Bayesian inference for an emerging arboreal epidemic in the presence of control

Matthew Parry1,2, Gavin J. Gibson3, Stephen Parnell4, Tim R. Gottwald5, Michael S. Irey1, Timothy C. Gast1, and Christopher A. Gilliganb,1

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The spread of Huanglongbing through citrus groves is used as a case study for modeling an emerging epidemic in the presence of a control. Specifically, the spread of the disease is modeled as a susceptible-exposed-infectious-detected-removed epidemic, where the exposure and infectious times are not observed, detection times are censored, removal times are known, and the disease is spreading through a heterogeneous host population with trees of different age and susceptibility. We show that it is possible to characterize the disease transmission process under these conditions. Two innovations in our work are (i) accounting for control measures via time dependence of the infectious process and (ii) including seasonal and host age effects in the model of the latent period. By estimating parameters in different subregions of a large commercially cultivated orchard, we establish a temporal pattern of invasion, host age dependence of the dispersal parameters, and a close to linear relationship between primary and secondary infectious rates. The model can be used to simulate Huanglongbing epidemics to assess economic costs and potential benefits of putative control scenarios.

Case study: Huanglongbing

Huanglongbing (HLB), also known as Citrus Greening, is the most destructive citrus pathosystem worldwide (10). It causes severe chlorosis of foliage and dieback, leading to tree death in some cases, and is associated with fruit drop as well as misshapen, discolored, and ill-flavored fruit. There is no known source of resistance to HLB within commercial citrus cultivars or genetically accessible citrus relatives, and there is no known commercially viable cure for an infected tree. In the last decade, HLB has inflicted increasingly severe economic losses on growers in some of the world’s key citrus-growing areas (10).

HLB is associated with three bacteria (11): Candidatus Liberibacter asiaticus (Las), Candidatus Liberibacter africanus (Laf), and Candidatus Liberibacter americanus (Lam). In the western hemisphere, Las is now the most prevalent type. The major vector of HLB is the Asian citrus psyllid (ACP), Diaphorina citri. During feeding, Las-carrying psyllids pass the bacteria into the vascular system of a tree. There, the bacteria multiply and become increasingly distributed, although not uniformly, throughout the tree. Subsequently, a proportion of psyllids feeding on Las+ sites on the tree become Las carriers, completing the loop of the host-vector-pathogen system. Las appears to have minimal impact on ACP.

The timeline of HLB in citrus is measured in years, with dependence on age and variety of tree (10, 12, 13). Once symptoms appear...

Significance

Fast-moving and destructive emerging epidemics are seldom left to run their course because of the imperative to control further spread. Contemporaneous control measures, however, greatly complicate the characterization of the disease transmission process and the extraction of the epidemiological parameters of interest. The spread of Huanglongbing on orchard scales is used as a case study for modeling an emerging epidemic in the presence of control. We show that even with missing and censored data, and with seasonal and host age dependencies, it is possible to infer the parameters of a fully spatiotemporal stochastic model of disease spread. The value of the fitted model is to provide an engine for simulation studies of the costs and benefits of proposed disease control strategies.


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1To whom correspondence should be addressed. E-mail: cag1@cam.ac.uk.

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are evident, they become increasingly severe, and the commercial value of the infected tree rapidly diminishes. Of major concern, however, is the long incubation period of infection, which is between 6 mo and 3 y. As a consequence, visual inspection is not a reliable method to assess the health of a tree, and although the cryptic period appears to be only of order several months, it is clear an epidemic can be underway well before symptoms are first seen.

ACP were first identified in the US state of Florida, in the coastal regions near Miami, in 1998, and HLB was first detected in 2005 (10). Subsequently, ACP reached the commercially important citrus-growing regions of central Florida. HLB has been endemic there since 2005, resulting in huge losses for growers due to crop loss and the costs of detection efforts, control measures, and tree removals and replantings.

The aim of this work is to provide and fit a phenomenological model of the tree-to-tree spread of HLB on orchard scales while accounting for contemporaneous control measures. Such a model can then be used as the engine for simulations of HLB epidemics to assess the economic costs and potential benefits of putative control strategies. We used an extensive dataset, collected by Southern Gardens Citrus, relating to more than a quarter of a million trees in the Southern Gardens plantation in south Florida. In particular, observational sweeps for symptomatic trees, conducted over the course of several years, give a series of spatial “snapshots” of the progress of the epidemic. We propose a spatially explicit, stochastic susceptible-exposed-infectious-detected-removed (SEIDR) model and carry out Bayesian estimation of the model parameters. Specifically, we ask the following questions. Can the primary and secondary rates of infection? What is the range of control measures? If so, can we identify details of the parameters. Specifically, we ask the following questions. Can the primary and secondary rates of infection? What is the range of control measures? If so, can we identify details of the parameters.

The novelty of the current work lies in model comparison and chain Monte Carlo (MCMC) methods developed in refs. 15–18); indeed, likelihood estimation of dispersal and transmission parameters for stochastic models from spatial snapshots of disease has attracted previous attention (14–18). The estimation of dispersal and transmission parameters for stochastic models from successive snapshots for a continuous time stochastic model was first applied (14) to citrus tristeza disease in plantations. The current analysis is based on subsequent Markov chain Monte Carlo (MCMC) methods developed in refs. 15–17. The novelty of the current work lies in model comparison and inference for data from successive snapshots subject to interference by disease control strategies, variable host age, and periodic environmental forcing. A vital feature of the approach is the ability to handle latent and censored data.

