



Epidemiology of the Influenza A Virus Applied to Multi-Pen Pig Farms



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Abstract

Influenza A virus is a major concern in pork production. Influenza does not only affect our swine markets around the world, but it also affects swine care-givers as well as the greater population. In this study, the focus is on the dynamics of swine herds in order to understand the spread of influenza. Understanding the epidemiology of influenza will further support efforts in decreasing infected swine populations and preventing an ongoing pandemic. Matlab and ode45 have helped develop fully continuous mathematical models that represent a multi-pen pig farm. These models allow us to examine the differences in model dynamics between the populations of pigs separated by age, resulting in the use of one, two and four class models. Three basic SEIR models were constructed to understand the spread of influenza in swine herds and discovered that population size is inversely related to infection activity. To surmise the observations, greater swine populations resulted in less rapid influenza spread between the swine pens. With the three assortments of pig populations, additional experimentation dove deeper into sensitivity, vaccination and r0. For sensitivity, manipulation of parameters indicated each parameter's influence on population numbers and transmission rates of swine herds. Vaccination studies allowed the examination of how maternal immunity transferred to piglets affects model dynamics as well as the differences between homologous and heterologous vaccinations. The model predicts that piglets can obtain immunity from their mothers vaccination but is not lifelong and may lead to the piglets becoming susceptible again. The analysis of r0 guides research towards differentiating the severity of influenza in comparison to other viruses. Additionally, the results demonstrate the significant impact influenza A has in swine herds all over the country. Controlling the spread is vital in regards to public health and in understanding epidemiology.

Introduction

Among the zoonotic diseases known to modern science, influenza has been one of the most costly, deadly, and infectious diseases to date. Swine demand and consumption has led it to be one of the highest produced meats, maintaining a stable consumption rate of about 67 pounds per capita from 1999 to 2001 [3]. The rate at which pigs are infected with the influenza virus is subject to the swine's age, vaccination status, and whether or not they have antibodies in their system [6] In this study, models that mimic a swine farm are created to represent the characteristics of a swines' life cycle and the epidemiological barriers that are created from influenza. The goal of this experiment is to portray the evolution of infection within each population. By the nature of zoonoses, careful understanding of the organism capable of transmitting such a devastating infection to our population is the bare minimum.

Methods

- All models were run using ode45 in MATLAB on a Macbook Pro laptop.
- Multiple SEIR models were created to depict the changes in susceptible, exposed, infected and recovered pig populations of 2500 pigs within a 46 day time period.
 - One class
 - Four classes
- The SEIR models were then modified using vaccination and sensitivity strategies to determine details of transmission rates..
- Initial conditions and parameters come from a previous study.

Transmission Models

Figure 1

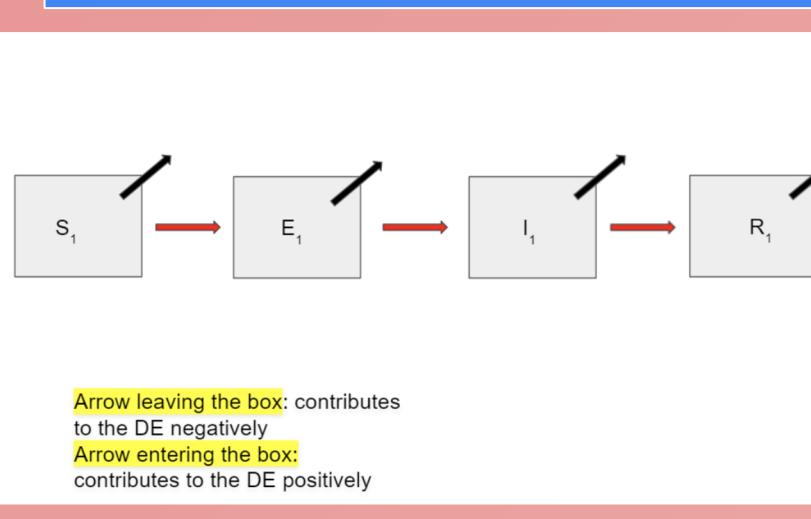
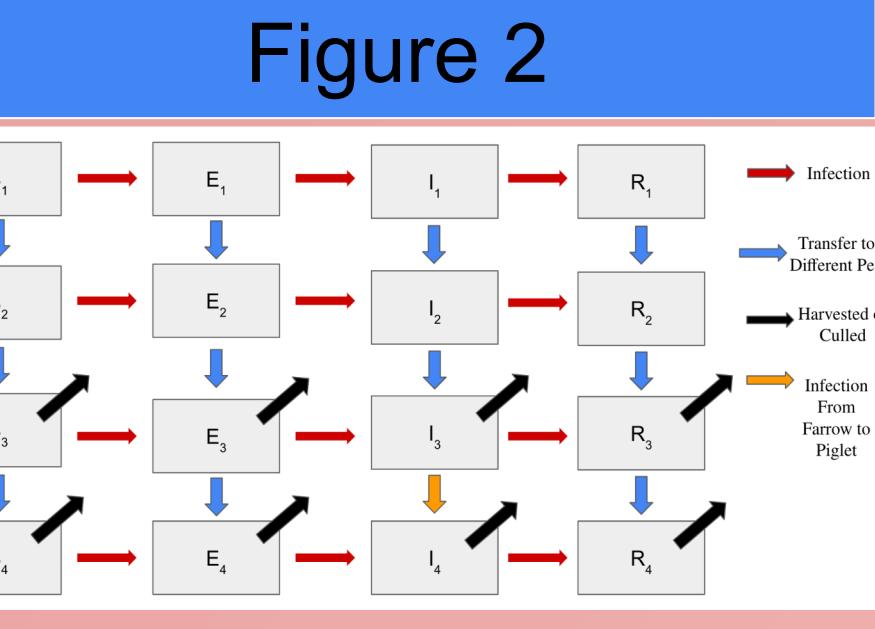


