Disease Dynamics in Hive Populations

Amalie McKee

August 13, 2010

Abstract

Colony Collapse Disorder has drawn attention to just how dependent we are on pollinators. However, while we are still trying to figure out what pathogen might cause the phenomenon, the dynamics and spread are still widely unknown.

We adapt the traditional SIR model for malaria ecology to model disease dynamics in a pollinator species. We then examine potential treatments, looking for a method of treatment that will enable hive survival and examining changes in long term behavior.

1 Introduction

In 2006, commercial bee keepers up and down the east coast noticed a phenomenon that came to be called Colony Collapse Disorder. CCD is unlike other known bee diseases in that it is characterized by the disappearance of adult bees while healthy brood and food stores remain in the hive.

Since 2006, CCD has spread nationwide and affects 30-40% of bees annually, in both commercial and non-commercial hives. Very little is known about the disease. Those dead bees that have been recovered are typically infected with numerous pathogens, so that it is difficult to determine the original infection. Suggested causes include the parasite Nosema ceranae and several viruses. Researchers are investigating these pathogens, as well as pesticide poisoning and a combination of environmental stresses as potential causes.

The flurry of panic triggered by colony collapse disorder has reminded us just how dependent we are on bees for pollination. Honeybees as commercial pollinators are a 15 billion dollar industry. They are the largest agricultural pollinators, pollinating 60% of all alfalfa, hay and seed among other crops.

Additionally, honeybees are generalist pollinators, pollinating almost every plant species. This provides a important stabilizing force for ecological networks, something that's likely especially important as habitat destruction and global temperature increase affect the life cycles of other pollinator species.

2 Methods

In this project, we adapted a traditional SIR model to model infection in bee populations. We used the Manipulate function in Mathematica to vary the parameters and look for changes in long-term dynamics. Those plots shown here are representative samples of the long-term behaviour, with parameters being uniform across all plots, with $\alpha = 0.1$, $\beta = 0.75$, $\delta_1 = 0.05$, $\delta_2 = 0.75$, $\delta_3 = 0.025$, $\gamma = 0.2$, $a = 0.25$, $b = 0.75$, $r = 0.97$ and $j = 0.3$.

3 SIR Model

An SIR model is a traditional model for disease dynamics. This is a model for disease dynamics within a population, where S stands for susceptible individuals - those who can catch the disease, I stands for infected individuals - those who can transmit the disease and R stands for resistant individuals - those who cannot catch the disease.

For example, traditional dynamics for a population infected with a virus that causes immunity in individuals who have recovered is $S \to I \to R$, represented by the equations:

$$\begin{aligned} \frac{dS}{dt} &= -rSI, \\ \frac{dI}{dt} &= rSI - aI, \\ \frac{dR}{dt} &= aI. \end{aligned}$$

Here, $1 > r > 0$ is the infection rate and $a > 0$ is the recovery rate.

Two things make this model unsuitable for our purposes. One is that the equations in the traditional SIR model sum to zero. This is because the model usually focuses on examining short term dynamics of human diseases. However, bees reproduce very quickly and we cannot assume them to have a constant population.

The next problem with the traditional model is the omission of vector dynamics. Typically this is done because any vector species reproduces relatively quickly compared to humans, so we can assume the vector population is at equilibrium with respect to the infected population. However, just as bees reproduce fairly quickly, they also have a relatively short individual life span, so probably will not outlive any vector population, so we must modify this model.

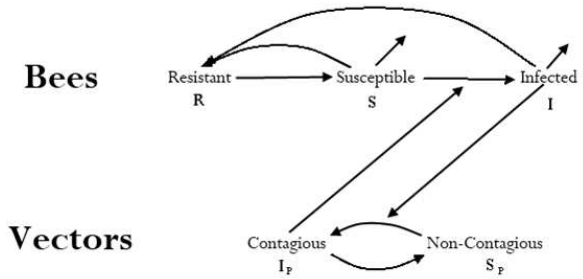
4 Our Model

Bees reproduce constantly. In species where only the queen reproduces, she does so at a rate proportional to the population of adults in the hive. Since the population of adult bees is rather large and grows fairly constantly, we assume it to be continuous.