Methods and Results

Data. Southern Gardens is a commercial citrus plantation consisting of ~4,900 ha of contiguous citrus plantings, located about 36 km south of Clewiston, Florida. The Southern Gardens dataset concerns more than 250,000 trees in a rectangular region of 180 contiguous blocks. Apart from its northeast corner, the region under consideration is surrounded by further blocks of citrus. The east-west dimension of the region is 3.5 km; the north-south dimension is 2.4 km. The blocks are arranged in six east-west rows of 30 blocks, with each block typically containing around 1,500 trees arranged in 14 north-south rows. The spacing between trees along rows is between 3 and 4 m; the spacing between rows is ~8 m; and the typical spacing between blocks is 25 m.

The blocks are all sweet orange, Citrus sinensis (L.) Osbeck, of two scion cultivars Hamlin and Valencia in nearly equal proportion, and grafted on Poncirus trifoliata (L.) Raf.; Citrus × limonia (Lam.) Osbeck × Poncirus trifoliata (L.) Raf.; and Volk, Citrus × swinglei Hara & Tan. Of the 180 blocks, 103 were planted in 1988 or 1989, and 77 were planted in 1999 or 2001–2003. Thus, the blocks naturally split into old and young blocks. The layout of the blocks is shown in Fig. 1.

Four sweeps for symptomatic trees were made through the blocks between November 2005 and July 2007, with each sweep taking approximately 3 mo to complete. The spread of HLB during the four sweeps is shown in SI Text. The epidemic first becomes apparent in young trees in the east and then spreads both westward and to older trees. More than 27,000 trees were found to be symptomatic. Symptomatic trees were fully enumerated using a differential global positioning system and tagged for subsequent removal. In most blocks, there was a sharp increase in detections in either the second or third observation cycle, followed by a decrease in detections in the fourth cycle. Symptomatic means HLB symptoms are readily apparent to visual inspection by trained and experienced personnel. Given the skill of the inspectors in detecting symptoms, the false-negative rate is small, and we take it to be zero. The false-positive rate is also small: PCR analysis of clippings taken from a random subsample of trees deemed to be symptomatic confirmed detection in 98% of cases.

These data therefore describe the presence of HLB symptoms but they do not account for cryptic or latent infection. Specifically, the dataset comprises symptomatic times and removal times, with times of transition to exposed and infectious classes unobserved. Data on the locations of host trees not exhibiting symptoms of HLB were inferred from a secondary dataset of block-by-block information and from satellite images. The secondary dataset gives tree count and plant date for each block, and the satellite images show where trees were and were not planted. Combined with the geometrical regularity of the orchard, this allows us to reconstruct reliably the locations of trees never seen to be symptomatic.

During the observational period, in addition to the removal of symptomatic trees, a spray program was initiated to control psyllid numbers in both the region under observation and the surrounding areas. The spray program was sufficiently intensive that most psyllids were eradicated by the end of 2006. Given the available data on the spraying schedule and psyllid trap counts, we estimate that the density of psyllids decreased linearly to zero during the course of 2006. Accounting for the control program is a crucial component in our modeling of the epidemic.

Modeling Approach. We model the spread of HLB on orchard scales as a spatially explicit, stochastic SEIDR epidemic in a fixed population of trees, where 5 denotes susceptible, E denotes exposed, I denotes infectious but not yet detectable, D denotes detectable or symptomatic, and R denotes removed. This compartmental model therefore gives a phenomenological description of the host-vector-pathogen system. In what follows, $t^*$ is used to denote the time at which tree $i$ enters class X. The key components of the model are the process of the exposure to HLB and the distribution of the latent period of the disease in the trees. These two components are the main innovations of our work. A primary, external source of infection is required to seed the epidemic, whereas secondary infection is mediated by a distance-dependent dispersal kernel. We consider both exponential and power law forms for the kernel. In addition, the rates of primary and secondary infection are allowed to be time dependent to account for control of the vector. The latent period is known to depend on the age of the tree at exposure. Furthermore, exploratory analysis of the data suggested...
a seasonal component, and we consider a variety of models to account for this. We also compare these with models without seasonality. Two assumptions of our model are that the primary infection is homogeneous in space, and the dispersal kernel is isotropic. Although these assumptions are usually considered reasonable on orchard scales, there is evidence of edge effects in the Southern Gardens dataset (Discussion).

**Initial Time.** Let \( t_0 \) denote the time the first infected psyllids arrive in a particular subregion. In other words, \( t_0 \) marks the onset of infectious challenge or, equivalently, the start time of the epidemic. In the absence of psyllid trap data from the earliest times, \( t_0 \) becomes a model parameter to be estimated.

**Modeling Exposure to Infection.** For a vectorial disease, to say a tree is infectious is to say it is transmitting inoculum to the vector. The rate at which inoculum is transmitted is proportional to the density of noninfected vectors in the vicinity of the infectious tree. On the other hand, the rate at which a tree becomes exposed is proportional to the density of infected vectors in its vicinity. In the absence of specific data on the spatial and temporal dependence of the densities of infected and noninfected psyllids—only a time sequence of spatially averaged total psyllid counts is available—it is necessary to make a number of approximations to model exposure.

