Modeling the spread of Curly Top Disease in tomato crops

Rachel Frantz*, Claudia Nischwitz**, Tyson Compton**, and Luis Gordillo*

*Utah State University, Department of Mathematics and Statistics, **Utah State University Department of Biology

Introduction

Curly Top Disease (CT) affects more than 300 species of plants. This includes economically important crops such as tomatoes, sugar beets, beans, and peppers in semi-arid areas across Utah, Arizona, California, and Nevada [1]. Tomatoes infected with CT display signs of stunted growth and upturned, choleric leaf margins. In most cases, infected tomatoes do not produce new fruit, and fruit that was set prior to infection fails to fully mature [2]. Historically, CT has caused extensive damage in tomato crops, resulting in substantial economic loss for the tomato industry [3]. Control methods for CT are scarce, and methods for predicting and assessing the scope of CT outbreaks are limited.

Goal: Accurately model the progression of Curly Top Disease in tomato crops and assess the variability of Curly Top Disease outbreaks.

Transmission Characteristics

Curtoviruses are transmitted by the beet leafhopper, an insect that acquires and transmits the viruses while feeding on plants [2]. A virus-free leafhopper acquires the pathogen from an infected plant only after a relatively long exposure [5]. Tomatoes are not a preferred food source for leafhoppers, and after biting a tomato, leafhoppers quickly move on [6]. Thus, it is believed that the spread of CT to unsuitable hosts, such as tomatoes, is a consequence of sample feeding by the leafhoppers [5, 6].
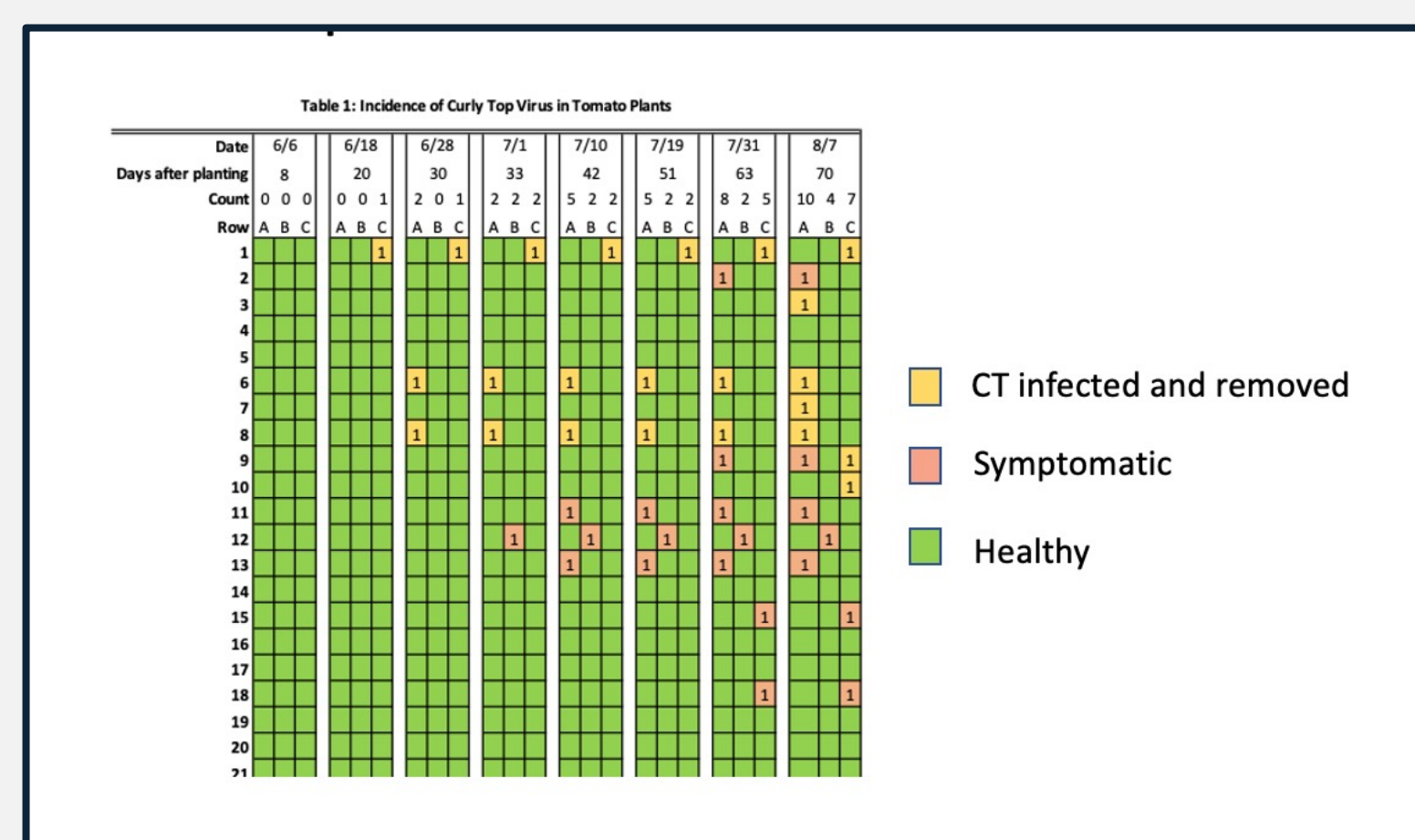

