*)[\xi'(r^*) - h'(r^*)] = \sigma S^* g'(S^*)\Big[h'(r^*) - \frac{h(r^*)}{r^*}\Big].$$

Here we have used (5.1.4). By the mean value theorem, $h(r^*)/r^* = h'(r)$ for some $r \in (0, r^*)$. Since h' is decreasing and $g'(S^*) < 0$, $\det J(S^*, r^*) \geq 0$. If h' is strictly decreasing on $(0, r^*]$ or $h'(r^*) = 0$, $\det J(S^*, r^*) > 0$.

Proposition 12.4.7. *The coexistence (or interior) equilibrium is not a saddle point. If h' is strictly decreasing on $(0, r^*]$ or $h'(r^*) = 0$, the eigenvalues of the Jacobian matrix are either complex conjugates or real and different from 0 with equal sign. If $\xi'(r^*) < 0$ in addition, then the coexistence equilibrium is locally asymptotically stable.*

Since the left hand side of $\frac{h(r^*)}{r^*} = \frac{\mu}{\sigma}$ is a decreasing function of r^*, we have that r^* is a decreasing function of μ and an increasing function of σ.

We also have that $g(S^*) = \mu r^*$; since g is decreasing and r^* is an increasing function of σ, S^* is a decreasing function of σ. Finally, since $g(S^*) = \sigma h(r^*)$ and g is decreasing and h is increasing and r^* an decreasing function of μ, S^* is a increasing function of μ.

12.4.1.2 Bifurcation Behavior of the Interior Equilibrium

Theorem 12.4.4 gives us $\sigma \in \left(\dfrac{\mu}{h'(0)}, \dfrac{g(0)}{h(g(0)/\mu)} \right)$ as necessary and sufficient condition for the existence of the interior equilibrium. Considering the limiting behavior we have

$$\lim_{\sigma \to \frac{\mu}{h'(0)}^+} S^* = K, \qquad \lim_{\sigma \to \frac{\mu}{h'(0)}^+} r^* = 0,$$

$$\lim_{\sigma \to \frac{g(0)}{h(g(0)/\mu)}^-} S^* = 0, \qquad \lim_{\sigma \to \frac{g(0)}{h(g(0)/\mu)}^-} r^* = \frac{g(0)}{\mu}.$$

The last equality follows from the fact that the limiting value for r^* satisfies $\sigma h(r^*) = \mu r^*$ for $\sigma = \frac{g(0)}{h(g(0)/\mu)}$. Thus as σ increases into this interval, the interior equilibrium bifurcates from the boundary equilibrium $(K, 0)$ which switches from a stable node to a saddle. As σ increases through the interval r^* increases and S^* decreases, until σ increases out of the interval and the interior equilibrium collides with a no-host boundary equilibrium, $(0, g(0)/\mu) = (0, r^\circ)$, at $\sigma = \frac{g(0)}{h(g(0)/\mu)}$. Since r^* is an increasing function of σ, we see that $r^* \leq \dfrac{g(0)}{\mu}$ so long as r^* exists. This inequality becomes strict if r^* is a strictly increasing function of σ.

The trace of the Jacobian matrix at the coexistence equilibrium is

$$T = S^* g'(S^*) + \sigma r^* \xi'(r^*). \tag{12.4.2}$$

Hence

$$\lim_{\sigma \to \frac{\mu}{h'(0)}+} T = Kg'(K) < 0, \qquad \lim_{\sigma \to \frac{g(0)}{h(g(0)/\mu)}} T = \sigma \frac{g(0)}{\mu} \xi'\Big(\frac{g(0)}{\mu}\Big). \tag{12.4.3}$$

If $\xi'\Big(\frac{g(0)}{\mu}\Big) > 0$, the trace T switches its sign and there is some σ where the Jacobian matrix has purely imaginary eigenvalues.

This motivates us to consider under what conditions we have a Hopf bifurcation. With a general incidence function there is too much difficulty, however we can make significant progress if we consider asymmetric frequency dependent incidence, with $p > q$, and consider the trace as a function of S^*.

Theorem 12.4.8. *If g is analytic, asymmetric frequency dependent incidence with $p > q$ will give us generic global Hopf bifurcation of periodic solutions.*

The theorem is proven in Section 12.13.2.

12.4.2 No-Host Boundary Equilibria

A boundary equilibrium $(0, r^\circ)$ with $r^\circ \in (0, \infty)$ satisfies the equation

$$\sigma \xi(r^\circ) = g(0) + \mu. \tag{12.4.4}$$

For the class of incidence function in Section 12.2, ξ is either constant, strictly monotone, or uni-modal (see the end of Section 12.3). This implies that there are at most two solutions of (12.4.4), except for the standard incidence in the exceptional case that ξ happens to be the constant function with value $(g(0) + \mu)/\sigma$ and all $r^\circ \in (0, \infty)$ are

solutions. However, we have not been able to derive this from the general assumptions for f or h.

A necessary and sufficient condition for existence of $(0,r^\circ)$ is

$$\frac{g(0)+\mu}{\sigma} \in \xi((0,\infty)). \tag{12.4.5}$$

The Jacobian matrix at a no-host boundary equilibrium $(0, r^\circ)$ is

$$\begin{pmatrix} g(0)-\sigma h(r^\circ) & 0 \\ \square & r^\circ \sigma \xi'(r^\circ) \end{pmatrix}.$$

We only record the following observation.

Proposition 12.4.9. *At a boundary equilibrium $(0, r^\circ)$, $0 < r^\circ < \infty$, the eigenvalues of the Jacobian matrix are both real. There is no zero eigenvalue if $g(0) \neq \sigma h(r)$ and $\xi'(r) \neq 0$ for all solutions $r \in (0,\infty)$ of $\sigma\xi(r) = g(0) + \mu$.*

If (S^*, r^*) is an interior equilibrium, r^* satisfies $\sigma\xi(r^*) = g(S^*) + \mu$. Since g is strictly decreasing, we have the following result.

Proposition 12.4.10. *Assume that the interior equilibrium (S^*, r^*) exists and that $(0, r^\circ)$ is an equilibrium with $r^\circ \in (0,\infty)$. Then $\xi(r^*) < \xi(r^\circ)$.*

Corollary 12.4.11. *Assume that the interior equilibrium (S^*, r^*) exists and that $(0, r^\circ)$ is an equilibrium with $r^\circ \in (0,\infty)$ and ξ is decreasing on (r^*,∞) or on $(0, r^\circ)$. Then $r^* > r^\circ$ and $g(0) - \sigma h(r^\circ) > 0$ for this eigenvalue of the Jacobian matrix at $(0, r^\circ)$. If $\xi'(r) < 0$ for all $r \in (0,\infty)$, $(0, r^\circ)$ is a saddle.*

Proof. If $r^\circ \geq r^*$, then $\xi(r^\circ) \leq \xi(r^*)$, a contradiction to Proposition 12.4.10. Since h is increasing and g strictly decreasing,

$$g(0) - \sigma h(r^\circ) > g(S^*) - \sigma h(r^*) = 0. \qquad \square$$

Proposition 12.4.12. *Assume that there is some $r_{\min} > 0$ such that ξ decreasing on $(0, r_{\min})$ and increasing on $(r_{\min}, \infty)$ and that there are two equilibria $(0, r_1)$ and $(0, r_2)$ with $0 < r_1 < r_2 < \infty$ and a coexistence equilibrium (S^*, r^*). Then $r_1 < r^* < r_2$. Further, $g(0) - \sigma h(r_1) > 0$ for this eigenvalue of the Jacobian matrix at $(0, r_1)$. If $\xi'(r) < 0$ for all $r \in (0, r_{\min})$, then $(0, r_1)$ is a saddle.*

Proof. By the monotonicity properties of ξ, $r_1 < r_{\min} < r_2$. If $r^* \geq r_2$, then $\xi(r^*) \geq \xi(r_2)$ contradicting Proposition 12.4.10. If $r^* \leq r_1$, then $\xi(r^*) \geq \xi(r_1)$ giving the same contradiction. Since h is increasing and g strictly decreasing,

$$g(0) - \sigma h(r_1) > g(S^*) - \sigma h(r^*) = 0. \qquad \square$$

12.5 Proportional Disease Persistence

Theorem 12.5.1. *Let $\sigma h'(0) \in (g(0)+\mu, \infty]$. Then the disease ratio persists uniformly in the sense that there is some $\epsilon > 0$ such that $r_\infty \geq \epsilon$ for any solution with $S(0) > 0$ and $r(0) > 0$.*

Proof. We assume that the disease does not persist uniformly. Choose $\epsilon \in (0, \epsilon_0/2)$ such that $h(\epsilon)/\epsilon > \mu + g(0) + \epsilon$. Then there exists a solution with $r(0) > 0$ and $S(0) > 0$ such that $r^\infty \geq \epsilon_0$ and $r_\infty < \epsilon$. Then we can find a sequence $t_n \to \infty$ such that $r(t_n) \to r_\infty$, $r(t_n) > 0$ and $r'(t_n) = 0$. Then

$$0 = r'(t_n) = r(t_n)[\xi(r(t_n)) - g(S(t_n)) - \mu]$$

Using our inequalities we see

$$0 = r(t_n)[\xi(r(t_n)) - g(S(t_n)) - \mu]$$
$$0 > r(t_n)[\xi(r(t_n)) - g(0) - \mu]$$
$$0 > r(t_n)\left[\frac{1+r}{r}h(r(t_n)) - g(0) - \mu\right].$$

For large n, $r(t_n) < \epsilon$ and so

$$\frac{1+r}{r}h(r(t_n)) - g(0) - \mu > \frac{h(r(t_n))}{r} - g(0) - \mu > 0,$$

a contradiction. □

12.6 Proportional Disease Boundedness

We start with an easy case of practical host persistence and proportional disease boundedness.

Theorem 12.6.1. *Let $\sigma h(\infty) < g(0)$. For any solution with $S(0) > 0$, $S_\infty \geq S_\diamond > 0$ where $S_\diamond > 0$ is the unique solution of $g(S^\diamond) = \sigma h(\infty)$. Further $r^\infty \leq K\frac{g(0)+\mu}{\mu S_\diamond}$.*

We speak about practical host persistence because we have an explicit bound for S_∞ (Cantrell and Cosner (1996)).

Proof. Let S be a solution with $S(0) > 0$. Then $S'(t) > 0$ if $S(t) < S_\diamond$. In particular, S is bounded away from zero and $S_\infty > 0$. By the fluctuation method, choose a sequence $t_n \to \infty$ with $S(t_n) \to S_\infty$ and $S'(t_n) \to 0$. Then

$$0 = \lim_{n\to\infty} S(t_n)[g(S(t_n)) - h(r(t_n))].$$

Then $0 = \lim_{n\to\infty}[g(S(t_n)) - h(r(t_n))]$ and $g(S_\infty) = \lim_{n\to\infty} h(r(t_n)) \le h(\infty)$. Since g is strictly decreasing, $S_\infty \ge S_\diamond$.

Since $r = I/S \le H/S$, the estimate for $r^\infty \le H^\infty/S_\infty$ follows from Theorem 9.1.1. □

Theorem 12.6.2. *Let $\sigma h(\infty) < g(0) + \mu$. Then there exists some $c > 0$ such that $r^\infty \le c$ for all solutions with $r(0) > 0$ (with $r(0) \ge 0$ if $h'(0) < \infty$ in addition).*

Proof. By Theorem 9.1.1, we can assume that $\sigma h(\infty) \ge g(0)$. We can absorb σ into h, i.e., we can assume that $\sigma = 1$. We first show the claim for r_∞. We can assume that $r(0) > 0$ and $S(0) > 0$.

Choose some $S^\flat > 0$ such that

$$\xi(\infty) = h(\infty) < g(S) + \mu, \qquad S \le S^\flat.$$

Since $g(S^\flat) < g(0) \le h(\infty)$, we can choose some $r^\flat > 0$ and $\delta > 0$

$$\xi(r) < g(S^\flat) + \mu - \delta, \quad g(S^\flat) < h(r) - \delta, \qquad r \ge r^\flat.$$

Suppose that $r_\infty > r^\flat$. After a shift in time, $r(t) > r^\flat$ for all $t \ge 0$. Then

$$g(S^\flat) < h(r(t)) - \delta, \qquad t \ge 0.$$

Since $S' = S(g(S) - h(r))$ and g is strictly decreasing, this implies that $S^\infty < S^\flat$. After another shift in time, $S(t) < S^\flat$ for all $t \ge 0$. Then

$$r' = (1 + r)h(r) - r(g(S) + \mu) < r(\xi(r) - (g(S^\flat) + \mu)) < r(-\delta).$$

So r decreases exponentially. This contradiction show that $r_\infty < r^\flat$.

Assume $h'(0) < \infty$. Then h is locally Lipschitz continuous on $\mathbb{R}_+$ and system (12.3.1) induces a semiflow on $\mathbb{R}^2_+$. Our previous consideration shows that the semiflow is uniformly weakly ρ-persistent for $\rho(S, r) = \frac{1}{1+r}$. By (Smith and Thieme, 2011,

Thm.4.13), with $B = [0, K] \times \mathbb{R}_+$, the semiflow is uniformly ρ-persistent. This implies the statement.

Without this assumption, we still have a semiflow on $X = [0, \infty) \times (0, \infty)$ which is uniformly weakly ρ-persistent for $\rho(S, r) = 1/r$. Now apply (Smith and Thieme, 2011, Thm.4.13), with $B = [0, K] \times (0, \infty)$ and notice that $B \cap \{\epsilon_1 \leq \rho(S, r) \leq \epsilon_2\} = [0, K] \times [1/\epsilon_2, 1/\epsilon_1]$ is compact whenever $0 < \epsilon_1 < \epsilon_2 < \infty$. □

No information about the host can be given without further assumptions.

12.7 Host Extinction

Recall that

$$\begin{aligned} S' =& S(g(S) - \sigma h(r)), \\ r' =& r[\sigma\xi(r) - (g(S) + \mu)], \end{aligned} \tag{12.7.1}$$

for any solution with $r(0) > 0$. By Corollary 9.1.2, $S(t) \to 0$ implies that $I(t) \to 0$ as $t \to \infty$.

Proposition 12.7.1. *If $r(t) \to \infty$ as $t \to \infty$, then $S(t) \to 0$, and $I(t) \to 0$ as $t \to \infty$.*

Proof. $r(t) = \dfrac{I(t)}{S(t)}$ implies $r(t)S(t) = I(t)$, and Theorem 9.1.1 provides us with a bound on I, call it c, which is independent of initial conditions. Thus, we have $r(t)S(t) = I(t) \leq c$, and $r(t) \to \infty$ as $t \to \infty$. Therefore, it must be the case that $S(t) \to 0$ which, by Corollary 9.1.2, implies the result. □

12.7.1 Initial-Value Dependent Host Extinction

Theorem 12.7.2. *Let there be some $r^\natural > 0$ such that $\sigma h(r^\natural) \geq g(0)$ and $\sigma\xi(r^\natural) \geq g(0) + \mu$.*

(a) Then there is initial-value dependent host extinction: If $r(0) \geq r^\natural$ *for such an* $r^\natural$*, then* $S(t) \to 0$*.*

(b) If, in addition, $\sigma h(r^\natural) > \mu r^\natural$*, all solutions with* $S(0) > 0$*,* $r(0) > 0$ *satisfy* $S(t) \to 0$ *or* $(S(t), r(t)) \to (K, 0)$ *or* $(S(t), r(t)) \to (S^*, r^*)$ *(provided the last one exists) as* $t \to \infty$*.*

Proof. Again, for ease of notation, we set $\sigma = 1$. Assume that there is some $r^\natural > 0$ such that $g(0) \leq h(r^\natural)$ and $\xi(r^\natural) \geq g(0) + \mu$. Let $r(0) \geq r^\natural$. We can assume that $S(0) > 0$. Then $S(t) > 0$ for all $t \geq 0$ and $\xi(r^\natural) > g(S(t)) + \mu$ for all $t \geq 0$. This implies that $r(t) > r^\natural$ for all $t > 0$. Since g is strictly decreasing, $S'(t) = S(t)(g(S(t)) - h(r(t)) < S(t)(g(0) - h(r^\natural)) \leq 0$. So S is decreasing, and $S(t)$ cannot converge to a strictly positive limit. So $S(t) \to 0$ as $t \to \infty$.

Assume $\sigma h(r^\natural) > \mu r^\natural$ in addition. If $r(t) \geq r^\natural$ for at least one $t \geq 0$, then $S(t) \to 0$ as $t \to \infty$. So we can assume that $r(t) < r^\natural$ for all $t \geq 0$. By Theorem D.1.1 (in Appendix D), $S(t)$ converges at $t \to \infty$ and, if the limit is not zero, $r(t)$ converges as well. The limit of $(S(t), r(t))$ is an equilibrium ((Thieme, 2003, Cor.A.19)). □

Corollary 12.7.3. *If* $\sigma h(g(0)/\mu) \geq g(0)$ *and* $r(0) \geq g(0)/\mu$*, then* $S(t) \to 0$*.*

Proof. We apply Theorem 12.7.2 with $r^\sharp = g(0)/\mu$. Then one condition is trivially satisfied. By definition of ξ,

$$\sigma\xi(g(0)/\mu) = \sigma h(g(0)/\mu)(1 + (\mu/g(0)) \geq g(0) + \mu.$$

So the other condition is also satisfied. □

Corollary 12.7.4. *Let* $\sigma h(r^\sharp) = g(0)$ *for some* $r^\sharp \in (0, \infty)$*. Then* $S(t) \to 0$*, if, for such an* $r^\sharp$*, we have* $\mu r^\sharp \leq g(0)$ *and* $r(0) \geq r^\sharp$*.*

Proof. Let $r^\sharp \in (0, \infty)$, $\sigma h(r^\sharp) = g(0)$, and $\mu r^\sharp \leq g(0)$. Then

$$\sigma\xi(r^\sharp) = \sigma h(r^\sharp)(1 + (1/r^\sharp)) = g(0)(1 + (1/r^\sharp)) \geq g(0) + \mu.$$

The claim now follows from Theorem 12.7.2. □

Corollary 12.7.5. *Let $\sigma h(\infty) > g(0) + \mu$. Then there exists some $r^\circ > 0$ such that $r(t) \to \infty$ and $S(t) \to 0$ if $r(0) \geq r^\circ$.*

Proof. Recall that $h(\infty) = \xi(\infty)$. By choosing $r^\circ > 0$ large enough, $r^\sharp = r^\circ$ satisfies the assumptions in Theorem 12.7.2. Thus $S(t) \to 0$ as $t \to \infty$.

Further we can achieve that $\sigma\xi(r) > g(0) + \mu$ for all $r \in [r^\circ, \infty)$. By System co:S-above-K-general, if $r(0) \geq r^\circ$, r is increasing and cannot converge to a finite limit. So $r(t) \to \infty$ as $t \to \infty$. □

Actually, we can do better than that.

Theorem 12.7.6. *Let $r^\circ \in (0, \infty)$ such that $\sigma\xi(r^\circ) = g(0) + \mu$ and assume that there is no larger solution of this equation and that $\xi'(r^\circ) > 0$.*

Then $r(t) \to \infty$ and $S(t) \to 0$ as $t \to \infty$, if $r(0) \geq r^\circ$.

Proof. Let $\tilde{r}$ be a solutions of $\tilde{r}' = \tilde{r}(\sigma\xi(\tilde{r}) - g(0) - \mu)$ with $\tilde{r}(0) > r^\circ$. Then $\tilde{r}(t) > r^\circ$ for all $t \geq 0$ and $\tilde{r}$ is bounded away from r°. If $\tilde{r}$ is not unbounded, $\tilde{r}_\infty = \liminf_{t\to\infty} \tilde{r}(t) \in (r^\circ, \infty)$. By the fluctuation method ((Thieme, 2003, Prop.A.22)), there exists a sequence $s_n \to \infty$, $\tilde{r}(s_n) \to \tilde{r}_\infty$, $\tilde{r}'(s_n) \to 0$ as $n \to \infty$. Then $0 = \tilde{r}_\infty(\sigma\xi(\tilde{r}_\infty) - g(0) - \mu)$, a contradiction. Thus $\tilde{r}_\infty = \infty$ and $\tilde{r}(t) \to \infty$ as $t \to \infty$.

Now consider a solution S, r of (12.7.1) with $S(0) > 0$ and $r(0) > r^\circ$. Then $S(t) > 0$ and $r(t) > r^\circ$ for all $t \geq 0$ and

$$r'(t) > r(t)(\sigma\xi(r(t)) - g(0) - \mu), \qquad t \geq 0.$$

Consider a solution $\tilde{r}$ of the differential equation with $r^\circ < \tilde{r}(0) < r(0)$. An elementary comparison argument shows that $r(t) > \tilde{r}(t) \to \infty$ as $t \to \infty$.

If $r(0) = r^\circ$, then $r'(0) > 0$ and $r(t_0) > r^\circ$ for some $t_0 > 0$ and the claim follows as well.

We claim that $\sigma h(\infty) \geq g(0) + \mu$. Suppose not; i.e. suppose $\sigma h(\infty) < g(0) + \mu$. This gives us $\sigma\xi(\infty) = \sigma h(\infty) < g(0) + \mu$, and thus, by the intermediate value theorem, there is some $r_c > r^\circ$ such that $\xi(r_c) = \mu + g(0)$, a contradiction to our hypothesis. Thus we have $\sigma h(\infty) \geq g(0) + \mu$.

Now we apply Theorem 12.7.1 for the final result. □

We use the previous result to describe another bistability scenario. An example is given in Section 12.13.

Theorem 12.7.7. *Let $r^\circ \in (0, \infty)$ be a unique solution of $\sigma\xi(r^\circ) = g(0) + \mu$ and assume $\xi'(r^\circ) > 0$, $g(0) \neq \sigma h(r^\circ)$. Further let $g(0) < \sigma h(\infty)$ and $\sigma h'(0) < \mu$.*

Then $r(t) \to \infty$ and $S(t) \to 0$ for all solutions with $r(0) \geq r^\circ$ and $S(t) > 0$.

For all solutions S, r with $S(0) > 0$ and $r(0) > 0$, there is the dichotomy that $(S(t), r(t)) \to (K, 0)$ or $S(t) \to 0$ as $t \to \infty$.

Proof. Let S, r be a solution of (12.7.1) with $S(0) > 0$ and $r(0) > 0$. By Theorem 12.7.6, we can assume that $r(t) < r^\circ$ for all $t \geq 0$. Since $\sigma h'(0) < \mu$, there is no interior equilibrium (Corollary 12.4.5) and no periodic orbit because it would surround an interior equilibrium. By the Poincaré-Bendixson theory, the ω-limit set of this solution contains an equilibrium. If the ω-limit set contains the locally asymptotically stable equilibrium $(K, 0)$ (cf. Theorem 12.4.2), the solution converges to this equilibrium.

The solution cannot converge to the saddle $(0, 0)$ because its stable manifold it the $S = 0$ axis (Theorem 12.4.1).

Suppose that the ω-limit set, let us call it Y, contains the saddle $(0, 0)$. Then $M = \{(0, 0)\}$ is an isolated compact invariant set which is a proper subset of Y. By the Butler-McGehee lemma (Smith and Thieme, 2011, Thm.8.8), there is solution of (12.7.1), defined on $\mathbb{R}$ and with range in $Y \setminus M$, which converges to $(0, 0)$ as

$t \to -\infty$. Since the unstable manifold of $(0,0)$ is the $r = 0$ axis, the range of this solution is contained in the $r = 0$ axis and the solution converges to $(K,0)$ as $t \to \infty$. This implies that $(K,0) \in Y$ and $Y = \{(K,0)\}$ because this equilibrium is locally asymptotically stable. This contradiction shows that Y cannot contain $(0,0)$.

If the solution converges to $(0, r^\circ)$, then $S(t) \to 0$ as $t \to \infty$.

Suppose that Y contains $(0, r^\circ)$ but also other elements. Using the Butler-McGehee lemma as before, we obtain that Y contains $(0,0)$ which we have ruled out before. So this cannot happen and our claim is proved. □

12.7.2 Global Host Extinction

Proposition 12.7.8. *Let $r^\sharp \in (0,\infty)$ and $\sigma\xi(r) \geq g(0) + \mu$ for all $r \in [0, r^\sharp]$. Then $r_\infty = \liminf_{t\to\infty} r(t) \geq r^\sharp$ or $S(t) \to 0$ as $t \to \infty$ for all solutions with $r(0) > 0$.*

Proof. Without loss of generality, we can consider a solution with $S(t) > 0$ for all $t \geq 0$ and $r(t) > 0$ for all $t \geq 0$. For simplicity, we absorb σ into h and ξ. Then $\xi(r) > g(S) + \mu$ for all $r \in [0, r^\sharp]$ and $S > 0$. By System (12.7.1), if $r(s) \geq r^\sharp$ for some $s \geq 0$, then $r(t) \geq r^\sharp$ for all $t \geq s$ and $r_\infty \geq r^\sharp$.

Suppose that $r(t) < r^\sharp$ for all $t \geq 0$. Since g is strictly decreasing, by System (12.7.1) r is strictly increasing and $r_\infty = \lim_{t\to\infty} r(t) \in (0, r^\sharp)$. Again by System (12.7.1), r' is uniformly continuous and $r'(t) \to 0$ as $t \to \infty$ by Barbalat's lemma ((Thieme, 2003, Cor.A.18)). Then

$$0 = \lim_{t\to\infty} r(t)(\xi(r(t)) - g(S(t)) - \mu) = r_\infty \lim_{t\to\infty} (\xi(r_\infty) - g(S(t)) - \mu).$$

Since $r_\infty > 0$,

$$\lim_{t\to\infty} g(S(t)) = \xi(r_\infty) - \mu \geq g(0).$$

Since g is strictly decreasing, $S(t) \to 0$ as $t \to \infty$. □

Theorem 12.7.9. *Assume there exists some $r^\sharp \in (0,\infty)$ such that $\sigma h(r^\sharp) \geq g(0)$ and $\sigma\xi(r) \geq g(0)+\mu$ for all $r \in [0,r^\sharp]$. Then the disease drives the host into extinction.*

Proof. For the ease of notation we absorb σ into h and set $\sigma = 1$. By proposition 12.7.8, we can assume that $r_\infty \geq r^\sharp$.

By the fluctuation method, there exists a sequence $t_n \to \infty$ such that $S(t_n) \to S^\infty = \limsup_{t\to\infty} S(t)$ and $S'(t_n) \to 0$. Then

$$0 = \lim_{n\to\infty} S(t_n)(g(S(t_n) - h(r(t_n))) = S^\infty \lim_{n\to\infty}(g(S(t_n) - h(r(t_n))).$$

So either $S^\infty = 0$ or

$$0 = g(S^\infty) - \lim_{n\to\infty} h(r(t_n)).$$

Since h is increasing and $g(S^\infty) < g(0)$,

$$g(0) > g(S^\infty) = \lim_{n\to\infty} h(r(t_n)) \geq h(r_\infty) \geq h(r^\sharp),$$

a contradiction. So $S^\infty = 0$ and $S(t) \to 0$ as $t \to \infty$. □

Corollary 12.7.10. *Let $\sigma\xi(r) \geq g(0)+\mu$ for all $r \in (0, g(0)/\mu]$. Then the disease drives the host into extinction.*

Since ξ is bounded away from 0, this condition can be satisfied by choosing σ large enough.

Proof. We apply Theorem 12.7.9 with $r^\sharp = g(0)/\mu$. By definition of ξ,

$$\sigma h(g(0)/\mu) = \sigma\xi(g(0)/\mu)\frac{g(0)/\mu}{1+(g(0)/\mu)} \geq (g(0)+\mu)\frac{g(0)}{\mu+g(0)} = g(0).$$ □

Corollary 12.7.11. *Let $\sigma h(\infty) \geq g(0)$ and $\sigma h(r^\sharp) = g(0)$ for some $r^\sharp \in (0,\infty]$. Further assume that, for such an $r^\sharp$, ξ is increasing on $[0,r^\sharp)$. Then the disease drives the host into extinction if $\sigma h'(0) \geq g(0)+\mu$.*

Proof. If ξ is increasing on $[0, r^\sharp)$, the condition $\sigma\xi(r) \geq g(0) + \mu$ in Theorem 12.7.9 is satisfied for all $r \in [0, r^\sharp]$ if it is satisfied for $r = 0$. Recall that $\xi(0) = h'(0)$. □

This implies the next result.

Corollary 12.7.12. *Assume that there is some $r^\sharp \in (0, \infty)$ such that $\sigma h(r^\sharp) = g(0)$, ξ is decreasing on $[0, r^\sharp)$ and $\mu r^\sharp \leq g(0)$. Then the disease drives the host into extinction.*

Proof. Since ξ is decreasing on $[0, r^\sharp)$, the condition $\sigma\xi(r) \geq g(0) + \mu$ in Theorem 12.7.9 is satisfied for all $r \in [0, r^\sharp)$ if it is satisfied for $r = r^\sharp$. By our other assumptions,

$$\sigma\xi(r^\sharp) = \sigma h(r^\sharp)(1 + (1/r^\sharp)) = g(0)\Big(1 + \frac{1}{r^\sharp}\Big) \geq g(0) + \mu. \qquad \square$$

For the next result, recall Section 12.4.2 which motivates us to assume that there are at most two no-host boundary equilibria.

Theorem 12.7.13. *Let $\sigma h(\infty) \geq g(0)$ and $\mu < \sigma h'(0) < \infty$.*

Let $\sigma h(\infty) \neq g(0) + \mu$ or, if $\sigma\xi(\infty) = \sigma h(\infty) = g(0) + \mu$, let there exist some $r^\sharp \in (0, \infty)$ such that $\sigma\xi(r) \geq g(0) + \mu$ for all $r \in (r^\sharp, \infty)$.

Assume that $g(0) < \sigma h(r)$ for any $r > 0$ with $\sigma h(r) = \mu r$. Further assume that there are at most two $r > 0$ with $\sigma\xi(r) = g(0) + \mu$ and that $\xi'(r)$ exists and $\xi'(r) \neq 0$ for any of those. Then the disease drives the host into extinction if $r(0) > 0$.

Proof. Consider a solution with $S(0) > 0$ and $r(0) > 0$. Then $S(t) > 0$ and $r(t) > 0$ for all $t \geq 0$. If $\sigma h(\infty) < g(0) + \mu$, then the solution is bounded by Theorem 12.6.2. If $\sigma h(\infty) > g(0) + \mu$, by Corollary 12.7.3, the solution is either bounded or $S(t) \to 0$ as $t \to \infty$.

If $\sigma h(\infty) = g(0) + \mu$, our assumptions imply those of Theorem 12.7.2 and $S(t) \to 0$ as $t \to \infty$, or there is some $r\sharp \geq 0$ such that $r(t) \leq r^\sharp$ for all $t \geq 0$.

So we can assume that the solution is bounded and thus has an ω-limit set, ω. There is no interior equilibrium by Theorem 12.4.3 and therefore also no periodic orbit. By the Poincaré-Bendixson theorem, ω contains an equilibrium $(0, r^\circ)$, and any equilibria in ω are of that form because we have uniform disease persistence by Theorem 12.5.1. If ω does not consist of just one equilibrium, it contains a cycle consisting of equilibria and orbits connecting them in a cyclic way (Thieme (1994)).

By Proposition 12.4.9, the eigenvalues of the Jacobian matrix at an equilibrium $(0, r^\circ)$ with $r^\circ \in (0, \infty)$ are both real and, by our assumptions, both different from 0.

None of the equilibria can be a stable or unstable node because they belong to a cyclic cycle. So, they are saddles.

Such a cycle cannot be contained in the union of the positive axes and must contain a point in $(0, \infty)^2$. These cycles are either homoclinic orbits connecting one boundary equilibrium to itself with the connecting orbit being contained in the interior of the positive quadrant or heteroclinic orbits cyclically connecting two boundary equilibria. One connecting orbit would be contained in the $S = 0$ axis and the other in the interior of the positive quadrant.

Either way, the cycle surrounds a point in $(0, \infty)$. Consider the full orbit starting at this point and its ω-limit and α-limit sets. By uniqueness (obtained via remark 12.3.1) the orbit cannot touch one of the connecting orbits. It cannot converge to any of the equilibria in forward or backward time because these are saddles and our interior point would be in the stable or unstable manifold. So both the ω-limit set and the α-limit set would be that cycle which cannot happen without violating uniqueness. So ω consists just of one equilibrium which means that the solution we started with (which is in the interior of the positive quadrant) converges towards an equilibrium $(0, r)$ with $r > 0$. □

Theorem 12.7.14. *Let h be differentiable on $\big(0, \frac{g(0)}{\mu}\big)$ and $h'(r) > 0$ for all $r \in \big(0, \frac{g(0)}{\mu}\big)$.*

Let one of the following two assumptions be satisfied:

(a) $\sigma h\big(\frac{g(0)}{\mu}\big) > g(0)$

or

(b) $\sigma h\big(\frac{g(0)}{\mu}\big) \geq g(0)$ *and* $h(r)/r$ *strictly decreasing in* $r \in (0, \frac{g(0)}{\mu}]$.

Then the disease drives the host into extinction.