A summary of the full analysis carried out in SI Text is as follows: we account for psyllid control in Southern Gardens by estimating the relative psyllid density as a piecewise linear function

\[
\rho(t) = \begin{cases} 1, & t \leq t_0 - 1 \\ 2 - t, & 1 < t \leq 2 \\ 0, & t > 2 \end{cases}
\]

where \( t \) is measured in years and \( t = 0 \) corresponds to the start of 2005. Then the instantaneous rate of infection at time \( t \) for susceptible tree \( i \) is written as

\[
\Phi_i(t) = \rho(t) \int \Phi(t', t < t' < t) \, dt',
\]

where \( 1 \) is the indicator function that returns 1 when its argument is true and 0 otherwise. The function \( \rho(\cdot) \) represents an isotropic dispersal kernel—isotropic because \( R_0 \) is the Euclidean distance between tree \( i \) and tree \( j \)—and we consider decaying exponential models, \( k(u) = \exp(-\alpha u) \), and power law models expressed in the form \( k(u) = (1 + u^2)^{-1/a} \). The parameters to be estimated are the primary or external rate of infection \( \beta_1 \), the secondary rate of infection \( \beta_2 \), and the dispersal length scale \( a \).

**Modeling the Latent Period.** It is useful to think of the latent period as a time to failure. Modeling the latent period then amounts to specifying a hazard rate \( h(t) \), which gives rise to the probability density function for the infection time given the exposed time: \( f(t|t') = h(t) e^{-\int_{t'}^t h(u) \, du} \). We suppose a tree moves from the exposed class to the infectious class independently of other trees but that the transition rate depends on intrinsic properties of the tree and has a seasonal component. We use an age-dependent hazard rate with yearly oscillations

\[
h(t, a) = 2A(a) \sin^2 \pi at,
\]

where \( A(a) \) is the amplitude fixed by a combination of in-orchard and in-orchard nursery observations, and \( a \) is the age of the tree when it became exposed. We call this the cyclic model. The cyclic model can be easily made much more flexible without sacrificing analytic tractability: both the period and phase of the oscillation can be changed and an overall constant positive can be added to the hazard rate. (This last change might be appropriate in regions of less marked seasonality, for example.) In what follows, we also consider the effect of 6-mo oscillations.

We also compare the cyclic model with three alternative models for the latent period: (i) the exponential model is the default model in many epidemiological applications: a constant hazard rate \( h(t, a) = A_0(a) \), leading to an exponentially-distributed latent period; (ii) the gamma model posits a gamma-distributed latent period, \( t^2 - t'^2 \sim \gamma \text{amma} \left( \alpha, \beta \right) \), corresponding to a hazard rate that starts at zero, grows, and then tapers to \( \alpha \); it has no cyclic behavior; and (iii) the cyclic Weibull model has a hazard rate \( h(t, a) = 2A_0(a)(t - t_0)^{\alpha - 1} \sin^2 \pi at \); this incorporates seasonality but with an amplitude that starts at zero and increases indefinitely.

The motivation for the gamma and cyclic Weibull models is to capture in-orchard observations of a hazard rate that is initially small and then increases. The parameter values for the cyclic model and the three alternative models in three age categories are given in Table 1.

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>A (y^{-1})</th>
<th>A_0 (y^{-1})</th>
<th>\beta (y^{-1})</th>
<th>\alpha (y^{-1})</th>
<th>A_0 (y^{-1})</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 &lt; a ≤ 3</td>
<td>2.4 0.47 0.29</td>
<td>2.4 0.27 0.19</td>
<td>12 0.64 0.56</td>
<td>0.37 0.32 0.23</td>
<td>0.15 0.21 0.16</td>
</tr>
<tr>
<td>3 &lt; a ≤ 10</td>
<td>1.1 0.67 0.60</td>
<td>0.70 0.65 0.62</td>
<td>0.65 0.70 0.61</td>
<td>0.60 0.59 0.41</td>
<td>0.38 0.44 0.33</td>
</tr>
<tr>
<td>a &gt; 10</td>
<td>0.8 0.67 0.64</td>
<td>0.70 0.65 0.62</td>
<td>0.69 0.70 0.64</td>
<td>0.42 0.53 0.38</td>
<td>0.18 0.24 0.12</td>
</tr>
</tbody>
</table>

Note that in the Southern Gardens dataset, there were no trees in the age range of 0–3 y.

**Modeling the Cryptic Period.** The times for the cryptic period, which measures the time from initiation of infectiousness to the detection of visible symptoms, are taken to be gamma distributed, \( t^2 - t'^2 \sim \gamma \text{amma} \left( \lambda, \nu \right) \), with parameters fixed by in-orchard observations. Specifically, we use \( \lambda = 100 \) and \( \nu = 500 \, y^{-1} \). Note that when combined with the cyclic model for the latent period transition, this suggests a peak in detections in the autumn months.

We do not attempt to estimate the latent period and cryptic period parameters. It has previously been noted (19) that the issue of identifiability arises in epidemiological models with unobserved compartments. This problem is further aggravated by the use of uninformative priors.

**Parameter Estimation.** We adopt a Bayesian approach to parameter estimation. We use data augmentation (15–17) and reversible-jump (20) MCMC techniques, with uninformative exponential priors, to obtain, after a burn-in period, a joint posterior density for the parameters \( \alpha, \beta \), and \( t_0 \). The technical details are provided in SI Text.

**Estimation in Subregions.** Model parameters were estimated in 16 subregions of the Southern Gardens dataset. Carrying out separate analyses of the data over disjoint subregions enables investigation of the spatial dependence of the parameters—notably, the initial time \( t_0 \) and ensures estimation via MCMC remains computationally feasible. The choice of subregions was motivated by the need to balance the competing requirements that the number of symptomatic trees in a subregion was sufficiently large to obtain reliable parameter estimates and that the subregion was small to afford reasonable spatial resolution of the epidemic in Southern Gardens. Ideally, subregions would comprise either young or old trees, but unavoidably some subregions are of mixed age. The mixed-age subregions, however, do provide a check of robustness of the model: typically, their inferred parameters interpolate between those for young and old subregions. The subregions are shown in Fig. 1. The smaller subregions consist of 7,000–8,000 trees, the larger subregions consist of 25,000–30,000 trees.