This continuous growth is important, however. Bee populations typically crash during the winter, a time when food is relatively scarce and the environment is hostile. In order for a hive to survive the winter, it must have a large population compared to the previous spring, so exponential growth during the pollination season is important. For this reason, we do not include a carrying capacity in our model.

One last assumption that makes our model different from the standard SIR model is the resistant class. To explain why only adult bees are afflicted, we propose flowers as vectors with no disease transmission between bees, only between bees and flowers during the pollination interaction. The resistant class is then the brood, which eventually matures into adults. However, between birth and maturity, young bees are fed nearly constantly, up to 10,000 times during that period. This means brood can die of neglect.

Figure 1: A representation of our model. Susceptible individuals become infected through interaction with a contagious plant. A plant becomes infected through interaction with an infected individual. Both populations of adults - the susceptibles and infecteds - reproduce at a constant rate to produce the resistant class, which eventually matures into the susceptible class.



The system of equations representing this is:

$$\begin{aligned}
\frac{dS}{dt} &= \alpha R - \beta S I_P - \delta_1 S, \\
\frac{dI}{dt} &= \beta S I_P - \delta_2 I, \\
\frac{dR}{dt} &= \gamma(S+I)(S_P+I_P) - \alpha R - \delta_3 \frac{R}{S+I}, \\
\frac{dS_P}{dt} &= a I_P - b S_P I, \\
\frac{dI_P}{dt} &= b S_P I - a I_P.
\end{aligned}$$

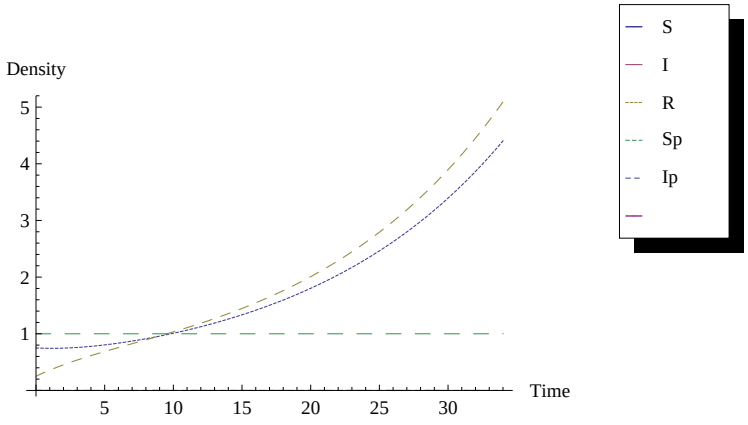
Here, S is the population of adult susceptible bees, I is the population of adult infected bees, R is the resistant class, or the brood, S_P is the non-contagious flowers and I_P are the contagious flowers. Resistant bees mature at a constant rate α, and die of neglect at a rate $\delta_3 \frac{R}{S+I}$. This last term gets large as R gets large relative to $S+I$ and gets small as $S+I$ get large relative to R, so it makes intuitive sense as a rate at which young die of neglect. Susceptible bees die of unspecified causes at a rate δ_1 and become infected at a proportion β of their interactions with contagious plants. Infected bees die at a rate δ_2, presumably

greater than δ_1. Lastly, both adult populations, S and I reproduce at a constant rate γ, also dependent on the available food supply.

The sum of the plant populations in these equations, S_P+I_P, is constant. This is because this is a seasonal model of colony collapse disorder, intended to reflect dynamics within a single pollination period, when the population of plants is relatively constant. That said, non-contagious plants do become infected at a fraction b of their interactions with infected bees, but contagious plants become non-contagious at a rate a.