Figure 1: Weekly disease maps for CT progression in tomatoes observed from June 6 - August 7, 2019.

Methods

We implemented two theoretical models, deterministic and stochastic, which we proposed within the classical susceptible-infected framework. The models assume a mixed plant population of beets (preferred hosts) and tomatoes (unsuitable hosts). The models consist of four classes, one susceptible class and one infected class, for each plant type. Due to the mechanisms of transmission, we consider only the beets to be infective agents. We parameterize the model using data collected from an experimental tomato field (Figure 1), in which tomatoes were planted next to beets. We then use these parameters to study the evolution of disease incidence variability over time.

Deterministic Model:

The deterministic model is given by a set of coupled ordinary differential equations. This model assumes two transmission parameters for "between suitable host transmission" (beet-to-beet) and "from suitable host to non-suitable host" (beet-to-tomato) transmission. We begin by defining the following state and transmission parameters:

β: Rate of transmission from beets to tomatoes $\quad$ S_b: Susceptible beets
β': Rate of transmission from beets to beets $\quad$ I_m: Infected tomatoes
S_m: Susceptible tomatoes $\quad$ I_b: Infected beets

Then, the model is given by:

$$\frac{d}{dt}I_m = \beta S_m I_b \qquad \text{and} \qquad \frac{d}{dt}I_b = \beta' S_b I_b,$$

Stochastic Model:

For the stochastic model, we assume two possible transitions:

$$\text{infection of a tomato plant:} \quad (S_m, I_m, S_b, I_b) \to (S_m - 1, I_m + 1, S_b, I_b) \quad \text{with rate } \beta S_m I_b$$
$$\text{infection of a beet plant:} \quad (S_m, I_m, S_b, I_b) \to (S_m, I_m, S_b - 1, I_b + 1) \quad \text{with rate } \beta' S_b I_b$$