Proof. Consider a solution with $S(0) > 0$ and $r(0) > 0$. Then $S(t) > 0$ and $r(t) > 0$ for all $t \geq 0$. Since the system is autonomous, by Corollary 12.7.3, $S(t) \to 0$ as $t \to \infty$ if $r(t) \geq \frac{g(0)}{\mu}$ for some $t \geq 0$. So we can assume that $r(t) \in (0, \frac{g(0)}{\mu})$ for all $t \geq 0$. In particular, the solution is bounded and its ω-limit set contains an equilibrium or a periodic orbit by the Poincaré-Bendixson theory. Since there is no interior equilibrium by Theorem 12.4.3 or Theorem 12.4.5, there is also no periodic orbit, and the ω-limit set contains a boundary equilibrium. By Theorem 12.5.1, it contains an equilibrium $(0, r^\circ)$ with $r^\circ \in (0, \infty)$. This implies that $S_\infty = \liminf_{t\to\infty} S(t) = 0$.

Now apply Theorem D.1.1 (in Appendix D) with $r^\sharp = g(0)/\mu$. Both (a) and (b) imply that $h(r)/r > \mu$ for all $r \in (0, r^\sharp)$. It follows that S converges as $t \to \infty$ and that its limit is 0. □

Remark 12.7.15. Since f is homogeneous,

$$h(g(0)/\mu) = f(1, g(0)/\mu) = (g(0)/\mu) f(\mu/g(0), 1).$$

So the condition $\sigma h\big(\frac{g(0)}{\mu}\big) > g(0)$ is equivalent to $f(\mu/g(0), 1) > \mu/\sigma$. Cf. Theorem 8.1.1.

12.8 Host Persistence

The following result will tells us that the combined conditions for initial-value dependent host extinction in Theorem 12.7.2 and Corollary 12.7.5 are almost sharp (with the case $\sigma h(\infty) = g(0) + \mu$ possibly not covered).

Theorem 12.8.1. *Let $\sigma h(\infty) < g(0) + \mu$ and $h'(0) < \infty$. Assume that either there is no $r \in (0, \infty)$ with $\sigma\xi(r) = g(0) + \mu$ or, if there is, it satisfies $g(0) > \sigma h(r)$.*

Then the host persists uniformly.

Proof. We absorb σ into h, i.e., without loss of generality $\sigma = 1$.

Since $h'(0) < \infty$, h is locally Lipschitz continuous on $\mathbb{R}_+$ and System (12.3.1) induces a continuous semiflow on $\mathbb{R}^2_+$. By Theorem 12.6.2, the semiflow has a compact attractor. We apply (Smith and Thieme, 2011, Sec.8.3) choosing the persistence function $\rho(S, r) = S$. Let Ω be the union of all ω-limit sets of solutions starting with $S(0) = 0$. Let A_0 be the compact attractor of bounded sets for the semiflow induced by the differential equation $r' = (1 + r)h(r) - r(g(0) + \mu)$. Then A_0 is invariant and connected ((Smith and Thieme, 2011, Prop.2.24)), and A_0 is a compact interval. Further A_0 is isolated and acyclic and $\Omega \subseteq A_0$. Let r° be the right endpoint of A_0. Since A_0 is invariant, there exist a total solution r of the equation with range in A_0 and $r(0) = r^\circ$. This implies that $r'(0) = 0$, i.e., $r^\circ = 0$ or $\xi(r^\circ) = g(0) + \mu$.

We show that, in either case, A_0 is uniformly weakly ρ-repelling.

Case 1: $r^\circ = 0$.

Then $A_0 = \{(0, 0)\}$. Choose some $\epsilon > 0$ such that $g(\epsilon) - h(\epsilon) > \epsilon$. This is possible because g and h are continuous and $g(0) > 0 = h(0)$. Since g is decreasing and h is increasing, $g(S) - h(r) > \epsilon$ for all $S, r \in [0, \epsilon]$. Suppose there is a solution with $S(0) > 0$ and $\limsup_{t\to\infty} d((S(t), r(t)), (0, 0)) < \epsilon$. After a shift in time, $0 < S(t) < \epsilon$

and $r(t) < \epsilon$ for all $t \geq 0$. By choice of ϵ, $S'(t) \geq \epsilon S(t)$ and S increases exponentially to ∞, a contradiction.

Case 2: $r^\circ > 0$ and $\xi(r^\circ) = g(0) + \mu$.

By assumption, $g(0) > h(r^\circ)$. To show that A_0 is uniformly weakly ρ-repelling, choose some $\epsilon > 0$ such that $g(S) > h(r^\circ + \epsilon) + \epsilon$ for all $S \in [0, \epsilon]$. Since h is increasing, $g(S) > h(r) + \epsilon$ for all $S \in [0, \epsilon]$ and $r \in [0, r^\circ + \epsilon]$. Assume that there exists a solution such that $S(0) > 0$ and $\limsup_{t\to\infty} d((S(t), r(t)), A_0) < \epsilon$. Then, after a shift in time, $S(t) \in [0, \epsilon]$ and $r(t) \in [0, r^\circ + \epsilon]$ for all $t \geq 0$. So $g(S(t)) \geq h(r(t)) + \epsilon$ for all $t \geq 0$ and S grows to infinity, a contradiction. By (Smith and Thieme, 2011, Thm.8.20), the induced semiflow is uniformly weakly ρ-persistent and thus uniformly persistent by (Smith and Thieme, 2011, Thm.4.13). □

The next conditions may appear crude, but will turn out to be sharp for the minimum function.

Corollary 12.8.2. *Let $\sigma h(\infty) < g(0)$ or $\sigma\xi(r) < g(0) + \mu$ for all $r \in (0, \infty]$. Then the host persists uniformly.*

Proof. If $\sigma h(\infty) < g(0)$, the assertion follows from the practical persistence result in Theorem 12.6.1. If the second condition holds, it follows from Theorem 12.8.1. Recall that $\xi(\infty) = h(\infty)$ and $h'(0) = \xi(0)$. □

Theorem 12.8.3. *Let $g(0) \leq \sigma h(\infty) < g(0) + \mu$. Assume that for any $r^\sharp > 0$ with $g(0) = \sigma h(r^\sharp)$ we have $\sigma\xi(r) \neq g(0) + \mu$ for all $r \in [r^\sharp, \infty)$.*

Then the host persists uniformly.

Proof. We check the assumption of Theorem 12.8.1 via a contradiction argument. Without loss of generality let $\sigma = 1$.

Assume that there are $r > 0$ with $\xi(r) = g(0) + \mu$ and $g(0) \leq h(r)$. Let r° be the largest of those $r > 0$ with $\xi(r) = g(0) + \mu$. Then $\xi(r^\circ) = g(0) + \mu$. By assumption, we have that $r^\circ < r^\sharp$ for any $r^\sharp$ with $g(0) = h(r^\sharp)$. Since h is increasing, $g(0) > h(r^\circ)$ and also $g(0) > h(r)$ for all $r > 0$ with $\xi(r) = g(0) + \mu$ because r° was the largest of such r, a contradiction. □

12.9 Disease Extinction

In particular, the host persists if the disease goes extinct. We study the conditions for the latter to happen. We start with an auxiliary result which allows us to concentrate on the situation where $S(t) < K$ for all $t \geq 0$. We have Corollary 9.1.7, however we prove a more precise result concerning r.

Proposition 12.9.1. *If $S(t) \geq K$ for all $t \geq 0$, then $\sigma \int_0^\infty h(r(t))dt \leq \ln \frac{S(0)}{K}$ and $r(t) \to 0$ as $t \to \infty$.*

Proof. Let $S(t) \geq K$ for all $t \geq 0$. We integrate the differential equation for S and use that $g(S(s)) \leq 0$ for all $s \geq 0$,

$$\ln S(t) - \ln S(0) = \int_0^t g(S(s))ds - \sigma \int_0^t h(r(s))ds \leq -\sigma \int_0^t h(r(s))ds.$$

We regroup and take the limit as $t \to \infty$ and obtain the first inequality. From the differential equation for r, we obtain

$$\frac{d}{dt}(r(t) + 1) = r'(t) = \sigma h(r(t))(r(t) + 1) - r(t)[g(S(t)) + \mu].$$

Since $S(t) \to K$ as $t \to \infty$, after a shift in time, we can assume that $g(S(t)) + \mu \geq \mu/2$ for all $t \geq 0$. We integrate the last differential equation and obtain,

$$r(t) + 1 \leq [r(0) + 1] \exp\Big(\int_0^t \sigma h(r(s))ds \Big) \leq [r(0) + 1]S(0)/K =: c.$$

We integrate the differential equation for r, using the variation of parameters formula

$$r(t) \leq r(0)e^{-t\mu/2} + \sigma c \int_0^t h(r(s))e^{-(t-s)\mu/2}ds \to 0, \qquad t \to \infty.$$

The convergence to 0 of the integral follows from Lebesgue's dominated convergence theorem or directly from

$$\int_0^t h(r(s))e^{-(t-s)\mu/2}ds \leq \int_{t/2}^t h(r(s))ds + \int_0^{t/2} h(r(s))e^{-t\mu/4}ds. \qquad \square$$

The conditions for disease extinction in the next result should be compared to those for host persistence in Theorem 12.8.1.

Theorem 12.9.2. *Assume $\sigma h(\infty) < g(0) + \mu$ and that there is some $\delta > 0$ such that $\sigma\xi(r) \leq \mu$ for all $r \in (0, \delta)$. Assume that either there is no $r \in (0, \infty)$ with $\sigma\xi(r) = g(0) + \mu$ or, if there is, it satisfies $g(0) > \sigma h(r)$.*

Then $r(t) \to 0$ as $t \to \infty$ for all solutions with $S(0) > 0$.

Proof. Under these conditions all solutions are bounded and, if $S(0) > 0$, their S components are bounded away from 0 due to Theorems 12.6.2 and 12.8.2. By the Poincaré-Bendixson theory, their ω-limit sets contain the equilibrium $(K, 0)$. So, for any solution, $r_\infty = 0$.

Suppose that $r^\infty > 0$. By the fluctuation theory ((Thieme, 2003, Lemma A.20)), there exists a sequence $t_n \to \infty$ such that $0 < r(t_n) \to 0$ as $n \to \infty$ and $r'(t_n) = 0$ for all $n \in \mathbb{N}$. From the differential equation of r,

$$0 = \sigma\xi(r(t_n)) - (g(S(t_n)) + \mu).$$

By Proposition 12.9.1, we only need to consider the case $S(t_n) < K$ and $g(S(t_n)) > 0$, possibly after choosing a subsequence. For sufficiently large $n \in \mathbb{N}$, $r(t_n) \in (0, \delta)$ and $\sigma\xi(r(t_n)) \leq \mu$. Thus we have

$$0 = \sigma\xi(r(t_n)) - (g(S(t_n)) + \mu) \leq -g(S(t_n)) < 0,$$

a contradiction.

This shows $r^\infty = 0$. □

12.10 The Minimum Incidence

Consider $f(S, I) = \min\{S, \alpha I\}$. Since we have chosen to normalize $f(1, 1) = 1$, we let $\alpha \geq 1$. This is consistent with this incidence being the same as the constant risk incidence unless the number of infective is substantially less than the number susceptibles. Then $h(r) = \min\{1, \alpha r\}$ for $r \geq 0$. Expressed by cases,

$$h(r) = \begin{cases} \alpha r, & 0 \leq r \leq 1/\alpha, \\ 1, & r \geq 1/\alpha, \end{cases}$$

and

$$\xi(r) = \begin{cases} \alpha(1 + r), & 0 \leq r \leq 1/\alpha, \\ 1 + r^{-1}, & r \geq 1/\alpha. \end{cases}$$

ξ takes its maximum, $1 + \alpha$, at $r = 1/\alpha$. Further

$$\xi(0) = h'(0) = \alpha, \quad \xi(\infty) = h(\infty) = 1, \qquad \xi_{\max} = 1 + \alpha = \xi(1/\alpha).$$

We first determine a coexistence equilibrium and the conditions under which it exists. Since f is defined by cases, we do this from scratch rather than using the general results in Section 12.4.1 though we follow the same procedure

We first look for a coexistence equilibrium with $r^* \leq 1/\alpha$. The second equation in System (12.4.1) becomes

$$\sigma\alpha r^* = \mu r^*, \qquad r^* \leq 1/\alpha.$$

This has a solution if and only if $\sigma\alpha = \mu$ in which case every $r^* \in (0, 1/\alpha]$ is a solution. The condition for solving the first equation in system (12.4.1) becomes $g(0) > \mu r^*$ which can be satisfied if $r^* < g(0)/\mu$.

We now look for a solution $r^* > 1/\alpha$. Then $r^* = \sigma/\mu$ which comes with the condition $\sigma > \mu/\alpha$. The condition for solving the first equation in (12.4.1) becomes $g(0) > \mu r^*$ which is satisfied if and only if $\sigma < g(0)$. Since $\xi'(r^*) < 0$ and $h'(r^*) = 0$, the equilibrium is locally asymptotically stable by Proposition 12.4.7. We summarize.

Theorem 12.10.1. *A coexistence equilibrium exists in exactly the following two cases:*

$$\sigma\alpha = \mu$$

In this case, there exists a line of coexistence equilibria (S^*, r^*) *with* $0 < r^* \leq 1/\alpha$ *and* $r^* < g(0)/\mu$.

$$\mu/\alpha < \sigma < g(0)$$

In this case, there exists a unique coexistence equilibrium with $r^* = \sigma/\mu < 1/\alpha$. *This equilibrium is locally asymptotically stable.*

We look for conditions for initial-condition-dependent disease-mediated host extinction.

Let $\sigma h(\infty) \geq g(0)$, i.e., $\sigma \geq g(0)$.

To apply Theorem 12.7.2, we look for some $r^\natural > 0$ such that $g(0) \leq \sigma h(r^\natural)$ and $\sigma\xi(r^\natural) \geq g(0) + \mu$.

Since both h and ξ take their maximum at $1/\alpha$, we try $r^\natural = 1/\alpha$ and obtain $g(0) \leq \sigma$ and $\sigma(1 + \alpha) \geq g(0) + \mu$.

Theorem 12.10.2. *Let* $\sigma \geq g(0)$ *and* $\sigma \geq \frac{g(0)+\mu}{1+\alpha}$. *Then* $S(t) \to 0$ *for all solutions with* $r(0) \geq 1/\alpha$.

This result is almost sharp because Corollary 12.8.2 gives the following host persistence result. Recall that $h(\infty) = 1$ and $\xi_{\max} = \sigma(1 + \alpha)$.

Theorem 12.10.3. *Let $\sigma < g(0)$ or $\sigma(1 + \alpha) < g(0) + \mu$. Then the host persists uniformly.*

If $g(0) \leq \sigma$, we can also try $r^\sharp = \frac{g(0)}{\sigma\alpha} \leq 1/\alpha$ and get a larger range for $r(0)$ to lead to host extinction.

Theorem 12.10.4. *Let $g(0) \leq \sigma$ and $\sigma\alpha \geq \mu$. Then $S(t) \to 0$ if $r(0) \geq \frac{g(0)}{\sigma\alpha}$.*

To find conditions for global disease-mediated host extinction, we apply Theorem 12.7.13.

Assume $g(0) \leq \sigma h(\infty) = \sigma$ and $\mu < \sigma h'(0) = \sigma\alpha$.

If $g(0) + \mu = \sigma\xi(\infty)$, then $g(0) + \mu < \sigma\xi(r)$ for all $r \in [1/\alpha, \infty)$.

Now solve $\sigma h(r) = \mu r$. If $r \geq 1/\alpha$, this yields $r = \sigma/\mu$ which is consistent with our assumption $\mu < \sigma\alpha$. So $g(0) < \sigma h(r)$ is satisfied if we assume $g(0) < \sigma$.

If $r \leq 1/\alpha$, then $\sigma\alpha r = \mu r$ which is excluded.

By the form of ξ, there are at most two solutions of $\sigma\xi(r) = g(0) + \mu$.

If there are exactly two solutions, they are both different from $1/\alpha$ and $\xi' \neq 0$ at both of them.

We have exactly one solution, $r^\circ = 1/\alpha$, if $g(0) + \mu = \sigma\xi_{\max} = \sigma(1 + \alpha)$. This equality cannot hold because we have assumed $g(0) < \sigma$ and $\mu < \alpha\sigma$.

Theorem 12.10.5. *Let $g(0) < \sigma$ and $\mu < \sigma\alpha$. Then the disease drives the host into extinction.*

This global host extinction result is almost sharp because of Theorem 12.10.1.

Theorem 12.10.6. *Let $\sigma\alpha < \mu$. Then the equilibrium $(K, 0)$ is locally asymptotically stable.*

This follows from Theorem 12.4.2 and $h'(0) = \alpha$.

Bistability between the equilibrium $(K, 0)$ and host extinction is possible.

Corollary 12.10.7. *Let $g(0) < \frac{\mu}{\alpha}$ and $\frac{g(0)+\mu}{1+\alpha} \leq \sigma < \frac{\mu}{\alpha}$. Then $(K, 0)$ is locally asymptotically stable and $S(t) \to 0$ as $t \to \infty$ whenever $r(0) \geq 1/\alpha$. Actually, any solution with $S(0) > 0$ and $r(0) > 0$ satisfies $(S(t), r(t)) \to (K, 0)$ or $S(t) \to 0$ as $t \to \infty$.*

Proof. The first condition makes the second feasible. The second condition is equivalent to $g(0) + \mu \leq \sigma(1 + \alpha)$ and $\sigma\alpha < \mu$. This implies $g(0) < \sigma$. Combine Theorems 12.10.2 and 12.10.6 to obtain the first statement. The second statement follows from Theorem 12.7.2 with $r^\natural = 1/\alpha$. □

12.11 Homogeneous Power Incidence

In Section 10, we have already established that for any power incidence $\sigma S^q I^p$ with $q \in (0, 1)$ there is initial-data dependent host extinction. We have also established global disease-mediated host extinction for the constant risk incidence σS if $\sigma \geq g(0)$.

So we concentrate on global host extinction for $p + q = 1$ and $0 < p < 1$. We have $h(r) = r^p$. We obtain the following result from Theorem 12.7.14.

Theorem 12.11.1. *Let $\sigma > g(0)^{1-p}\mu^p$. Then the disease always drives the host into extinction.*

This result is almost sharp.

Theorem 12.11.2. *Let $\sigma < g(0)^{1-p}\mu^p$. Then there exists a unique coexistence equilibrium.*

Proof. We apply Theorem 12.4.3 (b). Notice that $h'(0) = \infty$. The equation $\sigma h(r^*) = \mu r^*$ is solved by $r^* = (\mu/\sigma)^{\frac{1}{p-1}}$. The condition $g(0) > \mu r^*$ is equivalent to the condition of our theorem. □

12.12 Decreasing ξ

As an example, the per unit ratio growth rate ξ is decreasing if f is the asymmetric frequency-dependent incidence with $p \leq q$. Other examples of decreasing ξ are presented in Section 12.3.2. We recall that for Kuang and Beretta (1998) this is equivalent to $1 \leq m$.

We can completely categorize the dynamics of a system in terms of σ compared to some critical values in the case that $h(r)/r$ is strictly decreasing and ξ is decreasing.

We establish their order in the following lemma.

Throughout this section, we assume without further saying that ξ is decreasing, and $\dfrac{h(r)}{r}$ is strictly decreasing.

Lemma 12.12.1. *Then* $\frac{\mu}{h'(0)} < \frac{\mu+g(0)}{h'(0)} \leq \frac{g(0)}{h(g(0)/\mu)} \leq \frac{\mu+g(0)}{h(\infty)}$. *If ξ is strictly decreasing, all inequalities are strict.*

Proof. The first inequality is trivial. Since ξ is decreasing, we have

$$\xi(\infty) \leq \xi(g(0)/\mu) \leq \xi(0).$$

By definition of ξ, (5.1.4),

$$h(\infty) \leq h(g(0)/\mu)\left(1 + \frac{1}{(g(0)/\mu)}\right) \leq h'(0).$$

We divide by $g(0) + \mu$:

$$\frac{h(\infty)}{g(0)+\mu} \leq \frac{h(g(0)/\mu)}{g(0)} \leq \frac{h'(0)}{g(0)+\mu}.$$

Inverting the inequality gives the result. □

For Kuang and Beretta (1998), the values are as follows (recalling $g(S) = a(1 - S/K)$ and $h(r) = (m+1)r/(1+mr)$):

$$\begin{aligned}
\frac{\mu}{h'(0)} &= \frac{d}{1+m}, \\
\frac{\mu + g(0)}{h'(0)} &= \frac{d+a}{1+m}, \\
\frac{g(0)}{h(g(0)/\mu)} &= \frac{d+ma}{1+m}, \\
\frac{\mu + g(0)}{h(\infty)} &= \frac{(d+a)(1+m)}{m}.
\end{aligned}$$

Lemma 12.12.2. *There are no periodic orbits and, if there is a bounded solution with ω-limit set in $(0,\infty)^2$, this solution converges to the interior equilibrium.*

Proof. Let f be the vector field, i.e. $f_1(S,r) = Sg(S) - S\sigma h(r), f_2(S,r) = r\sigma\xi(r) - r\mu - rg(S)$. Letting $\rho = \frac{1}{Sr}$, we use Dulac's Criterion to get

$$\rho f_1(S,r) = \frac{g(S)}{r} - \frac{\sigma h(r)}{r}, \qquad \rho f_2(S,r) = \frac{\sigma\xi(r)}{S} - \frac{\mu - g(S)}{S}.$$

and

$$\bigtriangledown \cdot (\rho f) = \frac{g'(S)}{r} + \frac{\sigma\xi'(r)}{S} \leq \frac{g'(S)}{r} < 0.$$

Since the axes are invariant, every periodic orbit is contained in $(0,\infty)^2$ and does not exist by the Dulac criterion. Let $\omega \subseteq (0,\infty)^2$ be the ω-limit set of a bounded solution. By the Poincaré Bendixson theorem, ω contains an equilibrium which lies in $(0,\infty)^2$. Since the interior equilibrium is unique when it exists, it is contained in the ω-limit set. If ω contains other points, it must contain a cyclic connection of interior equilibria, i.e., a homoclinic orbit in $(0,\infty)^2$ connecting the interior equilibrium to itself. But such a homoclinic orbit is also ruled out by the Dulac criterion. □

Theorem 12.12.3. *Let $\sigma \leq \frac{\mu}{h'(0)}$. Then the disease dies out, $r(t) \to 0$, and $S(t) \to K$ as $t \to \infty$ if $S(0) > 0$.*

Proof. We observe that the only equilibria are (0,0) and $(0, K)$. Our hypothesis implies

$$\sigma h(\infty) = \sigma \xi(\infty) \leq \sigma \xi(r) \leq \sigma \xi(0) = \sigma h'(0) \leq \mu < g(0) + \mu$$

for all $r \geq 0$. We invoke Corollary 12.8.2 for host persistence and Theorem 12.6.2 for proportional disease boundedness. Therefore, all solutions are bounded and thus converge to an equilibrium by Lemma 12.12.2. In particular, $r(t) \to 0$ as $t \to \infty$. Since $(0, 0)$ is a saddle with the $S = 0$ axis being the stable manifold (Section 12.4), all solutions with $S(0) > 0$ satisfy $S(t) \to K$ as $t \to \infty$. □

Theorem 12.12.4. *Let $\frac{\mu}{h'(0)} < \sigma \leq \frac{\mu+g(0)}{h'(0)}$. Then (S^*, r^*) attracts all solutions with $S(0) > 0$ and $r(0) > 0$. $(0, 0)$ attracts all solutions with $S(0) = 0$ and $r(0) > 0$. $(K, 0)$ attracts all solutions with $S(0) > 0$ and $r(0) = 0$.*

Proof. If $r(0) = 0$, then $r(t) \equiv 0$. Then $S' = Sg(S)$, so $S(t) \to K$ as $t \to \infty$. If $S(0) = 0$, then $S(t) \equiv 0$, so $\frac{r'}{r} = \sigma \xi(r) - \mu - g(0)$. We refer to equation (12.4.5) for the existence of the no host equilibrium, which in this case simplifies to

$$\sigma \in \left(\frac{\mu + g(0)}{\xi(0)}, \frac{\mu + g(0)}{\xi(\infty)} \right).$$

Thus, for our current σ we do not have the no host equilibrium, which implies that there is no r° such that $\sigma \xi(r^\circ) = \mu + g(0)$. Therefore $\xi(r) < \mu + g(0)$, so we have that $r(t) \to 0$ as $t \to \infty$.

Now we consider the case where both $S(0)$ and $r(0)$ are positive. Our hypothesis satisfies Corollary 12.4.4, so the coexistence equilibrium exists. Since r° does not exist, host persistence follows from Theorem 12.8.1. Notice that $h(\infty) = \xi(\infty) \leq \xi(0) = h'(0)$.

We also easily check the assumptions of Theorems 12.5.1 and 12.6.2. Combining these three theorems we have that there is some $\varepsilon, M, T > 0$ such that for solutions with initial conditions $S(0) > 0$ and $r(0) > 0$ we have $(S(t), r(t)) \in [\varepsilon, M]^2$ for all $t > T$. By Lemma 12.12.2, these solutions converge to the interior equilibrium. □

Theorem 12.12.5. *Let* $\frac{\mu+g(0)}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)}$. *Then* (S^*, r^*) *attracts all solutions with* $S(0) > 0$ *and* $r(0) > 0$. *Solutions with* $S(0) = 0$ *and* $r(0) > 0$ *converge to an equilibrium* $(0, r^\circ)$ *with* $0 < r^\circ < r^*$. $(K, 0)$ *attracts all solutions with* $S(0) > 0$ *and* $r(0) = 0$.

Proof. If $r(0) = 0$, then $r \equiv 0$. Then $S' = Sg(S)$, so $S(t) \to K$ as $t \to \infty$ if $S(0) > 0$. If $S(0) = 0$, then $S \equiv 0$, so $\frac{r'}{r} = \sigma\xi(r) - \mu - g(0)$. Since ξ is decreasing, condition (12.4.5) for the existence of no-host equilibria becomes $h'(0) = \xi(0) < \frac{g(0)+\mu}{\sigma} < \xi(\infty) = h(\infty)$ which is satisfied by our assumptions, as seen with help from Lemma 12.12.1. So no-host boundary equilibria exist and $r(t)$ converges to one of them as $t \to \infty$ if $r(0) = 0$. ξ decreasing and Corollary 12.4.11 imply any such r° will be less than r^*.

Our hypothesis satisfies Corollary 12.4.4, so the coexistence equilibrium exists, and now we also have existence of the no-host boundary equilibrium. Nevertheless, the same arguments as for Theorem 12.12.4 imply that all solutions with $S(0) > 0$ and $r(0) > 0$ converge to the interior equilibrium (S^*, r^*). □

Theorem 12.12.6. *Let* $\frac{g(0)}{h(g(0)/\mu)} \leq \sigma < \frac{g(0)+\mu}{h(\infty)}$. *Then* $S(t) \to 0$ *as* $t \to \infty$ *if* $r(0) > 0$. *A boundary equilibrium* $(0, r^\circ)$ *exists with* $r^\circ > 0$. *The* ω*-limit set of a solution with* $r(0) > 0$ *has the form* $\{0\} \times [r_1, r_2]$ *with* $0 < r_1 \leq r_2 < \infty$ *and all* $(0, r)$ *with* $r \in [r_1, r_2]$ *being boundary equilibria. If such a boundary equilibrium is unique,* $g(0)/\mu \leq r^\circ$, *and* $(0, r^\circ)$ *is globally stable for all solutions* $S(0) \geq 0$ *and* $r(0) > 0$.

Proof. We prepare to apply Corollary 12.7.12. Since h is increasing and $h(0) = 0$, $g(0) \leq \sigma h(g(0)/\mu)$ and the intermediate value theorem imply that there is some $r^\sharp \in \left(0, \frac{g(0)}{\mu}\right]$ such that $\sigma h(r^\sharp) = g(0)$. Thus we have $r^\sharp \mu \leq g(0)$ and can use Corollary 12.7.12 to obtain that $S(t) \to 0$ as $t \to \infty$ with any solution with $r(0) > 0$

Combined with Theorems 12.5.1 and 12.6.2 we have disease persistence, disease boundedness, and host extinction. Since the ω-limit set of a bounded solution is connected and contained in the $S = 0$ axis by our considerations, the ω-limit set for a bounded solution with $r(0) > 0$ has the form $\{0\} \times [r_1, r_2]$ for some $0 < r_1 \leq r_2 < \infty$. Since this set is invariant, $(0, r_1)$ and $(0, r_2)$ are boundary equilibria, $\sigma\xi(r_j) = g(0)+\mu$. Since ξ is decreasing, $\sigma\xi(r) = g(0) + \mu$ for all $r \in [r_1, r_2]$. So the ω-limit set is a special continuum of no-host boundary equilibria. If such a boundary equilibrium is unique, $r_1 = r_2 = r^\circ$, then $r(t) \to r^\circ$ as $t \to \infty$. We have that $\sigma = \dfrac{g(0)}{h(g(0)/\mu)}$ implies $\sigma h(g(0)/\mu) = g(0)$, which in turn gives us $\sigma\xi(g(0)/\mu) = g(0) + \mu$. Since ξ is decreasing, $\sigma > \dfrac{g(0)}{h(g(0)/\mu)}$ implies $r^\circ > \dfrac{g(0)}{\mu}$. □

Theorem 12.12.7. *Let* $\dfrac{\mu + g(0)}{h(\infty)} \leq \sigma$. *Then* $S(t) \to 0$ *as* $t \to \infty$ *if* $r(0) > 0$. *If in addition* ξ *is strictly decreasing or* $\dfrac{\mu + g(0)}{h(\infty)} < \sigma$, *then* $r(t) \to \infty$ *as* $t \to \infty$ *if* $r(0) > 0$ *as well.*

Proof. Since ξ is decreasing, we have

$$\mu + g(0) \leq \sigma h(\infty) = \sigma\xi(\infty) \leq \sigma\xi(r),$$

for all $r \in [0, \infty)$. Applying Theorem 12.7.10 gives extinction of S.

Suppose that $\dfrac{\mu + g(0)}{h(\infty)} < \sigma$. Then we set $\varepsilon = \sigma\xi(\infty) - \mu - g(0) > 0$, and

$$\frac{r'}{r} = \xi(r) - \mu - g(S) \geq \xi(\infty) - \mu - g(0) > \varepsilon,$$

a contradiction.

Suppose that ξ is strictly decreasing and $\dfrac{\mu + g(0)}{h(\infty)} \leq \sigma$. Then,

$$\frac{r'}{r} = \sigma\xi(r) - \mu - g(S) > \sigma\xi(\infty) - \mu - g(0) \geq 0,$$

thus r is increasing, and either increases without bound or converges to a finite limit. Suppose toward contradiction that r converges to some value c. Then we set $\varepsilon = \sigma\xi(c) - \mu - g(0) > 0$, and we see

$$\frac{r'}{r} = \sigma\xi(r) - \mu - g(S) \geq \xi(c) - \mu - g(0) > \varepsilon,$$

implying that $r(t) \to \infty$ as $t \to \infty$, contradicting that it is bounded above. □

We compare our results to those of Kuang and Beretta (1998). With the simplifications $c = f$ and $1 \leq m$, we can show sharp results for the dynamics of the system, making slight improvements to Kuang and Beretta (1998).

We summarize the results in the following table. By $(r(t) \to \infty)^*$, we mean that this event only occurs if $\dfrac{\mu + g(0)}{h(\infty)} < \sigma$ or ξ is strictly decreasing, and GAS stands for "globally asymptotically stable."

Parameter Values	Dynamics	Theorem
$\sigma \leq \frac{\mu}{h'(0)}$	$r(t) \to 0, S(t) \to K.$	(12.12.3)
$\frac{\mu}{h'(0)} < \sigma \leq \frac{\mu+g(0)}{h'(0)}$	no $(0, r^\circ)$, (S^*, r^*) GAS for $(0, \infty)^2$	(12.12.4)
$\frac{\mu+g(0)}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)}$	$\exists(0, r^\circ)$, (S^*, r^*) GAS for $(0, \infty)^2$.	(12.12.5)
$\frac{g(0)}{h(g(0)/\mu)} \leq \sigma < \frac{\mu+g(0)}{h(\infty)}$	$\exists(0, r^\circ)$, $r(0) > 0 \Rightarrow S(t) \to 0$.	(12.12.6)
$\frac{\mu+g(0)}{h(\infty)} \leq \sigma$	$r(0) > 0 \Rightarrow S(t) \to 0, (r(t) \to \infty)^*.$	(12.12.7)

Table 12.1: Dynamics When ξ is Decreasing

The following table summarizes the situation for $f(S, I) = \frac{SI}{pS+qI}$ with $0 < p < q < 1$, $p + q = 1$, where $h(r) = \frac{r}{p+qr}$ is strictly increasing and $\xi(r) = \frac{1+r}{p+qr}$ is strictly decreasing.

Parameter ranges	Dynamics	Thm.
$\sigma \leq p\mu$	$r(t) \to 0, S(t) \to K$	(12.12.3)
$p\mu < \sigma \leq p(\mu + g(0))$	no $(0, r^\circ)$, (S^*, r^*) GAS for $(0, \infty)^2$	(12.12.4)
$p(\mu + g(0)) < \sigma < p\mu + qg(0)$	$\exists(0, r^\circ)$, (S^*, r^*) GAS for $(0, \infty)^2$	(12.12.4)
$p\mu + qg(0) \leq \sigma < q(\mu + g(0))$	$\exists(0, r^\circ)$, $r(0) > 0 \Rightarrow S(t) \to 0$	(12.12.6)
$q(\mu + g(0)) \leq \sigma$	$r(0) > 0 \Rightarrow r(t) \to \infty, S(t) \to 0$	(12.12.7)

Table 12.2: Dynamics when $p < q$

Finally, we show the table for symmetric frequency dependent incidence $f(S, I) = \frac{SI}{pS+qI}$ with $p = q = 1/2$ where $h(r) = 2\frac{r}{1+r}$ is strictly increasing and $\xi \equiv 2$ is constant.