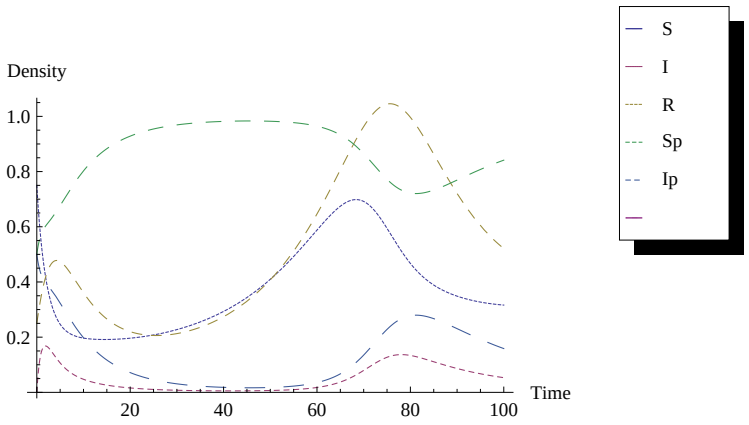
When the population is uninfected, both R and S grow exponentially, which is what we expect to see. Exponential growth is good for the hive, because the population will crash when winter comes.

Figure 2: A sample trajectory of the exponential growth of populations R and S when the population is uninfected.



However, when the population is infected, all populations eventually reach a stable equilibrium, at . This is bad news for the hive as approaching a stable equilibrium means that when winter comes, the population will not have grown enough for the hive to survive, and the hive will collapse.

Figure 3: A sample trajectory of the populations reaching a stable equilibrium in the presence of infection.



5 Treatment

One of the advantages to having a simple mathematical model for disease dynamics is that it can be used to model treatment. In this case, we examined various realistic treatment methods, looking for one that returned the behaviour of the system to that of its uninfected state. However, the population will crash every winter, which is not included in our model. Since evidence suggests that the disease acts as an immunodeficiency disease, it's reasonable to assume that during the population crash, infected bees will die first and the infection will die off. As such, a successful treatment might be one that only temporarily restores exponential growth, since we do not have a good idea of how long the season is.

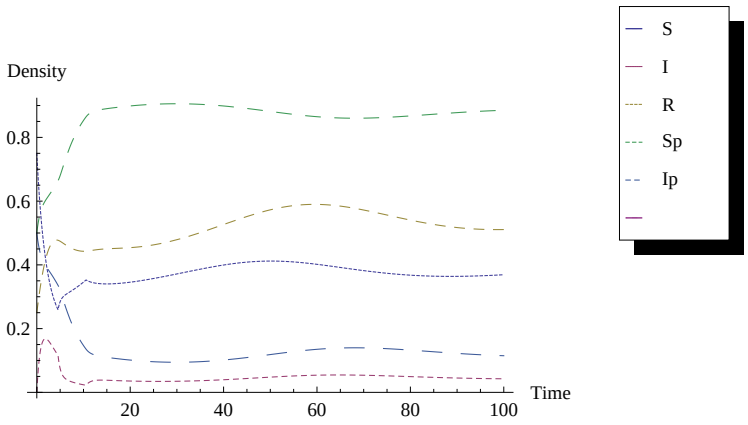
In order to model actual treatment, treatment takes effect when an infection is noticed. In this case, we say that an infection is noticed when the total adult population reaches some low fraction of the initial adult population.

The first and most intuitive treatment for an infection is to treat infected individuals. Our model for this treatment is:

$$\begin{aligned}
\tfrac{dS}{dt} &= \begin{cases} \alpha R - \beta S I_P - \delta_1 S, & \text{if } S+I > j(S_0+I_0) \\ \alpha R - \beta S I_P - \delta_1 S + rI, & \text{if } S+I \le j(S_0+I_0) \end{cases} \\
\tfrac{dI}{dt} &= \begin{cases} \beta S I_P - \delta_2 I, & \text{if } S+I > j(S_0+I_0) \\ \beta S I_P - \delta_2 I - rI, & \text{if } S+I \le j(S_0+I_0) \end{cases} \\
\tfrac{dR}{dt} &= \gamma(S+I)(S_P+I_P) - \alpha R - \delta_3 \tfrac{R}{S+I}, \\
\tfrac{dS_P}{dt} &= aI_P - bS_P I, \\
\tfrac{dI_P}{dt} &= bS_P - aI_P.
\end{aligned}$$

This is not an effective treatment. Rather than restoring the dynamics to that of the uninfected system, this treatment causes the system to stabilize faster, providing no opportunity for the population to grow.

Figure 4: A sample trajectory of this treatment. The sharp corners occur where the treatment takes effect. In this graph, $j = 0.5$, for ease of visibility.



