LETTERS IN BIOMATHEMATICS
An International Journal

Intercollegiate Biomathematics Alliance

RESEARCH ARTICLE

OPEN ACCESS

Modeling the Spread of Curly Top Disease in Tomatoes

Rachel Frantz[a], Claudia Nischwitz[b], Tyler Compton[b], Luis F. Gordillo[a]

[a]Department of Mathematics and Statistics, Utah State University, Logan, UT; [b]Department of Biology, Utah State University, Logan, UT

ABSTRACT

Curly Top disease (CT), caused by a family of *curtoviruses*, infects a wide variety of agricultural crops. Historically, CT has caused extensive damage in tomato crops resulting in substantial economic loss for the tomato industry. Control methods for CT are scarce, and methods for predicting and assessing the scope of CT outbreaks are limited. In this paper, we formulate a stochastic model for the spread of CT in a heterogeneous environment, which consists of beet plants, the preferred hosts, and tomato plants. The model is composed of two susceptible classes and two infected classes, where the beet plants are the primary reservoir of the pathogen. We parameterize the model using data from a field experiment and assess the variability of CT incidence in tomato plants at any point in time through extensive simulations.

ARTICLE HISTORY
Received May 22, 2022
Accepted November 23, 2022

KEYWORDS
Curly Top Disease, compartmental models, stochastic simulation algorithm, susceptible-infected model

1 Introduction

Curly Top (CT) disease is caused by a group of viruses known as *Curtoviruses*. These viruses affect more than 300 species of plants including economically important crops such as tomatoes, sugar beets, beans, and peppers in semi-arid areas across Utah, Arizona, California, and Nevada (Chen and Gilbertson, 2011). In the early 1900's, CT was identified as the cause of extensive agricultural crop loss (Stenger and McMahon, 1997). While studies have continued to investigate control methods such as row covers, double planting, and the development of CT resistant tomato varieties, CT still poses as an economic threat to the tomato industry (Heflebower and Schalau, 2014). In 2013, an outbreak of CT occurred in Southern California costing over 100 million dollars in economic loss for the tomato industry (Chen et al., 2017).

The goal of this paper is to describe the variability in the size of outbreaks produced by a stochastic model for the spread of CT in tomato plants. The model that we propose follows the classical susceptible-infected framework applied to two populations, beet and tomato plants. The distinctive characteristic of the model is that the pathogen is transmitted from beet to tomato plants but the converse is unlikely. The simulations allow us to assess the variability of the disease incidence in tomato plants at any point in time, a task that otherwise would be impractical due to the challenges in collecting CT data in infected hosts. We use data collected from an experimental tomato field to parameterize the deterministic model, information that is later used in the stochastic framework to simulate the evolution of the disease incidence over time.

2 Biology of Curly Top Transmission

In tomato plants, CT presents as stunted growth and chlorotic leaves with purple veins or upturned leaf margins. In many instances, infected plants are no longer able to produce new fruit and fruit that was already set fails to fully mature (Heflebower and Schalau, 2014). In Southern Utah, CT has been reported to cause losses of up to 90% in tomato crops (Douglass and Cook, 1954). This level of damage emphasizes the need to develop cost and time effective methods of management. The model that we propose simulates disease output and might help managers to assess the extent of damage caused by the pathogen.

Curtoviruses are transmitted by the beet leafhopper, *Circulifer tennellus*, an insect that acquires and transmits the viruses while feeding on plants (Heflebower and Schalau, 2014). Transmission of CT to crops occurs, in part, as a consequence of the leafhopper's migration patterns. After overwintering on rangeland vegetation, leafhoppers seek new feeding and breeding grounds. The migration occurs in the spring and aligns with the emergence of new crops (Douglass and Cook, 1954). The number of leafhoppers infected with CT in the first spring generation after overwintering varies greatly from year-to-year with as