Figure 2



One Class of Pigs

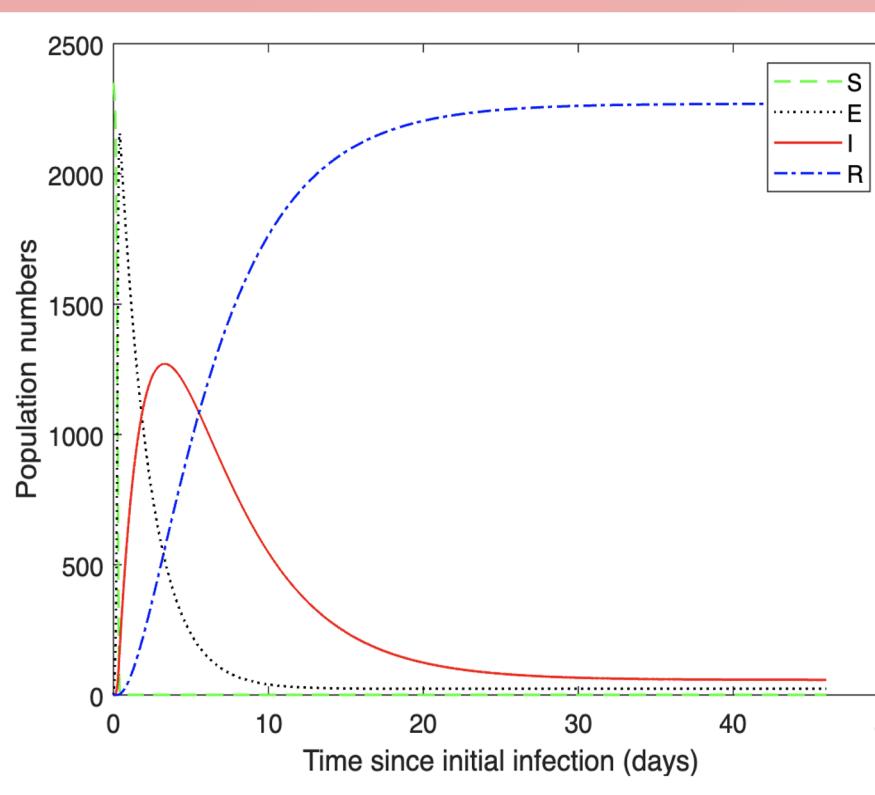
$$\frac{dS_1}{dt} = BC - \beta * S_1 * I_1 - \mu * S_1$$