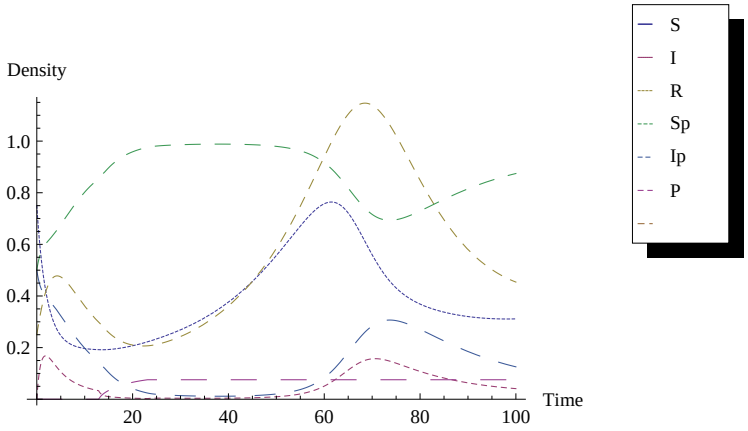
Depending on the type of pathogen and method of treatment, treated bees might be immunized against further infection. For example, individuals treated for a viral infection develop an immunity to further infection by that virus. In this case, that can be modelled by:

$$\begin{aligned}
\tfrac{dS}{dt} &= \alpha R - \beta S I_P - \delta_1 S \\
\tfrac{dI}{dt} &= \begin{cases} \beta S I_P - \delta_2 I, & \text{if } S + I > j(S_0 + I_0) \\ \beta S I_P - \delta_2 I - rI, & \text{if } S + I \leq j(S_0 + I_0) \end{cases} \\
\tfrac{dR}{dt} &= \gamma(S + I)(S_P + I_P) - \alpha R - \delta_3 \tfrac{R}{S+I}, \\
\tfrac{dP}{dt} &= \begin{cases} 0, & \text{if } S + I > j(S_0 + I_0) \\ rI, & \text{if } S + I \leq j(S_0 + I_0) \end{cases} \\
\tfrac{dS_P}{dt} &= aI_P - bS_P I, \\
\tfrac{dI_P}{dt} &= bS_P - aI_P,
\end{aligned}$$

where P is the population of bees that is immune to reinfection.

Unlike just treating bees, this method of treatment is not stabilizing, but the population still approaches an equilibrium eventually. In the meantime, however, it spikes.

Figure 5: A sample trajectory of the bee immunization treatment. The sharp corners occur where the treatment takes effect.

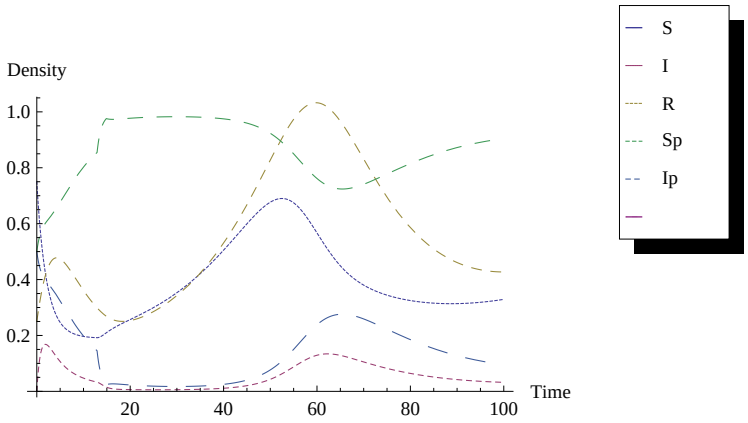


However, when fighting a vector-borne illness, treating infected individuals is not the only approach. Treating vectors is also possible. In this case, treating the vectors might actually be easier because beekeepers could spray entire fields with treatment. I modelled this form of treatment by:

$$\begin{aligned}
\tfrac{dS}{dt} &= \alpha R - \beta S I_P - \delta_1 S, \\
\tfrac{dI}{dt} &= \beta S I_P - \delta_2 I, \\
\tfrac{dR}{dt} &= \gamma(S+I)(S_P+I_P) - \alpha R - \delta_3 \tfrac{R}{S+I}, \\
\tfrac{dS_P}{dt} &= \begin{cases} aI_P - bS_P I, & \text{if } S+I > j(S_0+I_0) \\ aI_P - bS_P I + rI_P, & \text{if } S+I \le j(S_0+I_0) \end{cases} \\
\tfrac{dI_P}{dt} &= \begin{cases} bS_P I - aI_P, & \text{if } S+I > j(S_0+I_0) \\ bS_P I - aI_P - rI_P, & \text{if } S+I \le j(S_0+I_0) \end{cases}
\end{aligned}$$