Parameter Values	Dynamics	Thm.
$2\sigma \leq \mu$	no (S^*, r^*) or $(0, r^\circ)$, $r(t) \to 0, S(t) \to K$	(12.12.3)
$\mu < 2\sigma < \mu + g(0)$	(S^*, r^*) globally stable for $(0, \infty)^2$, $(0, r^\circ)$ DNE	(12.12.4)
$\mu + g(0) = 2\sigma$	$\forall r^\circ \geq 0, \quad \exists (0, r^\circ), \quad r(0) > 0 \Rightarrow S(t) \to 0$	(12.12.7)
$\mu + g(0) < 2\sigma$	$r(0) > 0 \Rightarrow r(t) \to \infty, S(t) \to 0$	(12.12.7)

Table 12.3: Dynamics when $p = q = \frac{1}{2}$

12.13 Frequency-Dependent Incidence with $p > q$

Here we delve deeper into asymmetric frequency-dependent incidence with $p > q$. Since $p + q = 1$, we get that $0 < q < \frac{1}{2} < p < 1$. Classification again is done in terms of σ, but is not so clear-cut as in the case $q \geq p$. Table 12.4 contains condensed results. We have

$$h(r) = \frac{r}{p + qr}, \qquad \xi(r) = \frac{1 + r}{p + qr}, \tag{12.13.1}$$

and ξ is strictly increasing,

$$\xi'(r) = \frac{p - q}{(p + qr)^2} > 0, \qquad r \in [0, \infty),$$

$$h'(0) = \xi(0) = \frac{1}{p}, \qquad h(\infty) = \xi(\infty) = \frac{1}{q}, \qquad h(g(0)/\mu) = \frac{g(0)}{p\mu + qg(0)}.$$

We recall that this is the case of $m < 1$ in Kuang and Beretta (1998). By Theorem 12.6.2, all solutions are bounded if $\sigma < q(g(0) + \mu)$. We will sometimes use the results on the (non)existence of equilibria and their stability in Section 12.4 without further reference.

- $\sigma \leq p\mu$ and $\sigma \leq q(\mu + g(0))$

The interior equilibrium does not exist by Theorem 12.4.6, and there is no parasite boundary equilibrium either. By the Poincaré-Bendixson theory, every ω-limit set contains $(K, 0)$ or $(0, 0)$. $(K, 0)$ is locally stable and attracts all solutions with $S(0) > 0$ and $r(0) \geq 0$. (0,0) attracts all solutions with $S(0) = 0$ and $r(0) \geq 0$.

- $q(\mu + g(0)) < \sigma \leq p\mu$

This case requires $q(\mu + g(0)) < p\mu$ which causes the exclusion of the next case. The interior equilibrium does not exist. $(K, 0)$ is locally stable. The parasite boundary equilibrium $(0, r^\circ)$ exists and is a saddle point with its unstable manifold being the $S = 0$ axis and stable manifold having a non-zero S component. Since $\xi(r) > g(0)+\mu$, if $r(t) > r^\circ$ for ant $t \geq 0$, we have $r(t) \to \infty$ as $t \to \infty$ Otherwise, solutions are bounded. By Theorem 12.7.7, we have bi-stability, with either $r(t) \to 0, S(t) \to K$ or $S(t) \to 0$ if $S(0) > 0$. It is suggestive that the stable manifold of $(0, r^\circ)$ acts as a separatrix between the domain of attractions of $(K, 0)$ and $(0, \infty)$.

- $p\mu < \sigma \leq q(\mu + g(0))$

This case requires $q(\mu + g(0)) > p\mu$ which causes the exclusion of the previous case. The parasite boundary equilibrium does not exist. $(K, 0)$ becomes and stays a saddle with the stable manifold being formed by the $r = 0$ axis. We have r and S both persist (Section 12.5 and 12.8) and are both bounded. The interior equilibrium comes into existence; however, its locally stability cannot be established without information about g and g' except that it is not a saddle (Proposition 12.4.7). The ω-limit sets of solutions with $S(0) > 0$ and $r(0) > 0$ are contained in $(0, \infty)^2$ and, by Poincarré-Bendixson theory, contain the interior equilibrium or a periodic orbit.

- $p\mu < \sigma < p\mu + qg(0)$ and $q(\mu + g(0)) < \sigma$

Both the interior equilibrium and parasite boundary equilibrium exist (Theorem (12.4.4), (12.4.5)), with $r^\circ > r^*$ (Proposition 12.4.10).

$(0, r^\circ)$ is a saddle, with the stable manifold pointing into the plane and the unstable manifold being formed by the $S = 0$ axis. Any trajectory which crosses the line $r = r^\circ$ will immediately begin $S(t) \to 0$ and $r(t) \to \infty$ as $t \to \infty$. The stability of (S^*, r^*) is indeterminable without information about g and g'; however, we do know that it is not a saddle (Proposition 12.4.7). Note: bi-stability is still possible if the interior equilibrium is locally unstable. This can occur if a stable limit cycle surrounds the interior equilibrium.

The equilibrium $(0, 0)$ is still a saddle with the $S = 0$ axis as the stable manifold and the $r = 0$ axis the unstable manifold. So there is the possibility of a cyclic heteroclinic orbit connecting the boundary equilibria $(0, 0)$, $(K, 0)$ and $(0, r^\circ)$.

- $p\mu + qg(0) \leq \sigma$

All solutions with $S(0) \geq 0$ and $r(0) > 0$ satisfy $S(t) \to 0$ as $t \to \infty$ (Theorem 12.7.14).

Parameter ranges	Dynamics
$\sigma \leq p\mu$ and $\sigma \leq q(\mu + g(0))$	no (S^*, r^*) or $(0, r^\circ)$, $r(t) \to 0, S(t) \to K$
$q(\mu + g(0)) < \sigma \leq p\mu$	no (S^*, r^*), $(0, r^\circ)$ saddle. Bi-stability: either $(S(t), r(t)) \to (K, 0)$ or $S(t) \to 0$
$p\mu < \sigma \leq q(\mu + g(0))$	r and S both persist and bounded (S^*, r^*) exists, no $(0, r^\circ)$; inconclusive global dynamics; periodic orbits possible
$p\mu < \sigma < p\mu + qg(0)$ and $q(\mu + g(0)) < \sigma$	(S^*, r^*) exists and $(0, r^\circ)$ saddle; initial-condition-dependent host extinction; periodic orbits, heteroclinic cycle possible
$p\mu + qg(0) \leq \sigma$	$S(t) \to 0$

Table 12.4: Dynamics as $t \to \infty$ when $p > q$ and $S(0) > 0$ and $r(0) > 0$.

12.13.1 Fixed Point and Hopf Bifurcations

Here we will consider the creation and destruction of fixed points. We have that both $(0, 0)$ and $(K, 0)$ exist for all parameter values. The interior equilibrium exists when $p\mu < \sigma < p\mu + qg(0)$, and for r° to exist σ must be between $p(\mu + g(0))$ and $q(\mu + g(0))$.

As we saw above, some of the dynamics get tough in the case of $p > q$; however, when we consider how the interior equilibrium comes into and out of existence, some light is shed on local behavior near birth and destruction of the fixed point.

Recall that (S^*, r^*) exists if and only if $p\mu < \sigma < p\mu + qg(0)$. We see that when $\sigma = p\mu$, $r^* = 0$, so it is birthed from $(K, 0)$ as σ crosses that threshold. Recall that

$(K, 0)$ is stable when $\sigma < p\mu$, and a saddle with stable manifold formed by the $r = 0$ axis and unstable manifold pointing into the plane when $\sigma > p\mu$. Thus, we expect there to be some $\varepsilon > 0$ such that when $\sigma \in (p\mu, p\mu + \varepsilon)$, the interior equilibrium inherits the stability and that there is a trajectory from $(K, 0)$ to (S^*, r^*) via $(K, 0)$'s unstable manifold. This can be seen mathematically via the trace of the Jacobian matrix:

$$\begin{aligned}\lim_{\sigma \to p\mu^+} T = &\lim_{\sigma \to p\mu^+} \left(S^* g'(S^*) + \frac{\mu^2 r^*}{\sigma}(p - q) \right) \\ =&Kg'(K) + \frac{\mu^2 \cdot 0}{\sigma}(p - q) = Kg'(K) < 0.\end{aligned}$$

We see that when $\sigma = p\mu + qg(0)$, $S^* = 0$, so as σ grows, the interior fixed point collides with $(0, r^\circ)$. For $\sigma < p\mu + qg(0)$ we have that $(0, r^\circ)$ is a saddle with stable manifold pointing into the plane and unstable on the $S = 0$ axis, and for $\sigma > p\mu + qg(0)$ it is an unstable node. Therefore, we will have that for some $\varepsilon > 0$ that if $\sigma \in (p\mu + qg(0) - \varepsilon, p\mu + qg(0))$ that there will be a trajectory connecting (S^*, r^*) to $(0, r^\circ)$, implying that the interior equilibrium is unstable for that parameter range. Again we look at the limit of the trace of the Jacobian matrix and we see that

$$\begin{aligned}\lim_{\sigma \to p\mu + qg(0)^-} T = &\lim_{\sigma \to p\mu + qg(0)^-} \left(S^* g'(S^*) + \frac{\mu^2 r^*}{\sigma}(p - q) \right) \\ =&0 \cdot g'(0) + \frac{\mu^2 r^\circ}{\sigma}(p - q) = \frac{\mu^2 r^\circ}{\sigma}(p - q) > 0.\end{aligned}$$

Recall that the Jacobian is always positive (Proposition 12.4.7). Since we have a stability change, the trace of the Jacobian matrix will also change signs, thus, by the intermediate value theorem, there exists an $\sigma_0 \in (p\mu, p\mu + qg(0))$ such that the trace

is zero at σ_0. Note that

$$T = S^* g'(S^*) + \frac{\mu^2 r^*}{\sigma}(p-q) = S^* g'(S^*) + \frac{\mu^2(\frac{\sigma}{q\mu} - \frac{p}{q})}{\sigma}(p-q)$$
$$= S^* g'(S^*) + \frac{\mu}{q}\Big(1 - \frac{p\mu}{\sigma}\Big)(p-q).$$

If S^* is an analytic function of σ and g' is analytic, there exists a so-called global Hopf bifurcation of periodic solutions at σ_0 (Chow and Mallet-Paret (1978)).

Now we consider the boundary equilibrium $(0, r^\circ)$. As σ exceeds $q(\mu + g(0))$, r° descends from infinity. Initially it is a saddle; the stable manifold points into the plane and the unstable manifold is the $S = 0$ axis. As σ passes $p\mu + qg(0)$, $(0, r^\circ)$ collides with and swallows the interior equilibrium (which is unstable) and becomes an unstable node. As σ passes $p(\mu + g(0))$, $(0, r^\circ)$ merges with the origin. This results in the change of stability of the origin from a saddle to an unstable node.

We note that the parameters determine which of the interior equilibrium and host extinction equilibrium come into existence first. If $p\mu < q(\mu + g(0))$, then the interior equilibrium comes into existence first and the reverse inequality yields the reverse result. If equality holds, then they come into existence simultaneously.

12.13.2 Generic Global Hopf Bifurcation

Theorem 12.13.1. *If $g \in C^2(0, K)$, asymmetric frequency dependent incidence with $p > q$ will give us generic global Hopf bifurcation of periodic solutions.*

Proof. Asymmetric frequency dependent incidence function gives us $h(r) = \dfrac{r}{p + qr}$, which implies $r^* = \dfrac{\sigma - p\mu}{q\mu}$. Using $g(S^*) = \mu r^* = \dfrac{\sigma - p\mu}{q}$, we can simplify to see $\sigma = qg(S^*) + p\mu$. A final observation we make is $\xi'(r) = (p-q)\dfrac{\mu^2}{\sigma^2}$.

We rewrite the trace as a function of S^*, and we call it $\hat{T}$. We perform some algebra and we have

$$\begin{aligned}
\hat{T}(S^*) &= S^* g'(S^*) + \sigma r^* \xi'(r^*) \\
&= S^* g'(S^*) + \sigma \frac{g(S^*)}{\mu}(p-q)\frac{\mu^2}{\sigma^2} \\
&= S^* g'(S^*) + g(S^*)(p-q)\frac{\mu}{\sigma} \\
&= S^* g'(S^*) + g(S^*)(p-q)\frac{\mu}{qg(S^*)+p\mu} \\
&= \frac{1}{qg(S^*)+p\mu}\left(S^* g'(S^*)[qg(S^*)+p\mu] + g(S^*)(p-q)\mu\right) \\
&= \frac{1}{qg(S^*)+p\mu}\left(S^* g'(S^*)qg(S^*) + S^* g'(S^*)p\mu + g(S^*)(p-q)\mu\right) \\
&= \frac{S^* g'(S^*)qg(S^*)\mu}{qg(S^*)+p\mu}\left(\frac{1}{\mu} + \frac{S^* g'(S^*)p + g(S^*)(p-q)}{S^* g'(S^*)qg(S^*)}\right).
\end{aligned}$$

We note that in this form, $\hat{T} : (0, K) \to \mathbb{R}$. Define $L(S) = \dfrac{Sg'(S)p + g(S)(p-q)}{Sg'(S)qg(S)}$. $g \in C^2$ gives us $L \in C^1$, which allows us to use Sard's Lemma (Deimling (1985), lemma 1.4). Sard's Lemma implies that the set $\varphi := \{\mu | L(S) + \frac{1}{\mu} = 0, L'(S) = 0, S \in (0, K)\}$ has Lebesgue measure zero. For all $\mu \notin \varphi$, we have $\hat{T}'(S) \neq 0$ for all S with $\hat{T}(S) = 0$, and so we have a transversal crossing of eigenvalues of the imaginary axis (as opposed to a tangential touching of the imaginary axis). Therefore, for almost all $\mu > 0$, there is a Hopf bifurcation. By Chow and Mallet-Paret (1978), there is a generic global Hopf bifurcation of periodic solutions. □

From Chow and Mallet-Paret (1978) we have that either the size of the periodic orbit will tend to infinity, the period will tend to infinity, S^* will be outside any compact subset of $\mathbb{R}$, or that another Hopf bifurcation will occur. Since $S^* \in (0, K)$, it is easily within a compact subset of $\mathbb{R}$. We also know that the periodic orbit's size is bounded in S by 0 and K. If r is unbounded, we have that there is initial condition

dependent host extinction, so if $r(t) > r^\circ$ for any t, we have that $r(t) \to \infty$, thus the orbits are bounded in r as well. Finally, since $\hat{T}(0) > 0$ and $\hat{T}(K) < 0$, there must be an odd number of transversal crossings, therefore at least 1 such bifurcation will not be absorbed by another. In this case we must have that the period of the periodic orbit tends to infinity, which supports the idea of a heteroclinic bifurcation eliminating the periodic orbit. The proposed heteroclinic orbit is between (0,0), (K,0), and $(0, r^\circ)$. This means that as periodic orbits are formed, they will either disappear via another Hopf Bifurcation, or they will disappear as their period tents toward infinity, probably in approach of a heteroclinic orbit.

12.13.3 A Hopf Bifurcation Example

To highlight the potential for rich dynamics with this system, we consider the growth function

$$g(S) = \frac{\kappa}{b + S^\alpha} - \theta.$$

By our previous observations, we know that a Hopf bifurcation must occur; however, we could not determine if it will be supercritical or subcritical. In fact, we cannot show it in general because both are possible. Using the above g we can vary only alpha (and sigma, as it is our bifurcation parameter) and obtain both sub and super critical bifurcations.

In both scenarios we use the parameter set $p = .8, q = .2, \kappa = 5, b = 1, \mu = 5$, and $\theta = 1$. We varied α, then used a Newton solver to determine σ_0 and hunted for the bifurcation. However since $g'(0)$ does not exist if $\alpha < 1$, our simulations only seek to prove the Hopf bifurcation and do not apply to some of the theory above.

The numerical evidence suggests that a supercritical bifurcation occurs when $\alpha = .2$ and $\sigma_0 = 4.050732988644327$. There is a small window, approximately 7×10^{-5}, for which a periodic orbit can occur when σ is greater than σ_0. Choosing $\sigma =$

4.050742988644327 we generate Figure 12.13.1, where the trace of the Jacobian matrix of the interior equilibrium at this value is $T = 0.000031581$.

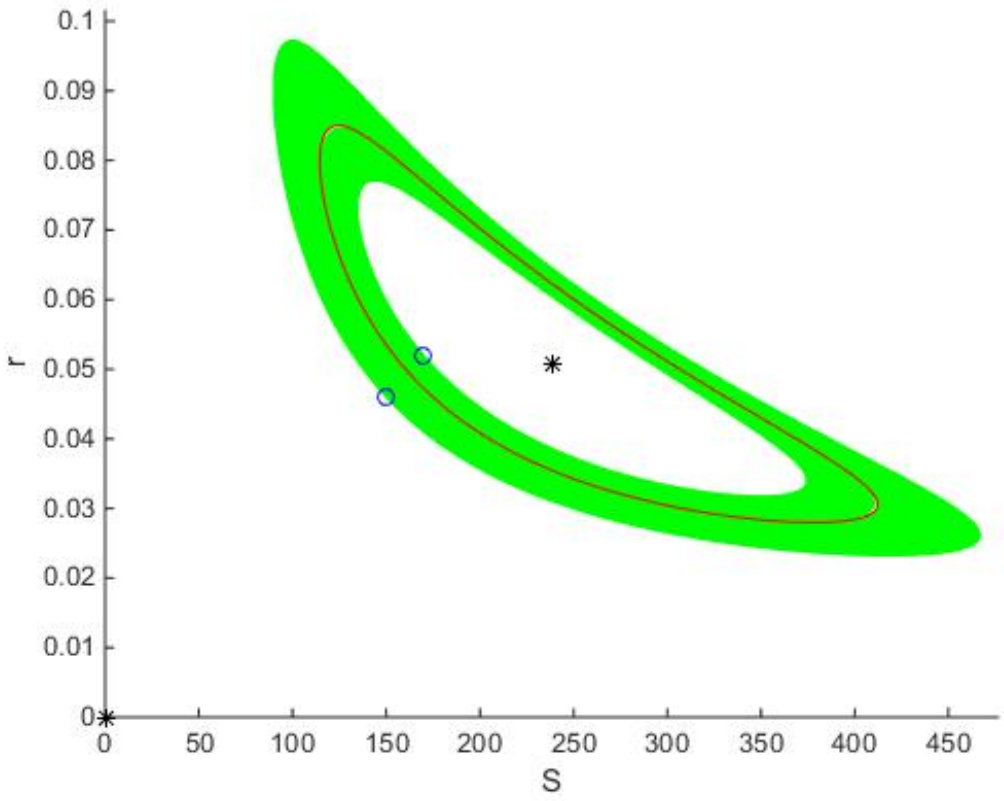

Figure 12.13.1: This is generated using $\sigma = 4.050742988644327$. The blue circle represents an initial condition, the green line is the trajectory from said initial condition, and the red line represents the stable periodic orbit. The black asterisk is the equilibrium (S^*, r^*), which is unstable. This suggests that the bifurcation which birthed this orbit is supercritical.

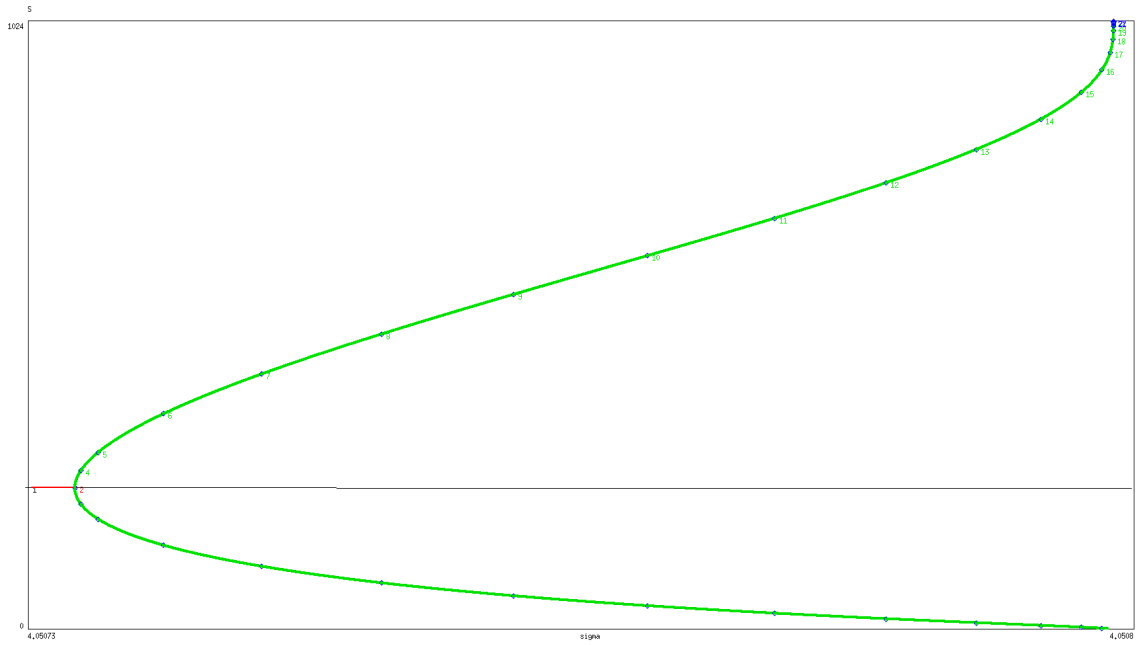

Figure 12.13.2: This figure is from an AUTO analysis, using $\alpha = .2$, and choosing σ as the bifurcation parameter. The vertical axis is the value of S, and the horizontal axis is σ. The top horizontal line of the box indicates the carrying capacity, K, which in this case is 1024. The horizontal line inside the box indicates the S value of the interior equilibrium as σ varies. The bottom horizontal line of the box is zero, which represents values at two equilibria; namely the origin and the parasite-only equilibrium. The pair of curves which branch off indicate the maximum and minimum of the stable periodic orbit which bifurcates from the equilibrium. The interior equilibrium is stable before the intersection and unstable after. We observe the curves reaching the upper and lower limits of the axis, which are K and 0 respectively, thus we believe that the periodic orbit disappears via a so called heteroclinic bifurcation. This supports the idea of a supercritical Hopf bifurcation.

For $\alpha = .3$ we found $\sigma_0 = 4.082811276721079$ supplies us with what appears to be a subcritical bifurcation. Here the window for a periodic orbit is still small,

approximately $2.5 \times 10^{-}4$ in this case, however this time it is for σ less than σ_0. Choosing $\sigma = 4.08280127672108$ we generate Figure 12.13.3, where the trace of the interior equilibria here is $T = -0.000029478$.

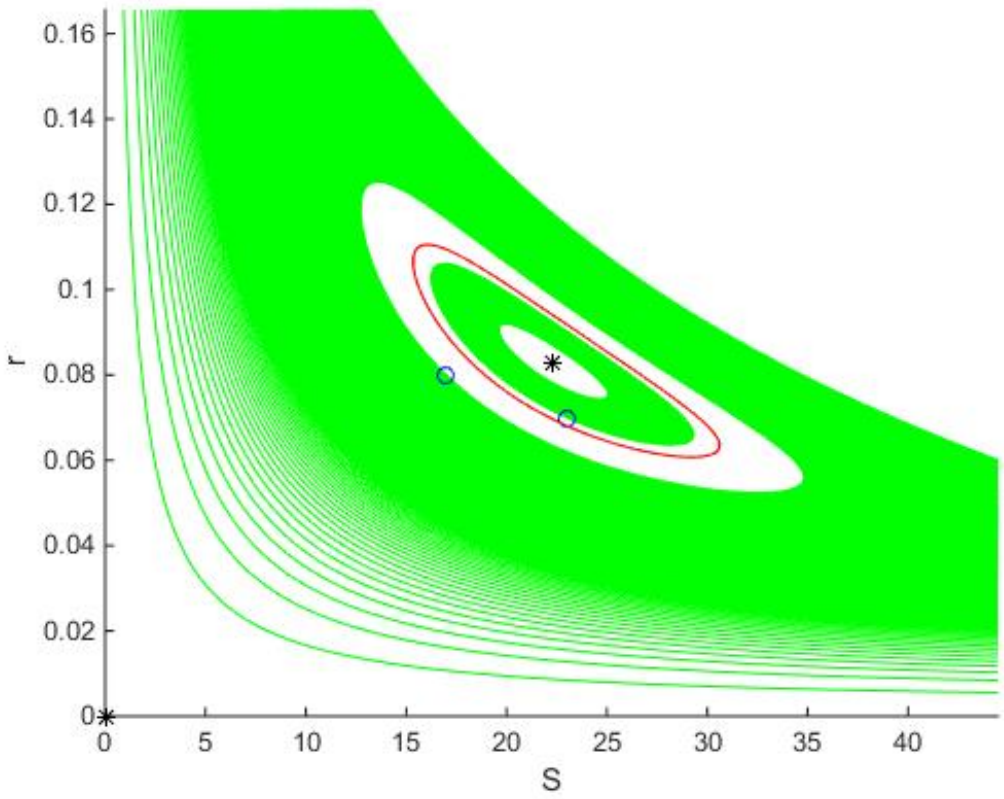

Figure 12.13.3: This is generated using $\sigma = 4.08280127672108$. The blue circle represents an initial condition, the green line is the trajectory from said initial condition, and the red line represents the unstable periodic orbit. The black asterisk is the equilibrium (S^*, r^*), which is stable. This suggests that the bifurcation which birthed this orbit is subcritical.

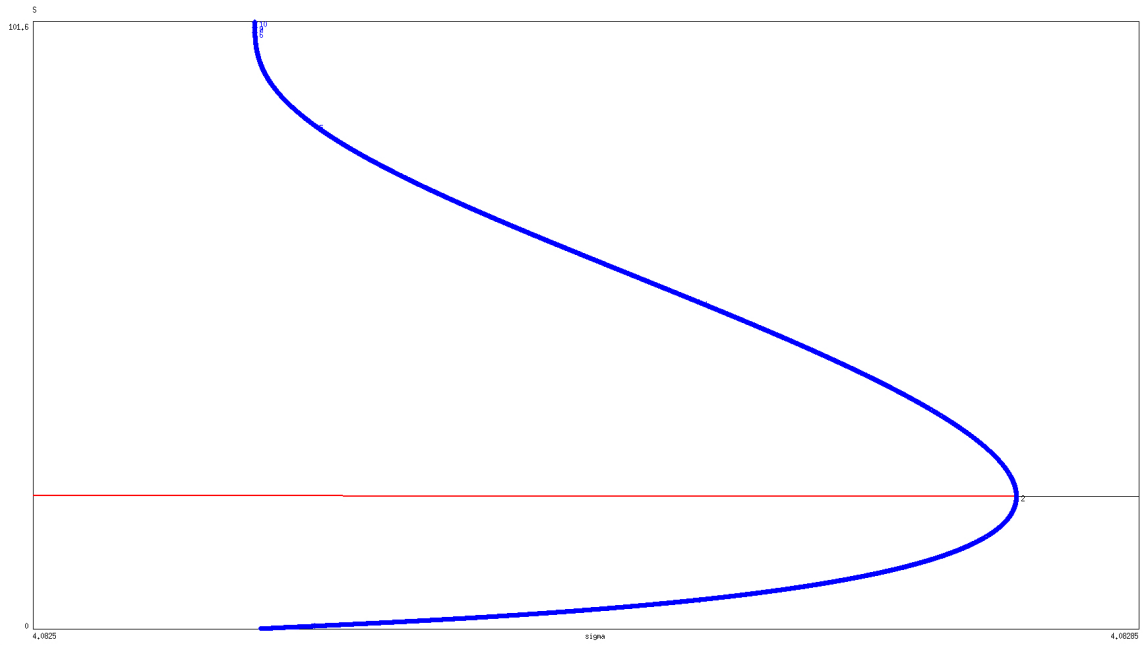

Figure 12.13.4: This figure is from an AUTO analysis, using $\alpha = .3$, and choosing σ as the bifurcation parameter. The vertical axis is the value of S, and the horizontal axis is σ. The top horizontal line of the box indicates the carrying capacity, K, which in this case is approximately 101.593667. The horizontal line inside the box indicates the S value of the interior equilibrium as σ varies. The bottom horizontal line of the box is zero, which represents values at two equilibria; namely the origin and the parasite-only equilibrium. The pair of curves which branch off indicate the maximum and minimum of the unstable periodic orbit which bifurcates from the equilibrium. The interior equilibrium is stable before the intersection and unstable after. We observe the curves reaching the upper and lower limits of the axis, which are K and 0 respectively, thus we believe that the periodic orbit disappears via a so called heteroclinic bifurcation. This supports the idea of a subcritical Hopf bifurcation.

The numerical and analytical data suggest that in the case of a supercritical (subcritical) bifurcation we have that the periodic orbit is absorbed (is born from)

the heteroclinic orbit between the fixed points $(0,0),(K,0)$ and $(0,r^{\circ})$. It was shown in Kuang and Beretta (1998) that, for their model, a periodic orbit could not exist around a stable equilibrium. In our model, however, it is possible! This is due to the change in growth function, which follows the biological rules of an endemic system rather than a predator-prey system.

Chapter 13

DISCUSSION

13.1 Comparison to Epidemic Model

Comparing these findings to the epidemic case, we see that for homogeneous incidences, the endemic model and epidemic model share outcomes: parameters determine whether or not the disease drives the host into extinction, as well as whether or not this outcome depends on the initial conditions. Endemic and epidemic outcomes agree when using upper density-dependent incidences as well: the disease cannot drive the host into extinction.

As for power incidences, only those of the form $f(S, I) = S^q I^p$ are considered in the endemic model, rather the more general form $f(S, I) = \theta(S) I^p$ which was considered in the epidemic model. If $p \in (0, 1]$ and $q \in (0, 1)$, then no matter the size of the transmission coefficient σ, the disease drives the host into extinction if the number of initial susceptibles is sufficiently small and the number of initial infectives is sufficiently large. It depends on σ though, how small or large these initial values have to be. If $p + q - 1 > 0$ as is suggested by Tables 3.1 and 3.4, there is also an endemic equilibrium which is locally asymptotically stable for small σ and unstable for large σ. Such an endemic equilibrium does not exist for the epidemic model. Numerical computations suggest that, for large σ, the disease drives the host into extinction unless the initial values are exactly those of the endemic equilibrium, as seen in Figure 10.2.1. Though the numerical evidence is strong, these findings could not be verified analytically.

If considering a power law incidence with $p \in (0,1)$ and $1 \leq q$, we see radically different behaviors as compared to both the epidemic model (for any parameters) and endemic model with $p \in (0,1]$ and $q \in (0,1)$. We will have an interior equilibrium for all σ, which is always globally asymptotically stable in $(0,\infty)^2$. This is in stark contrast to the global extinction found in the epidemic model, which holds for all initial conditions. It also is a sharp change from the endemic dynamics with $p \in (0,1]$ and $q \in (0,1)$, where there is always initial condition dependent extinction and for large σ the interior equilibrium is unstable.

13.2 Homogeneous Incidence Functions

In Section 12.3.1 we see some examples of homogeneous functions and their corresponding hazard functions. Using the large class of homogeneous functions as described by Equation (12.2.1), we have many unimodal ξ functions at our disposal, with limiting behavior that matches other desirable functions. Section 12.3.2 shows how easy it is to create homogeneous incidence functions, and, in particular, a decreasing ξ.

If ξ is strictly decreasing, then we have our dynamics neatly summed up by global stability of an equilibrium, and global asymptotic stability for almost all σ. Table 12.1 shows existence and globally stable of equilibria depending on the value of σ. An additional note is that the extinction equilibrium r° will be unique. If we consider when ξ is decreasing (not strictly), then our global stability results will still hold for almost all σ.

We also uncovered the versatility of the asymmetric frequency function. It yields a strictly decreasing ξ if $p < q$, a constant ξ and (symmetric) frequency dependence if $p = q = 1/2$, and a strictly increasing ξ if $p > q$. Table 12.4 provides the results for $p > q$, which have exciting dynamics including periodic orbits, heteroclinic orbits, and

bi-stability. The heteroclinic orbit will form between (0,0), $(K, 0)$, and $(0, r^{\circ})$ in the $S - r$ plane, and (0,0) and $(K, 0)$ in the $S - I$ plane. The Hopf bifurcation will occur for any $g(S)$ that is strictly decreasing and strictly increasing ξ. Numerically, the periodic orbit produced by the bifurcation has the potential to be stable or unstable. Similarly exciting dynamics can be seen in the broader case of ξ strictly increasing.

Although it is true for all of Part 2, the expected biological relation of $g(S)+\mu \geq 0$ does not seem to be important to the analysis we did, although it can provide an important change in dynamics. Comparing the results from Kuang and Beretta (1998) to our results, we see the possible changes that can occur without this relation in mind. Kuang and Beretta (1998) prove that, for their model, a periodic orbit cannot surround a stable interior equilibrium, while in our model, we have strong numerical evidence of an unstable periodic orbit surrounding a stable interior equilibrium. We also note that our analysis holds for a special case seen in Kuang and Beretta (1998), giving a greater understanding of the dynamics in that case.