$$\frac{dE_1}{dt} = \beta * S_1 * I_1 - (\mu + \sigma) * E_1$$

$$\frac{dI_1}{dt} = \sigma * E_1 - (\mu + \gamma) * I_1$$

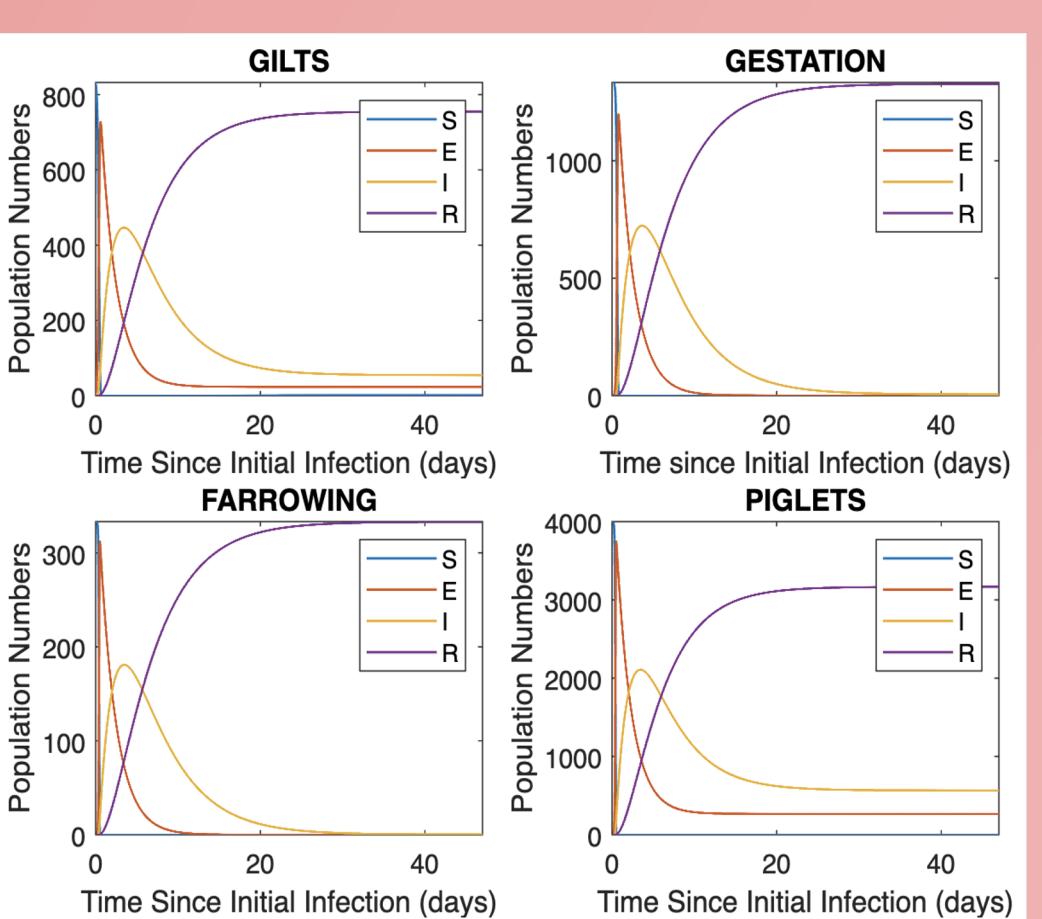
$$\frac{dR_1}{dt} = \gamma * I_1 - (\mu) * R_1$$

Figure 3



Four Classes of Pigs

Figure 4



$$\mu = \frac{12}{2500} \quad \gamma = 0.2$$

$$\frac{dS_1}{dt} = BC - \beta_1 S_1 I_1 - \beta_2 S_1 (I_2 + I_3 + I_4) - BC \frac{S_1}{N_1}$$

$$\frac{dE_1}{dt} = \beta_1 S_1 I_1 + \beta_2 S_1 (I_2 + I_3 + I_4) - BC \frac{E_1}{N_1} - \sigma_1 E_1$$

$$\frac{dI_1}{dt} = \sigma_1 E_1 - \frac{BC}{N_1} I_1 - \gamma_1 I_1$$

$$\frac{dR_1}{dt} = \gamma_1 I_1 - \frac{BC}{N_1} R_1$$

$$\frac{dS_2}{dt} = -\beta_1 * S_2 * I_2 - \beta_2 * S_2 * (I_1 + I_3 + I_4) + BC * \frac{S_1}{N_1} - BC * \frac{S_2}{N_2}$$

$$\frac{dE_2}{dt} = \beta_1 * S_2 * I_2 + \beta_2 * S_2 * (I_1 + I_3 + I_4) - BC * \frac{E_2}{N_2} + BC * \frac{E_1}{N_1} - \sigma_1 * E_2$$

$$\frac{dI_2}{dt} = \sigma_1 * E_2 + \frac{BC}{N_1} * I_1 - \frac{BC}{N_2} * I_2 - \gamma_1 * I_2$$

$$\frac{dR_2}{dt} = \gamma_1 * I_2 + \frac{BC}{N_1} * R_1 - \frac{BC}{N_2} * R_2$$

$$\frac{dS_3}{dt} = -\beta_1 * S_3 * (I_3 + I_4) - \beta_2 * S_3 * (I_1 + I_2) + BC * \frac{S_2}{N_2} - BC * \frac{S_3}{N_3}$$