**Temporal Pattern of Invasion.** The posterior estimates for \( t_0 \) by subregion are summarized in Fig. 2. Although it was not possible to estimate \( t_0 \) in the southeast corner, there is a clear east-to-west spread of the disease. Note
that the old blocks tend to have earlier values of the start time even though HLB is first clearly evident in young blocks, because of the shorter latent period for young trees. Furthermore, $t_0$ is not correlated with the first detection time in each subregion.

**Age Dependence of the Dispersal and Transmission Parameters.** The dependence of the dispersal and transmission parameters on age can only be interpreted in an averaged sense. Consider the impact of infectious tree $i_j$ on susceptible tree $j$: age dependence means the rate of secondary transmission, $\beta$, for example, depends on the ages of both trees and varies during the interval $[t_0^j, t_0^j + \beta]$, where of course both $t_0^j$ and $t_0^j + \beta$ are unknown. As a consequence, the imputed value of $\beta$ will be the average over all pairwise interactions and over time. In addition, there is some uncertainty in the age in which trees were planted out in the orchard. Thus, there is an intrinsic uncertainty in specifying a quantity like $\beta(a)$, and the same argument applies to $\alpha(a)$ and $\epsilon(a)$. Nevertheless, a reasonable proxy for age is the average age of trees in the subregion of interest at the start of the epidemic. Note that the uncertainty in the inferred value of $t_0$ is small in comparison with the intrinsic uncertainty. In the boxplots below, the subregions are color-coded by age: young (green), old (blue), and mixed (red).

The length scale of the dispersal kernel, shown in Fig. 3, grows approximately linearly with the age of the trees in the subregion. More precisely, a linear model for the mean posterior length scale, $\pi_k$, in terms of the average age of the $k$th subregion, $\bar{a}_k$, is

$$\pi_k = c + m \bar{a}_k + e_k,$$

where $c$ and $m$ are, respectively, the intercept and slope terms, and $e_k \sim \mathcal{N}(0, \sigma^2)$ is a Normal error term capturing between-subregion variability. Standard analysis shows 67% of the variation in the mean length scale is explained by the linear trend, and the distribution of residuals is consistent with the modeled error with $\sigma = 1.5$ m. One explanation as to why this linear relationship should hold is that psyllids use the new growth and open structure of young trees to the mature and tight canopy structure of old trees. Consequently, psyllids travel further in old-aged subregions.

The age dependence of the secondary and primary rates of infection is shown in SI Text. Both infection rates tend to decrease with age, and there appears to be rather sharp behavioral difference between subregions less than 8 y old and those more than 8 y old. We attribute this to the fact that 8 y is the approximate age at which tree canopies begin to touch and intermingle along rows. Before this age, host canopies are isolated. The conclusion is that tight canopies are a barrier to infection, in the sense that infection rates are reduced. This reduction is partial, however, by the fact that the dispersal kernel length scale is increased. Note that canopies are not allowed to intermingle across rows; this implies that the actual dispersal kernel in old-aged subregions will be anisotropic.

**Linear Relationship Between Primary and Secondary Infectious Rates.** A log-log plot of the infection rates is shown in Fig. 4. The clustering about the 45° line shown in red suggests that the ratio of primary and secondary infection rates is roughly constant across subregions. To make this statement more rigorous, we first note that the marginal posterior distributions for log $\alpha$ and log $\beta$ are well approximated by a Normal distribution in each subregion. We are then justified in fitting a linear model to the infection rates across subregions

$$\log(e/\beta) = c + m \log \beta_k + e_k,$$

where $e_k \sim \mathcal{N}(0, \sigma^2)$, Standard analysis shows 77% of the observed variation is explained by the linear trend, and the distribution of residuals is consistent with the modeled error with $\sigma = 0.44$. The 95% CI for the slope $m$ is $[0.52, 1.18]$. The fact this interval contains 1 supports the contention that the ratio $\epsilon/\beta$ is invariant over subregions. We show in SI Text that the observed invariance can be understood as a consequence of certain biological and physical assumptions concerning the exposure process. Note that subregions with both young and old trees have been omitted because psyllid density is likely to be uneven in these cases.

**Model Checking and Model Comparison.** To carry out model checking and model comparison, we focus on subregion 13a. This subregion was chosen because the course of the epidemic was the most consistent with the assumptions of homogeneity of primary infection and isotropy of the dispersal kernel. The subregion comprises five blocks and 7,700 trees.

We compare eight different models, a model being specified by a choice of dispersal kernel and latent period model: (a) exponential kernel plus cyclic model with yearly oscillations; (b) exponential kernel plus exponential latent period; (c) Cauchy kernel ($r^{-4}$ power law kernel) plus yearly cyclic model; (d) exponential kernel plus gamma model; (e) $r^{-4}$ power law kernel plus yearly cyclic model; (f) exponential kernel plus cyclic Weibull model; (g) $r^{-4}$ power law kernel plus yearly cyclic model; and (h) exponential kernel plus two-yearly cyclic model.