Unlike treatment of bees, treatment of plants is not stabilizing, instead briefly increasing exponential growth, although not doing so by a large amount.

Figure 6: A sample trajectory of plant treatment.



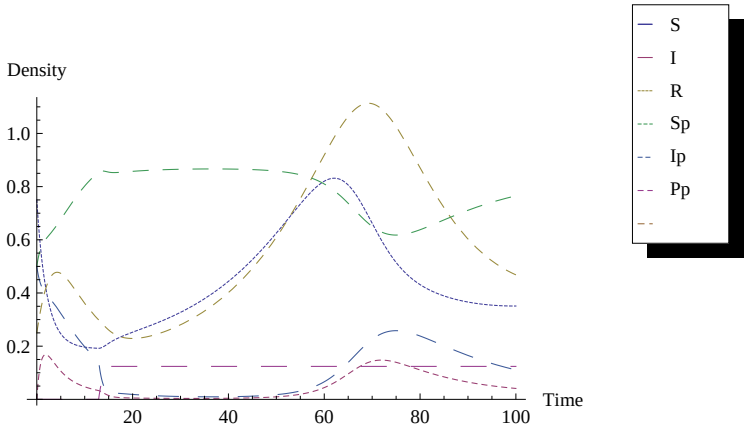
However, spraying fields with treatment might make the fields a bad vector for the pathogen, effectively immunizing the plants from becoming contagious. I modelled this effect with the equations:

$$
\begin{aligned}
\tfrac{dS}{dt} &= \alpha R - \beta S I_P - \delta_1 S, \\
\tfrac{dI}{dt} &= \beta S I_P - \delta_2 I, \\
\tfrac{dR}{dt} &= \gamma (S+I)(S_P + I_P) - \alpha R \delta_3 \tfrac{R}{S+I}, \\
\tfrac{dS_P}{dt} &= a I_P - b S_P I, \\
\tfrac{dI_P}{dt} &= \begin{cases} b S_P I - a I_P, & \text{if } S + I > j(S_0 + I_0) \\ b S_P - a I_P - r I_P, & \text{if } S + I \leq j(S_0 + I_0) \end{cases} \\
\tfrac{dP_P}{dt} &= \begin{cases} 0, & \text{if } S + I > j(S_0 + I_0) \\ r I_P, & \text{if } S + I \leq j(S_0 + I_0) \end{cases},
\end{aligned}
$$

where P_P is the fraction of plants that can no longer become contagious.

As immunizing bees was much better than simply treating them, so immunizing plants was much better than simply treating them. This makes some intuitive sense, since reducing the population of susceptible individuals is a common way of fighting epidemics - it's the rationale behind the whole vaccination movement.

Figure 7: A sample trajectory of the dynamics of plant immunization.

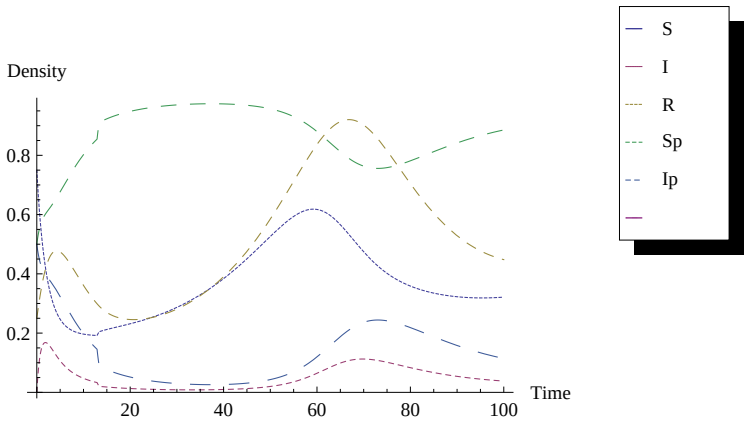


If a treatment were developed that cured any infected bees but did not cause an immunity, it might make sense to apply that treatment to both bees and plants. I modelled that with the equations:

$$
\begin{aligned}
\tfrac{dS}{dt} &= \begin{cases} \alpha R - \beta S I_P - \delta_1 S, & \text{if } S+I > j(S_0+I_0) \\ \alpha R - \beta S I_P - \delta_1 S + rI, & \text{if } S+I > j(S_0+I_0) \end{cases} \\
\tfrac{dI}{dt} &= \begin{cases} \beta S I_P - \delta_2 I, & \text{if } S+I > j(S_0+I_0) \\ \beta S I_P - \delta_2 I - rI, & \text{if } S+I \le j(S_0+I_0) \end{cases} \\
\tfrac{dR}{dt} &= \gamma(S+I)(S_P+I_P) - \alpha R - \delta_3 \tfrac{R}{S+I}, \\
\tfrac{dS_P}{dt} &= \begin{cases} aI_P - bS_P I, & \text{if } S+I > j(S_0+I_0) \\ aI_P - bS_P I + rI_P, & \text{if } S+I \le j(S_0+I_0) \end{cases} \\
\tfrac{dI_P}{dt} &= \begin{cases} bS_P I - aI_P, & \text{if } S+I > j(S_0+I_0) \\ bS_P I - aI_P - rI_P, & \text{if } S+I \le j(S_0+I_0) \end{cases}
\end{aligned}
$$

This treatment actually is more effective than just treating bees, but not any more effective than just treating the field. This is due to the stabilizing effect of treating the bee population.

Figure 8: A sample trajectory of treatment of both plant and bee infected populations.

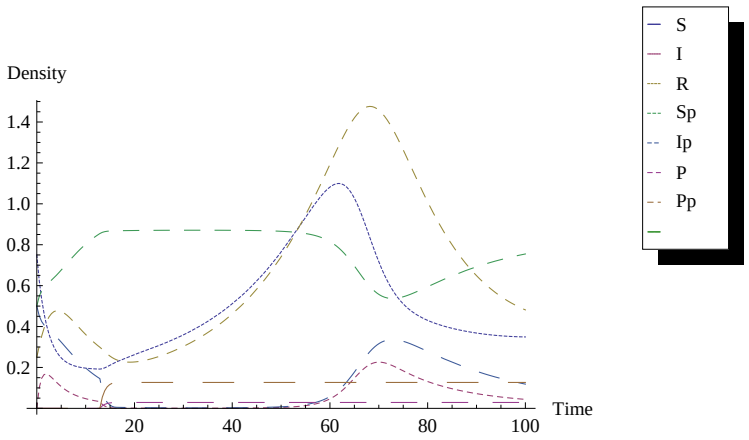


Lastly, we could immunize both plants and bees. Intuitively, this method seems like it would be effective as both component methods were effective. I modelled this with the following equations:

$$
\begin{aligned}
\tfrac{dS}{dt} &= \alpha R - \beta S I_P - \delta_1 S \\
\tfrac{dI}{dt} &= \begin{cases} \beta S I_P - \delta_2 I, & \text{if } S+I > j(S_0+I_0) \\ \beta S I_P - \delta_2 I - rI, & \text{if } S+I \le j(S_0+I_0) \end{cases} \\
\tfrac{dR}{dt} &= \gamma(S+I)(S_P+I_P) - \alpha R - \delta_3 \tfrac{R}{S+I}, \\
\tfrac{dP}{dt} &= \begin{cases} 0, & \text{if } S+I > j(S_0+I_0) \\ rI, & \text{if } S+I \le j(S_0+I_0) \end{cases} \\
\tfrac{dS_P}{dt} &= aI_P - bS_P I, \\
\tfrac{dI_P}{dt} &= \begin{cases} bS_P I - aI_P, & \text{if } S+I > j(S_0+I_0) \\ bS_P I - aI_P - rI_P, & \text{if } S+I \le j(S_0+I_0) \end{cases} \\
\tfrac{dP_P}{dt} &= \begin{cases} 0, & \text{if } S+I > j(S_0+I_0) \\ rI_P, & \text{if } S+I \le j(S_0+I_0) \end{cases}
\end{aligned}
$$

Out of the six options so far, this was the best, with the biggest exponential growth.