$$\frac{dE_3}{dt} = \beta_1 * S_3 * (I_3 + I_4) + \beta_2 * S_3 * (I_1 + I_2) + BC * \frac{E_2}{N_2} - BC * \frac{E_3}{N_3} - \sigma_1 * E_3$$

$$\frac{dI_3}{dt} = \sigma_1 * E_3 - \gamma_1 * I_3 + BC * \frac{I_2}{N_2} - BC * \frac{I_3}{N_3}$$

$$\frac{dR_3}{dt} = \gamma_1 * I_3 + BC * \frac{R_2}{N_2} - BC * \frac{R_3}{N_3}$$

$$\frac{dS_4}{dt} = BRP - \beta_1 * S_4 * (I_3 + I_4) - \beta_2 * S_4 * (I_1 + I_2) - BRP * \frac{S_4}{PP}$$

$$\frac{dE_4}{dt} = \beta_1 * S_4 * (I_3 + I_4) + \beta_2 * S_4 * (I_1 + I_2) - BRP * \frac{E_4}{PP} - \sigma_1 * E_4$$

$$\frac{dI_4}{dt} = \sigma_1 * E_4 - \gamma_1 * I_4 - BRP * \frac{I_4}{PP}$$

$$\frac{dR_4}{dt} = \gamma_1 * I_4 - BRP * \frac{R_4}{PP}$$

Pig Farm Life Cycle

Figure 5

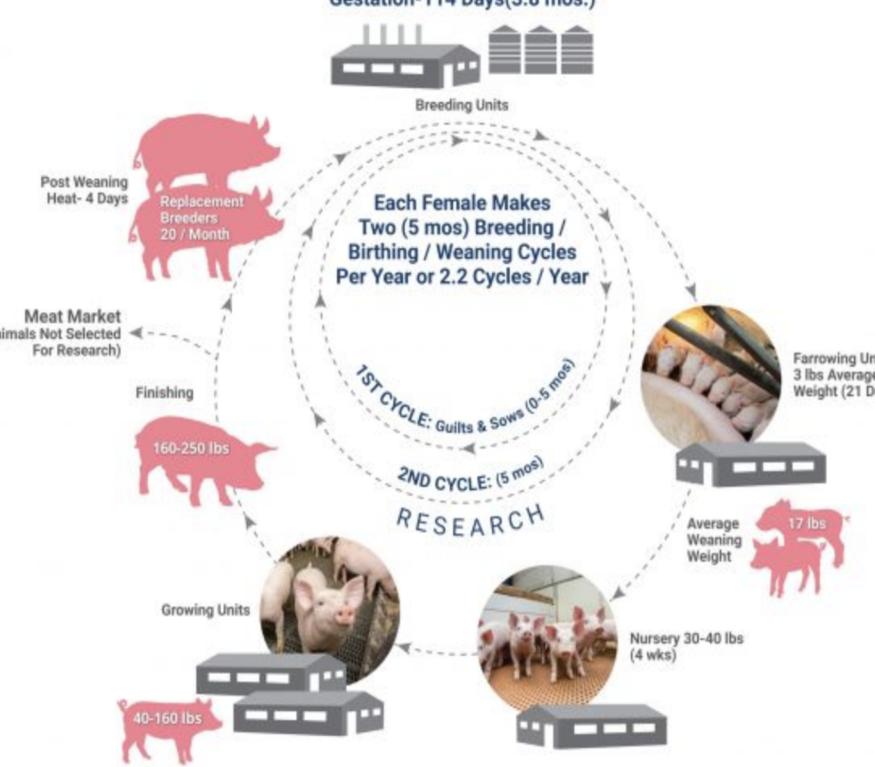


Figure 5

Image From Reference [7]