CONTACT Rachel Frantz rachel.frantz@usu.edu

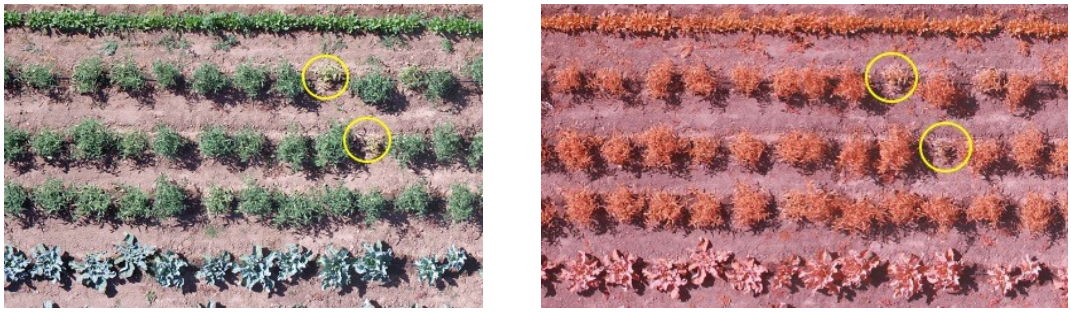

Figure 1: Identification of infected tomato plants using aerial images. A row of beets is shown at the top of each image and a row of cauliflowers are shown at the bottom of each image. **Left:** Picture taken with normal lighting. **Right:** Picture taken with near infrared imaging. The tomato plants circled in yellow were confirmed to be infected with Curly Top.

few as 4% carrying CT and reports of up to 80%, (Douglass and Cook, 1952). This percentage increases as the summer progresses due to the inability of leafhoppers to shed the virus. CT is carried in the circulatory system of leafhoppers and in the phloem of infected plants. Once the saliva of the leafhopper and the phloem of a plant come in contact with one another, the disease can be transmitted from an infected to a non-infected host in as little as one minute (Heflebower et al., 2012). Leafhoppers can reproduce in beets; however, they are unable to breed in the majority of other crops. This causes the leafhoppers to prefer sugar beets as a host. It has been documented that leafhopper's overall health declines with continual feeding on plants such as tomatoes (Douglass and Cook, 1954). A virus-free leafhopper acquires the pathogen from an infected plant only after a relatively long exposure. After sampling a tomato plant and finding out it is not their preferred food source, leafhoppers move quickly away from tomato plants. Thus, it is believed that the spread of CT to non-suitable hosts is a consequence of sample feeding by the leafhoppers (Thomas and Boll, 1977; Heflebower and Schalau, 2014).

3 Methods

3.1 Data collection

In early June, six-week-old tomato plants were transplanted to a field plot. The tomato plants were planted in three beds (running west to east) with 50 plants per bed. Within a row, tomato plants were spaced 20-35 inches apart, and the rows were planted 20 inches apart. Three beds of beets were planted to the south of the tomato plants. The beet plantings were 100 ft long with beets planted approximately two inches apart. To the north of the tomato plants three columns of cauliflowers were planted. Drone imaging captured aerial images of the field plot. Using near infrared (NIR) imaging, tomato plants suspected of being infected were identified. In NIR imaging the chlorophyll of the tomato plants (which gives them there distinctive green coloring) appears red. Plants that are not healthy do not produce the same amount of chlorophyll, causing the sick plants to appear white/yellow in the NIR image. Figure 1, shows the differences in appearance of tomato plants under normal daylight (top) versus NIR (bottom). At intervals ranging from three to ten days, disease maps (Figure 2) were drawn showing the location of infected plants. Symptomatic plants were collected and DNA was extracted using the Qiagen DNeasy Plant Mini Kit (Qiagen, Germantown, MD) following manufacturer's instructions. The reaction mixture for the PCR with a total volume of 50 μl contained 25 μl Phusion® High-Fidelity PCR Master Mix with HF Buffer (New England Biolabs Ipswich, MA), 2.5 μl each of primers curtovirus 1 and curtovirus 2 (10 μmol/μl) (Nischwitz and Olsen, 2010), 2 μl total DNA extract. Nuclease free distilled water was added to obtain the total final volume. The PCR products were visualized in a 1% agarose gel stained with ethidium bromide. Samples were considered positive if the expected 388bp band was observed.

All symptomatic plants identified with the unmanned aerial vehicle (UAV) tested positive for CT. Ten randomly selected non-symptomatic plants were also tested and all tested negative. Infected plants could be detected as soon as a slight chlorosis of leaves started to develop. Infected tomato plants will develop symptoms. No tomato variety has any resistance to the disease (Chen et al., 2010). Using UAVs for plant disease detection is novel but has been successfully used to detect Wheat mosaic virus in sweet corn (Nischwitz, 2020).

3.2 The model

First we set up a deterministic model, that we fit to field-collected data to obtain parameter estimates. Then we use these parameters to write the propensity functions for the stochastic model that we use to assess the variability of disease incidence 70 days after the initial planting.