We then use a stochastic simulation algorithm and the estimated transmission rates from the deterministic model to produce 10,000 realizations of the process.
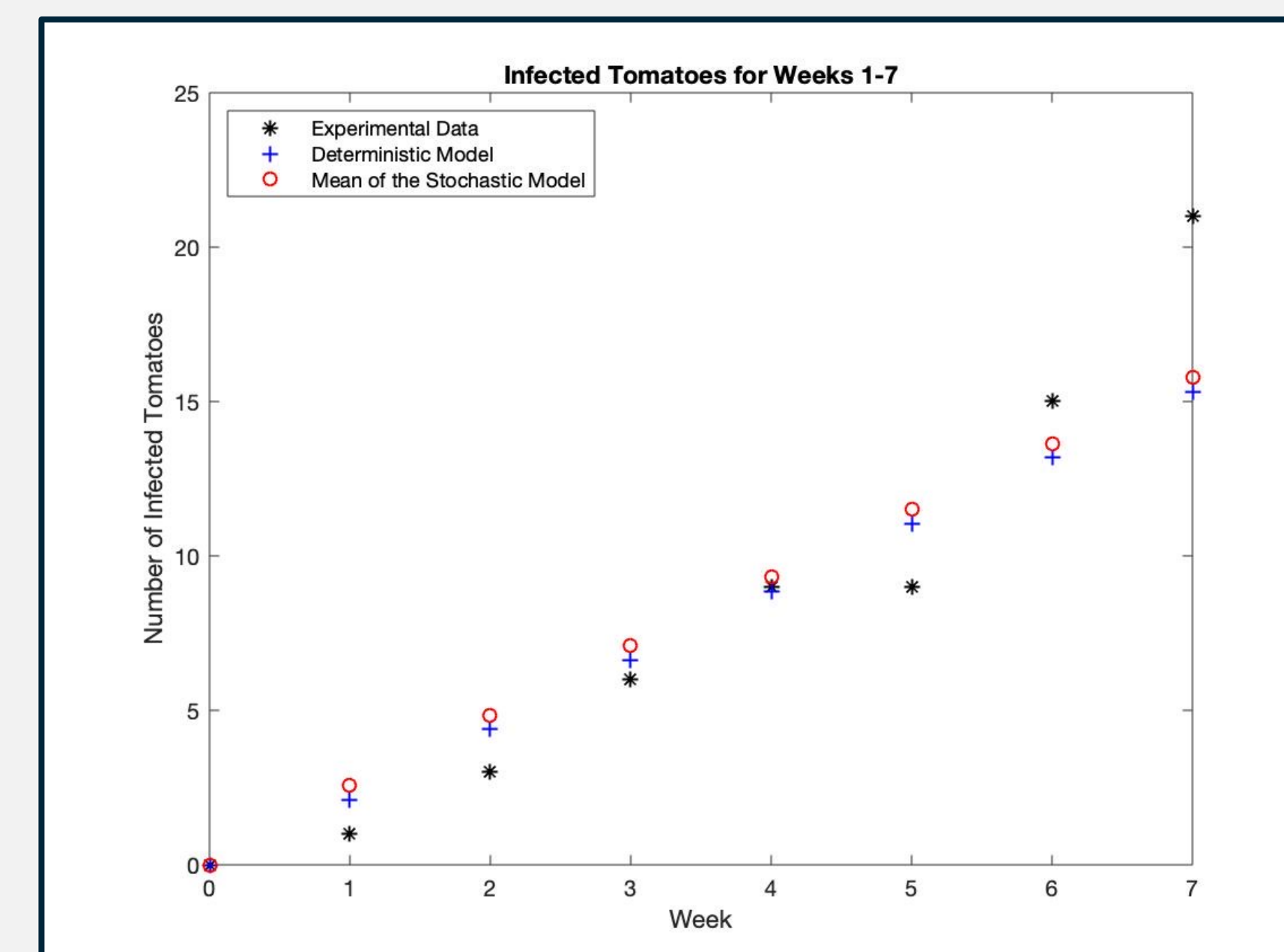