Model Modification: Vaccination

One Class of Pigs

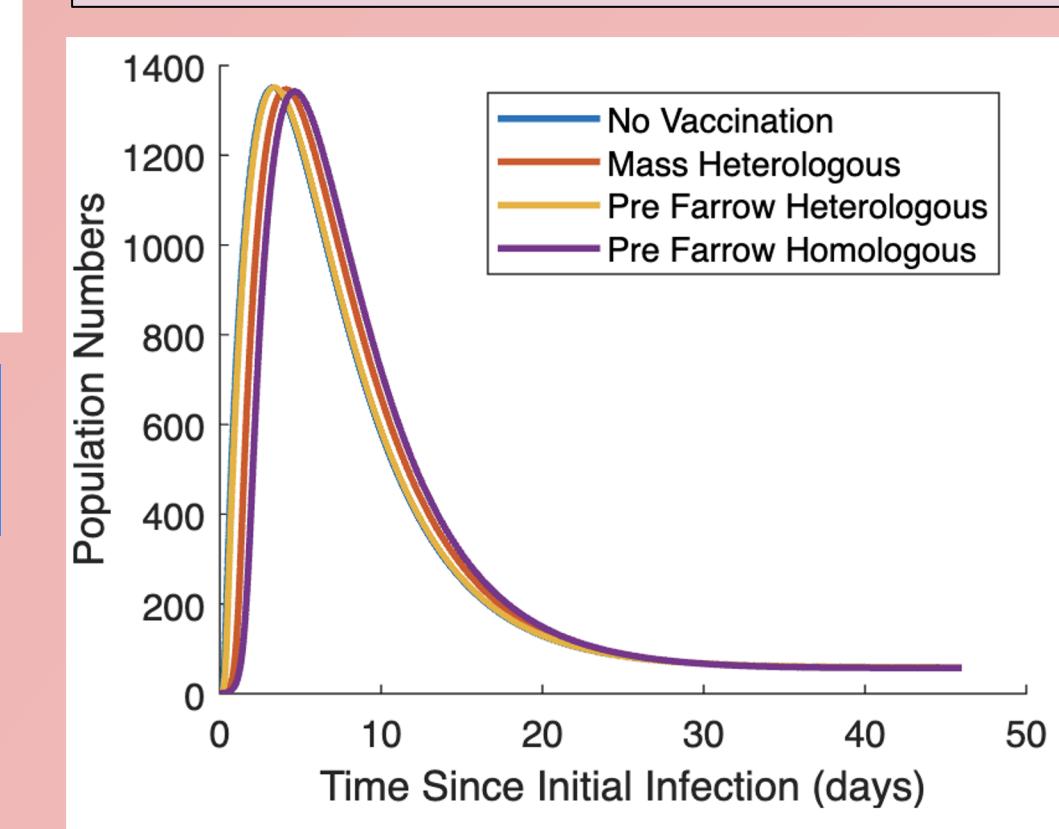


Figure 6

- Types of Vaccination:
- Original: 0.285
 - Pre-Farrow Homologous: 0.014
 - Pre-Farrow Heterologous: 0.174
 - Mass Heterologous: 0.0275