Table 1: Incidence of Curly Top Virus in Tomato Plants

Date	6/6			6/18			6/28			7/1			7/10			7/19			7/31			8/7		
Days after planting	8			20			30			33			42			51			63			70		
Count	0	0	0	0	0	1	2	0	1	2	2	2	5	2	2	5	2	2	8	2	5	10	4	7
Row	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C	A	B	C
1						1			1			1			1			1			1			1
2																			1			1		
3																						1		
4																								
5																								
6							1			1			1			1			1			1		
7																						1		
8							1			1			1			1			1			1		
9																			1			1		1
10																								1
11													1			1			1			1		
12											1			1			1			1			1	
13													1			1			1			1		
14																								
15																					1			1
16																								
17																								
18																					1			1
19																								
20																								
21																								
22																								
23																			1			1		
24											1			1			1			1			1	
25																								
26																								
27																								
28																								
29												1			1			1			1			1
30																								
31																								
32																								
33																								
34																					1			1
35																								
36																								
37																								
38																								
39													1			1			1			1		
40																							1	
41																								
42																							1	
43																								
44																								
45																								
46																								
47																								
48																								
49																								
50																								
51																								

Figure 2: Disease maps observed from June 6, 2019 to August 7, 2019. Green cells represent healthy plants, yellow cells are CT infected (infected and removed), pink cells are symptomatic and red cells are plants that were no longer visible in the aerial imaging. The beet plants were located to the left of three columns of tomato plants.

The deterministic model for the spread of CT in tomato plants consists of two differential equations representing susceptible-infected (SI) disease interactions. The model uses the mass action paradigm for modeling contacts between tomato and beet plants, which are produced by the leafhopper's bites. Mass action is widely used in modeling disease spread and in particular it has been successful in plant disease models (see for instance Madden et al., 2007). We assume that the spread of CT in tomato plants only occurs due to contact with infected beet plants; leafhoppers preferentially feed on beets, with tomato plants becoming infected when leafhoppers sample on them. Infections from an infected tomato plant to a healthy host, tomato or beet, are not taken into account because the time that leafhoppers spend on tomato plants is relatively small and the probability of pathogen transmission is consequently negligible (Klein, 1992). Once infected, a plant will not recover or gain immunity.

Under the assumptions stated above, the equations

$$\frac{d}{dt}I_t = \beta S_t I_b \qquad \text{and} \qquad \frac{d}{dt}I_b = \beta' S_b I_b, \tag{1}$$

with S_t and I_t (S_b and I_b) representing the number of susceptible and infected tomato (beet) plants, respectively, completely describe the disease transmission dynamics. The parameters β and β' are positive and represent the disease transmission rates. Let us write $T = S_t(0)$ and $B = S_b(0)$ the initial amounts of susceptible tomato and beet plants, respectively, and assume that $I_t(0) = 0$ and $I_b(0) > 0$. The equations in (1) can be integrated to obtain the relationship

$$\frac{I_t}{T} = 1 - \left(\frac{B - I_b}{B - I_b(0)}\right)^a, \tag{2}$$

where $a = \beta/\beta'$. In terms of the fractions of susceptible population of tomato plants, $x = S_t/T$, and beets, $y = S_b/B$, this is equivalent to $x = Cy^a$, where $C = (1/y(0))^a$. As expected, if $a < 1$ the proportion of susceptible beets will decrease faster (relatively) than that of susceptible tomato plants, which will not be reduced significantly until most of the beet population has been infected.

3.2.1 Stochastic model

Let us assume that S_t, I_t, S_b, and I_b are non-negative, integer-valued random variables. These random variables are the components of the state vector of our system, (S_t, I_t, S_b, I_b). According to the previously stated assumptions on the transmission mechanisms of the pathogen only the following two transitions of the state vector are permissible,

$$\begin{aligned} &\text{(i)} \quad (S_t, I_t, S_b, I_b) \to (S_t - 1, I_t + 1, S_b, I_b) \quad \text{with rate } \beta S_t I_b \\ &\text{(ii)} \quad (S_t, I_t, S_b, I_b) \to (S_t, I_t, S_b - 1, I_b + 1) \quad \text{with rate } \beta' S_b I_b \end{aligned}$$

The first is the infection of a tomato plant and the second is the infection of a beet plant. We use the stochastic simulation algorithm (SSA) (see for instance Gillespie, 1977; Higham, 2008), which consists in simulating the disease transmission events at exponentially distributed time points, with corresponding propensity functions (rates) $\beta S_t I_B$ and $\beta' S_b I_b$. At each event, the state vector is updated accordingly and the process continues until the total computation time is reached. The parameters in the propensity functions are those estimated from fitting the deterministic model (1) to the field data, see Section 3.