We fit each model using reversible jump MCMC with data augmentation. From the resulting joint posterior for $\Theta$, we randomly drew 100 parameter sets and simulated epidemics using the Selke algorithm. We then compared the temporal and spatial structure of simulated outcomes with those of the actual outcome. Specifically, we considered the counts of symptomatic trees in each of the four sweeps and the two-point spatial correlation of all symptomatic trees observed up to and including the final sweep.

The counts of symptomatic trees are shown in SI Text. Only in models a and c are all actual counts well within the distribution of simulated counts. Model d, the only model that favors a long latent period, is completely ruled out.

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Fig. 3. Length scale of the dispersal kernel, $\pi_1$, by average age of subregion at estimated epidemic start time. The line in black is the linear model fitted to the mean length scale in terms of the mean age.

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The two-point correlation function we use is a modified Moran’s I statistic for presence-absence data and is detailed in *SI Text*. Each model gives correlation functions that are consistent with the vanishing of the observed correlation function for $r > 80$ m. Conversely, only model a accommodates the observed correlation function for $r > 80$ m. Models c, d, and e fit reasonably well except for a lack of power in the range of 30–40 m. Once again, model d appears to be completely ruled out.

Model c gives widely varying outcomes in terms of counts of symptomatic trees and spatial correlation. The reason for this is that the favored dispersal length functions that are consistent with the vanishing of the observed alone is responsible for disease transmission. We consider this model to be highly implausible from a biological viewpoint.

**Discussion**

When faced with a fast-moving and destructive emerging epidemic, there is often a conflict between the need to observe the epidemic to estimate the key epidemiological parameters and the need to deploy control measures to contain the epidemic (21). We used the spread of HLB on orchard scales as a case study to test the feasibility of estimating parameters for models of an emerging epidemic that is subject to perturbation by control. Using just four censored successive snapshots of disease, we demonstrated that the disease transmission process can be characterized despite contemporaneous control measures. The statistical analyses have yielded previously unidentified aspects of the epidemiology of HLB. Our analyses were able to establish the temporal pattern of invasion, including the likely sequence of invasion through uneven aged groves of citrus. The analyses successfully demonstrated how dispersal and transmission parameters for HLB varied with host age and supported seasonal changes in host susceptibility. We also confirmed a linear relationship between primary infection of citrus by HLB, driven by inoculum arriving from outside the study area, and secondary infection from tree to tree within the groves.

Our analyses demonstrated the power of computational methods to extract these insights about disease spread through a spatially and temporally heterogeneous environment despite the high degree of censoring present in the observations. Given the severity of the challenge to extract meaningful and consistent results, we commend the use of these statistical methods to aid understanding of other pathosystems of similar complexity and scale. Although this paper is focused on demonstrating the robustness of the methodology for estimation, the power of the parameterized epidemiological models to predict the likely future of epidemics is shown in *SI Text*. The parametrized models may also be used to compare the effectiveness of alternative control strategies. The use of the model to predict future outcomes of disease spread, as well as the effectiveness of control, together with the consistency of results, for example, with respect to the effects of host age, all provide means to check the longer-term value of the deductions from the model.

Two of the principal innovations within the general strategy for model fitting in our work are modeling time dependence in a vectorized disease due to control of the vector, and incorporating seasonal and host age effects during the latent period. Recent work by Chiyaka et al. (22) has also incorporated seasonal forcing into a model for HLB spread within trees. Chiyaka et al. also modeled the dynamics of vector infection on a single tree. Here we treated the tree as the unit of interest within large populations of 7,000–30,000 trees, for which we inferred transmission and important dispersal parameters from censored maps of disease spread. There is scope in future research to assess the potential to use within-tree dynamics to assess the dynamics of the force of infection on individual trees for tree-to-tree spread.

The models used in the current analysis of HLB assume homogeneous distribution of primary infections. This assumption could easily be dropped to allow for a directional or spatially decaying source of external infection, when there is evidence for such effects. The models also assume isotropic secondary infection. By assuming isotropic spread, our estimates have utility by setting an upper bound for the worst case scenario for unimpeded spread of HLB. Future research will address anisotropic secondary infection to test for differences in transmission rates along and between rows and for edge effects, where vectors congregate, for example, along trees at the edge of a grove.

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Supporting Information

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SI Text

Data. The spread of Huanglongbing (HLB) during the four sweeps is shown in Fig. S1. The epidemic first becomes apparent in young trees in the east and then spreads both westward and to older trees. Removal of symptomatic trees usually occurred 1–3 mo after detection.

Modeling Exposure to Infection. Because the regions of interest are of suborchard scale and the timescale of the observations allows the vector to move a considerable distance, for the purposes of the model, we assume that the total psyllid density is roughly constant in space. Letting \( \tilde{\rho} \) denote the total psyllid density, \( \kappa \) denote, the fraction that are infected, and \( \Lambda \) denote the intrinsic infection rate per psyllid, the rate at which a susceptible tree becomes exposed will be \( \Lambda \tilde{\rho} \). It is convenient for modeling purposes, however, to distinguish between vectors coming from outside and inside the observed region. Let \( \rho_0 \) denote the density of external psyllids (assumed constant over the region of interest) and \( \rho_i \) denote the density of psyllids at tree \( i \) arriving from tree \( j \). The density fraction \( \tilde{\rho}_i \) can be further modeled via a dispersal kernel, namely, \( \tilde{\rho}_i = R K(x_i, x_j) \), where \( R \) is a scaling constant and \( x \) denotes the position of tree \( i \), etc. The dispersal kernels we consider are isotropic, \( K(x_i, x_j) = k(r_{ij}/\alpha) \), where \( r_{ij} \) is the Euclidean distance between tree \( i \) and tree \( j \) and \( \alpha \) is a length scale to be determined.

