TREATMENT THRESHOLDS FOR THE GLASSY-WINGED SHARPSHOOTER BASED ON THE LOCAL EPIDEMIOLOGY OF PIERCE'S DISEASE SPREAD (A STAGE-STRUCTURED EPIDEMIC MODEL)

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ABSTRACT
The conditions for the successful invasion of a vineyard by Pierce's disease (PD) are not well understood. To help integrate what knowledge we do have and indicate areas where research is needed we are developing a more biologically detailed model than has been previously available. Fortunately there is a large ensemble of literature from epidemiology regarding this problem, and in addition, much has been done toward solving the kinds of equations that arise in this work in terms of both mathematics and software. Here we outline very briefly our progress to date, and the ways in which these sorts of models can help us to better manage and understand the PD system. Here we describe a system of delay equations for modeling the dynamics of PD vectored by the glassy-winged sharpshooter (GWSS). We will analyze and study this system to derive threshold conditions for the invasion of a vineyard by PD and GWSS. Thresholds for disease outbreaks are common among epidemiological systems and a large literature exists on this subject. In addition new software (not commercially released yet) has been made available to us for solving these kinds of systems. We will attempt to use our model system to bring this methodology to the PD/GWSS problem and find new ways of controlling this disease.

INTRODUCTION
Last year we presented a model to evaluate how the threshold might change in relation to various biological and ecological factors (Perring et al. 2003). It was designed to determine the number of GWSS required to cause a single PD infection in grape. The primary model parameters were the proportion of GWSS carrying PD, GWSS transmission efficiency of PD, proportion of GWSS that will move from citrus to grape, the number of grapevines that a single GWSS will visit, grape varietal susceptibility, and the probability of an infection event resulting in disease. Our recent work, reported in this progress report, is an extension of the previous efforts and is more biologically detailed, allowing us to address more complicated biological processes affecting the epidemiology of PD in grapes. Over eighty years of research in epidemiology has shown that epidemics tend to be triggered when the generation reproductive factor of the pathogen becomes greater than 1.0 (Kermack and McKendrick 1927, Anderson 1978, Diekmann and Heesterbeek 2000, van den Driessche and Watmough 2002, Wonham et al. 2003). This fortunate result is useful in management since it provides us with a target threshold that will trigger a PD epidemic in grapes. More than just a threshold, this approach will provide a function for the basic generation factor of increase of the pathogen, R_0, as a parameter function from the model. The pathogen will grow into an epidemic or decline to zero according to whether R_0 is greater or less than 1.0. It is particularly helpful that this threshold indicator is a function of all of the model parameters, since this indicates what parameters the threshold is most sensitive to and therefore how management can be most effectively focused. Some of the things that we intuitively expect to be important are density of GWSS, pathogen titer of the insects, and their dispersal rate and feeding rate.

OBJECTIVES
1. Develop a model to describe the epidemiology of GWSS transmission of PD to provide a framework for organizing data and examining relationships between data from different research projects.
2. Use the model to develop field-specific treatment thresholds to prevent GWSS transmission of PD.

RESULTS AND CONCLUSIONS
Our results consist of a model system of state equations describing the progress of PD in a vineyard vectored by GWSS. Here we develop our basic model as set of four balance equations, two equations for the GWSS and two equations for grapes.

The state variables, process functions and parameters are defined in Table 1. We emphasize that this model is in an early development stage, and undoubtedly will evolve and improve as we develop it further. We used the delay-differential equation (DDE) formalism developed by Murdoch et al. (1987) and Murdoch et al. (2003) for stage structured insects, and to their formulation we will add time dependence (temperature forcing) of the developmental delays (although for simplicity we will not elaborate on this here). The time dependence in the delays can be incorporated according to the mathematical recipes developed by Nisbet and Gurney (1983), Gurney et al. (1983), Gurney and Nisbet (1998) and Nisbet (1998). Methods for setting up the initial history for starting the models are outlined well in Gurney et al. (1983). We will solve our set of equations using a new delay differential equation (DDE) solver, ddesd.m, (with time and system varying delays) developed for The Mathworks (Matlab) by L. F. Shampine (Shampine & Thompson 2001, Shampine 2004). The solver is not yet a part of Matlab itself, but a version is available on the Web at: http://faculty.smu.edu/lshampin/current.html .