4 Results

4.1 Parameter estimation

Parameter estimation was performed using the MATLAB function `fminsearch`, which uses the Nelder-Mead Simplex Algorithm (Lagarias et al., 1998) to perform a least squares fitting. We minimized the difference between the observed number of infected tomato plants and the predicted number of infected tomato plants produced by the deterministic model. The initial number of infected beets was fit in this process to account for the unknown initial infections in the beet population. For each trial we set the initial number of beet and tomato plants to $S_b = 800$ and $S_t = 150$, with the former estimated from aerial images. The estimates obtained for the transmission rates β and β', as well as the initial number of infections in the beet population are shown in Table 1.

The numerical solution for the model (1) is illustrated in Figure 3, where the numerical results are compared to the experimental data.

4.2 Numerical simulations

Repeated simulations with the stochastic model presented in Section 3.2 provide us with plausible measure of the disease incidence variability in the tomato plants over time. The parameters for the propensity functions are those estimated in Section 3.1.

Table 1: Parameter descriptions and estimated values obtained by fitting the deterministic model to experimental field data.

Model Variables		
Notation	Description	
S_t	Number of susceptible tomato plants	
S_b	Number of susceptible beet plants	
I_t	Number of infected tomato plants	
I_b	Number of infected beet plants	
Model Parameters		
Notation	Description	Value
$S_{t(0)}$	Initial number of susceptible tomato plants	150
$I_{t(0)}$	Initial number of infected tomato plants	0
$S_{b(0)}$	Initial number of susceptible beet plants	800
$I_{b(0)}$	Initial number of infected beet plants	7
β	Beet to tomato transmission rate	0.000003204 (day^{-1})
β'	Beet to beet transmission rate	0.0005199 (day^{-1})

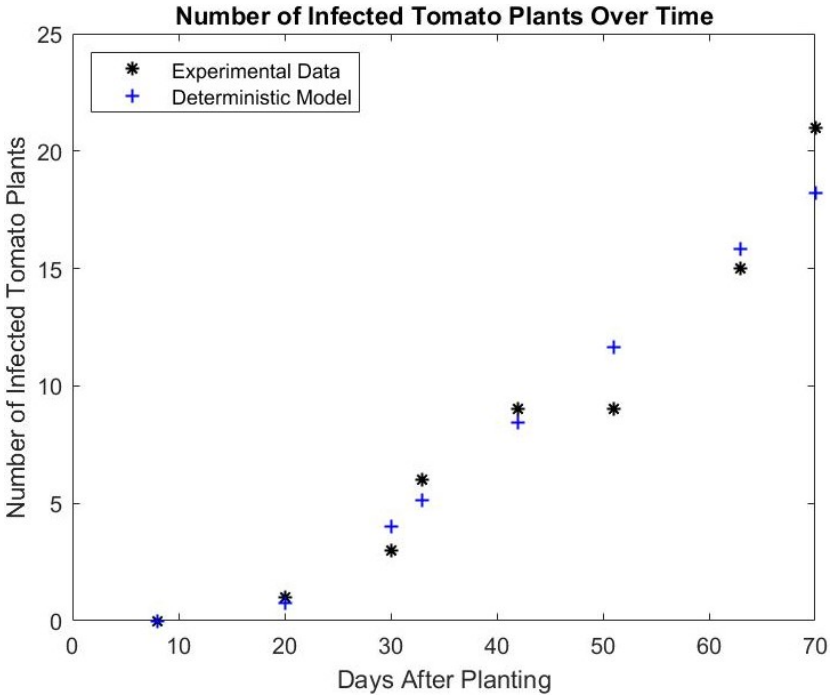

Figure 3: Disease counts from the experimental field data (black stars) and number of infected tomato plants at each time point as output by the deterministic model (blue plus signs)