Figure 9: A sample trajectory of immunization of both plant and be infected populations.



Unrealistically, there are other potential treatments. One is to simply add susceptible plants so that the bee reproduction rate goes up. We can model this by:

$$\begin{aligned}
\frac{dS}{dt} &= \alpha R - \beta S I_P - \delta_1 S, \\
\frac{dI}{dt} &= \beta S I_P - \delta_2 I, \\
\frac{dR}{dt} &= \gamma(S+I)(S_P+I_P) - \alpha R - \delta_3 \frac{R}{S+I}, \\
\frac{dS_P}{dt} &= \begin{cases} aI_P - bS_P I, & \text{if } S+I > j(S_0+I_0) \\ aI_P - bS_P I + r(S_P+I_P), & \text{if } S+I \leq j(S_0+I_0) \end{cases} \\
\frac{dI_P}{dt} &= bS_P I - aI_P.
\end{aligned}$$

In this model, when the population of adult bees dips sufficiently far below the initial population, plants are added continuously until the population rises again. This promotes exponential growth in the bee population, however, it doesn't eliminate the infection. Additionally, this is unlikely to be how the population would actually respond to an increase in plants.

6 Conclusions and Further Work

If it turns out that plants are a vector for CCD or another bee pathogen, it's clear from this model that we should treat the plants instead of the bees. However, there is much missing from this model.

The SIR model presents a very idealized model for disease ecology. Without data, it's difficult to say whether this model realistically models a disease like CCD. But such data is difficult to collect. Even commercial bee keepers do not keep constant watch over their hives, because hives are generally robust communities. As such, little is known about the onset of colony collapse disorder.

Additionally, it's still a mystery why workers are the only ones afflicted and why commercial hives are the most at risk. Stress is a good suggestion and a known cause of disease in other organisms. However, it's possible that commercial hives are more at risk because they travel more and pollinate more fields. If flowers really are vectors, than it would make sense that the more hives whose pollination area a hive overlaps with, the more at risk that hive is for disease.

Not only is this model idealized, but it deals with seasonal dynamics within a single hive. Ideally, the model would look at hives as the population, and examine yearly dynamics. This is made somewhat easy by the fact that hives collapse in winter, so a stochastic model in discrete time could accurately model these conditions. Another factor too include in this model would be the ability of different plants to be a medium for disease. Are some plants better vectors? Do pollinating more species make a hive more or less at risk for infection? Both of these would be good things to know going forward.

Finally, CCD was first observed in 2006, the same year white nose was first observed in bats. It would be interesting to study the disease interactions of insects and their predators and examine whether or not the two diseases are related.

7 Acknowledgements

I would like to thank SFI and the NSF for the support and Jennifer Dunne, Jeremy van Cleve and the other REUs for making this project possible.

References

[1] B.T. Grenfell and A. P. Dobson. *Ecology of Infectious Diseases in Natural Populations*. Cambridge University Press, 1995.

[2] Bernd Heinrich. *Bumblebee Economics*. Harvard University Press, 1979.

[3] Renee Johnson. Honey bee colony collapse disorder. *Congressional Research Service*, 2010.

[4] Matt J. Keeling and Pejman Rohani. *Modeling Infectious Diseases in Humans and Animals*. Princeton University Press, 2008.

[5] Robin F.A. Moritz and Edward E. Southwick. *Bees as Superorganisms: an Evolutionary Reality*.

[6] Cheryl J. Briggs Murdoch, Willian W. and Roger M. Nisbet. *Consumer - Resource Dynamics*. Princeton University Press, 2003.

[7] J.D. Murray. *Mathematical Biology*. Springer - Verlag, 1989.

[8] Mercedes Pascual and Jennifer Dunne. *Ecological Networks: Linking Structure to Dynamics in Food Webs*. Oxford University Press, 2006.