In Section 12.8, we have conditions for persistence of the host species. Conditions for initial condition dependent and global extinction of the host are given in Sections 12.7.1 and 12.7.2. For the parasite ratio, extinction is found in Section 12.9 and persistence is found in Section 12.5 (more precisely Theorem 12.5.1). For persistence of the *infective hosts*, we combine persistence results of both the host and parasite. Finally, boundedness of the parasite ratio is given in Section 12.6. Between these sections we can understand the dynamics of the system as a whole given a homogeneous incidence function and parameters.

PART 3 -

SIP MODEL: PREDATOR-PREY-INFECTIVE

Chapter 14

INTRODUCTION TO THE 3-D MODEL

Starting with the 2 dimensional prey-parasite endemic model, with homogeneous incidence (considered in Chapter 12), we expand it to a 3 dimensional system by adding a predator to the system, creating a so-called eco-epidemiological model. There is a large literature base on this, using a variety of infection functions, and predation functions, see Chattopadhyay and Arino (1999); Han and Pugliese (2009); Haque *et al.* (2009); Hethcote *et al.* (2004); Xiao and Chen (2002, 2001a,b); Venturino (1994); Bairagi *et al.* (2007); Chattopadhyay *et al.* (2003); Chen and Wen (2016); Ghosh and Li (2016); Arino *et al.* (2004); Khan *et al.* (2016); Yongzhen *et al.* (2011); Mukherjee (2016) and the references therein. Other authors have analyzed similar models from the viewpoint of a food chain/web: Ruan and Freedman (1991); Hsu *et al.* (2016).

Predator prey models which have disease in both prey and predator have been considered by Han *et al.* (2001); Bera *et al.* (2015); Gani and Swift (2013); Hadeler and Freedman (1989); Das and Chattopadhyay (2015). Each of Ghosh and Li (2016); Hethcote *et al.* (2004); Haque *et al.* (2009) use symmetric frequency incidence and logistic growth, where Ghosh and Li (2016); Hethcote *et al.* (2004) include infectives in the growth term, and growth in Haque *et al.* (2009) is from susceptibles only. Like us, Haque *et al.* (2009); Hethcote *et al.* (2004) use mass action to describe predation while Ghosh and Li (2016) uses a Holling Type II predation function. Both Haque *et al.* (2009) and Ghosh and Li (2016) use varational matrices to analyze behavior near the origin. Haque *et al.* (2009) is more focused on bifurcations, while we focus on persistence. In Hethcote *et al.* (2004), a transformation using the population total

($H = S + I$) and the ratio of infectives to the total (I/H) allows a standard analysis of the origin. However, Hethcote *et al.* (2004); Ghosh and Li (2016) do not have host extinction for solutions with positive initial conditions. In Hethcote *et al.* (2004), this is due to the lack of disease related death. Ghosh and Li (2016) include disease death, however, they do not to consider the possibility of host extinction. Outside of the differences mentioned above, our results align well with those of Ghosh and Li (2016); Hethcote *et al.* (2004); Haque *et al.* (2009).

The addition of another species adds a variety of interesting dynamics. We expect to see the following phenomena, depending on the choice of the parameters:

(i) Predator-mediated extinction of the parasite and survival of the prey and predator

(ii) Parasite-mediated extinction of the predator and survival of the prey and parasite

(iii) Parasite-mediated persistence of the predator

(iv) Predator-mediated survival of all three species at high initial predator levels and parasite-mediated extinction of all three species at low initial predator levels

(v) Predator-mediated extinction of the parasite and survival of the prey and predator at high initial predator levels and parasite-mediated extinction of all three species at low initial predator levels

(vi) Parasite-mediated extinction of all three species at all initial predator levels

(vii) Persistence of all three species.

The phenomena (i), (ii), (iii), and (vii) have been observed in other models. Phenomena (iv), (v), and (vi) can be observed because we have chosen a homogeneous

incidence function. They occur when the parasite would drive its host (which is also the prey of the predator) and itself into extinction if there were no predator. In (iv) and (v), we have a bistable situation: in one scenario the predator is initially at a high enough level that it eats enough of infective prey to keep the parasite from driving the host/prey species into extinction, along with itself and the predator. In the other scenario, the predator level is too low to make this happen. The difference in (iv) and (v) is that in (iv), the non-extinction scenario has solutions tending toward a state with all three species continuing to exist together, while in (v) we have solutions tending toward a parasite free state. In (vi), the parameters are such that the predator cannot rescue the ecosystem even if it is a high levels.

Phenomenon (vii) occurs when the parasite cannot drive the host to extinction in the absence of the predator, the parasite can invade the predator-prey subsystem, and the predator can invade the host-parasite subsystem.

Phenomena (i), (ii), (iii) occur if the parasite cannot drive the host/prey species into extinction in the absence of the predator. They can also be observed in models with density-dependent incidence.

Phenomenon (i) occurs if the predator can persist on susceptible prey alone, but also eats infective prey and infective prey is of similar nutritional value as susceptible prey. (Haque *et al.* (2009); Hethcote *et al.* (2004))

Phenomenon (ii) can occur in two distinct ways. First is if infective prey is of less nutritional value than susceptible prey and if a predator that has eaten infective prey is too tired or satiated to kill susceptible prey as well. Second, we have the case where the parasite causes very low levels of prey to be available, so even if infected prey is of similar nutritional value, there will not be enough biomass available for the predator to persist. (Haque *et al.* (2009); Hethcote *et al.* (2004))

Phenomenon (iii) occurs if the predator cannot persist on susceptible prey alone. It can survive on infective prey if infective prey is easier to catch and of similar nutritional value as susceptible prey. (Chattopadhyay and Arino (1999); Hethcote *et al.* (2004); Xiao and Chen (2001a, 2002, 2001b))

We also have both possible two dimensional dynamics if a species has an initial population of zero: prey and predator coexisting in absence of the parasite, and host and parasite coexisting in absence of the predator.

Chapter 15

THE 3-D MODEL

15.1 Initial Model

Recall S and I denote the biomass of the prey that are susceptible to and infected by the disease respectively. Let P denote the biomass of the predator population. Predation is modeled by mass action kinetics.

The disease dynamics between host and disease are as in Part 2. Otherwise, we assume that the parasite is not transmitted between predators. These are the model equations,

$$\begin{aligned} S' =& Sg(S) - \sigma f(S,I) - \kappa_1 SP, \\ I' =& \sigma f(S,I) - \kappa_2 IP - \mu I, \\ P' =& \gamma_1 SP + \gamma_2 IP - \nu P. \end{aligned} \tag{15.1.1}$$

The parameters have the following meaning:

$g(S)$ denotes the growth function for the susceptible population.

$\sigma f(S,I)$ denotes the *disease incidence*, i.e., the number of new infections per unit of time. f is called the *incidence function* and σ the *incidence coefficient*.

$\mu > 0$ per unit biomass capita mortality rate of infected prey

$\nu > 0$ per unit biomass natural mortality rate of predators

$\kappa_1 \geq 0$ rate at which one unit of susceptible prey is killed by one unit of predator.

$\kappa_2 > 0$ rate at which one unit of infected prey is killed by one unit of predator.

$\gamma_1 \geq 0$ rate of per unit predator biomass increase by killing (and eating) one unit of susceptible prey.

$\gamma_2 \in \mathbb{R}$ rate of per unit predator biomass change by killing (and eating) one unit of infected prey.

We assume g is locally Lipschitz, continuous, strictly decreasing, and that $g(0) > 0$. This assumption implicitly assumes that the infectives are too weak to compete for vital resources, or reproduce.

For f, we assume that it is locally Lipschitz, continuous, increasing and concave in both variables, $f(0, I) = 0$ for all $I \geq 0$, $f(S, 0) = 0$ for all $S \geq 0$, and homogeneous, i.e.

$$f(\alpha S, \alpha I) = \alpha f(S, I), \qquad \alpha, I, S \geq 0,$$

and we normalize f such that $f(1, 1) = 1$. When f is homogeneous, the *incidence coefficient*, σ, has the units of day^{-1}.

All constants are non-negative with the possible exception of γ_2 which is positive if eating infected prey has a positive effect on a predator and negative if eating infected prey has a negative effect.

We assume $\kappa_2 > \kappa_1$, i.e., infective prey are more easily caught than susceptible prey by the predator. Additionally, we assume $\kappa_1 + \kappa_2 > 0$, and $\kappa_1 = 0$ if and only if $\gamma_1 = 0$. Additionally, we assume if $\gamma_1 = 0$, then $\gamma_2 > 0$, so the predator has food available that provides it sustenance.

As before, we will perform some preliminary analysis in this form of the model, then introduce the ratio term to analyze behavior when S or I are zero.

Theorem 15.1.1. *If $S(0) \geq 0, I(0) \geq 0$ and $P(0) \geq 0$, then there exists a unique solution to System (15.1.1), such that $S(t) \geq 0, I(t) \geq 0$ and $P(t) \geq 0$ for all $t \geq 0$.*

Proof. We will use Theorem A.4 from Thieme (2003). Define $x(t) = (S(t), I(t), P(t))$, and the vector field via system 15.1.1 such that $x' = F(x)$. We assumed that all functions in our vector field are locally Lipschitz, so we just need to show $F_j(x) \geq 0$ when $x_j = 0$. For F_1, we have $F_1(x) = x_1 g(x_1) - \sigma f(x_1, x_2) - \kappa_2 x_1 x_3$, which is 0 when $x_1 = 0$. With our formulation, one can easily see that this result will hold for each F_j. □

Notice that γ_i/κ_i, $i = 1, 2$, are the nutritional values of susceptible and infective prey, respectively. We define

$$\alpha = \max_{i=1}^{2} \frac{\gamma_i}{\kappa_i} \tag{15.1.2}$$

as the maximum nutritional value, where $\gamma_i/\kappa_i := 0$ if $\gamma_i = 0$ (even if $\kappa_i = 0$ as well). Since we assume that $\gamma_1 = 0$ implies $\gamma_2 > 0$ and $\kappa_2 > 0$, we have that $\alpha > 0$.

Theorem 15.1.2. *For all non-negative solutions S, I, and P, we have $S(t) \leq \max\{K, S(0)\}$ for all $t \geq 0$ and $S^\infty \leq K$. For a weighted total population size $N(t) = S(t) + I(t) + (1/\alpha)P(t)$,*

$$N(t) \leq \max\{K, S(0)\}\frac{g(0)+\beta}{\beta} + H(0)e^{-\mu t}, \qquad \beta = \min\{\mu, \nu\}$$

and

$$\begin{aligned} N^\infty \leq & \sup\{S(g(S)+\beta)/\beta; S \in [0, S^\infty]\} \leq \sup\{S(g(S)+\beta)/\beta; S \in [0, K]\} \\ \leq & S^\infty \frac{g(0)+\beta}{\beta} \leq K\frac{g(0)+\beta}{\beta}. \end{aligned}$$

In particular, $P(t) \leq \alpha N(t)$, $I(t) \leq N(t)$, and

$$P^\infty \leq \sup\left\{\frac{\alpha S(g(S)+\beta)}{\beta}; S \in [0, K]\right\} =: P^\odot \leq \alpha K \frac{g(0)+\beta}{\beta}$$

and $I^\infty \leq K\dfrac{g(0)+\beta}{\beta}$, independent of initial conditions.

Proof. Our choice of α gives us $(\gamma_i/\alpha) - \kappa_i \leq 0$. Let $\beta = \min\{\mu, \nu\}$. Set $N = S + I + \alpha^{-1}P$.

$$\begin{aligned} N' &= Sg(S) - \mu I - (\nu/\alpha)P + ((\gamma_1/\alpha) - \kappa_1)SP + ((\gamma_2/\alpha) - \kappa_2)IP \\ &\leq Sg(S) - \mu I - (\nu/\alpha)P \\ &= S(g(S) + \beta) - \mu I - (\nu/\alpha)P - \beta S \\ &\leq S(g(S) + \beta) - \beta(S + I + \alpha^{-1}P) \\ &\leq \sup\{S(g(S) + \beta); S \in [0, S^\infty]\} - \beta N. \end{aligned}$$

From here we follow the same steps as Theorem 9.1.1 to give our result. □

Corollary 15.1.3. *If $S(t) \to 0$, as $t \to \infty$, then $N(t) \to 0$.*

Corollary 15.1.4. *For all non-negative solutions S, and I, let $M(t) = S(t) + I(t)$.*

If the function $S(g(S) + \mu)$ is an increasing function of S for all $S \in (0, K)$, then $M^\infty \leq K$.

Proof. With a slight modification to the calculation in 15.1.2, we have

$$M^\infty \leq \sup\{S(g(S) + \mu)/\mu; S \in [0, S^\infty]\}.$$

Using the hypothesis $S(g(S) + \mu)$ is an increasing function of S for all $S \in (0, K)$, we get

$$\sup\{S(g(S) + \mu)/\mu; S \in [0, K]\} = K\frac{g(K) + \mu}{\mu} = K\frac{0 + \mu}{\mu} = K.$$

Further we have

$$\sup\{S(g(S) + \mu)/\mu; S \in [0, S^\infty]\} \leq \sup\{S(g(S) + \mu)/\mu; S \in [0, K]\} = K,$$

which is our claim. □

This corollary implies that if $S(g(S)+\mu)$ is increasing on $(0,K)$ then the total number of hosts (susceptible and infectives) will be less than the carrying capacity of the species, which we may expect from a biological perspective.

Theorem 15.1.5. *If there is an $\varepsilon > 0$ such that $\limsup_{t\to\infty} S(t) =: S^\infty > \varepsilon$ for all solutions with $S(0) > 0$, then there is some $\delta > 0$ such that $\liminf_{t\to\infty} S(t) =: S_\infty > \delta$ for all solutions with $S(0) > 0$.*

Proof. Using the persistence function $\rho(S, I, P) = S$, we note that once ρ is zero, it will stay zero. Via Theorem 15.1.2 we define $B = [0, K] \times [0, I^\infty] \times [0, P^\infty]$, with I^∞ and P^∞ independent of initial conditions. Now we apply Theorem 4.2 from Thieme (2003), using B, which tell us that uniform weak persistence implies uniform strong persistence. □

Corollary 15.1.6. *If there is an $\varepsilon > 0$ such that $\limsup_{t\to\infty} P(t) =: P^\infty > \varepsilon$ for all solutions with $P(0) > 0$, then there is some $\delta > 0$ such that $\liminf_{t\to\infty} P(t) =: P_\infty > \delta$ for all solutions with $P(0) > 0$.*

Proof. Using the persistence function $\rho(S, I, P) = P$, we can repeat the above proof. □

15.2 The Ratio Model

Following the homogeneous incidence considerations above, we consider the ratio of infectives to susceptibles, $r = \dfrac{I}{S}$, replacing I in our system with r. We define $h(r) = f(1, r)$, and $\xi(r) = \dfrac{1+r}{r}h(r)$. Our assumptions on f imply that h is strictly increasing, concave, and $h(0) = 0$. We additionally assume $h(\infty) := \lim_{r\to\infty} h(r) <$

$\infty, \quad h'(0) < \infty$, and h' is strictly decreasing, so h is strictly concave. Our assumptions on ξ are that it is differentiable and either monotone, strictly decreasing then strictly increasing, or strictly increasing then strictly decreasing.

This gives us a new system of differential equations

$$\begin{aligned} \frac{S'}{S} =& g(S) - \sigma h(r) - \kappa_1 P, \\ \frac{r'}{r} =& \sigma\xi(r) + (\kappa_1 - \kappa_2)P - \mu - g(S), \\ \frac{P'}{P} =& \gamma_1 S + \gamma_2 r S - \nu. \end{aligned} \tag{15.2.1}$$

In addition, it may be worthwhile to keep the equation for $I = rS$,

$$I' = I\Big(\sigma\frac{h(r)}{r} - \kappa_2 P - \mu\Big). \tag{15.2.2}$$

Corollary 15.2.1. *Suppose that there is some $\varepsilon > 0$ such that $S^\infty > \varepsilon$ for all solutions with $S(0) > 0$. Then there is some c such that $\limsup_{t\to\infty} r(t) =: r^\infty < c$ for all solutions with $r(t) \geq 0$.*

Proof. Theorem 15.1.5 will imply that there is some $\delta > 0$ such that $S_\infty > \delta$. Using definition of r and theorem 15.1.2 we see $r = I/S \leq N/S$, which implies $r^\infty \leq N^\infty/S_\infty$. □

Theorem 15.2.2. *If $r(t) \to \infty$ as $t \to \infty$, then $S(t) \to 0, I(t) \to 0$, and $P(t) \to 0$ as $t \to \infty$.*

Proof. $r(t) = \dfrac{I(t)}{S(t)}$ implies $r(t)S(t) = I(t)$, and Theorem 15.1.2 provides us with a bound on I, call it c, which is independent of initial conditions. Thus, we have $r(t)S(t) = I(t) \leq c$, and $r(t) \to \infty$ as $t \to \infty$. Therefore, it must be the case that $S(t) \to 0$ which, by Corollary 15.1.3, implies the result. □

Proposition 15.2.3. *Suppose that for all solutions $r(t)$ with $r(0) \geq 0$ that there is some $c > 0$ such that $\liminf_{t\to\infty} r(t) =: r_\infty < c$. Then there is some $d > 0$ such that $\limsup_{t\to\infty} r(t) =: r^\infty < d$.*

Proof. Corollary A.33 in Thieme (2003) states that if a semiflow has the property $\liminf_{t\to\infty} \|\Phi_t(x)\| < C$ for some C, then there exists some C such that $\limsup_{t\to\infty} \|\Phi_t(x)\| < C$. We note that using the standard Euclidean norm, the semiflow induced by system 15.2.1 will have norm bounded in the limit inferior if and only if r_∞ is bounded, since by Theorem 15.1.2 S and P are bounded. □

Proposition 15.2.4. *If $S(t) \to K$ as $t \to \infty$, then $r(t) \to 0$ and $P(t) \to 0$ as $t \to \infty$.*

Proof. Let $S(t) \to K$ as $t \to \infty$. Then $\liminf_{t\to\infty} S(t) =: S_\infty = K$. We wish to apply Barbalat's Lemma, which requires differentiability of the vector field and uniform continuity of $S'(t)$. Looking at our assumptions of the system, we recall that g, h, and ξ are all differentiable. Now we show start by showing the uniform continuity of $S'(t)$.

Since $S^\infty = K > 0$, Corollary 15.2.1, can be combined with Theorem 15.1.2 to tell us that S, r, and P are bounded, which implies that S', r', and P' are bounded as well, thus S, r, and P are all uniformly continuous. Taking the derivative of S', r', and P' with respect to time, and using the boundedness of S, r, P, S', r', and P', we can see that S'', r'', and P'' will be bounded as well. Therefore S', r', and P' will be uniformly continuous.

Now we can apply Barbalat's Lemma, which implies that $S'(t) \to 0$ as $t \to \infty$. We see

$$0 = \lim_{t\to\infty} S(t)(g(S(t)) - \sigma h(r(t)) - \kappa_1 P(t)) = \lim_{t\to\infty} K(-\sigma h(r(t)) - \kappa_1 P(t)).$$

Thus $0 = \sigma h(\lim_{t\to\infty} r(t)) + \kappa_1 \lim_{t\to\infty} P(t)$. This implies $r(t) \to 0$ and $P(t) \to 0$ as $t \to \infty$. □

Corollary 15.2.5. *If $S(t) \geq K$ for all $t \geq 0$, then $r(t) \to 0$ and $P(t) \to 0$ as $t \to \infty$.*

Proof. Note that for all $S(t) > K$, we have $S'(t) < 0$, and if $S(t) = K$, then $S'(t) \leq 0$. So, if $S(t) \geq K$ for all $t \geq 0$, then $S(t) \to K$ as $t \to \infty$. □

Theorem 15.2.6. *If $\sigma h(\infty) < g(0)$, then there exists some c such that $S^\infty > c$ for all solutions with $S(0) > 0$.*

Proof. We can assume that $r(0) > 0$ and $S(0) > 0$. We can choose $S^\flat$ such that $\sigma h(\infty) < g(S)$ for all $S \leq S^\flat$. Now, we choose some $\varepsilon > 0$ such that $\sigma h(\infty) < g(S^\flat) - 2\varepsilon$. Let $\gamma = \max\{\gamma_1, \gamma_2\} \geq 0$. We consider when $\gamma = 0$, and $\gamma > 0$.

If $\gamma > 0$, we recall the definition of $N(t)$, and β from Theorem 15.1.2 we perform a time shift such that $N(t) \leq N^\infty + \dfrac{\nu}{3\gamma}$ for all $t \geq 0$. Note that this implies $S(t)+I(t) \leq N^\infty + \dfrac{\nu}{3\gamma}$ for all $t \geq 0$. Choose $S^\sharp$ such that

$$0 < S^\sharp < \min\left\{\frac{\beta\nu}{3\gamma(g(0)+\beta)}, S^\flat\right\}.$$

We will suppose toward contradiction that $S^\infty < S^\sharp$.

Then we see from the P' equation which has S and I that

$$\begin{aligned} \frac{P'}{P} = \gamma_1 S + \gamma_2 I - \nu &\leq \gamma(S(t) + I(t)) - \nu \\ \leq \gamma N^\infty + \frac{\nu}{3} - \nu &\leq \gamma S^\infty \frac{g(0)+\beta}{\beta} - \frac{2\nu}{3} < -\frac{\nu}{3}, \end{aligned}$$

which implies P will decline exponentially.

After another time shift, we have $\kappa_1 P(t) < \varepsilon$ for all $t \geq 0$. Then we see

$$\frac{S'}{S} = g(S) - \sigma h(r) - \kappa_1 P > g(S^\sharp) - \sigma h(\infty) + 2\varepsilon - \varepsilon > \varepsilon$$

which implies that S increases exponentially, a contradiction.

Therefore we always have $S^\infty > S^\sharp$. By Theorem 15.1.5, we have our result.

If $\gamma = 0$, then we omit the consideration of the first term from $S^\sharp$. We see $\dfrac{P'}{P} \geq -\nu$, so $P(t) \to 0$, so we can reuse the calculations concerning $\dfrac{S'}{S}$ to show $S^\infty > S^\sharp$, and apply Theorem 15.1.5. $\square$

Theorem 15.2.7. *If $\sigma h(\infty) < \mu + g(0)$, then there exists some c such that $r^\infty < c$ for all solutions with $r(0) > 0$.*

Proof. By Theorem 15.2.6 combined with Theorem 15.2.1, we have our result for $\sigma h(\infty) < g(0)$. Thus, we only need to consider when $\sigma h(\infty) \geq g(0)$. We can assume that $r(0) > 0$ and $S(0) > 0$. Via Proposition 15.2.3 it is sufficient to prove $r_\infty < d$ for some d. Thus, we assume that $r_\infty > d$ for all $d \in \mathbb{R}$, and arrive at a contradiction.

Choose some $S^\flat > 0$ such that

$$\xi(\infty) = h(\infty) < g(S) + \mu, \qquad S \leq S^\flat.$$

Since $g(S^\flat) < g(0) \leq h(\infty)$, we can choose some $r^\flat > 0$ and $\delta > 0$

$$\xi(r) < g(S^\flat) + \mu - \delta, \quad g(S^\flat) < h(r) - \delta, \qquad r \geq r^\flat.$$

Suppose that $r_\infty > r^\flat$. After a shift in time, $r(t) > r^\flat$ for all $t \geq 0$. Then

$$g(S^\flat) - h(r(t)) - \kappa_1 P(t) < g(S^\flat) - h(r(t)) < -\delta, \qquad t \geq 0.$$

Since $S' = S(g(S) - h(r) - \kappa_1 P)$ and g is strictly decreasing, this implies that $S^\infty < S^\flat$. After another shift in time, $S(t) < S^\flat$ for all $t \geq 0$. Then

$$r' = (1+r)h(r) - r(g(S) + \mu) + r(\kappa_1 - \kappa_2)P < r(\xi(r) - (g(S^\flat) + \mu)) < r(-\delta).$$

So r decreases exponentially. This contradiction shows that $r_\infty < r^\flat$, a contradiction to our initial assumption. $\square$

Corollary 15.2.8. *If $\sigma h(\infty) < \mu + g(0)$, the state $(0, \infty, 0)$ is a source.*

Chapter 16

2-DIMENSIONAL SUBSYSTEMS

16.1 Host-Parasite Subsystem

This is fully considered in Chapter 12.

16.2 The Prey-Predator Subsystem

$$\begin{aligned} \frac{S'}{S} &= g(S) - \kappa_1 P, \\ \frac{P'}{P} &= \gamma_1 S - \nu. \end{aligned} \tag{16.2.1}$$

Theorem 16.2.1. *(a) Let $\gamma_1 K \leq \nu$ (i.e. $g(\nu/\gamma_1) \leq 0$). Then $P(t) \to 0$ as $t \to \infty$.*

(b) Let $g(\nu/\gamma_1) > 0$. Then, if $S(0) > 0$ and $P(0) > 0$,

$$S(t) \to \frac{\nu}{\gamma_1} =: S^\diamond, \qquad P(t) \to \frac{g(\nu/\gamma_1)}{\kappa_1} =: P^\diamond.$$

Proof. (a) We start by looking into the equilibrium. $S' = 0 \implies$ either $S^\diamond = 0$ or $g(S^\diamond) = \kappa_1 P^\diamond$, and $P' = 0 \implies$ either $P = 0$ or $S^\diamond = \frac{\nu}{\gamma_1}$. So we see there are three possible fixed points, $(0,0), (K,0)$, (recalling that $g(K) = 0$) and $\left(\frac{\nu}{\gamma_1}, \frac{g\left(\nu/\gamma_1\right)}{\kappa_1}\right)$. We notice the condition for existence of the third equilibrium and its positivity is $\frac{g\left(\nu/\gamma_1\right)}{\kappa_1} > 0 \implies g\left(\frac{\nu}{\gamma_1}\right) > 0 \implies \frac{\nu}{\gamma_1} < K$. Thus for part a, our conditions leave us with only (0,0) and $(K,0)$. The Poincar-Bendixson trichotomy tells us that the omega limit set contains at most collection of fixed points, periodic orbits, and orbits connecting fixed points (possibly to themselves). If there were periodic orbits then

there must be an interior equilibrium somewhere inside the periodic orbit. Therefore we have the P value of both fixed points is $P = 0$, so all remaining possibilities result in $P \to 0$, since solutions are bounded. □

Proof. (b) We now have all three equilibria! Now need only to show that there are no periodic orbits, the interior fixed point is stable, and that no solutions from the interior converge to a boundary equilibrium.

We turn to Dulac, using $\phi = \dfrac{1}{SP}$ and we get that

$$\rho f_1(S,P) = \frac{g(S)}{P} - \kappa_1$$

$$\rho f_2(S,P) = \gamma_1 - \frac{\nu}{S}$$

Thus we have that $\bigtriangledown(\rho f) = \dfrac{g'(S)}{P} < 0$. Therefore there are no periodic orbits in $(0,\infty)^2$. For stability, we look at the Jacobian matrix and we see that

$$J(S^*,P^*) = \begin{pmatrix} g(S^*) + S^* g'(S^*) - \kappa_1 P^* & -\kappa_1 S^* \\ \gamma_1 P^* & \gamma_1 S^* - \nu \end{pmatrix}$$

At (0,0) we have

$$J(0,0) = \begin{pmatrix} g(0) & 0 \\ 0 & -\nu \end{pmatrix}$$

Since the matrix is upper triangular, we get $\lambda_1 = g(0) > 0$ and $\lambda_2 = -\nu < 0$ as our eigenvalues. Since it is a diagonal matrix as well, we can see that the stable eigenvector is for λ_2, and it is $(0,1)$, thus no solutions from $(0,\infty)^2$ are removed.

At $(K,0)$ we get

$$J(K,0) = \begin{pmatrix} Kg'(K) & -\kappa_1 K \\ 0 & \gamma_1 K - \nu \end{pmatrix}$$

Again we can see that $\lambda_1 = Kg'(K) < 0$ and $\lambda_2 = \gamma_1 K - \nu$, which is positive since we have $g(\nu/\gamma_1) > 0$. Similarly we see that the stable eigenvector is $(0,1)$, thus we have the solutions in $(0,\infty)^2$ are untouched yet again.

At $(S^\diamond, P^\diamond)$ we have

$$J(S^\diamond, P^\diamond) = \begin{pmatrix} S^\diamond g'(S^\diamond) & -\kappa_1 S^\diamond \\ \gamma_1 P^\diamond & 0 \end{pmatrix}$$

Here we see that the trace is $Tr(J) = S^\diamond g(S^\diamond) < 0$ and that the Jacobian is $Det(J) = (\gamma_1 P^\diamond)(\kappa_1 S^\diamond) > 0$. Therefore we have both eigenvalues negative, and thus this fixed point is globally stable for $S(0) > 0, P(0) > 0$. □

Here we see that comparing the incoming predator biomass to the exiting predator biomass will determine if the predator can invade. If incoming predator biomass exceeds outgoing predator biomass, i.e. $\gamma_1 K > \nu$, we have that the predator can invade and persist. If not, i.e. $\gamma_1 K \leq \nu$, then the predator eventually will die, and the equilibrium, $(K, 0)$ is globally stable.

Remark 16.2.2. It may be interesting to compare $P^\diamond = \dfrac{g(S^\diamond)}{\kappa_1}$ to the large-time estimate $P^\odot$ that we have obtained in Theorem 15.1.2. Since the existence of $P^\diamond$ requires $\gamma_1 > 0$ and $S^\diamond = \nu/\gamma_1 < K$, we have $\alpha \geq \gamma_1/\kappa_1$ and $\beta \leq \nu$ and

$$P^\odot \geq \frac{\alpha S^\diamond (g(S^\diamond) + \beta)}{\beta} \geq \frac{\alpha(\nu/\gamma_1)(g(S^\diamond) + \nu)}{\nu} \geq \frac{1}{\kappa_1}(g(S^\diamond) + \nu) = P^\diamond + \frac{\nu}{\kappa_1}.$$

Chapter 17

DISEASE EXTINCTION

Theorem 17.1.1. *Let* $\sigma \sup_r \xi(r) \leq \mu$. *Then the disease goes extinct for all solutions* $r(t)$ *with* $r \geq 0$. *Additionally, if* $\nu < \gamma_1 K$ *solutions with* $S(0) > 0$ *and* $P(0) > 0$ *will converge to* $(S^\diamond, 0, P^\diamond)$, *and if* $\nu > \gamma_1 K$, *solutions with* $S(0) > 0$ *and* $P(0) > 0$ *will converge to* $(K, 0, 0)$.

Proof. By Corollary 15.2.5, and a time shift as needed, we only need to consider when $S(t) \leq K$. We can immediately see

$$\frac{r'}{r} = \sigma\xi(r) - \mu - g(S) + (\kappa_1 - \kappa_2)P \leq -g(S) + (\kappa_1 - \kappa_2)P \leq 0.$$

So r is decreasing, bounded above by $r(0)$, and bounded below by 0.

We can reuse the portion of the proof of Proposition 15.2.4 in which we show boundedness of r implies uniform continuity of S', r', and P'. Thus we can use Barbalat's Lemma here as well.

Suppose that $r(t)$ has a strictly positive limit, call it c. By Barbalat's Lemma, we have $r'(t) \to 0$ as $t \to \infty$. This is only possible if $P(t) \to 0$ and $S(t) \to K$ as $t \to \infty$. Now we apply Proposition 15.2.4, which implies $r(t) \to 0$ as $t \to \infty$, a contradiction to $r(t)$ having a strictly positive limit. Thus it is the case that $r(t) \to 0$ as $t \to \infty$.

Using this, can reduce our 3 dimensional system to an asymptotically autonomous 2 dimensional system. More precisely we have our initial system

$$S' = S(g(S) - \sigma h(r(t)) - \kappa_1 P),$$
$$P' = P(\gamma_1 S + \gamma_2 S r(t) - \nu),$$

which, as $t \to \infty$, has the limit system we saw in Section 16.2,

$$S' = S(g(S) - \kappa_1 P),$$
$$P' = P(\gamma_1 S - \nu). \qquad \text{(16.2.1 revisited)}$$

We will use Theorem 2.3 from Castillo-Chavez and Thieme (1995) to imply our result. Theorem 16.2.1 implies that if $\nu > \gamma_1 K$, then, in System 16.2.1, the equilibrium $(K, 0)$ is locally asymptotically stable and that the basin of attraction for $(K, 0)$ is $(0, \infty) \times [0, \infty)$, which in System 15.2.1 is the plane $(0, \infty) \times \{0\} \times [0, \infty)$. We see that for any solution from the full system with $S(0) > 0, r(0) \geq 0$, and $P(0) \geq 0$ will have its $\omega-$limit set be a subset of $[0, \infty) \times \{0\} \times [0, \infty)$, so showing that the $\omega-$limit set is a subset of $(0, \infty) \times \{0\} \times [0, \infty)$ will complete the proof for $\nu > \gamma_1 K$.

Suppose $\nu > \gamma_1 K$. Suppose that $S(t) < \varepsilon$ for all $t \geq 0$, where ε is chosen such that $\gamma_1 \varepsilon - \nu < 0$ for all $t \geq 0$. Then, since $P' = P(\gamma_1 S + \gamma_2 S r - \nu)$, and $r(t) \to 0$ as $t \to \infty$, we can perform a time shift such that $\gamma_1 S + \gamma_2 S r - \nu < 0$ for all $t \geq 0$. Then, we will have that P will decrease exponentially. Now we perform another time shift such that $\sigma h(r(t)) + \kappa_1 P(t) < g(\varepsilon)$. Now we will have that $S' = S(g(S) - \sigma h(r(t)) + \kappa_1 P(t))$, implying the exponential growth of S, a contradiction. Therefore we have that $S^\infty > \varepsilon$, which by Theorem 15.1.5 implies $S_\infty > \delta > 0$. This implies that the $\omega-$limit set of solutions from our initial system is a subset of $[\delta, \infty) \times \{0\} \times [0, \infty)$, which implies our result.