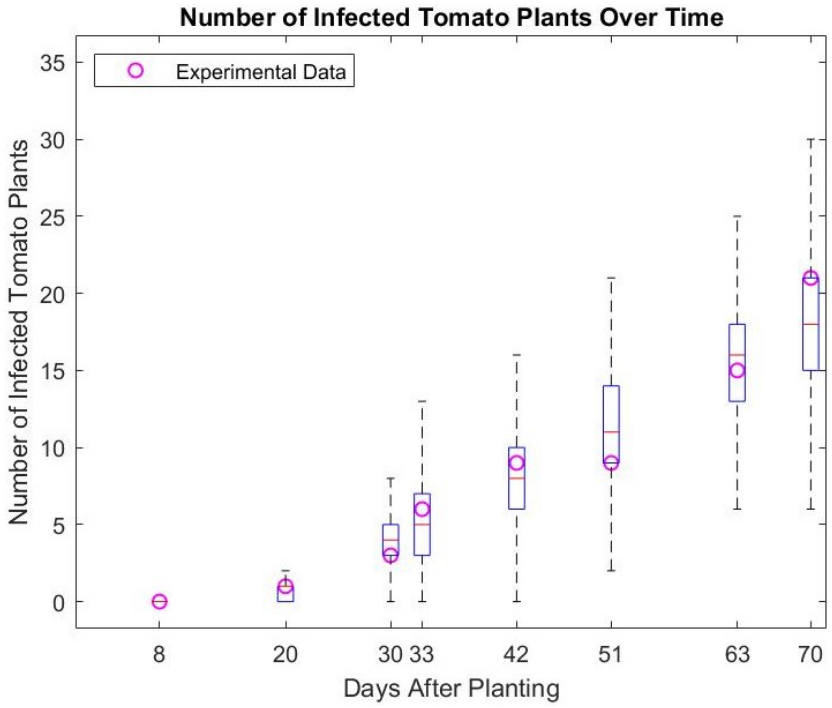

Figure 4: Box-plots of the simulated data, number of infected tomato plants, produced by the stochastic model. The box-plots were generated using data from 10,000 simulations. The experimental field data is shown in pink circles.

We produced 10,000 realizations of the process over 62 days and used the simulated data to generate the box-plot in Figure 4, where we also plot the experimental field data.

Figure 5 (left) shows ten sample paths corresponding to the infection process of the tomato plants. We also estimate the standard deviation for each point in time from the simulation of 10,000 paths in Figure 5 (right).

4.3 Model sensitivity

In order to assess the sensitivity of the model to the parameters β (beet-to-beet transmission rate) and β' (beet-to-tomato transmission rate) we investigate the effects of perturbations to each parameter on (i) the mean number of infected tomato plants and (ii) the variability of the outbreak after 70 days . We perturbed each parameter independent of the other. The results are shown in Figures 6 and 7, where the baseline values refer to the results using the estimated parameters.

Figure 6 shows how changes in the beet-to-tomato transmission rate have a larger impact on the mean number of tomato plants at day 70 than similar perturbations of the beet-to-beet transmission rate. Similarly, Figure 7 shows that the standard deviation at day 70 is more sensitive to changes of the beet-to-tomato transmission rate than equivalent changes of the beet-to-beet transmission rate.

5 Conclusions and Discussion

In this paper we used a susceptible-infected compartmental stochastic model to describe the spread of Curly Top disease in a population of beet and tomato plants. The model requires a pair of susceptible-infected compartments for each population, however the contagion events are only of the type beet-to-beet and beet-to-tomato. Experimental data gathered from a 2019 tomato planting in northern Utah was used to first parameterize a deterministic model. Once identified, the transmission parameters were used to determine the propensity functions for the stochastic model.

Extensive simulations with the stochastic model generated data that we used to approximate the variability of the number of infected tomato plants 70 days after planting. Figure 5 shows how the standard deviation of the number of infected tomato plants grows with respect to time. We also use simulations to address the sensitivity of the mean and standard deviation of the number of infected tomato plants to perturbations in the parameters. Figures 6 and 7 show that increasing (or decreasing) the beet-to-tomato transmission rate produces a larger impact on the number of infected tomato plants as compared to similar changes of the beet-to-beet transmission rate. This suggests that control measures aiming to reduce the beet-to-tomato pathogen