Four Classes of Pigs

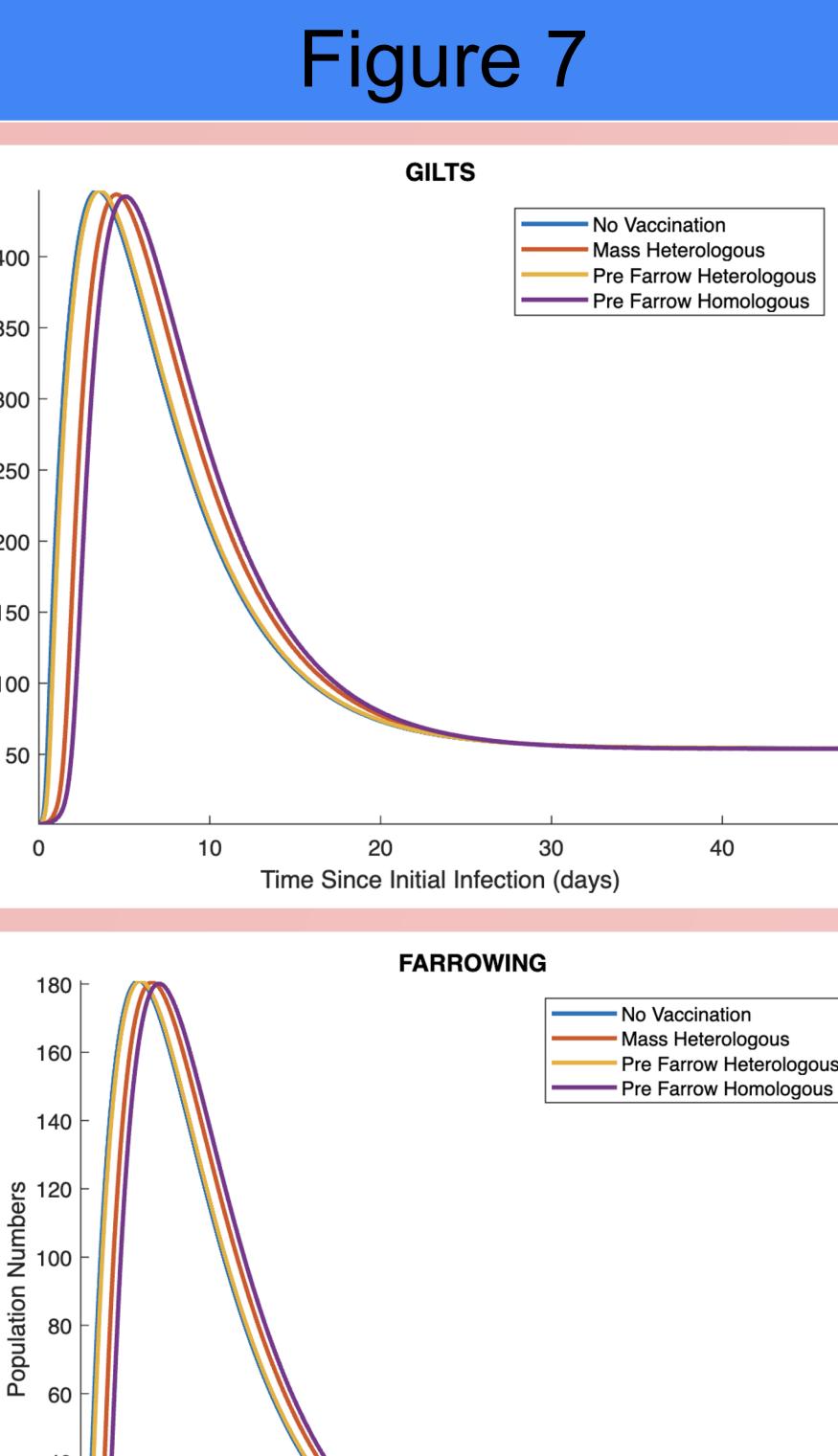


Figure 7

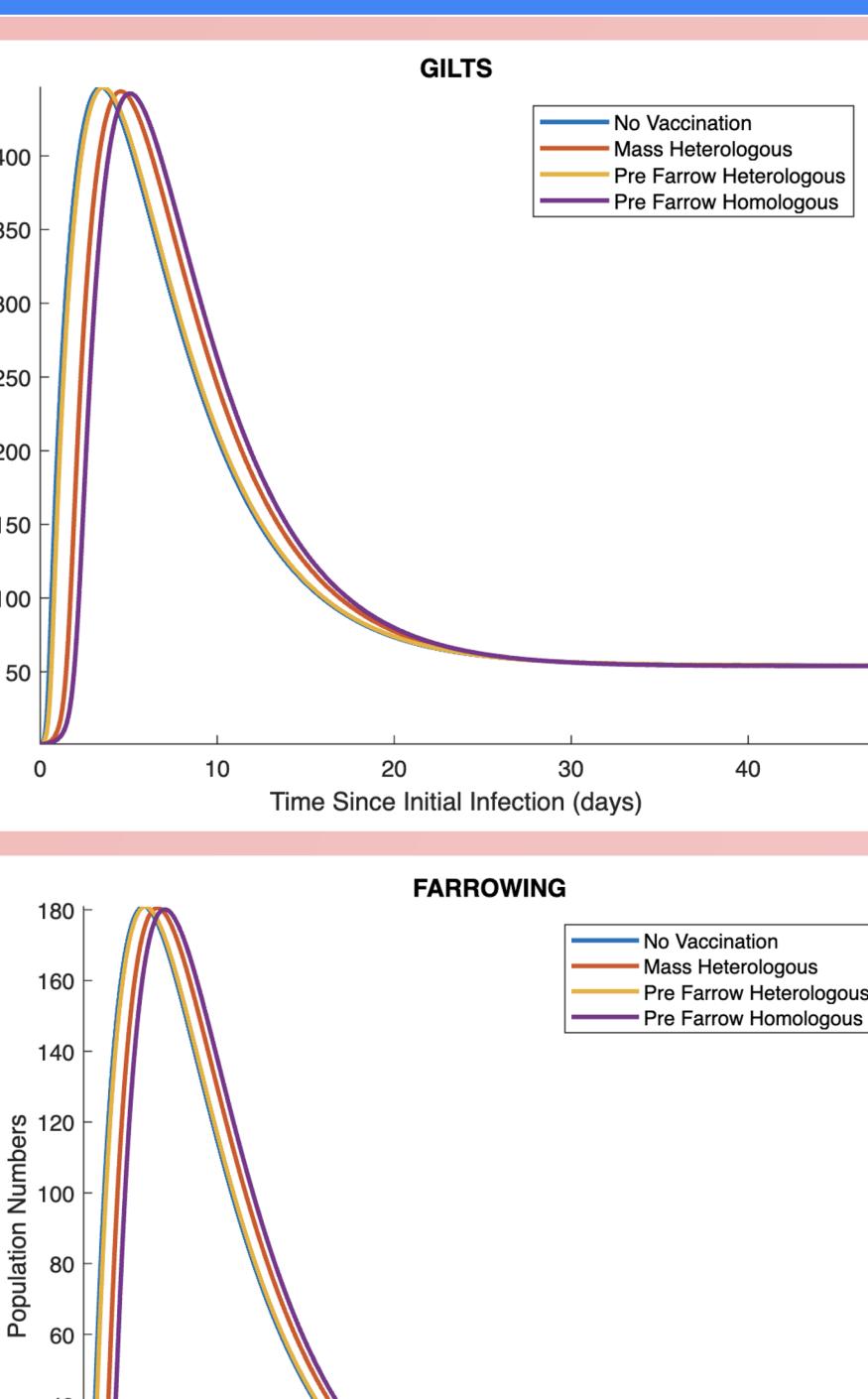


Figure 8

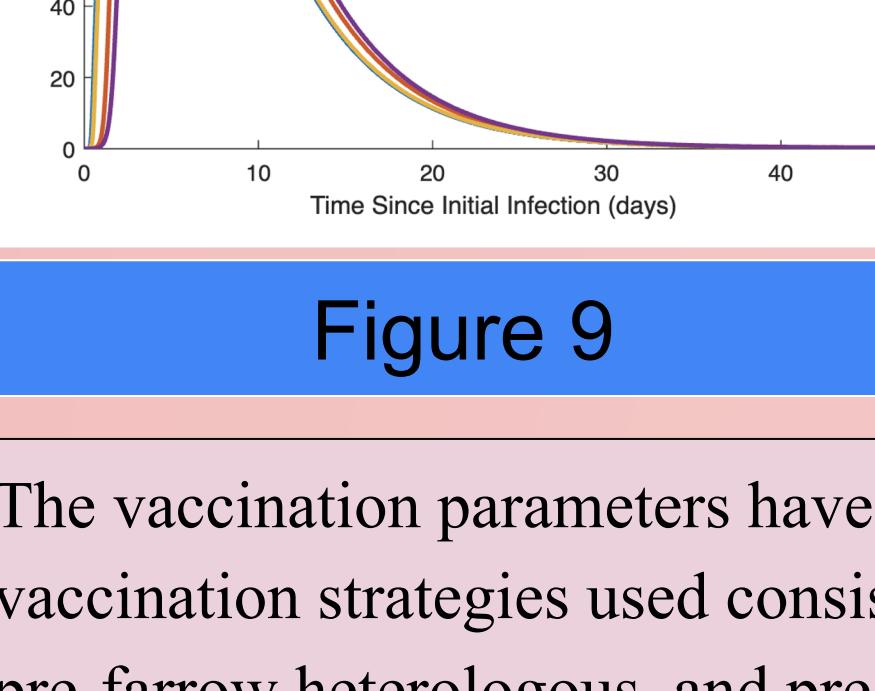


Figure 9

The vaccination parameters have been modeled in Figures 6-10. The vaccination strategies used consisted of no vaccination, mass vaccination, pre-farrow heterologous, and pre-farrow homologous. The results indicate that the peak of infection starts a little later with the mass vaccination and the pre-farrow homologous vaccination. Figure 10 shows infection rates happening at a slower rate and hitting the peak later than the other models shown. This is hypothesized to have to do with maternal immunity.