Now we consider when $\nu < \gamma_1 K$. Theorem 16.2.1 implies that if $\nu < \gamma_1 K$, then, in System 16.2.1, the equilibrium $(S^\diamond, P^\diamond)$ is locally asymptotically stable and that the basin of attraction for $(S^\diamond, P^\diamond)$ is $(0, \infty) \times (0, \infty)$, which in System 15.2.1 is the plane $(0, \infty) \times \{0\} \times (0, \infty)$. We see that for any solution from the full system with $S(0) > 0, r(0) \geq 0$, and $P(0) > 0$ will have its $\omega-$limit set be a subset of

$[0,\infty)\times\{0\}\times[0,\infty)$, so showing that the $\omega-$limit set is a subset of $(0,\infty)\times\{0\}\times(0,\infty)$ will complete the proof for $\nu < \gamma_1 K$.

We can still use the above to show $S_\infty > \delta$. By the fluctuation method, there is a sequence t_k, such that $t_k \to \infty$ as $k \to \infty$ with the property $S'(t_k) \to 0$ and $S(t_k) \to S_\infty$. This show us

$$\lim_{k\to\infty} S'(t_k) = \lim_{k\to\infty} \big[S(t_k)(g(S(t_k)) - \sigma h(r(t_k)) - \kappa_1 P(t_k)\big]$$
$$0 = S_\infty\Big(g(S_\infty) - \kappa_1 \lim_{k\to\infty} P(t_k)\Big),$$

which implies $P^\infty \geq P(t_k) = \dfrac{g(S_\infty)}{\kappa_1}$. If we have $S_\infty < K - \alpha$ for some $\alpha > 0$, then Corollary 15.1.6 implies $P_\infty \geq \delta_2 > 0$, so the $\omega-$limit set of solutions from our initial system is a subset of $[\delta,\infty)\times\{0\}\times[\delta_2,\infty)$, implying the result.

Choose α such that $\gamma_1(K-\alpha) > \nu$, and suppose $S_\infty > K - \alpha$. Observe that since $P(0) > 0$, $P(t) > 0$ for all $t \geq 0$. Since $r(t) \to 0$ as $t \to \infty$, we can perform a time shift such that $S(t) > K - \alpha$ and $\gamma_1(K-\varepsilon) + \gamma_2(K-\varepsilon)r(t) > \nu$ for all $t \geq 0$. We see that this implies $\dfrac{P'}{P} = \gamma_1 S + \gamma_2 S r - \nu > 0$ for all $t \geq 0$, implying exponential growth of P to infinity, a contradiction. □

Corollary 17.1.2. *Suppose ξ is strictly decreasing or constant, and $\sigma h'(0) \leq \mu$. Then the disease goes extinct for all solutions $r(t)$ with $r \geq 0$. Additionally, if $\nu < \gamma_1 K$ solutions with $S(0) > 0$, and $P(0) > 0$ will converge to $(S^\diamond, 0, P^\diamond)$, and if $\nu > \gamma_1 K$, solutions with $S(0) > 0$, and $P(0) > 0$ will converge to $(K, 0, 0)$.*

Chapter 18

LOCAL STABILITY OF BOUNDARY EQUILIBRIUM

We record the Jacobian matrix here for future use.

$$J = \begin{pmatrix} \alpha_1 & -S\sigma h'(r) & -\kappa_1 S \\ -rg'(S) & \alpha_2 & r(\kappa_1 - \kappa_2) \\ \gamma_1 P + \gamma_2 rP & P\gamma_2 S & \gamma_1 S + Sr\gamma_2 - \nu \end{pmatrix},$$

where $\alpha_1 = g(S) + Sg'(S) - \sigma h(r) - \kappa_1 P$, and

$\alpha_2 = \sigma r \xi'(r^*) + \sigma \xi(r) + (\kappa_1 - \kappa_2)P - \mu - g(S)$.

We note that the following local stability results will hold for any ξ considered so far, not just ξ strictly decreasing.

18.1 (0,0,0)

This equilibrium exists for all parameter values.

$$J = \begin{pmatrix} g(0) & 0 & 0 \\ 0 & \sigma h'(0) - \mu - g(0) & 0 \\ 0 & 0 & -\nu \end{pmatrix}.$$

We immediately have the eigenvalues are $\lambda_1 = g(0) > 0$, $\lambda_2 = \sigma h'(0) - \mu - g(0)$ and $\lambda_3 = -\nu < 0$. Thus the origin is a saddle so long as $\sigma h'(0) - \mu - g(0) \neq 0$.

18.2 (K,0,0)

This equilibrium exists for all parameter values.

$$J = \begin{pmatrix} Kg'(K) & -K\sigma h'(0) & -\kappa_1 K \\ 0 & \sigma h'(0) - \mu & 0 \\ 0 & 0 & \gamma_1 K - \nu \end{pmatrix},$$

The eigenvalues are

$$\lambda_1 = Kg'(K) < 0,$$

$$\lambda_2 = \sigma h'(0) - \mu,$$

$$\lambda_3 = \gamma_1 K - \nu.$$

The conditions for local stability in the 3-D system are the same as being stable in both of the 2 dimensional subsystems, as can be seen via the eigenvalues. Thus we have that this point is either a sink if both $\sigma h'(0) - \mu < 0$ and $\gamma_1 K - \nu < 0$, or a saddle otherwise so long as $\sigma h'(0) - \mu \neq 0$ and $\gamma_1 K - \nu \neq 0$. We take note that the eigenvectors of this equilibrium are two vectors of the form $(a, b, 0)$, which correspond to the eigenvectors from the $S - r$ subsystem, and a vector of the form $(c, 0, d)$. Thus there are no eigenvectors which point into the interior, so $(K, 0, 0)$ cannot take solutions from the interior if it is a saddle point.

18.3 (0,r°,0)

If ξ is decreasing, then an 'equilibrium' of the form $(0, r^\circ, 0)$ exists if and only if

$$\frac{\mu + g(0)}{h'(0)} < \sigma.$$

We say 'equilibrium' because the infinite extinction state is not a proper equilibrium.

18.3.1 $r^\circ < \infty$

For ξ decreasing, this equilibrium exists if and only if

$$\frac{\mu + g(0)}{h'(0)} < \sigma < \frac{\mu + g(0)}{h(\infty)}.$$

This can be a line of equilibrium if ξ is constant on an interval. If ξ is constant for all r, then this equilibrium exists only if $2\sigma = g(0) + \mu$. In this case, we have that the entire S-axis becomes a line of equilibiria.

The Jacobian matrix at this point is:

$$J = \begin{pmatrix} g(0) - \sigma h(r^\circ) & 0 & 0 \\ -r^\circ g'(0) & \sigma r^\circ \xi'(r^\circ) & r^\circ(\kappa_1 - \kappa_2) \\ 0 & 0 & -\nu \end{pmatrix}.$$

We have $\lambda_1 = g(0) - \sigma h(r^\circ), \lambda_2 = \sigma r^\circ \xi'(r^\circ)$, and $\lambda_3 = -\nu < 0$. Thus, we see that the sign of $g(0) - \sigma h(r^\circ)$ and $\xi'(r^\circ)$ determine the local stability. Recall that Theorem 12.12.6 shows us that if r° is unique, our current conditions imply $g(0)/\mu < r^\circ$, so $h(g(0)/\mu) \leq h(r^\circ)$, since h is increasing. Therefore, $\dfrac{g(0)}{h(g(0)/\mu)} < \sigma \implies g(0) < \sigma h(g(0)/\mu) \leq \sigma h(r^\circ)$. More precisely, we have local asymptotic stability of the point if

$$\frac{g(0)}{h(g(0)/\mu)} < \sigma \implies g(0) < \sigma h(r^\circ)$$

and $\xi'(r^\circ) < 0$, and the point is a saddle otherwise, as long as $\sigma \neq \dfrac{g(0)}{h(g(0)/\mu)}$ and $\xi'(r^\circ) \neq 0$. Additionally, recall that we may have a line of equilibria in the case of $\xi'(r^\circ) = 0$.

Corollary 18.3.1. *If $\xi'(r) < 0$ for all $r \geq 0$, the equilibrium (0,r°,0) exists and is locally asymptotically stable if and only if* $\dfrac{g(0)}{h(g(0)/\mu)} < \sigma < \dfrac{\mu + g(0)}{h(\infty)}$.

Proof. Suppose $\xi'(r) < 0$ for all $r \geq 0$. Then Lemma 12.12.1 can be applied, so $\frac{\mu + g(0)}{h'(0)} < \frac{g(0)}{h(g(0)/\mu)}$. Thus, the conditions for existence, $\frac{\mu + g(0)}{h'(0)} < \sigma < \frac{\mu + g(0)}{h(\infty)}$, and local asymptotic stability, $\frac{g(0)}{h(g(0)/\mu)} < \sigma$, can be combined into $\frac{g(0)}{h(g(0)/\mu)} < \sigma < \frac{\mu + g(0)}{h(\infty)}$. □

Further we note that the eigenvectors of the Jacobian matrix are of the form $(a, b, 0), (0, c, 0)$, and $(0, d, e)$. Thus there are no eigenvectors which point into the interior, so $(0, r^\circ, 0)$ cannot take solutions from the interior if it is a saddle point.

18.3.2 $r^\circ = \infty$

Here we consider the state in which there are no susceptible prey, or predators, and the ratio of infectives to susceptibles is infinite.

Theorem 18.3.2. *Suppose $\sigma h(\infty) \geq g(0) + \mu$. The state $(S, r, P) = (0, \infty, 0)$ is locally asymptotically stable. More precisely, there exists positive S_0, r_0 and P_0, with $I_0 = r_0 S_0$, such that the set $\{Sr \leq S_0 r_0\} \times \{r \geq r_0\} \times \{P \leq P_0\}$ is forward invariant for all solutions in the set. This implies that solutions in the set have the property $r(t) \to \infty$, $S(t) \to 0$, $I(t) \to 0$, and $P(t) \to 0$ as $t \to \infty$.*

If, in addition, $\sigma h(\infty) > g(0) + \mu$ and ξ is decreasing, or ξ is strictly decreasing, then there is no finite extinction equilibrium.

Proof. Choose positive I_0, S_0, P_0, r_0, with $I_0 = S_0 r_0$, such that

$$\begin{aligned} 0 &> g(0) + \mu - \sigma h(r_0), \\ 0 &> \frac{\sigma h(r_0)}{r_0} - \mu, \\ -(\kappa_1 - \kappa_2) P_0 &< \sigma h(r_0) - g(0) - \mu \\ 0 &> \gamma_1 S_0 + \gamma_2 r_0 S_0 - \nu. \end{aligned}$$

This means that we choose r_0 sufficiently large and $S_0 > 0$ and $P_0 > 0$ sufficiently small. Notice that we have $\dfrac{h(r)}{r}$ is decreasing to zero, thus the second condition will be satisfied for all r greater than some appropriately large r_0. Consider the set $\{Sr \leq S_0 r_0\} \times \{r \geq r_0\} \times \{P \leq P_0\}$. Recall $\xi(r) = \dfrac{1+r}{r} h(r) > h(r)$ for all r. On this set, by (15.2.1) and (15.2.2), we see that

$$\begin{aligned}
\frac{S'}{S} &= g(S) - \sigma h(r) - \kappa_1 P < g(0) - \sigma h(r_0) < 0, \\
\frac{I'}{I} &= \sigma \frac{h(r)}{r} - \kappa_2 P_0 - \mu < \sigma \frac{h(r_0)}{r_0} - \mu < 0, \\
\frac{P'}{P} &= \gamma_1 S + \gamma_2 I - \nu \leq \gamma_1 S_0 + \gamma_2 S_0 r_0 - \nu < 0, \\
\frac{r'}{r} &= \sigma \xi(r) - \mu + (\kappa_1 - \kappa_2) P - g(S) > \sigma h(r_0) - \mu + (\kappa_1 - \kappa_2) P_0 - g(0) > 0.
\end{aligned}$$

This implies that for all solutions in this set, we have the S, I, and P components have negative derivatives which are bounded away from zero, while the r components have a positive derivative which is also bounded from zero. Thus, this set is forward invariant, and solutions in in have the property $r(t) \to \infty$, $S(t) \to 0$, $I(t) \to 0$, $P(t) \to 0$ as $t \to \infty$.

If we have our additional assumptions, Section 12.12 will imply the results. □

The stability condition presented above is sharp, which we see via Corollary 15.2.8.

18.4 ($S^\diamond$,0,$P^\diamond$)

Theorem 18.4.1. *If $\gamma_1 K > \nu$, the equilibrium $(S^\diamond, 0, P^\diamond)$ exists. Recall*

$$S^\diamond = \frac{\nu}{\gamma_1}, \text{ and } P^\diamond = \frac{g(\nu/\gamma_1)}{\kappa_1} = \frac{g(S^\diamond)}{\kappa_1}.$$

If $\sigma h'(0) < \kappa_2 P^\diamond + \mu$ in addition, the equilibrium $(S^\diamond, 0, P^\diamond)$ is locally asymptotically stable.

If instead $\sigma h'(0) > \kappa_2 P^\diamond + \mu$, the equilibrium is a saddle point.

Proof. We notice that $\gamma_1 K > \nu$ implies the existence of $(S^\diamond, 0, P^\diamond)$. We consider the Jacobian matrix, and using the identity $\xi(0) = h'(0)$, our evaluation at $(S^\diamond, 0, P^\diamond)$ yields

$$J = \begin{pmatrix} S^\diamond g'(S^\diamond) & -S^\diamond \sigma h'(0) & -\kappa_1 S^\diamond \\ 0 & \sigma h'(0) - \kappa_2 P^\diamond - \mu & 0 \\ \gamma_1 P^\diamond & P^\diamond \gamma_2 S^\diamond & 0 \end{pmatrix}.$$

For ease we define $\delta = \sigma h'(0) - \kappa_2 P^\diamond - \mu$.

Considering $J - \lambda I$ and expanding along the middle row, we have that the characteristic polynomial is

$$(\delta - \lambda)[\lambda^2 - S^\diamond g'(S^\diamond)\lambda + \gamma_1 P^\diamond \kappa_1 S^\diamond].$$

This form gives us an obvious eigenvalue of $\lambda_1 = \delta$. We observe that the characteristic equation for the point $(S^\diamond, P^\diamond)$ in section 16.2 is the expression we have in brackets above. Thus we can conclude that the remaining two eigenvalues have negative real part, therefore the point is locally asymptotically stable if $\delta < 0$.

Similarly we see that this point is a saddle point if $\delta > 0$. In this case, the $S - P$ plane is the stable manifold and the unstable manifold is one dimensional and points into $(0, \infty)^3$. □

We recall that in the two dimensional host-parasite subsystem we have that the disease can invade if $\sigma h'(0) > \mu$. This means if the growth rate of the disease when the ratio is zero is greater than the death rate of infective individuals, the infection cannot invade the host species. Similarly here, with $\sigma h'(0) > \kappa_2 P^\diamond + \mu$, we see that if the disease growth rate at zero is greater than the total death rate, (in this case it is natural death from the infection plus death induced by the predator) then the disease cannot invade.

Further, we observe that $S^\diamond = \dfrac{\nu}{\gamma_1}$ and $P^\diamond = \dfrac{g(\nu/\gamma_1)}{\kappa_1}$ are independent of the term κ_2, so this parameter can be changed (without changing the predator prey dynamics) to allow or restrict the invasion of the parasite into the system. Notably, this allows the presence of the predator to prevent the parasite from invading the ecosystem, keeping the susceptible prey healthy, even in the case of the disease being strong enough to eliminate the host population if there were no predator. A concrete example can be found in Theorems 12.12.6 and 12.12.7 when ξ is decreasing, and $\sigma \geq \dfrac{g(0)}{h(g(0)/\mu)}$. Thus, if $\sigma h'(0) < \kappa_2 P^\diamond + \mu$, we observe the local case of the predator keeping the parasite from invading, i.e. predator-mediated parasite extinction.

In Haque *et al.* (2009), they propose a Hopf bifurcation of this equilibrium, however our analysis indicates that this cannot occur.

18.5 (S*,r*,0)

Theorem 18.5.1. *If*

$$\frac{\mu}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)},$$

$(S^*, r^*, 0)$ *exists. Recall that* S^* *and* r^* *satisfy*

$$g(S^*) = r^*\mu = h(r^*).$$

Two of the eigenvalues of the Jacobian matrix at this point are the eigenvalues of the Jacobian matrix of the point (S^*, r^*) *in the two dimensional plane. The third eigenvalue is* $S^*(\gamma_1 + \gamma_2 r^*) - \nu$.

Proof. The existence of (S^*, r^*) in the two dimensional case implies the existence of $(S^*, r^*, 0)$ in three dimensions.

Evaluating the Jacobian matrix at (S^*, r^*, P^*) yields

$$= \begin{pmatrix} S^* g'(S^*) & -S^* \sigma h'(r^*) & -\kappa_1 S^* \\ -r^* g'(S^*) & r^* \sigma \xi'(r^*) & r^*(\kappa_1 - \kappa_2) \\ 0 & 0 & \gamma_1 S^* + \gamma_2 S^* r^* - \nu \end{pmatrix}.$$

For ease we define $\psi = \gamma_1 S^* + \gamma_2 S^* r^* - \nu$.

Considering $J - \lambda I$ and expanding along the last row, we have that the characteristic polynomial is

$$(\psi - \lambda)[\lambda^2 - (S^* g'(S^*) + r^* \sigma \xi'(r^*))\lambda + S^* g'(S^*) r^* \sigma \xi'(r^*) - S^* \sigma h'(r^*) r^* g'(S^*)].$$

This form gives us an obvious eigenvalue of $\lambda_3 = \psi$. We observe that the characteristic equation for the point (S^*, r^*), which can be found in Section 12.4.1, is the expression we have in brackets above, thus we recover the eigenvalues from the two dimensional system here. □

Corollary 18.5.2. *Suppose ξ is decreasing.*

If $\nu > S^* (\gamma_1 + \gamma_2 r^*)$, the equilibrium $(S^*, r^*, 0)$ is locally asymptotically stable.

If instead $\nu < S^* (\gamma_1 + \gamma_2 r^*)$, the equilibrium is a saddle.

Proof. From 12.4.1, we have that ξ decreasing implies the eigenvalues of (S^*, r^*) have strictly negative real part.

Then, if $\nu > S^* (\gamma_1 + \gamma_2 r^*)$, all of our eigenvalues have negative real part, therefore the point is locally asymptotically stable if $\psi < 0$.

Similarly we see that if $\nu < S^* (\gamma_1 + \gamma_2 r^*)$, this is a saddle point. In this case, the $S - r$ plane is the stable manifold and the unstable manifold is one dimensional and points into $(0, \infty)^3$. □

An important note: we recall Proposition 12.4.7 tells us that if ξ is decreasing, then (S^*, r^*) will be locally asymptotically stable whenever it exists.

We again compare the dynamics of the two dimensional case to the three dimensional case. Considering the predator prey subsystem, we recall that invasion of the predator required $\nu < \gamma_1 K$, i.e. the expected predator biomass increase at prey equilibrium, which is the carrying capacity K times the predator biomass increase per unit prey consumed γ_1, is greater than biomass loss due to predator death. In the case of an infected system, our prey equilibrium now includes both healthy and infective prey, where S^* and r^* satisfy $g(S^*) = r^*\mu = h(r^*)$. Thus, when we consider the expected predator biomass increase, we consider it at the endemic host-parasite equilibrium, where we have differing values for predator biomass increase per healthy prey consumption and the predator biomass increase due to diseased prey consumption. We note that γ_2 can change without effecting $S^*, r^*, S^\diamond$, or $P^\diamond$ to either allow or restrict predator invasion. Moving back to the predator biomass increase, we recalculate at host-parasite-only equilibrium and we see our new formula is $\gamma_1 S^* + \gamma_2 I^* = \gamma_1 S^* + \gamma_2 S^* r^*$. Thus, we have that the predator can invade if $\gamma_1 S^* + \gamma_2 S^* r^* > \nu$, and cannot if $\gamma_1 S^* + \gamma_2 S^* r^* < \nu$.

We make note that in general there is no available comparison between $\gamma_1 S^* + \gamma_2 S^* r^*$ and $\gamma_1 K$, so there are four cases to consider.

- $\gamma_1 S^* + \gamma_2 S^* r^* < \nu$ and $\gamma_1 K < \nu$: Here the predator cannot invade either the healthy system or the infected system.

- $\gamma_1 S^* + \gamma_2 S^* r^* < \nu < \gamma_1 K$: In this case, the predator cannot invade the infected system, however it can invade the healthy system. This is due to the disease lowering the expected nutritional value of available prey, possibly by acting as a poison to the predator (as in the case of$\gamma_2 < 0$) and reducing the predator's biomass if it consumes an infected prey. Locally, this is parasite mediated extinction of the predator.

- $\gamma_1 K < \nu < \gamma_1 S^* + \gamma_2 S^* r^*$: This is the opposite of the second case, namely the predator cannot invade the healthy system, however it can invade the infected system. Here, the infection allows for the predator to survive where it would otherwise perish, by increasing the amount of prey captured, due to the infected prey being easier to catch than healthy prey (recall $\kappa_2 > \kappa_1$). This is the case of parasite mediated persistence of the predator.

- $\nu < \gamma_1 S^* + \gamma_2 S^* r^*$ and $\nu < \gamma_1 K$: In this case, the predator can invade and survive in both systems.

Chapter 19

UNIFORM PERSISTENCE

For this section, we only consider the case where ξ is strictly decreasing unless otherwise specified. We do this to simplify the dynamics in the predator-parasite-only subsystem to a point where we can apply persistence theorems and obtain our results. This also gives us uniqueness of the equilibrium $(0, r^\circ)$, in the predator-parasite subsystem.

We will begin with uniform persistence of all three species, then consider the cases where one species can survive without the others. We will only consider persistence in the case when all three species are initially present, as the two dimensional subsystems have already been discussed.

19.1 Uniform Persistence of all three species

We choose the state space

$$X = \mathbb{R}^3_+$$

and the functional $\rho(S, r, P) = \min\{S, r, P\}$. We assume $\sigma h(\infty) < g(0) + \mu$, so by Corollary 15.2.8, we have an asymptotic time bound on r. This is important, because if there is a solution with $r(t) \to \infty$ as $t \to \infty$, then Theorem 15.2.2 implies that solution will not have persistence of S or P. Then

$$X_0 = \{(S, r, P) \in X; S = 0 \text{ or } r = 0 \text{ or } P = 0\}.$$

From the form of our system of equations, it is clear that once S is zero, it will stay zero. This holds for r and P as well. Therefore we have that X_0 is forward

invariant. Additionally, we can rewrite X_0 as the finite union of closed sets, thus it is closed. Our two dimensional analyses have shown that all solutions in X_0 will converge to one of the equilibria $(0,0,0)$, $(K,0,0)$, $(S^*,r^*,0)$, $(0,r^circ,0),(0,\infty,0)$, or $(S^\diamond,0,P^\diamond)$ if they exist. Thus $\bigcup_{x\in X_0}\omega(x)$, where $\omega(x)$ is the omega limit set of a solution x, is equal to the union of these equilibria. Notice that both (0,r^circ,0) and (0,∞,0) cannot exist at the same time.

19.1.1 Dynamic Coexistence I

We assume that the prey-predator equilibrium exists,

$$\nu < \gamma_1 K, \tag{19.1.1}$$

and that the disease can invade the prey-predator equilibrium,

$$\sigma h'(0) > \mu + \kappa_2 P^\diamond. \tag{19.1.2}$$

We assume that the host-parasite equilibrium exists

$$\frac{\mu}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)} \tag{19.1.3}$$

and that the predator can invade it,

$$\nu < S^*(\gamma_1 + \gamma_2 r^*). \tag{19.1.4}$$

Recall that S^* and r^* satisfy $g(S^*) = r^*\mu = h(r^*)$, $S^\diamond = \dfrac{\nu}{\gamma_1}$, and $P^\diamond = \dfrac{g(S^\diamond)}{\kappa_1} = \dfrac{g(\nu/\gamma_1)}{\kappa_1}$. We also recall that ξ decreasing implies $(S^*,r^*,0)$ is always stable in the $S-r$ plane when it exists, as seen in Proposition 12.4.7. Note that Equations (19.1.1) and (19.1.3) share no parameters, so we can set up the predator-prey equilibrium and host-parasite equilibrium simultaneously. We also note that γ_2 does not appear in Equations (19.1.1), (19.1.3), or in the equilibrium equations for $(S^*,r^*,0)$ or

$(S^\diamond, 0, P^\diamond)$, so changing γ_2 will not alter the existence of either equilibrium. We can also see that κ_2 is not explicitly in either formula, however we have the constraint $\kappa_1 < \kappa_2$, thus decreasing κ_2 may require a decrease in κ_1. A change in κ_1 will only change the value of $P^\diamond$, and will not change the existence of $(S^\diamond, 0, P^\diamond)$, so to fulfill all these conditions, we can decrease κ_2 as much as needed and $(S^\diamond, 0, P^\diamond)$ will still exist. Since Equation (19.1.3) implies $\mu < \sigma h'(0)$, there exists some $\kappa_2 > 0$ small enough such that Equation (19.1.2) will hold. For Equation (19.1.4) we can choose γ_2 large enough this relationship to hold. Thus we see that it is possible to fulfill Equations (19.1.1),(19.1.2), (19.1.3), and (19.1.4)

Theorem 19.1.1. *Assume (19.1.1),(19.1.2),(19.1.3), (19.1.4), and ξ is either strictly decreasing or constant. Then there is dynamic prey-predator-parasite coexistence: There exists some $\epsilon > 0$ such that*

$$\liminf_{t\to\infty} S(t) \geq \epsilon, \quad \liminf_{t\to\infty} I(t) \geq \epsilon, \quad \liminf_{t\to\infty} P(t) \geq \epsilon$$

whenever $S(0) > 0$, $I(0) > 0$, $P(0) > 0$.

Proof. We apply the results in (Smith and Thieme, 2011, Sec.8.3) to give us uniform weak persistence, then use (Smith and Thieme, 2011, Sec.4.1) to upgrade to uniform persistence. Since we are considering when ξ is strictly decreasing or constant, we have $\dfrac{g(0)}{h(g(0)/\mu)} < \dfrac{g(0)+\mu}{h(\infty)}$, so assumption (19.1.3) allows us to apply Theorem 15.2.7, so we have a compact set B which attracts all positive solutions. By Theorem 8.20 in Smith and Thieme (2011), letting $M_1 = (0,0,0)$, $M_2 = (K,0,0)$, $M_3 = (S^*, r^*, 0)$, $M_4 = (0, r^\circ, 0)$, and $M_5 = (S^\diamond, 0, P^\diamond)$, we need to show that each M_i is compact, invariant, and isolated in X_0, $M_i \cap M_j = \emptyset$ if $i \neq j$, $\{M_1, M_2, M_3, M_4, M_5\}$ are acyclic, and M_i is a uniformly weakly ρ-repelling for $i = 1, 2, 3, 4, 5$. We observe that the second inequality in the condition 19.1.3 implies that $(0, \infty, 0)$ does not exist, so it is not considered. In the case when M_4 ceases to exist, we omit its argument.

We can see that since each M_i is a fixed point or a collection of fixed points, we have that for any solution with initial conditions within M_i, the solution will stay in M_i, so it is invariant. With the conditions we require, each of the M_i are saddles in 3 dimensional space, thus we have they are isolated in X_0. All that is left to be shown is uniformly weakly repelling and acyclicity.

To show the former condition, we suppose these points are not uniformly weakly repelling, that is for all $\varepsilon > 0$, we have that there exists a solution such that $\limsup_{t\to\infty} d((S(t), r(t), P(t)), M_i) < \varepsilon$ whenever $\rho(S(0), r(0), P(0)) > 0$ for i=1,2,3,4,5. Note that the solution need not be the same for each i. Further note that this implies that for large t, $d(S(t), M_i(1)) < \varepsilon$, $d(r(t), M_i(2)) < \varepsilon$, and $d(P(t), M_i(3)) < \varepsilon$, where $M_i(j)$ is the jth component of M_i. We define β_i^ε to be the ball of radius ε surrounding the point M_i.

For M_1, we choose ε such that $g(\varepsilon) > \sigma h(\varepsilon) + \kappa_1\varepsilon$. This implies for solutions within β_1^ε, $\frac{S'}{S} = g(S) - \sigma h(r) - \kappa_1 P > g(\varepsilon) - \sigma h(\varepsilon) - \kappa_1 e > 0$. Therefore, we will have exponential growth of S, a contradiction to staying $S < \varepsilon$.

At M_2, we begin by defining ε such that $\sigma h'(0) > \mu + g(K - \varepsilon)$ which we can do by the existence of the prey-parasite equilibrium. Then we can see that for solutions in β_2^ε, $\frac{r'}{r} = \sigma\xi(r) - \mu + (k_1 - k_2)P - g(S) > \sigma h'(0) - \mu - g(K - \varepsilon) > 0$. Thus we have exponential growth of r, a contradiction.

Considering M_3, we find an ε such that

$$\gamma_1(S^* + \varepsilon) + \gamma_2(r^* + \varepsilon)(S^* + \varepsilon) = \gamma_1 S^* + \gamma_2 r^* S^* + \varepsilon\gamma_1 + \varepsilon^2\gamma_2 > \nu.$$

Then we have that solutions in β_3^ε satisfy $\frac{P'}{P} = \gamma_1 S + \gamma_2 r S - \nu > 0$, implying that P will grow exponentially, a contradiction.

For M_5, we choose ε such that

$$\sigma h'(0) > (\kappa_1 - \kappa_2)P^\diamond + \mu + g(S^\diamond - \varepsilon) = \kappa_1 P^\diamond + \mu + g(S^\diamond - \varepsilon) - g(S^\diamond).$$

Then, for solutions within β_5^ε, we see $\frac{r'}{r} = \sigma\xi(r) + (\kappa_1 - \kappa_2)P - \mu - g(S) > 0$, implying that r increases exponentially, contradicting the assumption.

If M_4 exists, ξ decreasing implies we can use Corollary 12.4.11 and $g(0) - \sigma h(r^\circ) > 0$. We choose an ε such that $g(\varepsilon) - \sigma h(r^\circ + \varepsilon) - \kappa_1\varepsilon > 0$. Looking into the S' equation we see

$$\frac{S'}{S} = g(S) - \sigma h(r) - \kappa_1 P > g(\varepsilon) - \sigma h(r^\circ + \varepsilon) - \kappa_1\varepsilon > 0,$$

will hold for all solutions within β_4^ε, which implies that S grows exponentially, a contradiction. Observe that this equilibrium will not exist for our parameters if ξ' is constant.

Therefore we have that the M_i's are uniform weakly $\rho-$repelling.

To show acyclicity, we will follow all the unstable manifolds of equilibria, and we will show which equilibria are chained. From this, we will show that the chains do not connect back to themselves, so a cycle cannot occur. Throughout this we only consider solutions in X_0.

With our conditions we have $(S^*, r^*, 0)$ is globally asymptotically stable for solutions in $(0,\infty) \times (0,\infty) \times 0$, implying that if it is in a chain, it is the last point in a chain. Similarly, $(S^\diamond, 0, P^\diamond)$ is globally asymptotically stable in $(0,\infty) \times 0 \times (0,\infty)$, so it is also guaranteed to be the end of a chain.

(0,0,0) is chained to $(K, 0, 0)$ via solutions of the form $(S(t), 0, 0)$, with $S(0) \in (0, K)$. By our assumptions (K,0,0) is chained to both $(S^*, r^*, 0)$ and $(S^\diamond, 0, P^\diamond)$, as it has an unstable manifold pointing into both $(0,\infty)\times(0,\infty)\times 0$ and $(0,\infty)\times 0\times(0,\infty)$, and those fixed points are globally attracting on their respective sets.

If $(0, r^\circ, 0)$ exists, then $(0, 0, 0)$ is chained to it via the trajectory $(0, r(t), 0)$, with $r(0) \in (0, r^\circ)$. By our assumptions the unstable manifold of $(0, r^\circ, 0)$ points into the interior of the $S - r$ plane, so it is chained to $(S^*, r^*, 0)$, which ends the chain.

All the chains in X_0 are:

- $(0,0,0) \to (K,0,0) \to (S^*, r^*, 0)$,
- $(0,0,0) \to (K,0,0) \to (S^\diamond, 0, P^\diamond)$,
- $(0,0,0) \to (0, r^\circ, 0) \to (S^*, r^*, 0)$, if $(0, r^\circ, 0)$ exists.

We see that none of the chains connect to form a cycle, therefore, there are no cycles in X_0, and the system is uniformly weakly persistent.

We note that in this case, we can use Theorem 15.2.7 to give us a bounded set $B = (0,K) \times (0, r^\infty) \times (0, P^\infty)$, independent of initial conditions such that B is forward invariant, and all solutions with positive initial conditions will enter B. This consideration combined with Theorem 4.2 from Smith and Thieme (2011) gives us that uniform weak persistence implies uniform strong persistence. □

Looking ahead to existence of the interior equilibrium, we note that the assumptions (19.1.1),(19.1.2), (19.1.3), (19.1.4), and ξ is either strictly decreasing or constant, imply the existence of an interior equilibrium by Theorem 20.2.8, which can be seen in the fourth or fifth bullet.