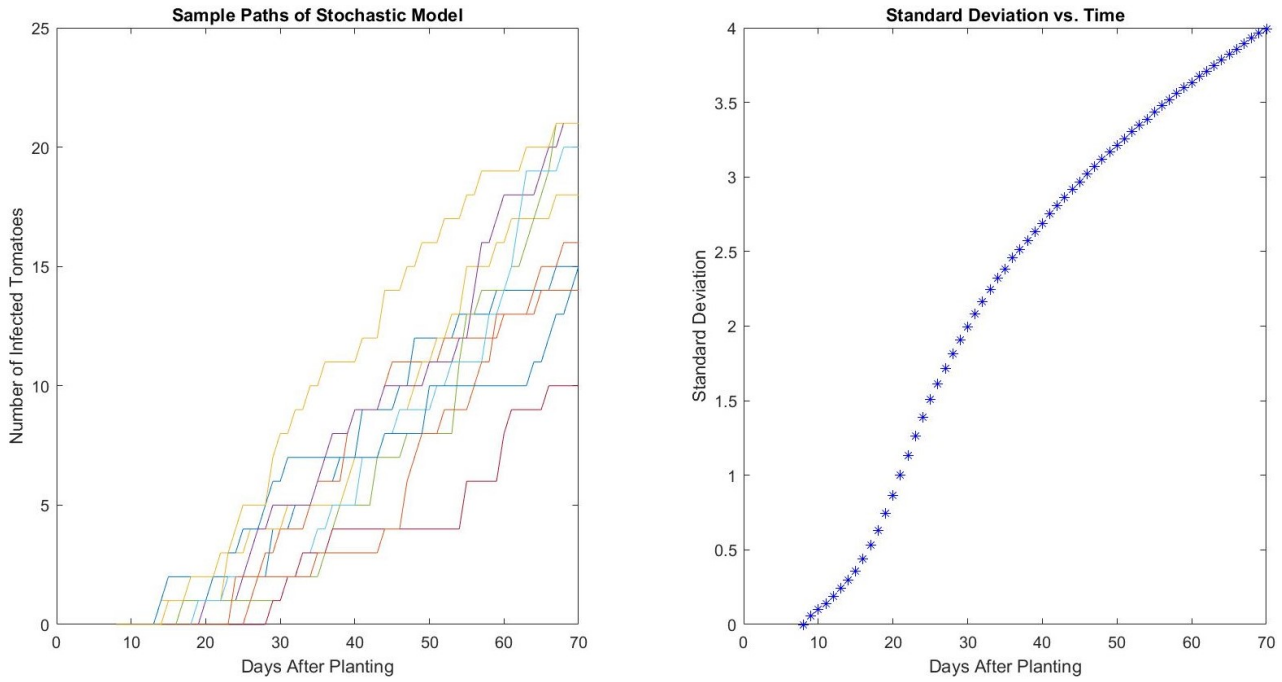

Figure 5: **Left:** Ten sample paths generated with the stochastic model. The parameter values used correspond to those fit from the deterministic model. **Right:** Plot of the standard deviation for the incidence distribution, obtained from 10,000 simulated sample paths, at each point in time.

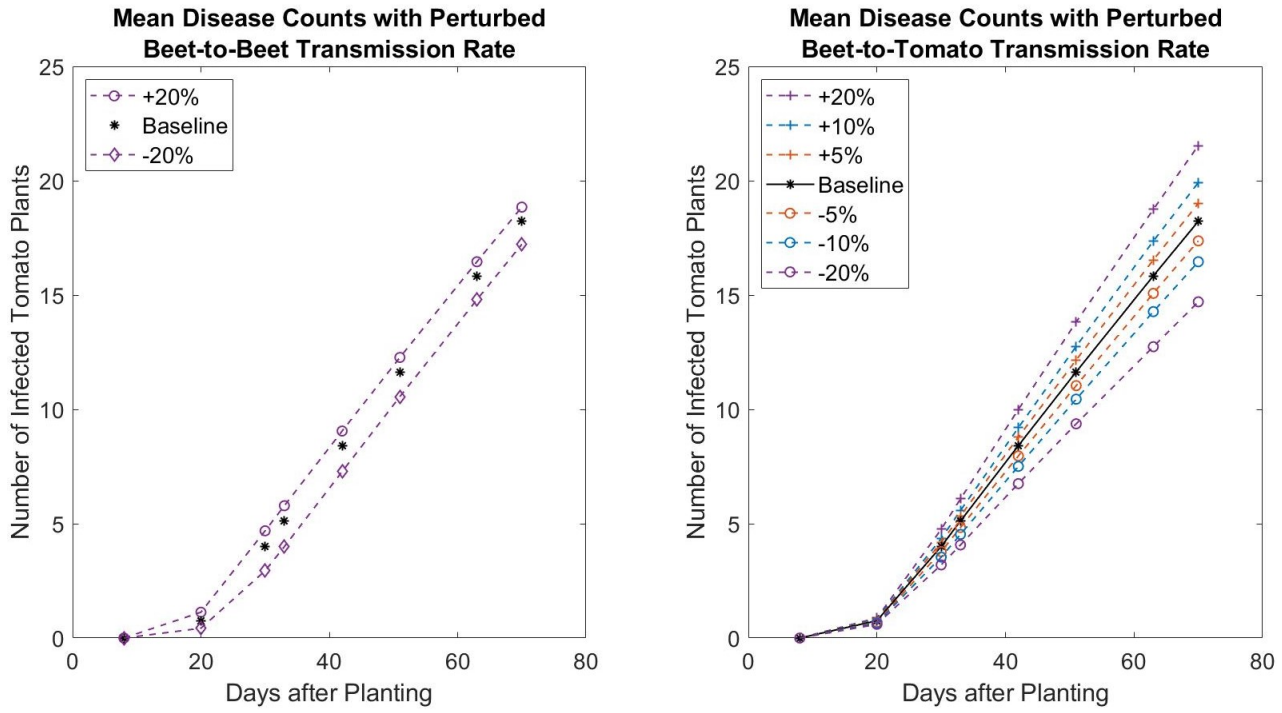

Figure 6: Sensitivity of the disease incidence from the transmission parameters. **Left:** Mean number of infected tomato plants after 10,000 simulations of the SSA algorithm with +20% (purple circles), 0% (black stars), −20% (purple squares) change to the beet-to-beet transmission **Right:** Mean number of infected tomato plants after 10,000 simulations of the SSA algorithm with +20% (purple pluses), +10% (blue pluses), +5% (orange pluses), 0% (black stars), −5% (orange circles), −10% (blue circles), −20% (purple circles) change to the beet-to-tomato transmission.