Figure 2: Disease counts from experimental data (black stars), outputs from the deterministic model (blue plus signs), and mean values of 10,000 paths generated using the stochastic simulation algorithm (red circles).
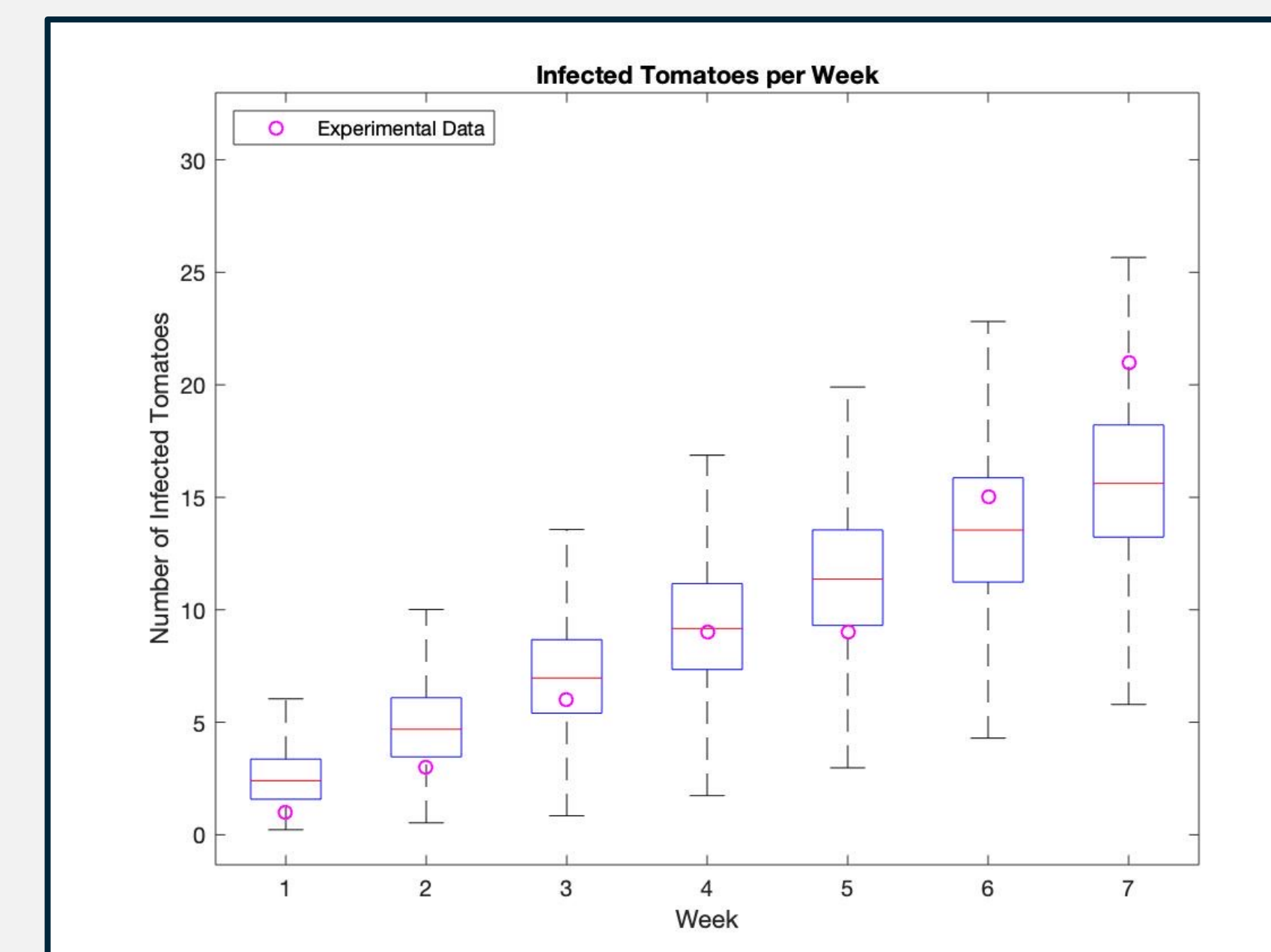

Figure 3: Box-plots of the simulated data for the number of infected tomatoes. The experimental data counts collected from the tomato planting are shown in pink circles.