To account for psyllid control in Southern Gardens, this was carried out in the region under observation and surrounding areas, it is necessary to let \( \tilde{\rho} = \rho_t \). We assume that the same function of time controls the time dependence of both \( \rho_t \) and \( \tilde{\rho}_i \), namely \( \rho_t = \rho_0(t)/\rho_{i0} = \tilde{\rho}_i(t)/\tilde{\rho}_{i0} \), and call it the relative psyllid density. We estimate the relative psyllid density as a piecewise linear function

\[
\rho(t) = \begin{cases} 
1, & t \leq 1 \\
2 - t, & 1 < t \leq 2 \\
0, & t > 2
\end{cases} \tag{S1}
\]

where \( t \) is measured in years and \( t = 0 \) corresponds to the start of 2005. Note that \( t_0 < 1 \) is implied.

Defining \( \epsilon = \Lambda \kappa \rho_0(t_0) \) and \( \beta = \Lambda \kappa R \), the instantaneous rate of infection at time \( t \) for susceptible tree \( i \) can be written as

\[
\Phi_i(t) = \rho(t) \left[ e + \beta \sum_j k \left( \frac{r_{ij}}{\alpha} \right) I \left( t' < t \leq t'' \right) \right], \tag{S2}
\]

where \( I \) is the indicator function that returns 1 when its argument is true and 0 otherwise. We identify \( e \) as the primary or external rate of infection and \( \beta \) as the secondary rate of infection; both are model parameters to be estimated.

Although the form of Eq. S2 is standard, the physical and biological reasoning underpinning the model of the exposure process leads to the expressions for \( e \) and \( \beta \) given above. As a direct consequence, the ratio of primary to secondary transmission rates is the same constant for all spatial regions of interest

\[
\frac{e}{\beta} = \frac{\rho_0(t_0)}{R} = \text{constant}. \tag{S3}
\]

Note that had we introduced the normalized secondary rate of infection per tree, \( \beta^* = \beta^* Z(\alpha) \), where \( Z(\alpha) = (1/N) \sum_j k(r_{ij}/\alpha) \) and \( N \) is the number of trees, this result would not have been apparent: the ratio \( e/\beta^* \) depends on \( \alpha \) and the details of the observed region.

Latent Period Parameters. The parameters of the cyclic, exponential, and cyclic Weibull models are estimated from in-orchard and in-nursery observations. Expert opinion, based on in-orchard observations, for the upper bound on the combined latent and cryptic periods was interpreted as the time for 95% of trees to move from exposed to symptomatic. (Our results do not depend sensitively on the assumption of 95%.) In-orchard observations of the cryptic period then allow us to estimate the \( T_{0.95} \), the time for 95% of trees to move from exposed to infectious. By itself, \( T_{0.95} \) is not sufficient to fix the parameters of the cyclic, exponential, and cyclic Weibull models, because these models describe a latent period that depends on \( \beta^* \). Fortunately, the dependence on \( \beta^* \) becomes small for latent periods longer than the seasonal period. The in-orchard data are given in Table S1.

On other hand, the gamma model is a two-parameter model. Its parameters are fixed by \( T_{0.95} \) and by expert opinion that the lower bound on the combined latent and cryptic periods is at least 6 mo and usually more than 1 y. The lower bound was interpreted as the time for 5% of trees to move from exposed to symptomatic. Recently, however, it has been realized that the latent period can be as little as 1 mo. This shortness of the latent period is in line with the conclusions of our study, being entirely consistent with the cyclic, exponential, and cyclic Weibull models.

It is also important to point out that seasonal effects were introduced to account for the increase in symptomatic counts during the second and third sweeps and the reduced counts in the fourth sweep. Having seasonality in the latent period was forced on us by a combination of the assumption of a compartmental model for the epidemic, the early appearance of symptomatic trees in Southern Gardens, the introduction of efficient psyllid control measures, and the relatively long incubation period for HLB.

Cryptic Period Parameters. The parameters are fixed by in-nursery observations showing that 5% (95%) of trees become symptomatic within 2 (3) mo of becoming infectious.

Note that both the latent period and cryptic period parameters could also have been estimated as part of the Bayesian inference. Because the in-orchard and in-nursery observations lead to rather strong priors on these parameters, it was the felt the extra computational complexity would have yielded limited additional information.

Parameter Estimation. We adopt a Bayesian approach to parameter estimation. We use Markov chain Monte Carlo (MCMC) techniques, with uninformative exponential priors, to obtain, after a burn-in period, a joint posterior density for the parameters, conveniently represented as a vector \((\alpha, \beta, e, t_0)\) in the parameter space \( \Theta_0 \).

Suppose there are initially \( N \) susceptible trees and that \( T \) is the final observation time. Let \( S \) be the index set of trees never exposed, \( E \) be those exposed but never infectious, \( I \) be those infectious but never symptomatic, and \( D \) be those that were symptomatic, up to time \( T \). It follows that \( S, E, I, \) and \( D \) are disjoint sets and that \( S \cup E \cup I \cup D = \{1, \ldots, N\} \). Then, taking the removal times as known, the theoretical joint probability density for the epidemic times is
\[ p((\ell', \ell') | \alpha, \beta, \epsilon, t_0) = \prod_{i \in \ell} e^{-\Phi(T_i)} \prod_{i \in \ell} \Phi(t_i) e^{-\Phi(x_i)} \Phi(T_i) \prod_{i \in \ell} \Phi(x_i) = \prod_{i \in \ell} \Phi(t_i) e^{-\Phi(x_i)} \Phi(T_i) \prod_{i \in \ell} \Phi(x_i) \]

where \( f(\ell'|\ell) \) is the probability density function for the latent period, \( F(T|\ell) := \int_T^\infty ds f(\ell'|\ell) \) is its (right) tail distribution, \( g(\ell'|\ell) \) is the probability density function for the cryptic period, and \( G(T|\ell) := \int_T^\infty ds g(\ell'|\ell) \) is its tail distribution.