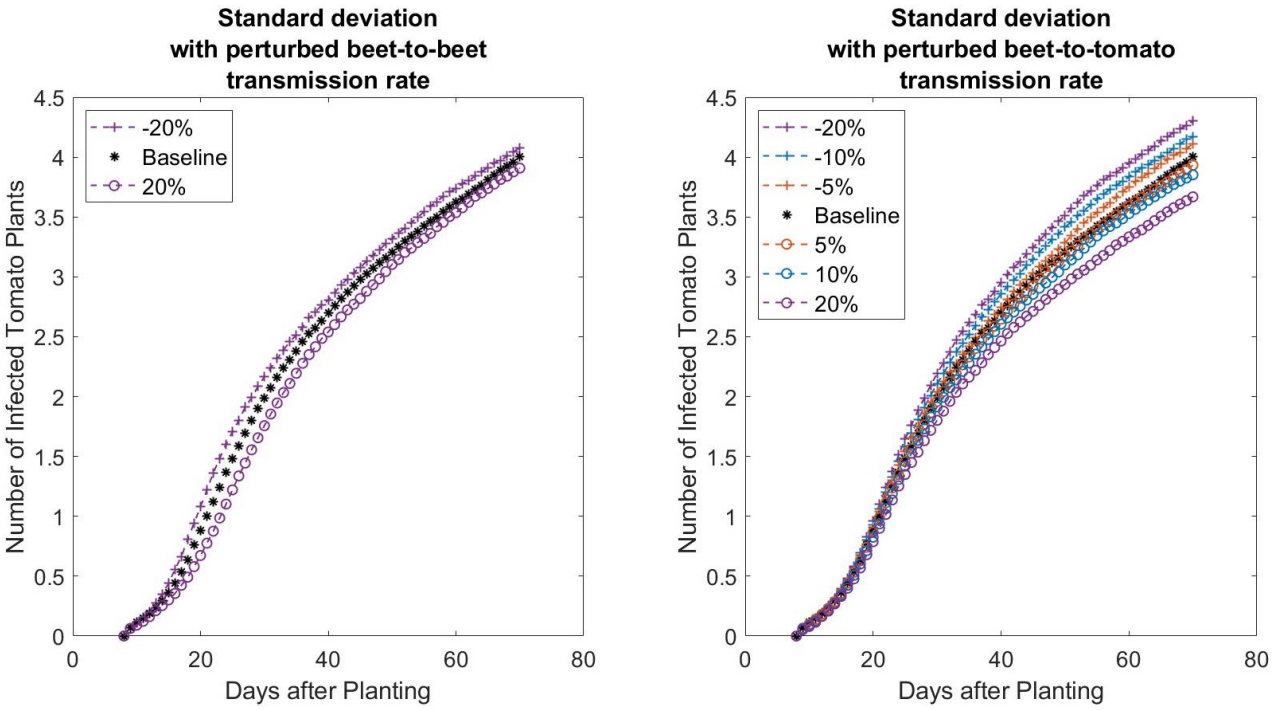

Figure 7: Sensitivity of the disease incidence from the transmission parameters. **Left:** Plot of the standard deviation for the incidence distribution, obtained from 10,000 simulated sample paths, at each point in time with +20% (purple circles), 0% (black stars), −20% (purple squares) changes to the beet-to-beet transmission **Right:** Plot of the standard deviation for the incidence distribution, obtained from 10,000 simulated sample paths, at each point in time with +20% (purple pluses), +10% (blue pluses), +5% (orange pluses), 0% (black stars), −5% (orange circles), −10% (blue circles), −20% (purple circles) change to the beet-to-tomato transmission.

transmission should be preferred. By assessing the variability, we gain insight into the risk associated with CT outbreaks in tomato plants. The variability highlights the range of possible outcomes from an outbreak. This information may be useful to individuals who manage tomato crops as they can better determine the risk associated with their field.