19.1.2 Dynamic Coexistence II: Parasite-Mediated Predator Survival

This is a scenario where the predator could not survive on healthy prey alone but needs the infected prey because it is easier to catch.

So the prey-predator equilibrium does not exist,

$$\gamma_1 = \kappa_1 = 0 \text{ or } g(\nu/\gamma_1) \leq 0, \text{ i.e. } \nu/\gamma_1 \geq K. \tag{19.1.5}$$

However, the host-parasite equilibrium exists,

$$\frac{\mu}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)} \tag{19.1.3 revisited}$$

and that the predator can invade it,

$$\nu < S^*(\gamma_1 + \gamma_2 r^*). \qquad \text{(19.1.4 revisited)}$$

Recall that S^* and r^* satisfy $g(S^*) = r^*\mu = h(r^*)$. We also recall that ξ decreasing implies $(S^*, r^*, 0)$ is always stable in the $S-r$ plane when it exists, as seen in Proposition 12.4.1. Equation (19.1.5) implies that $(S^\diamond, 0, P^\diamond)$ does not exist, so Equation (19.1.1) does not need to be satisfied. We note that Equation (19.1.3) and the equations for S^* and r^* are independent of γ_2, so changing γ_2 will not effect the existence or values of $(S^*, r^*, 0)$. For Equation (19.1.4) we can choose γ_2 large enough this relationship to hold. Thus we see that it is possible to fulfill Equations (19.1.3), (19.1.4), and (19.1.5).

Theorem 19.1.2. *Assume (19.1.3), (19.1.4), (19.1.5), and ξ is either strictly decreasing or constant. Then there is dynamic prey-predator-parasite coexistence: There exists some $\epsilon > 0$ such that*

$$\liminf_{t\to\infty} S(t) \geq \epsilon, \quad \liminf_{t\to\infty} I(t) \geq \epsilon, \quad \liminf_{t\to\infty} P(t) \geq \epsilon$$

whenever $S(0) > 0$, $I(0) > 0$, $P(0) > 0$.

Proof. The same proof as theorem 19.1.1 can be followed, using only i=1,2,3,4 as M_5 ceases to exist. This also causes a slight change to the acyclicity portion. In this case, the chains are

- $(0, 0, 0) \to (K, 0, 0) \to (S^*, r^*, 0)$,
- $(0, 0, 0) \to (0, r^\circ, 0) \to (S^*, r^*, 0)$, if $(0, r^\circ, 0)$ exists.

Again, no cycles are formed. □

By Theorem 6.2 in Smith and Thieme (2011), we have that there will be an interior equilibrium under the assumptions required for the above Theorem. Again, looking ahead shows us that Corollary 20.2.8 is satisfied, again proving the existence of an interior equilibrium.

19.2 Uniform Persistence of a Single Species

Note that for a given parameter set, Corollary 15.1.3 implies that P and I cannot persist uniformly for all positive initial conditions if there is one solution with positive initial conditions and S component such that $S(t) \to 0$ as $t \to \infty$. This restriction, however, does not extend to r, the *ratio* of infectives to susceptibles, which we recall is not the parasite itself.

The following Theorem is proven for general ξ, however the corollary below has the simplified terms for the case when ξ is strictly decreasing or constant.

19.2.1 Persistence of the Host

Theorem 19.2.1. *Let $\sigma h(\infty) < g(0) + \mu$. Assume that either there is no $r \in (0, \infty)$ with $\sigma\xi(r) = g(0) + \mu$ or, if there is, it satisfies $g(0) > \sigma h(r)$.*

Then the host persists uniformly, i.e. $S_\infty > \varepsilon$ for some $\varepsilon > 0$ for all solutions with $S(0) > 0$.

Proof. The system (15.2.1) induces a continuous semiflow on $\mathbb{R}^3_+$. By Theorem 15.2.7, the semiflow has a compact attractor. We will use the techniques in Sec.8.3 from Smith and Thieme (2011) and choose the persistence function $\rho(S, r, P) = S$ to prove our persistence result. Let Ω be the union of all ω-limit sets of solutions starting with $S(0) = 0$. Let A_0 be the compact attractor of bounded sets for the semiflow induced

by the differential system of equation

$$r' = \sigma(1+r)h(r) - r(g(0)+\mu) + (\kappa_1 - \kappa_2)P$$
$$P' = -\nu P.$$

Then A_0 is invariant and connected via Proposition 2.24 in Smith and Thieme (2011), and $A_0 = [r_1, r_2] \times \{0\}$, with $0 \leq r_1 \leq r_2 < \infty$, is a compact interval. Further A_0 is isolated in X_0 and acyclic and $\Omega \subseteq A_0$. Since A_0 is invariant, there exist a total solution r of the equation with range in A_0, $P(0) = 0$, and $r(0) = r_2$. This implies that $r'(0) = 0$, i.e., $r_2 = 0$ or $\sigma\xi(r_2) = g(0) + \mu$.

We show that, in either case, A_0 is uniformly weakly ρ-repelling.

Case 1: $r_2 = 0$.

Then $A_0 = \{(0,0,0)\}$. Choose some $\epsilon > 0$ such that $g(\epsilon) - \sigma h(\epsilon) - \kappa_1\varepsilon > \epsilon$. This is possible because g and h are continuous and $g(0) > 0 = \sigma h(0)$. Since g is decreasing and h is increasing, $g(S) - \sigma h(r) - \kappa_1 P > \epsilon$ for all $S, r, P \in [0, \epsilon]$. Suppose there is a solution with $S(0) > 0$ and

$$\limsup_{t\to\infty} d((S(t), r(t), P(t)), (0,0,0)) < \epsilon.$$

After a shift in time, $0 < S(t) < \epsilon, r(t) < \epsilon$, and $P(t) < \varepsilon$ for all $t \geq 0$. By choice of ϵ, $S'(t) \geq \epsilon S(t)$ and S increases exponentially to ∞, a contradiction.

Case 2: $r_2 > 0$ and $\sigma\xi(r_2) = g(0) + \mu$.

By assumption, $g(0) > \sigma h(r_2)$. To show that A_0 is uniformly weakly ρ-repelling, choose some $\epsilon > 0$ such that $g(S) > \sigma h(r_2 + \epsilon) + \epsilon + \kappa_1\varepsilon$ for all $S \in [0, \epsilon]$. Since h is increasing, $g(S) > \sigma h(r) + \epsilon + \kappa_1 P$ for all $S, P \in [0, \epsilon]$ and $r \in [0, r_2 + \epsilon]$. Assume that there exists a solution such that $S(0) > 0$ and $\limsup_{t\to\infty} d((S(t), r(t), P(t)), A_0) < \epsilon$.

Then, after a shift in time, $S(t), P(t) \in [0, \epsilon]$ and $r(t) \in [0, r_2 + \epsilon]$ for all $t \geq 0$. So $g(S(t)) \geq h(r(t)) + \epsilon + \kappa_1 \varepsilon$ for all $t \geq 0$ and S grows to infinity, a contradiction.

By Theorem 8.20 in Smith and Thieme (2011), the induced semiflow is uniformly weakly ρ-persistent and thus uniformly persistent by Theorem 4.13 in Smith and Thieme (2011). □

Comparing the conditions of Theorem 15.2.6 and Theorem 19.2.1, we note that Theorem 15.2.6 is in fact a special case of Theorem 19.2.1. Obviously we have $\sigma h(\infty) < g(0) < g(0) + \mu$, however $\sigma h(\infty) < g(0)$ also implies $\sigma h(r) < g(0)$ for all r, thus the conditions of Theorem 19.2.1 are satisfied.

Corollary 19.2.2. *Let ξ be strictly decreasing or constant and $\sigma < \dfrac{g(0)}{h(g(0)/\mu)}$. Then the host persists uniformly, i.e. $S_\infty > \varepsilon$ for some $\varepsilon > 0$.*

Proof. Section 12.12 shows that ξ strictly decreasing implies the rest of the conditions required to apply Theorem 19.2.1. □

19.2.2 Persistence of the Predator

Theorem 19.2.3. *Let either*

- $\sigma \leq \dfrac{\mu}{h'(0)}$ *and* $\nu < \gamma_1 K$.

  or

- $\dfrac{\mu}{h'(0)} < \sigma < \dfrac{g(0)}{h(g(0)/\mu)}$, *and* $\nu < \gamma_1 S^* + \gamma_2 S^* r^*$

Then the predator persists uniformly, i.e. there exists some $\varepsilon > 0$ such that $P_\infty > \varepsilon$ for all solutions with $P(0) > 0, S(0) > 0,$ and $r(0) > 0$.

We recall that S^* and r^* satisfy $\sigma h(r^*) = \mu r^* = g(S^*)$.

Proof. The first bullet can be seen from Corollary 17.1.2.

Consider the second bullet. If $\nu \geq \gamma_1 K$, Theorem 19.1.2 will give our result. Note $\nu \geq \gamma_1 K$ is only considered in the second bullet. Now we only need to consider when $\nu < \gamma_1 K$, $\nu < \gamma_1 S^* + \gamma_2 S^* r^*$, and $\dfrac{\mu}{h'(0)} < \sigma < \dfrac{g(0)}{h(g(0)/\mu)}$.

We choose the state space $X = \mathbb{R}^3_+$, and the persistence function $\rho(S, r, P) = \min\{S, P\}$. This gives us $X_0 = \{(S, r, P) \in X; S = 0 \text{ or } P = 0\}$ In this case, the equilibria in X_0 are M_1 :=(0,0,0), M_2 := (K,0,0), $M_3 := (S^*, r^*, 0)$, and $M_4 := (0, r^\circ, 0)$. We observe that we can reuse all calculations from Theorem 19.1.1 to show that M_1, M_3, and M_4 are isolated in X_0 and uniformly weakly repelling.

We define β_i^ε to be the ball of radius ε surrounding the point M_i. Since $\nu < \gamma_1 K$, there is some ε such that $\varepsilon < \gamma_1(K - \varepsilon) - |\gamma_2|\varepsilon(K + \varepsilon) - \nu$, which implies that for any solution in β_2^ε, $\dfrac{P'}{P} = \gamma_1 S + \gamma_2 S r - \nu > \varepsilon$, implying the exponential growth of P, a contradiction.

Recall from Section 12.12 that r° exists if $\dfrac{\mu + g(0)}{h'(0)} < \sigma < \dfrac{\mu + g(0)}{h(\infty)}$. If $(0, r^\circ, 0)$ exists, then $(0, 0, 0)$ is chained to it via the trajectory $(0, r(t), 0)$, with $r(0) \in (0, r^\circ)$. By our assumptions the unstable manifold of $(0, r^\circ, 0)$ points into the interior of the $S - r$ plane, so it is chained to $(S^*, r^*, 0)$, which ends the chain.

All the chains in X_0 are:

- $(0, 0, 0) \to (K, 0, 0) \to (S^*, r^*, 0)$,
- $(0, 0, 0) \to (0, r^\circ, 0) \to (S^*, r^*, 0)$, if $(0, r^\circ, 0)$ exists.

We see that none of the chains connect to form a cycle, therefore, there are no cyclic chains in X_0, and the system is uniformly weakly persistent.

Therefore we can conclude that there is some $\delta > 0$ such $\rho(S, r, P) = \min\{S, P\} > \delta$ for any solution with initial conditions $S(0) > 0$ and $P(0) > 0$, and so $P_\infty > \delta$ for solutions with $S(0) > 0$ and $P(0) > 0$. □

The change in conditions between bullet points may seem strange at first; However, we see from 12.4.1.2 and Theorem 12.4.4 that as for $\sigma \leq \dfrac{\mu}{h'(0)}$, the equilibrium $(S^*, r^*, 0)$ does not exist, and if $\sigma \to \dfrac{\mu}{h'(0)}$ from above, then $(S^*, r^*) \to (K, 0)$. This implies $\gamma_1 S^* + \gamma_2 S^* r^* \to \gamma_1 K$ as $\sigma \to \dfrac{\mu}{h'(0)}$ from above. With this observation, we can see the second bullet is the natural extension of the first.

19.2.3 Persistence of the Ratio of Infectives to Susceptibles

Theorem 19.2.4. *Let $S(0) > 0,\ P(0) > 0$ and $\sigma h'(0) > \mu$. Further, if $\gamma_1 K > \nu$, then we suppose the stronger condition of $\sigma h'(0) > \mu + \kappa_2 P^\diamond$.*

Then the ratio persists uniformly, i.e. $r_\infty > \varepsilon$ for some $\varepsilon > 0$ for all solutions with $r(0) > 0$.

Recall $\kappa_1 P^\diamond = g(S^\diamond) = g(\nu/\gamma_1)$.

Proof. In the case of $\sigma \in \left(\dfrac{\mu}{h'(0)}, \dfrac{g(0)}{h(g(0)/\mu)}\right)$, we may apply Theorem 19.1.1 or 19.1.2 depending on the relationship between ν and $\gamma_1 K$.

Now we may assume $\sigma \geq \dfrac{g(0)}{h(g(0)/\mu)}$, which, by Section 12.12, also implies $\sigma > \dfrac{g(0)+\mu}{h'(0)} > \dfrac{\mu}{h'(0)}$. We will follow the structure and notation of Theorem 19.1.1 to prove persistence of the ratio. For this Theorem, we will use $X = \mathbb{R}^3_+$ as our state space, $\rho(S, r, P) = r$ as our persistence function, and $B = [0, K] \times [0, \infty) \times [0, P^\odot]$, where $P^\odot$ is defined in Theorem 15.1.2, as our attracting set. Although B is not compact, $B \cap \{\rho \leq 1\} = [0, K] \times [0, 1] \times [0, P^\odot]$ is compact, which is all that is required of B for Theorem 8.20 from Smith and Thieme (2011). This Theorem will imply uniform weak persistence, which we will upgrade to uniform persistence by applying Theorem 4.2 from Smith and Thieme (2011), using the same B as above. We now split into the cases of $\gamma_1 K \leq \nu$ and $\gamma_1 K > \nu$.

Case 1: $\gamma_1 K \leq \nu$:

In this case, we have two equilibria in X_0, i.e. with $\rho(S, r, P) = r = 0$, which are $M_1 = (0, 0, 0)$ and $M_2 = (K, 0, 0)$. Since (0,0,0) is a saddle, our points are isolated in X_0. We define β_i^ε to be the ball of radius ε centered at M_i.

For M_1, $\sigma > \dfrac{\mu + g(0)}{h'(0)}$ implies that we can find an $\varepsilon > 0$ such that $\sigma\xi(\varepsilon) - \mu - g(\varepsilon) + (\kappa_1 - \kappa_2)\varepsilon > \varepsilon$, since $\xi(0) = h'(0)$, which in turn implies for any solution in β_1^ε, $\dfrac{r'}{r} = \sigma\xi(r) - \mu - g(S) + (\kappa_1 - \kappa_2)P > \varepsilon$. This inequality implies the exponential growth of r, a contradiction.

In the case of M_2, $\sigma > \dfrac{\mu}{h'(0)}$ allows us to get an $\varepsilon > 0$ such that $\sigma\xi(\varepsilon) - \mu - g(K - \varepsilon) + (\kappa_1 - \kappa_2)\varepsilon > \varepsilon$, and so for any solution in β_2^ε, $\dfrac{r'}{r} = \sigma\xi(r) - \mu - g(S) + (\kappa_1 - \kappa_2)P > \varepsilon$, leading to the exponential growth of r yet again.

Therefore all equilibria in X_0 are uniformly weakly $\rho-$repelling. We note that the only chain in X_0 is

- $(0, 0, 0) \to (K, 0, 0)$,

implying that there can be no cycles.

Thus we can apply the two Theorems from Smith and Thieme (2011), and we have uniform persistence or r.

Case 2: $\gamma_1 K > \nu$:

In this case we also have the equilibrium $M_5 = (S^\diamond, 0, P^\diamond)$ (named this way to use the same terms as in Theorem 19.1.1). Note that M_5 is locally asymptotically stable in X_0, so it is isolated in X_0. The proofs of M_1 and M_2 being uniformly weakly $\rho-$repelling still hold, we now only need to show that M_5 is uniformly weakly $\rho-$repelling, and show that there are still no cycles.

Considering M_5, we recall the case $\gamma_1 K > \nu$ provides us with the stronger assumption $\sigma h'(0) > \mu + \kappa_2 P^\diamond$. Further we observe $g(S^\diamond) = \kappa_1 P^\diamond$ implies $-\kappa_2 P^\diamond =$

$-g(S^\diamond) + (\kappa_1 - \kappa_2)P^\diamond$. Using $h'(0) = \xi(0)$, we find an $\varepsilon > 0$ such that $\sigma\xi(\varepsilon) - \mu - g(S^\diamond - \varepsilon) + (\kappa_1 - \kappa_2)(P^\diamond + \varepsilon) > \varepsilon$, which implies that any solution in β_5^ε, $\dfrac{r'}{r} = \sigma\xi(r) - \mu - g(S) + (\kappa_1 - \kappa_2)P > \varepsilon$, leading to the exponential growth of r one more time.

As for acyclicity, we see the only chain is

- $(0, 0, 0) \to (K, 0, 0) \to (S^\diamond, 0, P^\diamond)$,

implying that there can still be no cycles. Thus we will have uniform persistence in this case as well. □

19.2.4 Persistence of the Parasite

We note that in the above we observe uniform persistence of the *ratio* of infectives to susceptibles, not the infectives. A quick avenue to uniform persistence of the parasite, is uniform persistence of both the parasite ratio and the host, but in fact the conditions are both necessary and sufficient for uniform persistence of I. The result holds for any ξ.

Corollary 19.2.5. *Both S and r persist uniformly for all solutions with $S(0)$, $r(0) > 0$ if and only I persists uniformly for all solutions with $S(0)$, $r(0) > 0$.*

Proof. ($\Rightarrow$) Recall that $I = Sr$, and suppose that both S and r persist uniformly, with $S_\infty > \varepsilon > 0$ and $r_\infty > \delta > 0$. Then we see $\varepsilon\delta < S_\infty r_\infty \leq I_\infty$.

($\Leftarrow$) Suppose I persists uniformly, with $I_\infty > 2\varepsilon > 0$. Then, after a time shift, we have

$$\varepsilon < I(t) = S(t)r(t) < Kr(t),$$

so $\dfrac{\varepsilon}{K} < r(t)$, which implies r persists uniformly.

For S, we see from Theorem 15.1.2 that $I(t) < N(t)$ and so

$$I_\infty \leq I^\infty \leq N^\infty \leq S^\infty \frac{g(0) + \beta}{\beta}.$$

Thus, for all solutions, $S^\infty > I_\infty \dfrac{\beta}{g(0)+\beta}$, which by Theorem 15.1.5 implies our result. $\square$

Thus we can consider the intersection of the requirements for Theorems 19.2.1 and 19.2.4 for uniform persistence of I.

Chapter 20

THE INTERIOR EQUILIBRIUM

For this chapter we will still only consider the case where ξ is strictly decreasing unless otherwise specified. We do this to attempt to ease computation of the interior equilibrium and its stability.

20.1 Local Stability of Interior Equilibrium

Recall the full system

$$\begin{aligned}\frac{S'}{S} =& g(S) - \sigma h(r) - \kappa_1 P,\\ \frac{r'}{r} =& \sigma\xi(r) + (\kappa_1 - \kappa_2)P - \mu - g(S),\\ \frac{P'}{P} =& \gamma_1 S + \gamma_2 r S - \nu.\end{aligned}$$

Theorem 20.1.1. *If a coexistence equilibrium $(S^\dagger, r^\dagger, P^\dagger)$ exists, $\gamma_2 \geq 0$, and $(S^\dagger)^2 g'(S^\dagger)\gamma_2 + \nu\sigma h'(r^\dagger) \leq 0$, then it is locally asymptotically stable.*

Proof. Using $g(S^\dagger) - \sigma h(r^\dagger) - \kappa_1 P = 0$, $\frac{1+r^\dagger}{r^\dagger}\sigma h(r^\dagger) + (\kappa_1 - \kappa_2)P^\dagger - \mu - g(S^\dagger) = 0$, and $\gamma_1 S^\dagger + \gamma_2 r^\dagger S^\dagger - \nu = 0$ we can simplify the Jacobian matrix to

$$J = \begin{pmatrix} S^\dagger g'(S^\dagger) & -S^\dagger\sigma h'(r^\dagger) & -\kappa_1 S^\dagger \\ -r^\dagger g'(S^\dagger) & \sigma r^\dagger \xi'(r^\dagger) & r^\dagger(\kappa_1 - \kappa_2) \\ \gamma_1 P^\dagger + \gamma_2 r^\dagger P^\dagger & P^\dagger\gamma_2 S^\dagger & 0 \end{pmatrix}.$$

Observe that the trace is $S^\dagger g'(S^\dagger) + \sigma r^\dagger \xi'(r^\dagger) < 0$.

Using $\dfrac{\nu}{\gamma_1+\gamma_2 r^\dagger} = S^\dagger$, we rewrite the 3-1 entry as $\dfrac{P^\dagger \nu}{S^\dagger}$. By expanding along the last column we see

$$\begin{aligned} Det(J) &= -S^\dagger \kappa_1[-r^\dagger g'(S^\dagger)P^\dagger \gamma_2 S^\dagger - \frac{P^\dagger \nu}{S^\dagger}\sigma r^\dagger \xi'(r^\dagger)] \\ &\qquad - r^\dagger(\kappa_1-\kappa_2)[S^\dagger g'(S^\dagger)P^\dagger\gamma_2 S^\dagger + S^\dagger \sigma h'(r^\dagger)\frac{P^\dagger\nu}{S^\dagger}] \\ &= r^\dagger P^\dagger \left[\kappa_1(\nu\sigma\xi'(r^\dagger) - \sigma h'(r^\dagger)\nu) + \kappa_2((S^\dagger)^2 g'(S^\dagger)\gamma_2 + \sigma h'(r^\dagger)\nu)\right] \\ &= r^\dagger P^\dagger \left[\kappa_1\nu(\sigma\xi'(r^\dagger) - \sigma h'(r^\dagger)) + \kappa_2((S^\dagger)^2 g'(S^\dagger)\gamma_2 + \nu\sigma h'(r^\dagger))\right] \end{aligned}$$

Recall that since h' is strictly decreasing, $\xi'(r^\dagger) - h'(r^\dagger) < 0$. Thus, our hypothesis gives us the rest, and we have that $Det(J) < 0$. If $\kappa_1 > 0$, we observe an alternate form of the determinant for later use:

$$\kappa_1 r^\dagger P^\dagger \left[\nu\sigma\left(\xi'(r^\dagger) - h'(r^\dagger) + \frac{\kappa_2}{\kappa_1}h'(r^\dagger)\right) + \frac{\kappa_2}{\kappa_1}\gamma_2 (S^\dagger)^2 g'(S^\dagger)\right]. \tag{20.1.1}$$

In preparation for Routh-Hurwitz we calculate

$A_1 = -P^\dagger\gamma_2 S^\dagger r^\dagger(\kappa_1-\kappa_2)$,

$A_2 = \kappa_1 S^\dagger(\gamma_1 P^\dagger + \gamma_2 r^\dagger P^\dagger)$, and

$A_3 = S^\dagger g'(S^\dagger)\sigma r^\dagger \xi'(r^\dagger) - S^\dagger g'(S^\dagger) r^\dagger \sigma h'(r^\dagger) = S^\dagger g'(S^\dagger)\sigma r^\dagger(\xi'(r^\dagger) - h'(r^\dagger)) > 0.$

We also take note that $A_1 + A_2 = P^\dagger \gamma_2 S^\dagger r^\dagger \kappa_2 + \kappa_1 S^\dagger \gamma_1 P^\dagger > 0$. Thus we have that the Routh-Hurwitz coefficient is

$$\begin{aligned}
h &= Det(J) - Tr(J)(A_1 + A_2 + A_3) \\
&\geq Det(J) - Tr(J)(A_1 + A_2) \\
&= S^\dagger r^\dagger P^\dagger \left[\kappa_1 \frac{\nu}{S^\dagger} \sigma(\xi'(r^\dagger) - h'(r^\dagger)) + \kappa_2(\gamma_1 \sigma h'(r^\dagger) + \gamma_2[S^\dagger g'(S^\dagger) + \sigma r^\dagger h'(r^\dagger)]) \right] \\
&\quad - (S^\dagger g'(S^\dagger) + \sigma r^\dagger \xi'(r^\dagger))(P^\dagger \gamma_2 S^\dagger r^\dagger \kappa_2 + \kappa_1 S^\dagger \gamma_1 P^\dagger) \\
&= -\kappa_1 \gamma_1 (S^\dagger)^2 P^\dagger g'(S^\dagger) \quad + \quad (\kappa_2 - \kappa_1)\gamma_1 S^\dagger r^\dagger P^\dagger \sigma h'(r^\dagger) \quad + \\
&\quad + \sigma(\kappa_2 - \kappa_1)\gamma_2 S^\dagger (r^\dagger)^2 P^\dagger \big(h'(r^\dagger) - \xi'(r^\dagger)\big).
\end{aligned}$$

Since all terms in the above sum are positive, we have local asymptotic stability of the interior equilibrium! □

We note that the Routh-Hurwitz coefficient only used $\gamma_2 \geq 0$ from the hypothesis. This insight allows for a nice corollary.

Corollary 20.1.2. *Suppose an interior equilibrium exists, and $\kappa_1 > 0$.*

If $\gamma_2 \geq 0$ and Expression (20.1.1) is negative at that point, then the point is locally asymptotically stable.

If Expression (20.1.1) is positive at that point, then the point is unstable.

Corollary 20.1.3. *Suppose a coexistence equilibrium $(S^\dagger, r^\dagger, P^\dagger)$ exists and $\kappa_1 = \gamma_1 = 0$.*

If $S^\dagger g'(S^\dagger) + r^\dagger \sigma h'(r^\dagger) < 0$, then $(S^\dagger, r^\dagger, P^\dagger)$ is locally asymptotically stable.

If $S^\dagger g'(S^\dagger) + r^\dagger \sigma h'(r^\dagger) > 0$, then $(S^\dagger, r^\dagger, P^\dagger)$ is unstable.

Proof. We have $\gamma_2 > 0$ is required for an equilibrium to exist in this case. The local stability follows from Theorem 20.1.1.

Using these values and the determinant we found in the proof of Theorem 20.1.1, we have

$$Det(J) = S^\dagger r^\dagger P^\dagger \kappa_2 \gamma_2 [S^\dagger g'(S^\dagger) + \sigma r^\dagger h'(r^\dagger)],$$

which will be positive if $S^\dagger g'(S^\dagger) + r^\dagger \sigma h'(r^\dagger) > 0$. If we let the ith eigenvalue be λ_i, then we know that $Det(J) = \lambda_1 \lambda_2 \lambda_3$. Thus, if $Det(J) > 0$, then there is at least one non-negative eigenvalue, which implies that the interior equilibrium will be unstable. □

20.2 Existence of the Interior Equilibrium

A coexistence equilibrium of (15.2.1), which we denote $(S^\dagger, r^\dagger, P^\dagger)$, where susceptible and infective prey and the predator are present satisfies the following equations,

$$0 = g(S^\dagger) - \sigma h(r^\dagger) - \kappa_1 P^\dagger, \tag{20.2.1}$$

$$0 = \sigma \xi(r^\dagger) + (\kappa_1 - \kappa_2) P^\dagger - \mu - g(S^\dagger), \tag{20.2.2}$$

$$0 = \gamma_1 S^\dagger + \gamma_2 r^\dagger S^\dagger - \nu. \tag{20.2.3}$$

Adding (20.2.1) and (20.2.2), we can replace (20.2.2) with

$$0 = \sigma \frac{h(r^\dagger)}{r^\dagger} - \kappa_2 P^\dagger - \mu. \tag{20.2.4}$$

Solving (20.2.4) for $P^\dagger$, we have

$$P^\dagger = \frac{1}{\kappa_2} \left[\sigma \frac{h(r^\dagger)}{r^\dagger} - \mu \right]. \tag{20.2.5}$$

Solving for $S^\dagger$ in equation 20.2.3 will imply that $S^\dagger = \dfrac{\nu}{\gamma_1 + \gamma_2 r^\dagger}$. Thus, if $\gamma_1 > 0$ we have

$$S^\dagger \begin{cases} < \frac{\nu}{\gamma_1} & \text{if } \gamma_2 > 0 \\ = S^\diamond & \text{if } \gamma_2 = 0 \\ > S^\diamond & \text{if } \gamma_2 < 0 \end{cases} \tag{20.2.6}$$

We use $\dfrac{\nu}{\gamma_1}$ in the first case, as $S^\diamond$ may not exist in this case, however the condition $\dfrac{\nu}{\gamma_1} < K$ must be fulfilled in order for $S^\dagger < K$ in the other two cases. Thus we have if an interior equilibrium exists, $\gamma_2 \leq 0$ implies the predator-prey equilibrium exists as well.

We also make note that the conditions that follow only ensure than *an* interior equilibrium exists. It may be the case that it not unique, and an interior equilibrium may exist outside the specified parameter ranges.

Throughout consideration of the interior equilibrium, we use the notation r_1 and r_2 as the minimum and maximum values respectively for which the interior equilibrium can exist. However, as we will see, the upper and lower bounds for $r^\dagger$ can change depending on some parameters, thus we need to break our analysis into cases.

20.2.1 The Predator Consumes Infectives Only ($\kappa_1 = 0$)

This is the case of the predator eating infectives only. Since $\kappa_1 = 0$, we also have $\gamma_1 = 0$. Similar models have been considered in Xiao and Chen (2002, 2001a,b); Yongzhen *et al.* (2011); Chattopadhyay and Arino (1999). This is a viable scenario in some natural systems, e.g. if the behavior of the infected prey individuals is modified such that they live in parts of the habitat which are accessible to the predator (fish and aquatic snails staying close to the water's surface, ants staying on top of vegetation rather than under plant cover) Holmes and Bethel (1972). In some of these cases

(ant on top of vegetation), the predator (sheep) is another host which is required for parasite reproduction, which would not fit the model we have. Holmes and Bethel (1972) say that injured/infected prey exhibit different behavior, which predators can pick up on. This is supported by the observation that wolves will 'test' prey for such response, then will either quickly attack or give up. This case may be more in line biologically with our model than the above examples.

For this section only, we change the notation of the interior equilibrium to be $(S^\bullet, r^\bullet, P^\bullet)$.

Our new system of differential equations becomes:

$$\begin{aligned}\frac{S'}{S} =& g(S) - \sigma h(r),\\ \frac{r'}{r} =& \sigma\xi(r) - \kappa_2 P - \mu - g(S),\\ \frac{P'}{P} =& \gamma_2 r S - \nu.\end{aligned}$$

We also have

$$\begin{aligned}\frac{P'}{P} &= \gamma_2 I - \nu. \qquad (20.2.7)\\ \frac{I'}{I} &= \sigma\frac{h(r)}{r} - \kappa_2 P - \mu.\end{aligned}$$

Recall that

$$r = \frac{I}{S}.$$

For ease, we consider $I^\bullet$ as a parameter, where $I^\bullet = \dfrac{\nu}{\gamma_2}$ is the equilibrium solution to Equation (20.2.7). In the following, we use

$$P^\bullet = \frac{1}{\kappa_2}\Big(\sigma\frac{h(r)}{r} - \mu\Big), \qquad \text{and}$$

$$0 = g(I^\bullet/r^\bullet) - \sigma h(r^\bullet). \qquad (20.2.8)$$

Lemma 20.2.1. *Given $I^\bullet > 0$, there exists a coexistence equilibrium if and only if there is some $r^\bullet > 0$ such that*

$$g(I^\bullet/r^\bullet) = \sigma h(r^\bullet) > \mu r^\bullet.$$

A necessary condition for the inequality is $\sigma h'(0) > \mu$, so we continue this section with this assumption. Consider the set

$$\mathcal{I} = \{Sr; g(S) = \sigma h(r) \geq \mu r, r > 0, S > 0\}.$$

Since $h(0) = 0 < g(0)$, there is some $r > 0$ such that $g(0) > \sigma h(r) > \mu r$. Since $g(K) = 0$, by the intermediate value theorem there is some $S \in (0, K)$ such that $g(S) = \sigma h(r)$. So $\mathcal{I}$ is not empty.

$\mathcal{I}$ is bounded by $Kg(0)/\mu$ and thus has a supremum,

$$I^\sharp = \sup \mathcal{I}.$$

Theorem 20.2.2. *There is no coexistence equilibrium if $I^\bullet > I^\sharp$. If $I^\bullet \in (0, I^\sharp)$, there exists a coexistence equilibrium that is associated with some $r^\bullet < r^\sharp$ where $g(I^\bullet/r^\sharp) > \sigma h(r^\sharp) \geq \mu r^\sharp$.*

Proof. The first statement is evident.

For the second, we first notice that there exists $S^\sharp, r^\sharp > 0$ with $I^\sharp = S^\sharp r^\sharp$ and $\sigma h(r^\sharp) = g(S^\sharp)$ and $\sigma h(r^\sharp) \geq \mu r^\sharp$.

Indeed, we find r_n, S_n such that $r_n S_n \to I^\sharp$. Since $S_n \leq K$ and $r_n \leq g(0)/\mu$, $S_n \to S^\sharp$ and $r_n \to r^\sharp$ after choosing subsequences. Since $I^\sharp > 0$, $S^\sharp > 0$ and $r^\sharp > 0$.