In practice, however, the times \( t_i \) and \( t' \) are unobserved, and we observe only that \( t_i \in \Delta \), where \( \Delta \) is some time interval. Indeed, we cannot even specify the index sets \( S, \mathcal{E} \), or \( T \). Ordinarily, we would integrate the joint density with respect to the latent variables and over the censoring intervals to obtain the probability \( \mathbb{P}(t_i \in \Delta) | \alpha, \beta, \epsilon, t_0) \), but such an approach is not tractable here. The reason is that \( \Phi(t) \) depends on all infectious times \( t < t_i \), making it impossible to find a closed form expression for the integral of Eq. S4 over the unobserved times. To solve this problem, we use data augmentation and reversible-jump MCMC methods.

**Reversible-Jump MCMC with Data Augmentation.** Let \( \delta \) denote a partition of the set of trees that were never symptomatic into sets of trees that were not exposed, exposed but not infectious, and infectious: \( S^0 \cup E^0 \cup I^0 = \mathcal{A} \). MCMC with data augmentation (1–3) extends the parameter space from \( \Theta_0 \) to \( \Theta^\delta \), where \( \Theta^\delta \) contains \( \Theta_0 \) and all times consistent with the data and the partition \( \delta \). Given a partition \( \delta \), MCMC will generate an empirical joint posterior density on \( \Theta^\delta \). The marginal density on \( \Theta_0 \) is then the desired posterior.

The reversible-jump technique (4) enables MCMC to explore \( \Theta^\delta \) for different \( \delta \), a requirement that is clearly necessary because the actual partition is not known. Reversible-jump MCMC generates an empirical joint posterior density on the parameter space \( \Theta = \bigcup \{ \delta \} \times \Theta^\delta \). Because \( \Theta_0 \subset \Theta^\delta \) for all \( \delta \), we marginalize over all times and partitions to obtain the posterior for the parameter vector.

**Age Dependence in the Rates of Infection.** The age dependence in the rates of infection is shown in Fig. S2.

**Model Checking and Model Comparison.** For subregion 13a, we compare eight different models, a model being specified by a choice of dispersal kernel and latent period model: (i) exponential kernel plus cyclic model with yearly oscillations; (ii) exponential kernel plus exponential latent period; (iii) Cauchy kernel \( (r^{-2} \text{ power law kernel}) \) plus yearly cyclic model; (iv) exponential kernel plus gamma model; (v) \( r^{-2} \text{ power law kernel} \) plus cyclic model; (vi) exponential kernel plus cyclic Weibull model; (vii) \( r^{-8} \text{ power law kernel} \) plus cyclic model; and (viii) exponential kernel plus twice-yearly cyclic model.

The labeling has been chosen so that in Figs. S4 and S5, the first column shows the results of different dispersal kernels and the second column shows the results of different latent period models. We fit each model using reversible-jump MCMC with data augmentation. From the resulting joint posterior for \( \Theta_0 \), we randomly drew 100 parameter sets and simulated epidemics using the Selke algorithm. We then compared the temporal and spatial structure of simulated outbreaks with those of the actual outcome. Specifically, we considered the counts of symptomatic trees in each of the four sweeps, and the two-point spatial correlation of all symptomatic trees observed up to and including the final sweep.

**Dispersal kernel length scales.** The estimated length scale \( \alpha \) varies with choice of dispersal kernel and latent period model. Table S2 gives the explicit form of the kernel \( k(r/\alpha) \) in each of the eight models above and the posterior mean and 95% credible region for \( \alpha \) measured in units of meters. Except for model c, the marginal posterior densities for \( \alpha \) were roughly bell-shaped. For the Cauchy kernel in model c, the marginal posterior peaks at \( \alpha = 0 \). The interpretation when \( \alpha \) is vanishingly small is that external sources of infection are entirely responsible for driving the epidemic.

**Counts of symptomatic trees.** The counts of symptomatic trees are indicated in Fig. S3. The red line is the actual observed count; the frequency histogram of counts from the 100 simulations is in blue. Only in models a and c are all actual counts well within the distribution of simulated counts. Model d, the only model that favors a long latent period, is completely ruled out.

**Two-point correlation function.** The two-point correlation function we use is a modified Moran’s I statistic for presence-absence data. Letting \( s_i = 1 \) if tree \( i \) is found to be symptomatic before or at the final observation time and \( s_i = 0 \) otherwise, we define

\[ \xi_2(r_1, r_2) = \frac{\sum_{i,j} w_{ij}(r_1, r_2) s_i s_j - \bar{s}^2}{3 \bar{s} - \sum_{i,j} w_{ij}(r_1, r_2)}, \]

where \( \bar{s} = (1/N) \sum_{i=1}^N s_i \) and \( w_{ij}(r_1, r_2) = 1 (r_1 \leq |x_i - x_j| < r_2) \) is a ring weighting. Note that in the limit \( r \to 0 \), we have \( \xi_2(0, r) = 1 \).

