Uncertainty Quantification of Plant Disease Dynamics Using SIR Model V – High Inoculum Case

A mathematical modeling study of stem canker disease using parameter estimation, model fitting, and sensitivity analysis to quantify uncertainty in disease progression. This research was conducted under the supervision of the School of Mathematics and Statistics, University College Dublin(UCD)

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1. Introduction

Plant diseases like stem canker can seriously affect crop health, especially in potatoes. This disease, caused by the soil-borne fungus *Rhizoctonia solani.* This fungus hits early in the growing season and damages young stems, often leading to major yield losses if not managed properly.

To better understand how the disease spreads, we use **Model V-high inoculum density** which is an extended version of the classic SIR (Susceptible–Infected–Removed) model. Model V includes a built-in plant response to infection that changes with stem density. This makes it especially useful for **high inoculum density** scenarios, where infection pressure is high from the beginning.

A key feature of this model is the **host response function**:

$$f(I,S) = \frac{\alpha I}{\gamma + I + S},$$

Above equation captures how infection pressure builds up with more infected stems. It also reflects the plant's ability to respond eventually slows down as infection saturates.

We fitted the model to observed stem count data using numerical methods, and explored how sensitive the system is to changes in key parameters. This helps us evaluate both the accuracy and reliability of the model in describing real world disease dynamics.

2. Model & Equations

We used a compartmental **SIR** Model V- High density to explore how stem canker spreads in potato plants. Our model follows how stems move between being **Susceptible (S)**, **Infected (I)**, and **Removed (R)** over time. It also accounts for a decreasing infection pressure as plants age and includes a nonlinear response from the host. For the high inoculum density scenario, **Gilligan et al. (1997)** provided a set of parameter estimates specific to Model

Parameters description:

b – Stem production rate
κ – Carrying capacity
λ_0 – Initial infection force
μ – Decay rate of infection
α – Host response strength
γ – Saturation threshold
d – Infected stem death rate

Values for our model are:
b = 1.467 κ = 5.743, λ_0 = 0.499,
μ = 0.247, α =5.2.106, γ = 0.026,
d = 0.058, with a residual sum of squares (RSS) of 14.10 and 9 degrees of freedom.

Equations:

susceptible stems

$$\frac{dS}{dt} = b(\kappa - N) - \lambda S - f(I,S),$$

infected stems

$$\frac{dI}{dt} = \lambda S - dI,$$

removed stems

$$\frac{dR}{dt} = dI,$$

force of infection

$$\frac{d\lambda}{dt} = -\mu\lambda,$$

3. Data and models

1)We used a version of Model V-High density where the infection rate drops over time, since plants become less vulnerable as they grow. It is like the original study described in Gilligan.
2)In our model, we solved the system of ODEs(ordinal differential equations) using deSolve package in R.
3)Estimated parameters using **least squares optimization** via the optim() function with the **L-BFGS-B** method.
4)Evaluated the performance of model using **Residual Sum of Squares (RSS)** for both S and I compartments.
5)Conducted **local sensitivity analysis** by perturbing each parameter (±30%) to identify their impact on the output of model.
6)Visualized the model predictions and fit quality using **ggplot2**. We compared simulated and observed data to plot the graphs.
7)Implemented a modified SIR model accounting for nonlinear host response influenced by total stem density.
8)Our model gave a better fit (RSS = 2.99 vs. 14.10 in the paper), likely because we had cleaner data and carefully adjusted the parameters

time	S	I	R
0.00000000	0.00000000	0.00000000	0.00000000
0.10000000	0.76192550	0.01930508	0.00003814
0.20000000	1.37986100	0.07101795	0.00028651
0.30000000	1.87688840	0.14717114	0.00090914