Now let $I^\bullet \in (0, I^\sharp)$. Since g is strictly decreasing,

$$\sigma h(r^\sharp) = g(S^\sharp) = g(I^\sharp/r^\sharp) < g(I^\bullet/r^\sharp).$$

Since $g(I^\bullet/r_1) = 0$ for $r_1 = I^\bullet/K$, by the intermediate value theorem, there is some $r^\bullet \in (r_1, r^\sharp)$ with $\sigma h(r^\bullet) = g(I^\bullet/r^\bullet)$. Since $h(r)/r$ is strictly decreasing, $\sigma h(r^\bullet) > \mu r^\bullet$. We have a coexistence equilibrium by Lemma 20.2.1. □

Theorem 20.2.3. *Let $I^\bullet \in (0, I^\sharp)$. For almost all $\sigma > 0$, the coexistence equilibrium with the smallest r-value is locally asymptotically stable. Similarly, the coexistence equilibrium with the second smallest r-value is unstable (if it exists).*

Proof. By the quotient and chain rule,

$$\frac{d}{dr}\frac{g(I^\bullet/r)}{h(r)} = \frac{g'(I^\bullet/r)(-I^\bullet/r^2)h(r) - g(I^\bullet/r)h'(r)}{h(r)^2}.$$

At a solution $r = r^\bullet$,

$$\frac{d}{dr}\frac{g(I^\bullet/r)}{h(r)} = \frac{g'(I^\bullet/r^\bullet)(-I^\bullet/r^\bullet) - \sigma r^\bullet h'(r^\bullet)}{r^\bullet h(r^\bullet)} = -\frac{g'(S^\bullet)S^\bullet + \sigma r^\bullet h'(r^\bullet)}{r^\bullet h(r^\bullet)}.$$

Let $r_1^\bullet$ be the smallest solution. Since $\dfrac{g(I^\bullet/r_1)}{h(r_1)} = 0$ for $r_1 = I^\bullet/K$,

$$\frac{g(I^\bullet/r)}{h(r)} < \sigma = \frac{g(I^\bullet/r_1^\bullet)}{h(r_1^\bullet)}, \qquad r_1 < r < r_1^\bullet.$$

Hence

$$0 \le -[g'(S_1^\bullet)S_1^\bullet + \sigma r_1^\bullet h'(r_1^\bullet)].$$

By Sard's lemma, applied to $\dfrac{g(I^\bullet/r)}{h(r)}$ as a function of r,

$$0 > g'(S_1^\bullet)S_1^\bullet + \sigma r_1^\bullet h'(r_1^\bullet)$$

for almost all σ. Now we apply Corollary 20.1.3 for our result. □

Corollary 20.2.4. *If the interior equilibrium exists and is unique, it is locally asymptotically stable for almost all $\sigma > 0$.*

Note that $I^\bullet \to 0$ as $\gamma_2 \to \infty$. Thus, by the above, we have that sufficiently increasing γ_2 will result in the existence of an interior equilibrium.

We have proved uniform persistence (and a coexistence equilibrium) if $\sigma h(g(0)/\mu) < g(0)$ and $\gamma_2 S^* r^* > \nu$ by Theorem 19.1.5. We may hope for a stable interior equilibrium which attracts all interior solutions in this case. While we cannot show that, we do know that there is an interior equilibrium and it is locally asymptotically stable, which lends support to the idea. Recall that $S^* r^* = I^*$, and we can see $\gamma_2 S^* r^* > \nu$ is equivalent to $I^* > I^\bullet$. We also note that if I^* exists, $I^* \in \mathcal{I}$, so $I^* \le I^\sharp$.

Thus, we are interested in the cases if $\sigma h(g(0)/\mu) < g(0)$ and $\gamma_2 S^* r^* \geq \nu$, and if $\sigma h(g(0)/\mu) \geq g(0)$. For our next theorem, we note that $I^\bullet \in (I^*, I^\sharp)$ implies $I^* < I^\bullet$, which is equivalent to $\gamma_2 S^* r^* > \nu$.

Theorem 20.2.5. *Let* $\sigma \in \left(\dfrac{\mu}{h'(0)}, \dfrac{g(0)}{h(g(0)/\mu)}\right)$ *and* $I^* \neq I^\sharp$. *If* $I^\bullet \in (I^*, I^\sharp)$, *there exists at least two coexistence equilibria.*

Proof. By Theorem 20.2.2, there exists a coexistence equilibrium $(S_1^\bullet, r_1^\bullet, P_1^\bullet)$ with $\sigma h(r_1^\bullet) > \mu r_1^\bullet$. Further, $r^\bullet < r^\sharp$ where $g(I^\bullet/r^\sharp) > \sigma h(r^\sharp) \geq \mu r^\sharp$. Since $\dfrac{h(r)}{r}$ is strictly decreasing and $\sigma \dfrac{h(r^*)}{r^*} = \mu$, $r_1^\bullet < r^\sharp \leq r^*$.

Further, since g is strictly decreasing, for $r_2 = r^*$,

$$\sigma h(r_2) = g(S^*) = g(I^*/r_2) > g(I^\bullet/r_2).$$

By the intermediate value theorem, there exists some $r_2^\bullet \in (r^\sharp, r^*)$ such that $g(I^\bullet/r_2^\bullet) = \sigma h(r_2^\bullet) > \mu r_2^\bullet$. This $r_2^\bullet$ is associated with a second coexistence equilibrium. □

Recall that in this case, $\gamma_2 S r = \gamma_2 I$ represents the biomass increase of the predator, so $\gamma_2 I^\sharp$ is the maximum possible increase, $\gamma_2 I^*$ is the increase at the host-parasite equilibrium, and $\gamma_2 I^\bullet$ is the increase at the interior equilibrium, which by definition is equal to ν. Thus, if we have $\gamma_2 I^\sharp > \gamma_2 I^\bullet = \nu > \gamma_2 I^*$, the predator cannot invade, however it is still possible for it to survive at the interior equilibrium. This suggests a bistable situation, where trajectories can go to the smallest interior equilibrium or to $(S^*, r^*, 0)$.

In fact, this result can be extended to the case where the host-parasite-only equilibrium no longer exists.

Theorem 20.2.6. *Let* $\sigma h(g(0)/\mu) \geq g(0)$ *and* $I^\bullet \in (0, I^\sharp)$. *Then there exists at least two coexistence equilibria.*

Proof. By Theorem 20.2.2, there exists a coexistence equilibrium $(S_1^\bullet, r_1^\bullet, P_1^\bullet)$ with $\sigma h(r_1^\bullet) > \mu r_2^\bullet$. Further, $r^\bullet < r^\sharp$ where $g(I^\bullet/r^\sharp) > \sigma h(r^\sharp) \geq \mu r^\sharp$. This implies that $r_1^\bullet < r^\sharp \leq g(0)/\mu$.

Further, for $r_2 = g(0)/\mu$, $\sigma h(r_2) > g(I^\bullet/r_2)$. By the intermediate value theorem, there exists some $r_2^\bullet \in (r^\sharp, g(0)/\mu)$ such that $g(I^\bullet/r_2^\bullet) = \sigma h(r_2^\bullet) > \mu r_2^\bullet$. This $r_2^\bullet$ is associated with a second coexistence equilibrium. □

In the cases where we have multiple interior equilibria, we are interested in their stability. If $\sigma h(g(0)/\mu) \geq g(0)$, equivalently, $\sigma\xi(g(0)/\mu) \geq g(0) + \mu$, we have at least two equilibria. Generically, the one with the smallest proportion of infective to susceptible prey is locally asymptotically stable, and the one with the second smallest proportion is unstable. Recall that this is a situation where the parasite drives the host/prey and itself into extinction if there is no predator. This is a bistable situation because extinction also occurs if there are too few predators at the beginning, as the extinction state $(0, r^\circ, 0)$ with $r^\circ \in (0, \infty]$ is locally asymptotically stable ($r^\circ < \infty$ if $\sigma\xi(\infty) < g(0) + \mu$ and $r^\circ = \infty$ is $\sigma\xi(\infty) \geq g(0) + \mu$).

20.2.2 The Predator Consumes Susceptibles and Infectives $\kappa_1 \neq 0$

We now return to the use of $(S^\dagger, r^\dagger, P^\dagger)$ to denote an interior equilibrium. Here we will have that both κ_1 and γ_1 are non zero. If $\kappa_1 \neq 0$, we can substitute 20.2.5 into 20.2.1. Rearranging gives us

$$\sigma\varphi(r) := \sigma h(r^\dagger)\left(\frac{1}{r^\dagger} + \frac{\kappa_2}{\kappa_1}\right) = \mu + \frac{\kappa_2}{\kappa_1} g(S^\dagger). \tag{20.2.9}$$

From Equation (20.2.9) we get bounds on $r^\dagger$. We can only have $r^\dagger$ as a solution if $\sigma\varphi(r^\dagger) \in \left(\mu, \mu + \frac{\kappa_2}{\kappa_1} g(0)\right)$. One may see φ as a perturbation to ξ, more specifically, $\varphi(r) = \xi(r) + \frac{\kappa_2 - \kappa_1}{\kappa_1} h(r)$.

We observe

$$\varphi(0) := \lim_{r\to 0} \varphi(r) = h'(0). \tag{20.2.10}$$

Theorem 20.2.7. *Suppose $\kappa_1 > 0$, and $\gamma_1 > 0$. There exists an interior equilibrium if and only if there is some $r > 0$ such that $\mu r < \sigma h(r)$ and*

$$\sigma\varphi(r) = \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r}\Big).$$

Proof. From Equation (20.2.3) we can solve for $S^\dagger$ and we get $S^\dagger = \dfrac{\nu}{\gamma_1 + \gamma_2 r^\dagger}$. Substituting this into Equation (20.2.9) gives the equation
$\sigma\varphi(r) = \mu + \dfrac{\kappa_2}{\kappa_1} g\Big(\dfrac{\nu}{\gamma_1 + \gamma_2 r}\Big)$, thus our hypothesis provides us with $r^\dagger > 0$.

We see that the $r^\dagger$ which satisfies the equation will imply that $S^\dagger < K$. Suppose not, and that $S^\dagger \geq K$. Then $g(S^\dagger) \leq 0$, and we have

$$\begin{aligned}
\sigma\varphi(r^\dagger) = \sigma h(r^\dagger)\Big(\frac{1}{r^\dagger} + \frac{\kappa_2}{\kappa_1}\Big) &= \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r^\dagger}\Big) \\
&\leq \mu < \sigma \frac{h(r^\dagger)}{r^\dagger} \qquad \text{(by our hypothesis)} \\
&< \sigma \frac{\kappa_2}{\kappa_1} \frac{h(r^\dagger)}{r^\dagger},
\end{aligned}$$

a contradiction. Thus $S^\dagger < K$.

From here, we simply find the solution to Equation (20.2.5) to give us $P^\dagger$, which is guaranteed to be positive by our assumption $\mu r^\dagger < \sigma h(r^\dagger)$. □

We note here that the requirement $\mu r < \sigma h(r)$ implies $\sigma h'(0) > \mu$.

Corollary 20.2.8. *Suppose ξ is strictly decreasing or constant, $\kappa_1 > 0, \quad \gamma_1 > 0$ and*

$$\frac{\mu}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)}.$$

Recall that S^* and r^* satisfy the equations $g(S^*) = r^*\mu = \sigma h(r^*)$. Further recall that if $\gamma_1 K > \nu$, then $S^\diamond = \dfrac{\nu}{\gamma_1}$, and $P^\diamond = \dfrac{g(S^\diamond)}{\kappa_1} = \dfrac{g(\nu/\gamma_1)}{\kappa_1}$.

If any of the following conditions are met, there is an interior equilibrium:

- $\sigma h'(0) < \mu + \kappa_2 P^\diamond, \quad$ *and* $\quad 0 < \gamma_1 S^* + \gamma_2 S^* r^* < \nu < \gamma_1 K,$
- $\mu + \kappa_2 P^\diamond < \sigma h'(0), \quad$ *and* $\quad \nu < \min\{\gamma_1 K, \quad \gamma_1 S^* + \gamma_2 S^* r^*\},$
- $\gamma_1 K \leq \nu < \gamma_1 S^* + \gamma_2 S^* r^*.$

Proof. Note that $\dfrac{\mu}{h'(0)} < \sigma < \dfrac{g(0)}{h(g(0)/\mu)}$ implies $(S^*, r^*, 0)$ exists and that r^* is the unique solution to $\sigma \dfrac{h(r)}{r} = \mu$. $\dfrac{h(r)}{r}$ strictly decreasing and Equation (20.2.5) tell us $r^\dagger < r^*$ will hold if an interior equilibrium exists.

Further, we have if $\gamma_2 < 0$, then for any $0 < r < r^*, \quad 0 < \gamma_1 + \gamma_2 r^* < \gamma_1 + \gamma_2 r$, so $0 < \gamma_1 + \gamma_2 r$ is guaranteed whenever $\gamma_2 < 0$ and $0 < r < r^*$.

First item: $\sigma h'(0) < \mu + \kappa_2 P^\diamond, \quad$ and $\quad 0 < \gamma_1 S^* + \gamma_2 S^* r^* < \nu < \gamma_1 K$:

The assumption $\nu < \gamma_1 K$ will imply that $(S^\diamond, 0, P^\diamond)$ exists. We see that at $r = 0$, our assumptions and Equation 20.2.10 imply

$$\begin{aligned}
\sigma\varphi(0) &= \sigma h'(0) \\
&< \mu + \kappa_2 P^\diamond \\
&= \mu + \frac{\kappa_2}{\kappa_1} g(S^\diamond) \\
&= \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1}\Big) \\
&= \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 \cdot 0}\Big),
\end{aligned}$$

so $\sigma\varphi(0) < \mu + \dfrac{\kappa_2}{\kappa_1} g\Big(\dfrac{\nu}{\gamma_1 + \gamma_2 \cdot 0}\Big)$.

From our assumption $0 < \gamma_1 S^* + \gamma_2 S^* r^* < \nu$ we can see

$$\begin{aligned}
\gamma_1 S^* + \gamma_2 S^* r^* &< \nu \\
S^* &< \frac{\nu}{\gamma_1 + \gamma_2 r^*} \\
g(S^*) &> g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r^*}\Big) \\
\sigma h(r^*) &> g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r^*}\Big) \\
\mu + \frac{\kappa_2}{\kappa_1}\sigma h(r^*) &> \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r^*}\Big) \\
\sigma\varphi(r^*) &> \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r^*}\Big).
\end{aligned}$$

Then, by the intermediate value theorem, there is some $r^\dagger \in (0, r^*)$ such that $\sigma\varphi(r^\dagger) = \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r^\dagger}\Big)$. Now applying Theorem 20.2.7 provides the result.

Second item: $\mu + \kappa_2 P^\diamond < \sigma h'(0), \quad$ and $\quad \nu < \min\{\gamma_1 K, \quad \gamma_1 S^* + \gamma_2 S^* r^*\}$.

Reusing the calculations from the first item with the inequalities reversed, we have that there is some $r^\dagger \in (0, r^*)$ such that $\sigma\varphi(r^\dagger) = \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r^\dagger}\Big)$ in this case as well.

Third item: $\gamma_1 K \leq \nu < \gamma_1 S^* + \gamma_2 S^* r^*$:

Here we will apply Theorem 6.2 from Smith and Thieme (2011). Let $X = \mathbb{R}^3_+$ and $\rho(S, r, P) = \min\{S, r, P\}$, so we check its assumptions. First we check that ρ is concave. We do so by showing the minimum function is superadditive and homogeneous.

Let $x \in \mathbb{R}^n_+$, for some $n \in \mathbb{N}$, $n \geq 1$, with x_i the ith component of x. Let k be the index such that $\min x = \min_i x_i = x_k$. Then for any $\alpha \in \mathbb{R}$,

$$\alpha(\min x) = \alpha(x_k) = \alpha x_k = \min \alpha x,$$

thus the minimum function is homogeneous. Let $y \in \mathbb{R}^n_+$, with y_j as its minimal component, and define $z = x + y$, with z_m is its minimal component. Then

$$\min(x+y) = \min z = z_m = x_m + y_m \geq x_k + y_j = \min x + \min y,$$

so the minimum function is superadditive. Therefore the minimum function is concave, and so ρ is as well.

Next, we note that these the assumptions of the second item of this theorem are the same as the assumptions of Theorem 19.1.2, thus we have uniform ρ persistence. By Theorems 15.1.2 and 15.2.7, we have a bounded attracting set. Since we are considering a finite dimensional system of equations, a bounded attractor implies that the induced semiflow is condensing. Finally we have that once S, r, or P become 0, they will stay zero, so there is no possibility of ρ going from zero to a non-zero quantity. Thus all conditions of Theorem 6.2 from Smith and Thieme (2011) are met, so there is an equilibrium which we denote $(S^\dagger, r^\dagger, P^\dagger)$ with $\rho(S^\dagger, r^\dagger, P^\dagger) = \min\{S^\dagger, r^\dagger, P^\dagger\} > 0$, thus an interior equilibrium will exist. □

Recall from Corollary 18.5.2 and Theorem 18.4.1, that the conditions in the first item imply that both $(S^*, r^*, 0)$ and $(S^\diamond, 0, P^\diamond)$ are locally asymptotically stable, so we have bi-stability. This leads us to believe that the interior equilibrium is a saddle, and acts as a seperatrix between the basins of attraction for the two fixed points.

Theorem 20.2.9. *Suppose ξ is strictly decreasing or constant, $\kappa_1 > 0$, $\gamma_1 > 0$, $S^* + \gamma_2 S^* r^* < \nu < \gamma_1 K$ and*

$$\frac{\mu}{h'(0)} < \sigma < \max\left\{\frac{g(0)}{h(g(0)/\mu)}, \frac{\mu + \kappa_2 P^\diamond}{h'(0)}\right\}.$$

For almost all μ, there is an unstable interior equilibrium.

Proof. By the first item of Theorem 20.2.8 we have the existence of an interior equilibrium, so there is a solution to the equation $\sigma\varphi(r) = \mu + \dfrac{\kappa_2}{\kappa_1} g\Big(\dfrac{\nu}{\gamma_1 + \gamma_2 r}\Big)$. Let $\kappa = \dfrac{\kappa_2}{\kappa_1}$,

and define

$$E(r) := \sigma\xi(r) - \sigma h(r) + \kappa\sigma h(r) - \kappa g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r}\Big).$$

Note that if $E(r^\dagger) = \mu$, then we have an interior equilibrium. Further we note $E(0) = \kappa_1(\sigma h'(0) - \mu - \kappa_2 P^\diamond) < 0$, and $E(r^*) = \sigma\varphi(r^*) - \mu - \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r^*}\Big) > 0$, which can be seen in the proof of Theorem 20.2.8. We consider $E'(r)$, and we see

$$\begin{aligned} E'(r) &= \frac{d}{dr}\Big[\sigma\xi(r) - \sigma h(r) + \kappa\sigma h(r) - \kappa g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r}\Big)\Big] \\ &= \Big[\sigma\xi'(r) - \sigma h'(r) + \kappa\sigma h'(r) - \kappa g'\Big(\frac{\nu}{\gamma_1 + \gamma_2 r}\Big) \cdot \frac{-\gamma_2\nu}{(\gamma_1 + \gamma_2 r)^2}\Big] \\ &= \frac{1}{\nu}\Big[\sigma\nu(\xi'(r) - h'(r) + \kappa h'(r)) - \kappa\gamma_2 g'\Big(\frac{\nu}{\gamma_1 + \gamma_2 r}\Big) \cdot \Big(\frac{\nu}{\gamma_1 + \gamma_2 r}\Big)^2\Big]. \end{aligned}$$

We use $E(0) < \mu < E(r^*)$ to show that there must be some $r^\dagger$ with $E(r^\dagger) = \mu$ and $E'(r^\dagger) \geq 0$. By applying Sard's Lemma to E, we know $E'(r) \neq 0$ for almost all μ, thus we can conclude that $E'(r^\dagger) > 0$ for almost all μ. Finally, we see that

$$E'(r^\dagger) = \frac{1}{\nu}\Big[\sigma\nu(\xi'(r) - h'(r) + \kappa h'(r)) - \kappa\gamma_2 g'(S^\dagger)(S^\dagger)^2\Big].$$

We observe that $E'(r^\dagger) = \dfrac{\kappa_1 r^\dagger P^\dagger}{\nu} Det(J)$. This will imply Expression (20.1.1) is positive, and applying 20.1.2 will show us that the equilibrium is unstable. □

In a similar manner, the second item, Corollary 18.5.2, and Theorem 18.4.1 imply that both boundary equilibria are saddles, and in fact Theorem 19.1.1 implies that we have uniform persistence in this case. This leads us to believe that there is a stable interior equilibrium which attracts positive solutions. We cannot show this in its entirety, however by some slight modifications to the above, we can show that an interior equilibrium is stable, if we add a condition.

Corollary 20.2.10. *Suppose ξ is strictly decreasing or constant, $\kappa_1 > 0, \quad \gamma_1 > 0, \quad \gamma_2 \geq 0, \quad \nu < \min\{\gamma_1 K, \quad S^* + \gamma_2 S^* r^*\}$ and*

$$\frac{\mu + \kappa_2 P^\diamond}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)}.$$

For almost all μ, there is a locally asymptotically stable interior equilibrium.

Proof. Reversing the inequalities and applying Sard's lemma, we get that $E'(r^\dagger) < 0$ for almost all μ, which allows us to apply Corollary 20.1.2, giving us local asymptotic stability. □

Corollary 20.2.11. *Suppose ξ is strictly decreasing or constant, $\kappa_1 > 0, \quad \gamma_1 > 0, \nu < \gamma_1 K$, and*

$$\frac{g(0)}{h(g(0)/\mu)} \leq \sigma < \frac{\kappa_2 P^\diamond + \mu}{h'(0)}.$$

Here, S^* and r^* does not exist, however we still have $S^\diamond = \dfrac{\nu}{\gamma_1}$, and $P^\diamond = \dfrac{g(S^\diamond)}{\kappa_1} = \dfrac{g(\nu/\gamma_1)}{\kappa_1}$.

If $0 < \gamma_1 + \gamma_2 \dfrac{g(0)}{\mu}$, there is an interior equilibrium

Proof. We observe that $\dfrac{g(0)}{h(g(0)/\mu)} \leq \sigma$ is equivalent to $g(0) \leq \sigma h(g(0)/\mu)$, therefore $r_2 = \dfrac{g(0)}{\mu}$. Observe that $0 < \nu < K\gamma_1 + K\gamma_2 r_2$ implies $0 < \gamma_1 + \gamma_2 r$ all $r \in (0, r_2)$. Still considering $r < r_2$ and using $\dfrac{g(0)}{h(g(0)/\mu)} \leq \sigma$ if and only if $\sigma h(g(0)/\mu) \geq g(0)$, we also have

$$\begin{aligned}
\sigma\varphi(r_2) &= \sigma h\Big(\frac{g(0)}{\mu}\Big)\Big(\frac{1}{r_2} + \frac{\kappa_2}{\kappa_1}\Big) \\
&\geq g(0)\Big(\frac{1}{r_2} + \frac{\kappa_2}{\kappa_1}\Big) \\
&= \mu + \frac{\kappa_2}{\kappa_1} g(0) \\
&> \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 r}\Big).
\end{aligned}$$

Thus, since $\sigma\varphi(0) = \sigma h'(0) < \mu + \kappa_2 P^\diamond = \mu + \frac{\kappa_2}{\kappa_1} g\Big(\frac{\nu}{\gamma_1 + \gamma_2 \cdot 0}\Big)$, the intermediate value theorem guarantees some $r^\dagger \in \Big(0, \frac{g(0)}{\mu}\Big)$ such that the conditions of Theorem 20.2.7 are satisfied. □

This corollary extends the first item in Theorem 20.2.8 to the case where $(S^*, r^*, 0)$ no longer exists, however we now have an extinction 'equilibrium' which is locally asymptotically stable. This again suggests a bi-stable situation, where solutions are attracted either to $(S^\diamond, 0, P^\diamond)$, or $(0, r^\circ, 0)$, where r° could be infinity. We can reuse the calculations from Theorem 20.2.9 with $\frac{g(0)}{\mu}$ in place of r^* to prove an analogous result for our current parameter range.

Corollary 20.2.12. *Suppose ξ is strictly decreasing or constant, $\kappa_1 > 0$, $\gamma_1 > 0, \nu < \gamma_1 K$, and*

$$\frac{g(0)}{h(g(0)/\mu)} \leq \sigma < \frac{\kappa_2 P^\diamond + \mu}{h'(0)}.$$

For almost all μ, there is an unstable interior equilibrium.

These bi-stable cases are further discussed in Section 21.2.

Chapter 21

PARASITE MEDIATED COLLAPSE OF THE ECOSYSTEM

21.1 At All Predator Levels

Here we show conditions such that the disease will collapse the ecosystem no matter the initial condition of the predator. Recall from Theorem 15.1.2 that P has an asymptotic time bound $P^{\odot}$, which is independent of initial conditions, and independent of σ.

Theorem 21.1.1. *The state* $(S, r, P) = (0, \infty, 0)$ *attracts all solutions from* $(0, \infty)^3$ *if*

$$\frac{\mu + g(0) + (\kappa_2 - \kappa_1)P^{\odot}}{\inf_{r \geq 0} \xi(r)} < \sigma.$$

Proof. Choose $\varepsilon > 0$ such that $\dfrac{\mu + g(0) + (\kappa_2 - \kappa_1)(P^{\odot} + \varepsilon)}{\inf_{r \geq 0} \xi(r)} < \sigma$. Next, perform a time shift, such that $P(t) < P^{\odot} + \varepsilon$ for all $t \geq 0$. We observe that the hypothesis implies that $g(0) < \sigma h(r^{\sharp})$, for some finite $r^{\sharp}$.

Now, rearranging our hypothesis, we observe

$$\frac{r'}{r} = \sigma\xi(r) - \mu + (\kappa_1 - \kappa_2)P - g(S) > \sigma \inf_{r \geq 0} \xi(r) - \mu + (\kappa_1 - \kappa_2)(P^{\odot} + \varepsilon) - g(0) > 0$$

for all r. Thus, we have that $r \to \infty$ as $t \to \infty$. Therefore, there is some time τ such that $r(t) > r^{\sharp}$ for all $t \geq \tau$. This implies $\dfrac{S'}{S} = g(S) - \sigma h(r) - \kappa_1 P < g(0) - \sigma h(r^{\sharp}) < 0$, so $S(t) \to 0$ as $t \to \infty$.

Finally we use 15.1.3 to give us that the ecosystem collapses indeed. □

We can achieve this result by increasing σ as much as needed, since none of the parameters on the left hand side of the inequality depend on σ.

Recall that ξ strictly decreasing implies $\xi(r) \geq \xi(\infty) = h(\infty)$, i.e. $\inf_{r\geq 0} \xi(r) = h(\infty)$.

Corollary 21.1.2. *If ξ is decreasing, the state $(S, r, P) = (0, \infty, 0)$ attracts all solutions from $(0, \infty)^3$ if*

$$\frac{\mu + g(0) + (\kappa_2 - \kappa_1)P^{\odot}}{h(\infty)} < \sigma.$$

21.2 At Low Predator Levels

Recalling the interior equilibrium discussion from Section 20.2 and local stability of boundary equilibria from Section 18 we see the possibility of ecosystem survival at high predator levels and ecosystem extinction at low predator levels. This would create a bistable situation, where some initial conditions would lead to the collapse of the ecosystem, while others would lead to survival of the ecosystem.

We can create one such scenario via combining conditions for existence and local stability of the predator prey equilibrium $(S^{\diamond}, 0, P^{\diamond})$ and extinction equilibrium $(0, r^{\circ}, 0)$, so both will exist and be locally stable. Note that the bistable situation created here implies extinction of the parasite or extinction of the predator, prey, and parasite, via the parasite. Combining and simplifying the requirements from Theorem 18.4.1 and Corollary 18.3.1 we have

$$\frac{g(0)}{h(g(0)/\mu)} < \sigma < \min\left\{\frac{\mu + g(0)}{h(\infty)}, \frac{\kappa_2 P^{\diamond} + \mu}{h'(0)}\right\}, \qquad \gamma_1 K > \nu, \text{ and}$$

$$\xi \text{ strictly decreasing}, \qquad \text{where } P^{\diamond} = \frac{g(S^{\diamond})}{\kappa_1} = \frac{g(\nu/\gamma_1)}{\kappa_1}.$$

Comparing our current conditions to persistence requirements from Theorems 19.1.1, and 19.1.2, we see that Equation (19.1.3) tells us $\sigma < \dfrac{g(0)}{h(g(0)/\mu)}$, which is mutually

exclusive with our condition above, as we would expect. Since ξ is decreasing we have Lemma 12.12.1, which implies $\dfrac{g(0)}{h(g(0)/\mu)} < \dfrac{\mu + g(0)}{h(\infty)}$, thus our main concern is making $\dfrac{\kappa_2 P^\diamond + \mu}{h'(0)}$ large enough. To that end, we observe that no variables above depend on κ_2, with the obvious exception of κ_2 itself. Thus, the above can easily achieved by increasing κ_2 until the inequality $\dfrac{g(0)}{h(g(0)/\mu)} < \dfrac{\kappa_2 P^\diamond + \mu}{h'(0)}$ holds, without effecting anything else in the inequality. We also recall from Corollary 20.2.9 that for almost all μ, these conditions imply the existence of an unstable interior equilibrium. Given the bistable scenario, we suspect it will be a saddle point which acts as a seperatrix for the solutions between the two points.

However, if we want bistability between $(S^\diamond, 0, P^\diamond)$, and the state $(0, \infty, 0)$, we can accomplish this by meeting the requirements from Theorem 18.4.1 and Theorem 18.3.2. This simplifies to

$$\frac{\mu + g(0)}{h(\infty)} \leq \sigma < \frac{\kappa_2 P^\diamond + \mu}{h'(0)}, \qquad \gamma_1 K > \nu.$$

In this scenario we no longer require ξ to be strictly decreasing, due to the relaxed requirements in Theorem 18.3.2. This inequality can be made to hold in the same way as above, namely increasing κ_2 sufficiently. As above, Corollary 20.2.9 implies that for almost all μ, we have an unstable interior equilibrium, which we suspect will be a saddle point which acts as a seperatrix for the solutions between the two points.

This is the case of the predator preventing the disease from invading the predator-prey system, however if the parasite is introduced with large enough numbers, it can eliminate the host species.

Chapter 22

PREDATOR PREYS ON INFECTIVES ONLY

In this case we have $\gamma_1 = \kappa_1 = 0$. Preliminary discussion and analysis is presented in Section 20.2.1 We will only consider $\gamma_2 > 0$, otherwise the predator will go extinct for any initial conditions. Additionally, we require ξ strictly decreasing. This gives us the same ordering that is found in Section 12.12.

$$\frac{\mu}{h'(0)} < \frac{\mu + g(0)}{h'(0)} < \frac{g(0)}{h(g(0)/\mu)} < \frac{\mu + g(0)}{h(\infty)}. \tag{22.0.1}$$

For almost all parameters, we have an idea of the dynamics to expect. We recall that the point $(K, 0, 0)$ always exists, and it is unstable if $\frac{\mu}{h'(0)} < \sigma$, with the unstable manifold pointing into the $S - r$ plane, so it will draw no solutions from the interior, as seen in Section 18.2. We recall the existence conditions of some other boundary equilibria when ξ is strictly decreasing. The equilibrium $(S^*, r^*, 0)$ exists if $\frac{\mu}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)}$, with $\sigma h(r^*) = \mu r^* = g(S^*)$. Using that $I = Sr$, we have $I^* = S^* r^*$, which will be useful for categorizing the dynamics neatly. The equilibrium $(0, r^\circ, 0)$ exists if $\frac{\mu + g(0)}{h'(0)} < \sigma < \frac{\mu + g(0)}{h(\infty)}$. Also recall from Section 20.2.1 that $I^\sharp = \sup\{Sr; g(S) = \sigma h(r) \geq \mu r, r > 0, S > 0\}$, and if I^* exists, $I^* \leq I^\sharp$. Finally we recall, from Section 20.2.1 as well, that $I^\bullet := \frac{\nu}{\gamma_2}$.

22.1 $\sigma \leq \frac{\mu}{h'(0)}$

If $P(0) = 0$, our considerations in Chapter 12 imply all solutions with $S(0) > 0$ and $r(0) \geq 0$ will converge to $(K, 0, 0)$. If $P(0) > 0$, we next note that the predator requires infectives to survive, thus we know that there will be trouble in any case

where the parasite does not persist. We observe that $\sigma h'(0) > \mu$ is the requirement for Corollary 19.2.5, which gives the persistence of r, and we have $\sigma h'(0) \leq \mu$.

To see the extent of the trouble the predators will have, we use the fact that ξ strictly decreasing implies $h'(0) = \xi(0) = \sup \xi(r)$, and so Theorem 17.1.1 can be applied. This tells us that $S(t) \to K$, $r(t) \to 0$, and $P(t) \to 0$ as $t \to \infty$, i.e. (K,0,0) is globally stable.