The two-point correlation functions are shown in Fig. S4. We chose a sequence of ring radii, \( r_1, r_2, \ldots \), and plotted \( \xi(r) \). The red line is the actual observed two-point correlation; the two-point correlations from the 100 simulations for each model are in green. Each model gives correlation functions that are consistent with the vanishing of the observed correlation function for \( r > 80 \text{ m} \). Conversely, only model a accommodates the observed correlation function for \( r > 80 \text{ m} \). Models c and g fit reasonably well except in the range of 30–40 m. Once again, model d appears to be completely ruled out.

**Model predictions.** In Fig. S5, the cumulative counts of symptomatic trees are shown for 100 simulations of each model that have been run more than 3 y past the final observation time. Note that simulations are run from the randomly drawn initial times \( t_0 \in \Theta_0 \) and incorporate the removal of symptomatic trees. Although the simulations are not constrained by the actual detections at the four observational sweeps, they are in good agreement with the data, as evidenced by the red lines that meet at the total cumulative detection count at the final observation time. Alternatively, we could have sampled from the full posterior for \( \Theta \), which, for each sample, would have allowed us to infer a complete epidemiological state of subregion 13a at the final observation time. We could then have run each simulation forward from this time.

**Model limitations.** Our model for HLB spread is not uniformly applicable. In subregions with very few symptomatic trees, a very large length scale \( \alpha \) can be favored. This outcome reflects an underlying identifiability problem because in this case the dispersal kernel is essentially constant and mimics an external infection. Such a situation occurred in the large subregion in the southeast corner of Southern Gardens. Additionally, subregions with trees of differing ages can be problematic because the epidemic will proceed very differently in different parts of the subregion. Because the parameters are phenomenological quantities combining effects due to host and vector, dispersal parameters can only be understood in an averaged sense.
Fig. S1. (A) Sweep 1: November 2005 to March 2006, 1,748 detections, shown in blue. (B) Sweep 2: September 2006 to November 2006, 12,962 detections, shown in green. (C) Sweep 3: January 2007 to April 2007, 10,591 detections, shown in orange. (D) Sweep 4: May 2007 to July 2007, 2,626 detections, shown in red.
Fig. S2. Boxplots of the transmission parameter posteriors for 16 subregions; the subregions are color-coded by age: young (green), old (blue), and mixed (red). (A) Secondary rate of infection, $\beta$, by average age of subregion at estimated epidemic start time. (B) Primary rate of infection, $\epsilon$, by average age of subregion at estimated epidemic start time.
Fig. S3. Comparing models by observed detections in each of the four successive sweeps. The red line is the actual observed count; the frequency histogram (scaled to have unit area) of counts from 100 simulations is in blue. The vertical and horizontal scales are the same for each sweep.
Fig. S4. Comparing models by the two-point spatial correlation of all detections. The red line is the actual observed two-point correlation; the two-point correlations from 100 simulations are in green. Note that the actual correlation function goes to zero at about 80 m.
Fig. S5. Comparing models by predicted future detection counts. The red lines meet at the actual observed count at the final observation time; the counts from 100 simulations are in blue. White stripes in the plots are a result of the latent period models with cyclic hazard rates.

(a) Exponential kernel and yearly cyclic latent period

(b) Exponential kernel and exponential latent period

(c) Cauchy kernel and yearly cyclic latent period

(d) Exponential kernel plus gamma latent period

(e) $r^{-4}$ power law kernel and yearly cyclic latent period

(f) Exponential kernel and cyclic Weibull latent period

(g) $r^{-8}$ power law kernel and yearly cyclic latent period

(h) Exponential kernel and twice-yearly cyclic latent period

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Table S1. Expert opinion of upper bound on combined latent and cryptic periods for trees of different ages

<table>
<thead>
<tr>
<th>Age (y)</th>
<th>Upper combined latent and cryptic period (y)</th>
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</thead>
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<tr>
<td>$1 &lt; a \leq 3$</td>
<td>1.5</td>
</tr>
<tr>
<td>$3 &lt; a \leq 10$</td>
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</tr>
<tr>
<td>$a &gt; 10$</td>
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Table S2. Posterior mean and 95% credible region for $\alpha$ measured in units of meters

<table>
<thead>
<tr>
<th>Model</th>
<th>Dispersal kernel $k(u)$</th>
<th>Length scale $\alpha$ (m)</th>
</tr>
</thead>
<tbody>
<tr>
<td>a</td>
<td>$\exp(-u)$</td>
<td>$6.9_{-3.3}^{+7.7}$</td>
</tr>
<tr>
<td>b</td>
<td>$\exp(-u)$</td>
<td>$6.0_{-4.4}^{+8.2}$</td>
</tr>
<tr>
<td>c</td>
<td>$(1 + u^2)^{-1}$</td>
<td>$0.6_{-0.2}^{+0.7}$</td>
</tr>
<tr>
<td>d</td>
<td>$\exp(-u)$</td>
<td>$4.0_{-2.8}^{+7.7}$</td>
</tr>
<tr>
<td>e</td>
<td>$(1 + u^4)^{-1}$</td>
<td>$7.2_{-5.0}^{+11.1}$</td>
</tr>
<tr>
<td>f</td>
<td>$\exp(-u)$</td>
<td>$6.9_{-2.1}^{+8.3}$</td>
</tr>
<tr>
<td>g</td>
<td>$(1 + u^8)^{-1}$</td>
<td>$16.4_{-10.5}^{+22.1}$</td>
</tr>
<tr>
<td>h</td>
<td>$\exp(-u)$</td>
<td>$6.0_{-4.4}^{+8.2}$</td>
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</tbody>
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