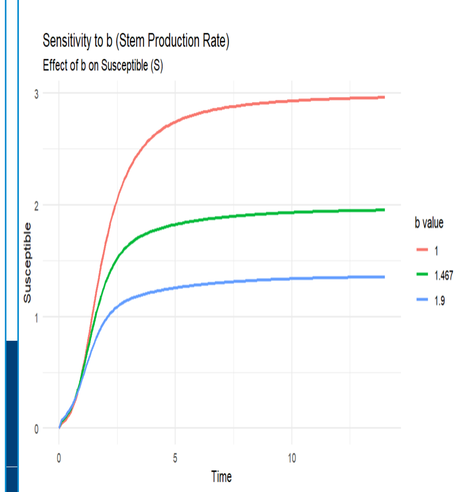
5. Sensitivity

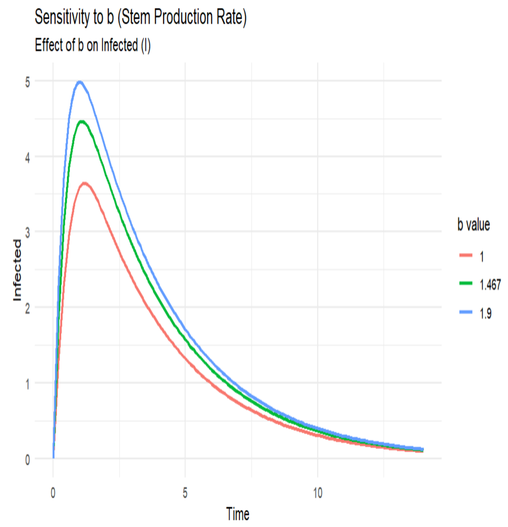
1)We explored how small changes in b affect the model. We kept other values fixed.

2)We noticed that with a higher b, the number of susceptible stems levels off lower this means more stems get infected during early stage.

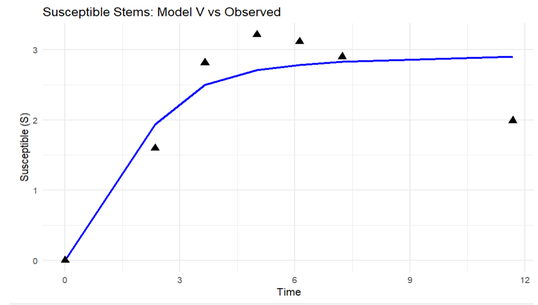
3)For infected stems, we observed that a higher b leads to a quicker rise and a higher peak.

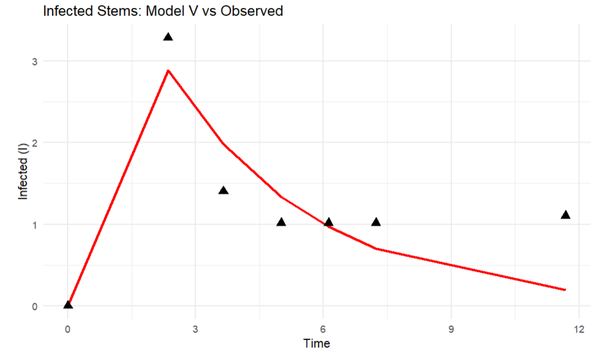
4)Overall, we saw that b has the biggest effect at the start of the outbreak, shaping how fast things take off.





The plots compare the model's predictions (solid lines) with observed data (black triangles) for both susceptible (S) and infected (I) stems under high inoculum density.





1)Susceptible stems (S) rise quickly in the early phase and then gradually decline, likely due to the effects of infection and the plant's natural response..
2)Infected stems (I) climb sharply at first and then slowly decrease , showing how the disease begins and fade over time.

Compared to the paper's RSS of 14.10 for Model V (High Density), our fitted model achieved a noticeably lower **total RSS of 2.99**, with **RSS for S = 1.4153** and **RSS for I = 1.53105**, indicating a closer fit to the observed data. This improved accuracy is likely due to the smaller number of data points and more precise parameter tuning based on our dataset.

6. Conclusion

1)Model V – High Density captured the disease dynamics well, especially the rise and fall of both susceptible and infected stems over the period of time.

2) When we tested how sensitive the model is to changes in the stem production rate b we found that it really matters because increasing b led to faster and more intense infections, while lowering it slowed everything down.

3) The final RSS value we got was **2.49**, which is much lower than the **14.10** reported in the original paper. This likely means that our fit was tighter, possibly because we had fewer data points and tuned the parameters more precisely.

4)Our final RSS was **3.33**, which is much lower than the paper's value of **14.10** for the same model.

5) The parameters that worked best for our model were: **b = 1.467**, **λ_0 = 0.499**, **α = 2.106**, **γ = 0.026**, **μ = 0.247**, **d = 0.058**, and **κ = 5.743**

6) Overall, this model gives a pretty realistic picture of how stem canker spreads when infection pressure is high, and helps us understand which factors have the biggest effect on that spread.