Our model system.
The state balance equations are written as a set delay-differential equations (DDEs) with functions for recruitment, infection and death rates as:

$$\begin{aligned}
\text{Susceptible Adults: } & \frac{dA_S(t)}{dt} = R(t-T_J)S_J - X(t) + X(t-T_1)S_1 - D_A(t) \\
\text{Infectious Adults: } & \frac{dA_I(t)}{dt} = X(t) - X(t-T_1)S_1 - D_A(t) \\
\text{Susceptible Vines: } & \frac{dS(t)}{dt} = D_V(t) - Y(t) \\
\text{Infectious Vines: } & \frac{dI(t)}{dt} = Y(t-T_2) - D_V(t)
\end{aligned} \qquad 1.1$$

We adopted and slightly modified the notation of Murdoch et al. (2003) by using $R(t)$, $X(t)$, $Y(t)$ and $D(t)$ to represent recruitment (R), infection of GWSS (X) infection of vines (Y) and death rate (D) functions for each stage, and we then define each of these for our case. These equations indicated that the rate of change of a stage is simply the input to that stage minus output from that stage. The interpretations for each equation are outlined below.

Susceptible adult equation.
The first equation says that susceptible adults have input from reproduction, one juvenile delay period (T_J) in the past times survival going through the juvenile stage, $R(t - T_J)S_J$. Another input to susceptible adults is (possible) recovery from an infectious adult class with a time delay, $X(t - T_1)S_1$ where T_1 is the time that the disease persists in an infected adult, and S_1 is the survival during the infectious period. Outputs from susceptible adults are infection by feeding on an infectious vine, $X(t)$, and death, $D_A(t)$.

Infectious adult equation.
The second equation says that infectious adults have input from the infection process, $X(t)$, (which was output from the susceptible class) and output to (possible) recovery from infection, $X(t - T_1)S_1$, and death, $D_A(t)$.

Susceptible vine equation.
The third equation says that susceptible vines have input equal to death rate of infectious vines, $D_V(t)$, that is, we assume that dead vines are replaced at the death rate. Output from susceptible vines is infection by infectious sharpshooters, $Y(t)$.

Infectious vine equation.
The last equation says that infectious vines have input from the infection process with a latent period time lag, $Y(t - T_2)$, where T_2 is the latent period of the disease in vines after becoming infected. We assume that all vines survive the latent period. Output from the infectious vine equation is by death of infected vines, $D_V(t)$.

Our model system of equations will allow us to simulate the introduction and progress of PD into a vineyard under different conditions and management strategies. What we would like is to see the disease die-out and not invade the vineyard effectively. What we do not understand at this point is how all of the factors influence this scenario and determine its progress and to which factors spread is most sensitive. By studying the dynamic behavior of this model system we can learn how different management options are likely to affect the disease progress in a vineyard, giving us new ideas and methods about how to best control and prevent disease outbreaks.

Table 1. State variables, process functions and parameters for GWSS-PD Model

Variables	Description	
$A_S(t)$	Susceptible GWSS Adults	
$A_I(t)$	Infectious GWSS Adults	
$S(t)$	Susceptible Vines	
$I(t)$	Infectious Vines	
Process Functions		**Process Sub-Models**
$R(t-T_J)S_J$	Recruitment into the adult stage	$R(t-T_J)S_J = b\left(A_S(t-T_J)+A_I(t-T_J)\right)S_J$
$D(t)$	Death rate for a stage	Linear constant death rate, e.g.: $D_A(t) = d_A A(t)$
$X(t)$	Infection rate for GWSS	$X(t) = \alpha I(t) A_S(t)$
$Y(t)$	Infection rate for vines	$Y(t) = \beta S(t) A_I(t)$
S_J	Survival of stage J with constant death rate	$S_J = \exp(-d_J T_J)$
Parameters		
b	Average birth rate	
T_i	Time in the ith stage or process	
d_i	Constant death rate for ith stage	
a	Transmission rate for GWSS	
β	Infection rate for vines	

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