Results

The stochastic and deterministic models provide reasonable approximations of the CT outbreak as compared to the observed disease counts (Figure 2). The standard deviation of the number of infected tomato plants over the first seven weeks is shown in Figure 4. The identification of the variability of CT outbreaks allows for important insight into the range of outcomes for any given outbreak.
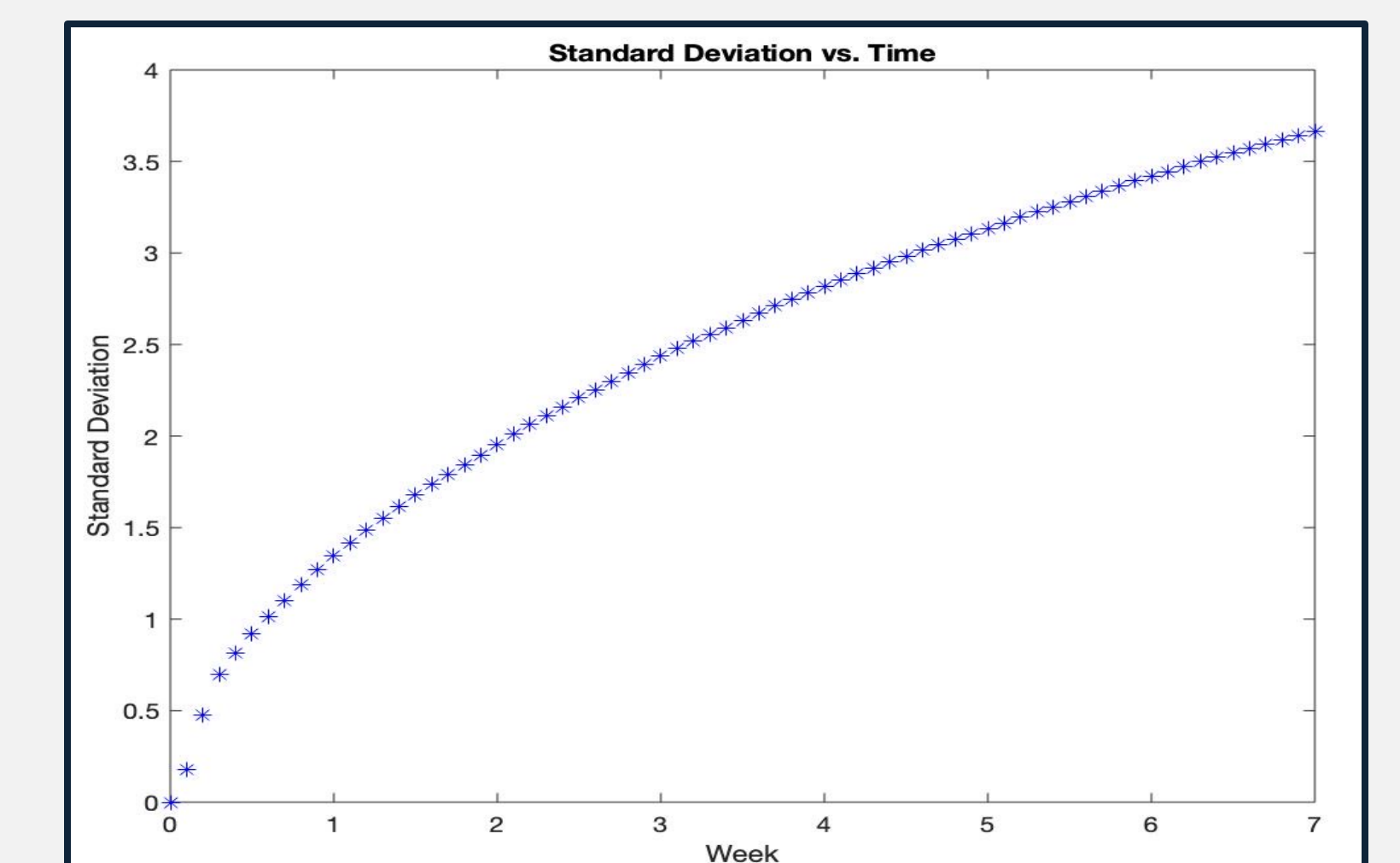

Figure 4: Standard deviation at each week of 10,000 simulations

Acknowledgements

References

[1] Chen, L.-F., and R.L.Gilbertson. 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 (2011), 547–555.
[2] Heflebower, R., and Schalau, J. Controlling Curly Top Virus of Tomato., 2014.
[3] Stenger, D. C., and McMahon, C. L. Genotypic Diversity of Beet Curly Top Virus Populations in the Western United States. Phytopathology 87, 7 (1997), 737–744.
[4] Heflebower, R., Reid, C., and Winward, D. Controlling Curly Top of Tomato Using Resistant Varieties and Row Covers . Journal of the NACAA 5, 2 (2012).
[5] Douglass, J. The beet leafhopper. US Department of Agriculture, 942 (1954)
[6] Thomas, P., and Boll, R. K. Effect of host preference on transmission of curly top virus to tomato by the beet leafhopper. Phytopathology 67, 7 (1977), 903–905.205