Figure 10

Model Modification: Sensitivity

One Class of Pigs

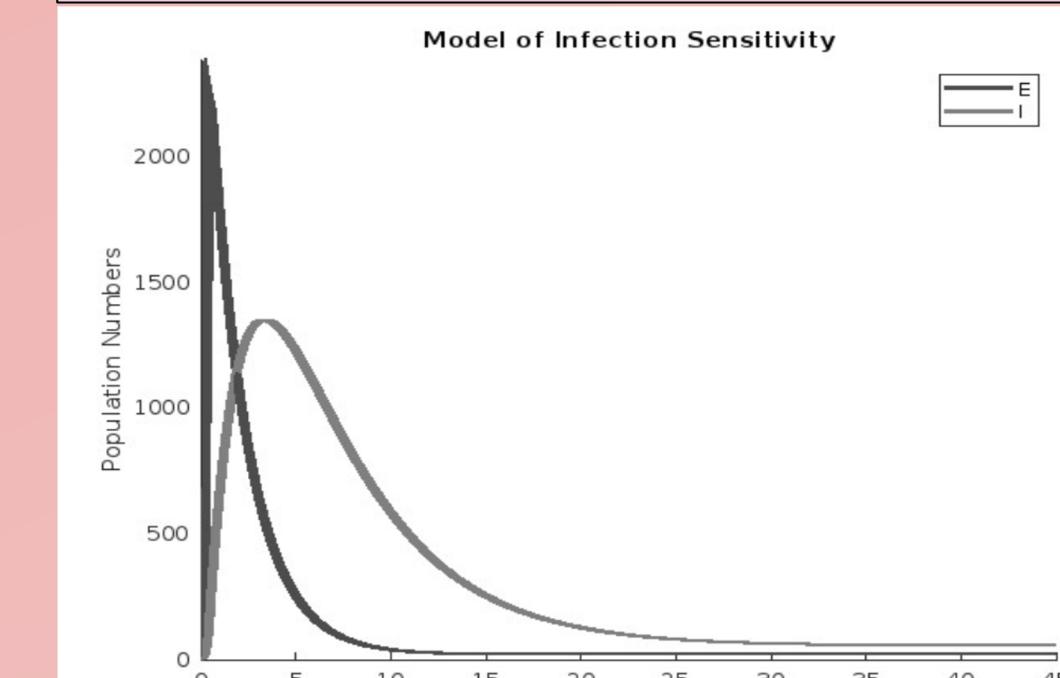


Figure 11

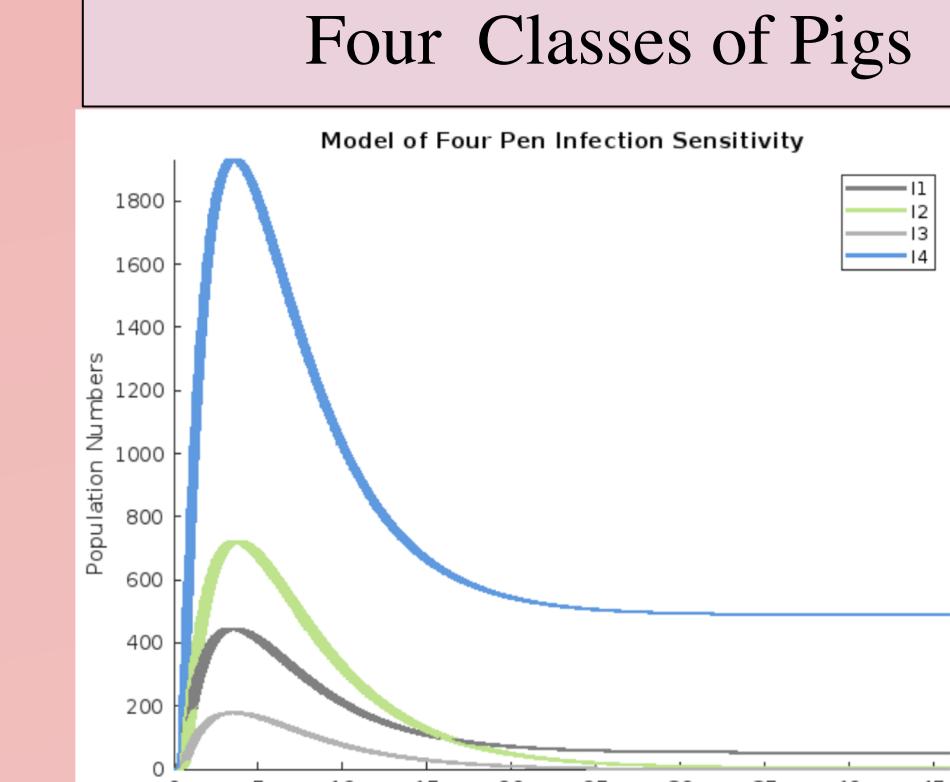


Figure 12

Our specific parameters have driven the models to take into account real life statistics to create a more realistic curve. Manipulating these parameters (specifically the infection rate among pigs) allows researchers to observe how each change impacts the dynamics of the model. With the help of ode45 inside MATLAB, we varied the beta values within a 95% confidence interval range in order to observe the effect on model dynamics of beta.

Discussion

Our multi-month simulation of influenza infection dynamics within swine farms displayed the multilayered relationships between pen separation, \$\beta\$ variation, and differing vaccination strategies. Firstly, the direct relationship between population size and time until complete population recovery supports previous research. Displayed by the pen separation, larger population sizes or less population separation results in delayed recovery time. The four pen model displays rapid recovery rates in comparison to the remaining two models. Sensitivity studies involving a varying \$\beta\$ within a controlled range presented predictable results. Increasing \$\beta\$ values, the infection constant, directly increased the infection population numbers over time. Additionally, vaccination strategies involving immunity from maternal swine being passed onto their offspring revealed interesting results. Though this immunity was effective, the immunity was not everlasting. After a short period of three weeks, the piglets were no longer protected by the parental immunity and were vulnerable to infection.

Acknowledgements

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