While simple in its construct, our model serve as a first step in developing further theoretical tools to describe the progression of CT in tomato crops. Our CT model takes into account heterogeneous plant populations, consisting of beet and tomato plants, a concept that is absent in the current CT literature. Furthermore, many wild plants can serve as reservoir for the CT virus for which the disease patterns are unknown.

Further experiments designed to collect data on the number of infections in tomato plants are needed, which are unfortunately expensive in terms of time and cost. A second experiment was conducted the following year but it failed due to unfavorable environmental conditions. Data collection for the disease vector is difficult to carry out because leafhoppers migrate quickly and their population waves pass through the fields in a few weeks. Incorporating the leafhopper's movement into the model remains a challenging task.

Our modelling assumptions are suitable for relatively small tomato plantations, but further considerations are needed in exploring large commercial tomato crops. In our model we assume susceptible plants (beets or tomatoes), are equally likely to become infected as any other plant of the same type. This assumption is permissible since we are working in a relatively small area. Additional work is needed to determine a size threshold for when this assumption is no longer valid. There is also a need to construct spatially explicit models that take into account the spatial distribution of plants. Spatial models could provide additional insight into the CT's dispersal characteristics as well as control methods that are focused on the spatial distribution of the plants.

Acknowledgments

Rachel Frantz received support from the National Science Foundation under Grant No. 1633756. The field experiment was supported by the Utah State University Extension grant. We are grateful to the reviewers whose feedback and insight improved this manuscript.

Data Availability

Source code available at https://github.com/rachelf23256/CT-Modeling-in-Tomatoes.

References

Chen, L.-F., O. Batuman, B. J. Aegerter, and R. L. G. J. Willems (2017). First report of curly top disease of pepper and tomato in California caused by the spinach curly top strain of beet curly top virus. *Plant Disease 101*(7), 1334. 53

Chen, L.-F., K. Brannigan, R. Clark, and R. L. Gilbertson (2010). Characterization of curtoviruses associated with curly top disease of tomato in California and monitoring for these viruses in beet leafhoppers. *Plant Disease 94*(1), 99–108. 54

Chen, L.-F. and R. L. Gilbertson (2011). Genetic diversity in curtoviruses: a highly divergent strain of beet mild curly top virus associated with an outbreak of curly top disease in pepper in Mexico. *Archives of Virology 156*(4), 547–555. 53

Douglass, J. R. and W. C. Cook (1952). The beet leafhopper. In *Insects; the yearbook of agriculture, 1952*, pp. 544–550. Washington: U.S.G.P.O. Accessible via https://archive.org/details/insectsyearbooko00unit/page/544/mode/2up. 54

Douglass, J. R. and W. C. Cook (1954). *The Beet Leafhopper*. Circular (United States. Department of Agriculture). U.S. Department of Agriculture. 53, 54

Gillespie, D. T. (1977). Exact stochastic simulation of coupled chemical reactions. *The journal of physical chemistry 81*(25), 2340–2361. 56

Heflebower, R., C. Reid, and D. Winward (2012). Controlling curly top of tomato using resistant varieties and row covers. *Journal of the NACAA 5*(2), 1–3. 54

Heflebower, R. and J. Schalau (2014). Controlling curly top virus of tomato. 53, 54

Higham, D. J. (2008). Modeling and simulating chemical reactions. *SIAM Review 50*(2), 347–368. 56

Klein, M. (1992). Role of *Circulifer/Neoaliturus* in the transmission of plant pathogens. In K. F. Harris (Ed.), *Advances in Disease Vector Research*, Chapter 5, pp. 150–175. Springer-Verlag. 56

Lagarias, J. C., J. A. Reeds, M. H. Wright, and P. E. Wright (1998). Convergence properties of the nelder–mead simplex method in low dimensions. *SIAM Journal on optimization 9*(1), 112–147. 56

Madden, L. V., G. Hughes, and F. van den Bosch (2007). *The Study of Plant Disease Epidemics*. APS Press, St. Paul, MN. 56

Nischwitz, C. (2020). Seed-transmitted wheat mosaic virus in sweet corn in Utah. Accessible via https://doi.org/10.1094/PHP-12-19-0092-BR. 54

Nischwitz, C. and M. W. Olsen (2010, January). First report of two curtoviruses in spinach and common beet in Arizona. *Plant Health Progress 11*(1), 57. 54

Stenger, D. C. and C. L. McMahon (1997). Genotypic diversity of beet curly top virus populations in the western United States. *Phytopathology 87*(7), 737–744. 53

Thomas, P. E. and R. K. Boll (1977). Effect of host preference on transmission of curly top virus to tomato by the beet leafhopper. *Phytopathology 67*(7), 903–905. 54