Neither $(S^*, r^*, 0)$ nor $(0, r^\circ, 0)$ exist in this case.

$$22.2 \quad \frac{\mu}{h'(0)} < \sigma \leq \frac{\mu + g(0)}{h'(0)}$$

Here $(S^*, r^*, 0)$ exists, however $(0, r^\circ, 0)$ does not exist. If $P(0) = 0$, Chapter 12 tells us all solutions with $S(0) > 0$ and $r(0) > 0$ will converge to $(S^*, r^*, 0)$. If $P(0) > 0$, Theorem 18.5.1, using $\gamma_1 = 0$, tells us that the local stability of $(S^*, r^*, 0)$ is linked to the sign of $\gamma_2 S^* r^* - \nu$; more specifically if this expression is negative, the point is locally asymptotically stable, and a saddle if the expression is positive, with the unstable manifold pointing into the interior. We see that $\gamma_2 S^* r^* - \nu$ can be rewritten as $\frac{1}{\gamma_2}\left(S^* r^* - \frac{\nu}{\gamma_2}\right) = \frac{1}{\gamma_2}\left(I^* - I^\bullet\right)$, Thus the sign of $I^* - I^\bullet$ can be used to determine the stability of $(S^*, r^*, 0)$ in the same way as $\gamma_2 S^* r^* - \nu$. Now we will consider cases, depending on the relationship between $I^\bullet$, and I^* and $I^\sharp$. Note that we can use Theorem 15.2.7, since by Equation 22.0.1 we have $\sigma < \frac{\mu}{h'(0)} < \frac{\mu + g(0)}{h(\infty)}$, thus r is bounded. Furthermore, Corollary 19.2.2 and Theorem 19.2.4 are both satisfied for all subcases, thus both S and r will persist uniformly for all positive initial conditions.

Case 1: $I^\bullet < I^*$

For almost all σ we will have a stable interior equilibria, as seen in Theorem 20.2.3. If there is more than one interior equilibrium, for almost all σ there will be an odd

number of interior equilibria, and the stability will alternate between unstable and stable, if they are ordered by r value from least to greatest. Additionally, Theorem 18.5.1 and $I^\bullet < I^*$ imply the boundary equilibrium $(S^*, r^*, 0)$ is a saddle, with the unstable manifold pointing into the interior.

These facts together lead us to believe that if the interior equilibrium is unique, all solutions with positive initial conditions will tend toward the stable interior equilibrium. If there are an odd number of equilibria, we believe that each unstable equilibrium will act as a seperatrix, dividing the interior into regions. Each region will contain a stable interior equilibrium, which will attract all solutions in its region. We note here that the conditions of Theorem 19.2.3 are met, thus P persists for all initial conditions with $S(0), r(0)$, and $P(0)$ positive.

Case 2: $I^* < I^\bullet < I^\sharp$

This case is not guaranteed, as we only know that $I^* \leq I^\sharp$. If equality holds, this case cannot occur.

If it does occur, we are guaranteed two interior equilibrium due to 20.2.5, and, as above, Theorem 20.2.3 gives us that for almost all σ, the point with the smallest r value will be stable, and the next will be unstable, continuing to alternate if there are more. Here, $I^* < I^\bullet$ and Theorem 18.5.1 implies the boundary equilibrium $(S^*, r^*, 0)$ will be locally asymptotically stable. Thus we believe that if there are two interior equilibria, the unstable equilibrium will act as a seperatrix, and solutions will tend toward either the interior equilibrium, or $(S^*, r^*, 0)$, depending on which region they begin in.

In a similar fashion as case 1, we expect that for almost all σ there will be an even number of interior equilibria. Each pair will have one stable and one unstable equilibrium. We expect each pair to split the interior into another region with its

own stable fixed point to attract all solutions within its region, with the exception of one region which contains $(S^*, r^*, 0)$ as its stable equilibrium. Thus, we have initial condition dependent persistence of the predator.

Case 3: $I^\sharp < I^\bullet$

Theorem 20.2.2 tells us that there is no interior equilibrium in this case. Note that $I^* \leq I^\sharp < I^\bullet$ implies the local asymptotic stability of $(S^*, r^*, 0)$ via Theorem 18.5.1. This leads us to believe that this point is in fact globally stable.

$$22.3 \quad \frac{\mu + g(0)}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)}$$

This parameter set tells us that $(S^*, r^*, 0)$ and $(0, r^\circ, 0)$ both exist! One can see from Section 18.3.1 that $(0, r^\circ, 0)$ will be a saddle, with its stable manifold lying on the boundary, thus taking no solutions from the interior. Theorem 15.2.7, which supplies us with the boundedness of r, still applies since $\sigma < \frac{g(0)}{h(g(0)/\mu)} < \frac{\mu + g(0)}{h(\infty)}$ holds, as seen in Equation 22.0.1. Besides this addition, the dynamics will not change from the above section, with the exception that we know that for some σ we are guaranteed to observe case 2, since $I^* \to 0$ as $\sigma \to \frac{g(0)}{h(g(0)/\mu)}$, which implies the interval $(I^*, I^\bullet) \to (0, I^\bullet)$ as $\sigma \to \frac{g(0)}{h(g(0)/\mu)}$.

$$22.4 \quad \frac{g(0)}{h(g(0)/\mu)} \leq \sigma < \frac{\mu + g(0)}{h(\infty)}$$

Now $(S^*, r^*, 0)$ no longer exists, and $(0, r^\circ, 0)$ exists and is locally asymptotically stable, via Theorem 18.3.1. The elimination of $(S^*, r^*, 0)$ also implies I^* does not exist, and so we will no longer see the case of $I^\bullet < I^*$. If $P(0) = 0$, we can see from Chapter 12 that all solutions with $S(0) \geq 0$ and $r(0) > 0$ will converge to $(0, r^\circ, 0)$.

If $P(0) > 0$, we can still use Theorem 15.2.7, since $\sigma < \dfrac{\mu + g(0)}{h(\infty)}$, to tell us that r is bounded and Theorem 19.2.4 for the uniform persistence of r, however we are not guaranteed persistence of S.

Case 1: $I^\bullet < I^\sharp$

Again, we are guaranteed two interior equilibria, however this time we will use Theorem 20.2.6 instead of Theorem 20.2.5. Theorem 20.2.3 still applies, so for almost all σ, the point with the smallest r value will be stable and the next will be unstable, continuing to alternate if there are more. Thus we believe that if there are two interior equilibria, the unstable equilibrium will act as a seperatrix, and solutions will tend toward either the stable interior equilibrium, or $(0, r^\circ, 0)$, depending on which region they begin in.

In a similar fashion as above, we expect that for almost all σ there will be an even number of interior equilibria. Each pair will have one stable and one unstable equilibrium. We expect each pair to split the interior into another region with its own stable fixed point to attract all solutions within its region, with the exception of one region which contains $(0, r^\circ, 0)$ as its stable equilibrium. Thus we will have initial condition dependent persistence of both S and P.

Case 2: $I^\sharp < I^\bullet$

Theorem 20.2.2 tells us that there is no interior equilibrium in this case. Since $(0, r^\circ, 0)$ is locally asymptotically stable via Theorem 18.3.1, we believe that this point is in fact globally stable.

$$22.5 \quad \frac{\mu + g(0)}{h(\infty)} \leq \sigma$$

In this case, neither $(S^*, r^*, 0)$ nor $(0, r^\circ, 0)$ exist, and r is no longer guaranteed to be bounded. Now if $P(0) = 0$, Chapter 12 gives us all solutions with $S(0) \geq 0$ and $r(0) > 0$ will converge to the state $(0, \infty, 0)$. For 3 dimensions, Theorem 18.3.2 informs us that the state $(0, \infty, 0)$ is locally asymptotically stable, by which we mean there exists positive S_0, r_0 and P_0, with $I_0 = r_0 S_0$, such that the set $\{Sr \leq S_0 r_0\} \times \{r \geq r_0\} \times \{P \leq P_0\}$ is forward invariant for all solutions in the set, which implies that solutions in the set have the property $r(t) \to \infty$, $S(t) \to 0$, $I(t) \to 0$, and $P(t) \to 0$ as $t \to \infty$. Since $(S^*, r^*, 0)$ does not exist I^* does not either and again we do not have the case of $I^\bullet < I^*$. Finally we see Theorem 19.2.4 still applies, and so r persists uniformly strongly.

Case 1: $I^\bullet < I^\sharp$

Once more, we are guaranteed two interior equilibria, and again we will use Theorem 20.2.6 to show it. Theorem 20.2.3 still applies, so for almost all σ, the point with the smallest r value will be stable, and the next will be unstable, continuing to alternate if there are more. Thus we believe that if there are two interior equilibria, the unstable equilibrium will act as a seperatrix, and solutions will tend toward either the stable interior equilibrium, or $(0, \infty, 0)$, depending on which region they begin in.

We still expect that for almost all σ there will be an even number of interior equilibria. Each pair will have one stable and one unstable equilibrium. We expect each pair to split the interior into another region with its own stable fixed point to attract all solutions within its region, with the exception of one region which

contains $(0, \infty, 0)$ as its stable 'equilibrium.' Thus we will have initial condition dependent persistence of both S and P.

Case 2: $I^\sharp < I^\bullet$

Theorem 20.2.2 tells us that there is no interior equilibrium in this case. Since $(0, \infty, 0)$ is locally asymptotically stable via Theorem 18.3.2 we believe that this state is in fact globally stable.

22.6 Predator Persistence Revisited

We note that with our parameters, we are only guaranteed predator persistence for all initial conditions by Theorem 19.2.3 if $\frac{\mu}{h'(0)} < \sigma < \frac{g(0)}{h(g(0)/\mu)}$ and $\nu < \gamma_2 S^* r^*$. The second condition can be rewritten as $\frac{\nu}{\gamma_2} < S^* r^*$, which is equivalent to $I^\bullet < I^*$. This provides us with a small, but very clear, set of parameters which will ensure that the predator does not die out. If either of these conditions are violated, there is the potential for predator extinction, however so long as $I^\bullet < I^\sharp$, there is always a possibility for the predator to persist, given the proper initial conditions.

Notably, even in cases where the parasite would drive the host to extinction without the predator (i.e. $P(0) = 0$), so long as $I^\bullet < I^\sharp$, there are always initial conditions which lead to an equilibrium without extinction thanks to the predator. Additionally, we recall $I^\sharp = \sup\{Sr; g(S) = \sigma h(r) \geq \mu r, r > 0, S > 0\}$, and $I^\bullet := \frac{\nu}{\gamma_2}$, so we can easily decrease $I^\bullet$ via either decreasing ν or increasing γ_2 without changing the value of $I^\sharp$, to ensure that the predator has the potential to rescue the system regardless of the value of σ.

Chapter 23

DISCUSSION

Many authors have analyzed predator-prey-parasite models using the density-dependent incidence (Arino *et al.* (2004); Bairagi *et al.* (2007); Chattopadhyay *et al.* (2003); Mukherjee (2016); Khan *et al.* (2016); Venturino (1994); Xiao and Chen (2001a, 2002, 2001b); Yongzhen *et al.* (2011), to name a few). Few authors (that we are aware of) use frequency dependent incidence: Haque *et al.* (2009); Hethcote *et al.* (2004); Ghosh and Li (2016). We focus on homogeneous incidences because they can cause extinction in the SI subsystem, and the relative ease in analysis when compared to power laws. For predator-prey interactions, we use a simple, density dependent term. For more complex predator-prey interactions, see Arino *et al.* (2004); Bairagi *et al.* (2007); Chen and Wen (2016); Ghosh and Li (2016); Mukherjee (2016); Khan *et al.* (2016); Xiao and Chen (2001a, 2002); Yongzhen *et al.* (2011), where, in some cases, the infection dynamics are simplified, When a specific incidence function is needed (i.e. for numerical simulations), we choose asymmetric frequency dependent incidence, due to the poor fit that frequency dependence had on the data from Greer *et al.* (2008).

A similar model (although with a different biological interpretation) is analyzed in Ruan and Freedman (1991), using general functions for predation and infection, where conditions for persistence are found. Thanks to Xiao and Chen (2001a, 2002, 2001b); Yongzhen *et al.* (2011) and the references therein, we found a biological basis for the analysis performed in Section 20.2.1 and Chapter 22, transforming it from a purely mathematical exercise, into an exercise with reasonable biological ties.

We recall that we expected to see the following phenomena:

(i) Predator-mediated extinction of the parasite and survival of the prey and predator

(ii) Parasite-mediated extinction of the predator and survival of the prey and parasite

(iii) Parasite-mediated persistence of the predator

(iv) Predator-mediated survival of all three species at high initial predator levels and parasite-mediated extinction of all three species at low initial predator levels

(v) Predator-mediated extinction of the parasite and survival of the prey and predator at high initial predator levels and parasite-mediated extinction of all three species at low initial predator levels

(vi) Parasite-mediated extinction of all three species at all initial predator levels

(vii) Persistence of all three species.

We also recall that for the predator-prey-only equilibrium, $P^\diamond = \frac{1}{\kappa_1} g(S^\diamond)$ and $S^\diamond = \frac{\nu}{\gamma_1}$. The parasite-prey-only equilibrium satisfies $g(S^*) = \sigma h(r^*) = \mu r^*$. Here we only consider when ξ is strictly decreasing or constant. Some of the phenomena were only observed locally, however there are still many interesting situations that may occur.

Locally we see (i) by Theorem 18.4.1. Here we have the existence of $(S^\diamond, 0, P^\diamond)$ if $\nu < \gamma_1 K$ and its local stability if $\sigma h'(0) < \mu + \kappa_2 P^\diamond$. The first condition is a comparison of predator death rate and expected biomass from the prey species at its carrying capacity. In order for the predator to persist, it needs to have more incoming biomass ($\gamma_1 K$) than outgoing biomass (ν). The second condition is a comparison between the

infective force of the parasite and the total death rate of infective individuals, including death by predator. If the infective force is too small, the number of new infective individuals will not be able to overcome the combined death rate by predator and parasite, and so the infective population will die out, and the parasite will die with it. The point $(S^\diamond, 0, P^\diamond)$ becomes a saddle if $\sigma h'(0) > \mu + \kappa_2 P^\diamond$, i.e. that the parasite's infectivity is strong enough to overcome the death rate of infective hosts due to the predator and parasite.

(ii) can be seen locally as well, but via Theorem 18.5.1 and Corollary 18.5.2 instead. The Theorem gives us existence of $(S^*, r^*, 0)$ if $\sigma \in \left(\frac{\mu}{h'(0)}, \frac{g(0)}{h(g(0)/\mu)}\right)$, and the Corollary implies local stability if $\nu > \gamma_1 S^* + \gamma_2 S^* r^*$. The first condition concerns the infectivity of the parasite being enough to become endemic in the host species, but not so strong as to cause extinction. More precisely, we mean to say that the condition $\sigma h'(0) > \mu$ allows the parasite to invade the equilibrium (K,0,0), and $\sigma h(g(0)/\mu) < g(0)$ prevents the disease from causing extinction in the absence of the predator. The second condition is that the expected biomass from a predator hunting ($\gamma_1 S^* + \gamma_2 S^* r^*$) is less than the predator's death rate (ν). Again, if the second inequality is reversed (i.e. $\nu < \gamma_1 S^* + \gamma_2 S^* r^*$) then the Corollary implies $(S^*, r^*, 0)$ is a saddle, since the predator has a net increase to biomass near the point.

We can achieve either, neither, or both of phenomenon (i) or (ii), since their conditions are not mutually exclusive. Conditions for (i) or (ii) globally have not yet been found. We see in the case of both having the same type of stability (i.e. both saddles or both stable), that an interior equilibrium is guaranteed to exist by Theorem 20.2.8. If both are saddle points, then an interior equilibrium will be stable (Theorem 20.2.10). While we do not know the global stability at this point, we suspect it is globally stable because Theorem 19.1.1 will give us uniform persistence of S, r, and P, which is phenomenon (vii). If both equilibria are stable then the interior equilibrium

will be unstable (Theorem 20.2.9), and we suspect that the interior equilibrium acts an a seperatrix for solutions, so solutions will either converge to $(S^*, r^*, 0)$ or $(S^\diamond, 0, P^\diamond)$.

Phenomenon (vii) is most easily seen by applying either Theorem 19.1.1 or Theorem 19.1.2. The assumptions of Theorem 19.1.1 are

$$\nu < \min\{\gamma_1 K, \gamma_1 S^* + \gamma_2 S^* r^*\} \quad \text{and} \quad \sigma \in \Big(\frac{\mu + \kappa_2 P^\diamond}{h'(0)}, \frac{g(0)}{h(g(0)/\mu)}\Big),$$

This is case, as described above, is where any boundary equilibrium can be invaded by the predator or the prey, and both can survive in the absence of the other. More precisely, if there are no predators, the parasite will persist, if there is no parasite, the predator will persist, and in all cases, the prey persists. Here, we appear to have the intersection of two healthy ecosystems resulting in coexistence of all three species. The assumptions of Theorem 19.1.2 are

$$\gamma_1 K < \nu < \gamma_1 S^* + \gamma_2 S^* r^* \quad \text{and} \quad \sigma \in \Big(\frac{\mu}{h'(0)}, \frac{g(0)}{h(g(0)/\mu)}\Big).$$

This is the case where the predator may still eat healthy prey, but can no longer survive on healthy prey alone, and requires the parasite to allow it to persist. The parasite allows the predator to persist by creating prey which is easier to catch, and likely of a similar nutritional value as healthy prey. This is a case of phenomenon (iii), however any time we witness phenomenon (iii), we also have phenomenon (vii).

Although by the above we have phenomenon (iii) via Theorem 19.1.2, we also have different cases with interesting dynamics. We begin by considering the case of the predator eating infective prey only, i.e. $\kappa_1 = \gamma_1 = 0$, and when $\sigma \in \Big(\frac{\mu}{h'(0)}, \frac{g(0)}{h(g(0)/\mu)}\Big)$. In this case, we require $I^\bullet := \dfrac{\nu}{\gamma_2} < \sup\{Sr; g(S) = \sigma h(r) \geq \mu r, r > 0, S > 0\} =: I^\sharp$ for an interior equilibrium to exist (Theorem 20.2.2). By Theorem 20.2.3 we also know that for almost all σ that an interior equilibrium will be stable. Defining $I^* = S^* r^*$, with S^* and r^* the values of the endemic equilibrium, we get differing scenarios depending on the relationship between I^* and $I^\bullet$. Note that $I^* \leq I^\sharp$ by the definition

of $I^\sharp$. Considering the case $I^\bullet < I^*$, we also have persistence of all three species from Theorem 19.1.2, which requires $\nu < \gamma_2 S^* r^*$, which can be written equivalently as $I^\bullet = \dfrac{\nu}{\gamma_2} < S^* r^* = I^*$. Thus we have a stable interior equilibrium, and persistence of all three species, so we suspect the interior equilibrium is globally stable for positive initial conditions. Again, we note that this persistence result is also a case of phenomenon (vii).

To begin with phenomenon (iv), we continue with $\kappa_1 = \gamma_1 = 0$ and we note, using the reverse of the inequality above and Theorem 18.5.2, that $I^\bullet > I^*$ is equivalent to local stability of $(S^*, r^*, 0)$ when $\gamma_1 = 0$. If $I^* < I^\bullet \leq I^\sharp$ then we are guaranteed a stable and an unstable interior equilibrium by Theorems 20.2.3 and 20.2.5. Therefore, if $I^* < I^\bullet \leq I^\sharp$, then $(S^*, r^*, 0)$ is locally stable, and we have two interior equilibria. We suspect in this case, the unstable interior equilibrium acts as a seperatrix between the stable interior equilibrium (where the predator persists thanks to the parasite) and $(S^*, r^*, 0)$. Having exhausted the possibilities when $\dfrac{\mu}{h'(0)} < \sigma < \dfrac{g(0)}{h(g(0)/\mu)}$, we consider what happens once $\sigma \geq \dfrac{g(0)}{h(g(0)/\mu)}$. Here the extinction state $(0, r^\circ, 0)$, with r° possibly being infinite, is locally asymptotically stable no matter the remaining parameters. Using Theorems 20.2.3 and 20.2.6, we will have a stable and an unstable interior equilibrium, just as above, and we expect the unstable interior equilibrium functioning as a seperatrix between extinction and persistence of the ecosystem. In fact, we see that the dynamics mostly remain the same, except that our bistability will be between extinction and coexistence of all three species, instead of between host-parasite-only dynamics and coexistence of all species. This is due to the collision of $(S^*, r^*, 0)$ with $(0, r^\circ, 0)$ when σ crosses (positively) over $\dfrac{g(0)}{h(g(0)/\mu)}$, and the change of stability of $(0, r^\circ, 0)$ after the collision occurs.

When $\kappa_1 > 0$, $\nu < \gamma_1 K$, and $\dfrac{g(0)}{h(g(0)/\mu)} \leq \sigma < \dfrac{\kappa_2 P^\diamond + \mu}{h'(0)}$ we can see phenomenon (v) occurring in a similar fashion as for phenomenon (iv). Corollaries 20.2.11 and

20.2.12 will imply the existence and instability of an interior equilibrium. Theorem 18.3.2 and Corollary 18.3.1 will imply the local stability of the state $(0, r^\circ, 0)$, and Theorem 18.4.1 will imply the local stability of $(S^\diamond, 0, P^\diamond)$. Thus, we believe the interior equilibrium is a seperatrix between solutions converging to $(0, r^\circ, 0)$ and solutions converging to $(S^\diamond, 0, P^\diamond)$.

Phenomenon (vi) can be seen in Theorem 21.1.1, which requires $\frac{\mu + g(0) + (\kappa_2 - \kappa_1)P^\odot}{h(\infty)} < \sigma$. In Section 21.1 and Theorem 15.1.2, we recall that $P^\odot$ is independent of σ. Thus, while the condition may be strong, the left hand side of the inequality is independent of σ, so it is possible for this inequality to hold. The inequality implies that the parasite is virulent enough to continue infecting new hosts, while withstand the death rates due to the predator and the parasite itself, as well as overcoming the decrease in susceptible hosts, until it drives all three species to extinction. In this case, the host-parasite ratio will grow without bound, which will drive the prey and parasite into extinction, and the predator must follow. We note that the occurrence of phenomenon (vi) excludes the occurrence of any other phenomena.

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APPENDIX A

EXTINCTION OF INFECTIVES

Proof of Theorem 2.4.1. Assume that I is a nonnegative bounded uniformly continuous function on $\mathbb{R}_+$ and S a nonnegative solution of (2.4.1) on $\mathbb{R}_+$. Since $f(S, I) \geq 0$, $S' \leq 0$ and S is decreasing and $S(t) \to S_\infty$ as $t \to \infty$ for some $S_\infty \geq 0$. Since S and I are bounded on $\mathbb{R}_+$ and f is continuous, S' is bounded on $\mathbb{R}_+$ by (2.4.1), and S is uniformly continuous on $\mathbb{R}_+$. Since I is uniformly continuous by assumption, $f(S(t), I(t))$ is a uniformly continuous function of $t \in \mathbb{R}_+$ and so is $S'(t)$ by (2.4.1). By Barbalat's Lemma Barbalat (1959) (see also (Thieme, 2003, Cor.A.18)), $S'(t) \to 0$ as $t \to \infty$. By (2.4.1), $f(S(t), I(t)) \to 0$ as $t \to \infty$. □

Proof of Theorem 2.4.2. Adding (2.4.1) and (2.4.3) yields $(S + I)' \leq 0$ which implies that $S(t) + I(t)$ are a decreasing and thus bounded function of $t \in \mathbb{R}_+$, since S and I are nonnegative. Equation (2.4.3) and the boundedness of S and I imply that I' is bounded, and thus I is uniformly continuous. So our previous consideration apply and $f(S(t), I(t)) \to 0$ as $t \to \infty$. By the fluctuation method, there is a sequence (t_n) in $\mathbb{R}_+$ with $t_n \to \infty$ such that $I'(t_n) \to 0$ and $I(t_n) \to I^\infty$. This implies that

$$0 = \lim_{n\to\infty} [f(S(t_n), I(t_n)) - \mu I(t_n)] = 0 - \mu I^\infty.$$

So $I(t) \to 0$ as $t \to \infty$. □

APPENDIX B

EPIDEMIC OUTBREAKS EX NIHILO FOR POWERS OF I

While it is clear that, in (4.1.3), for $I_0 = 0$,

$$I^{1-p}(t) = \int_0^t (1-p)\theta(S(t-r))e^{-(1-p)\mu r}dr \tag{B.1.1}$$

provides a solution I for given S, this may be not so clear when S is also given by a differential equation. For simplicity, let $\theta(S) = \sigma S$. Then $S(t) = S_0 \exp\big(-\sigma \int_0^t I^p(s)ds\big)$. We substitute this into (B.1.1),

$$I^{1-p}(t) = \int_0^t (1-p)\sigma S_0 \exp\Big(-\sigma \int_0^{t-r} I(s)ds\Big)e^{-(1-p)\mu r}dr.$$

We see that $I \equiv 0$ is not a solution of this equation. To see whether this has a solution at least, we set $x(t) = I^{1-p}(t)$ and obtain the following nonstandard Volterra integral equation for x:

$$x(t) = \int_0^t (1-p)\sigma S_0 \exp\Big(-\sigma \int_0^{t-r} (x(s))^{p/(1-p)}\, ds\Big)e^{-(1-p)\mu r}dr, \qquad t \geq 0.$$

This can be solved in the usual way by the Schauder fixed point principle. If $p/(1-p) > 1$, solutions are unique because the nonlinearities are Lipschitz continuous.

APPENDIX C

EXPECTATION OF REMAINING INFECTED LIFE IS UNBOUNDED FOR LOGNORMAL DISTRIBUTION

To prove Theorem 6.5.1, we need the following version of the mean value theorem which we could not find in any text book though its proof is implicitly used in proofs of some of the l'Hôpital's rules.

Theorem C.1.1. *Let $x_0 \in \mathbb{R}$ and $f_1, f_2 : (x_0, \infty) \to \mathbb{R}$ be differentiable and $f_j(x) \to 0$ as $x \to \infty$, $j = 1, 2$. Assume that $f_2'(x) \neq 0$ for $x > x_0$. Then, for every $x > x_0$, $f_2(x) \neq 0$ and there exists some $y > x$ such that*

$$\frac{f_1(x)}{f_2(x)} = \frac{f_1'(y)}{f_2'(y)}.$$

Proof. We define $\tilde{f}_j : [0, \infty)$ by

$$\tilde{f}_j(t) = f_j(x_0 + (1/t)), \quad t \in (0, \infty), \qquad \tilde{f}_j(0) = 0.$$

Then $\tilde{f}_j$ is continuous on $[0, \infty)$ and differentiable on $(0, \infty)$. By the mean value theorem, for any $t \in (0, \infty)$ there is some $s \in (0, t)$,

$$\tilde{f}_2(t) = \tilde{f}_2(t) - \tilde{f}_2(0) = \tilde{f}_2'(s)t = f_2'(x_0 + (1/s))(-s^2)t \neq 0.$$

So $f_2(x) \neq 0$ for all $x > x_0$.

By the Cauchy mean value theorem, for any $t \in (0, \infty)$ there exists some $s \in (0, t)$, such that

$$\frac{\tilde{f}_1(t)}{\tilde{f}_2(t)} = \frac{\tilde{f}_1'(s)}{\tilde{f}_2'(s)} = \frac{f_1'(x_0 + (1/s))(-s^{-2})}{f_2'(x_0 + (1/s))(-s^{-2})} = \frac{f_1'(x_0 + (1/s))}{f_2'(x_0 + (1/s))}.$$

Now any $x > x_0$ can be written as $x = x_0 + (1/t)$ for some $t \in (0, \infty)$. Then $x < x_0 + (1/s)$. This implies the second assertion. □

Proof of Theorem 6.5.1. If the time from infection to disease death is lognormally distributed, the survival function is given by (6.5.2) and its probability density by

$$-(2\pi)^{1/2}\mathcal{F}'(a) = e^{-b(a)^2/2}\frac{1}{\zeta a}, \qquad b(a) = (1/\zeta)\ln(a/m).$$

By the chain rule,

$$(2\pi)^{1/2}\mathcal{F}''(a) = e^{-b(a)^2/2}\Big(b(a)\frac{1}{(\zeta a)^2} + \frac{1}{\zeta a^2}\Big) = \frac{e^{-b(a)^2/2}}{\zeta^3 a^2}(\ln(a/m) + \zeta^2).$$

Notice that $\mathcal{F}(a)$, $\mathcal{F}'(a)$ and $\mathcal{F}''(a)$ are different from 0 if $a/m > e^{-\zeta^2}$. Further, $\mathcal{F}''(a) \to 0$, $\mathcal{F}'(a) \to 0$ and $\mathcal{F}(a) \to 0$ as $a \to \infty$. Since $\mathcal{F}$ is decreasing, actually $\mathcal{F}'(a) < 0$ for all $a > 0$, and $\int_0^\infty \mathcal{F}(a)da = me^{\zeta^2/2} < \infty$, $\int_a^\infty \mathcal{F}(r)dr \to 0$ as $a \to \infty$. We apply the mean value theorem C.1.1 twice and, for any $a > m$, find some $\tilde{a} > \hat{a} > a$ such that

$$D(a) = \frac{\int_a^\infty \mathcal{F}(r)dr}{\mathcal{F}(a)} = -\frac{\mathcal{F}(\hat{a})}{\mathcal{F}'(\hat{a})} = -\frac{\mathcal{F}'(\tilde{a})}{\mathcal{F}''(\tilde{a})} = \frac{\zeta\tilde{a}}{b(\tilde{a}) + \zeta} = \frac{\zeta^2\tilde{a}}{\ln(\tilde{a}/m) + \zeta^2}.$$

The right hand side of this equality is strictly increasing for $\tilde{a} \geq e^{1-\zeta^2}$ as one sees by taking the derivative. This implies the assertion. □

Remark C.1.2. If the time from infection to disease death is gamma distributed,

$$D(a) = \frac{1}{h(a)} \int_a^\infty h(s)da, \qquad h(s) = \int_s^\infty \tilde{h}(t)dt, \qquad \tilde{h}(t) = t^{\kappa-1}e^{-\theta t}.$$

By Theorem C.1.1,

$$D(a) = -\frac{h(s)}{h'(s)} = -\frac{\tilde{h}(t)}{\tilde{h}'(t)}$$

for some s, t with $a < s < t$. Now

$$-\frac{\tilde{h}(t)}{\tilde{h}'(t)} = \frac{1}{\theta - (\kappa-1)t^{-1}} \overset{t\to\infty}{\longrightarrow} \frac{1}{\theta}.$$

This implies that $D(a) \to \frac{1}{\theta}$ as $a \to \infty$.

Proof of Remark 6.5.2. We choose $j_0(a) = 0$ for $a \leq c$ and $j_0 = a^{-4/3}$ for ≥ 0. Then, $\int_0^\infty j_0(a)da = 3c^{-1/3}$. Without loss of generality, we can choose $c > 0$ so large $c \geq me^{1-\zeta^2}$. By (6.2.9) and (6.2.10),

$$C_0^\infty \geq \xi_0 \int_c^\infty j_0(a)D(a)da$$

By Theorem 6.5.1,

$$C_0^\infty \geq \xi_0 \int_c^\infty \frac{\zeta^2 a^{-1/3}}{\ln(a/m) + \zeta^2} da.$$

We substitute $a = me^x$,

$$C_0^\infty \geq \xi_0 \zeta^2 m^{2/3} \int_{\ln(c/m)}^\infty \frac{e^{(2/3)x}}{x + \zeta^2} dx.$$

There exists some $\delta > 0$ such that $\dfrac{e^{(2/3)x}}{x+\zeta^2} \geq \delta e^{(1/3)x}$ for all $x \geq 0$. This implies that $C_0^\infty = \infty$. □

APPENDIX D

CONVERGENCE FOR S

Theorem D.1.1. *Let $r^\sharp \in (0,\infty)$ and h be differentiable on $(0,r^\sharp)$,*

$$h'(r) > 0, \qquad \sigma h(r)/r > \mu, \qquad r \in (0,r^\sharp).$$

Let S, r be solutions of (12.3.2) with $S(0) > 0$ and $r(0) > 0$ such that $r(t) < r^\sharp$ for all $t \geq 0$.

Then $S(t)$ converges as $t \to \infty$. If the limit of S is not zero, $r(t)$ converges as well as $t \to \infty$.

Proof. Suppose that S does not converge, By the fluctuation theory (Thieme, 2003, Thm.A.20), there exists a sequence $s_n \to \infty$ with $S(s_n) \to S_\infty$ as $n \to \infty$ and $S'(s_n) = 0$ and $S''(s_n) \geq 0$. By (12.7.1), with σ absorbed into h and ξ,

$$S'(s_n) = 0 = g(S(s_n)) - h(r(s_n))$$

and

$$0 \leq S''(s_n) = -S(s_n)h'(r(s_n))r'(s_n).$$

Since $h'(r) > 0$ for $r \in (0,r^\sharp)$, $h'(r(s_n)) > 0$ for all $n \in N$. Again by (12.7.1), for all $n \in \mathbb{N}$,

$$0 \leq -r'(s_n) = r(s_n)\Big[g(S(s_n)) + \mu - h(r(s_n))\Big(1 + \frac{1}{r(s_n)}\Big)\Big].$$

After simplification, for all $n \in \mathbb{N}$,

$$0 \leq \mu - \frac{h(r(s_n))}{r(s_n)}.$$

Since $h(r)/r$ is decreasing in $r > 0$,

$$\mu \geq \limsup_{n\to\infty} \frac{h(r(s_n))}{r(s_n)}, \qquad r(s_n) \in (0,r^\sharp),$$

a contradiction. So $S(t)$ converges as $t \to \infty$.

Then $S'(t) \to 0$ as $t \to \infty$ by Barbalat's lemma. If $S_\infty = \lim_{t\to\infty} S(t) > 0$,

$$0 = \lim_{t\to\infty} S(t)[g(S(t)) - h(r(t))] = S_\infty \lim_{t\to\infty}[g(S_\infty) - h(r(t))].$$

So $h(r(t)) \to S_\infty$ as $t \to \infty$. Since h is continuous and strictly increasing on $[0,r^\sharp]$, $r(t)$ converges as $t \to \infty$. □