A Model for the Temporal Buildup of *Polymyxa betae*

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Polymyxa betae Keskin is an obligate root parasite of sugar beet that typifies an important class of plant parasites that have multiple infection cycles driven by host and parasite dynamics. The fungus occurs almost everywhere sugar beet is grown (25) and has recently been the focus of much research because it is the vector of beet necrotic yellow vein virus (BNYVV), the causal agent of rhizomania in sugar beet (14). Rhizomania may cause losses in sugar yield of up to 70% (26), rendering the growing of sugar beet uneconomic. Strategic planning for the control of rhizomania requires information on the dynamics of the vector, since BNYVV cannot survive in the absence of the fungus. Although much experimental work has been done on *P. betae* (1,5,7,21,30), little is known about the quantitative interactions of the individual components involving root growth, colonization, and the production of inoculum on the dynamics of infection. The life cycle of the fungus is comparatively complicated, and field experimentation with the fungus is both difficult and expensive because of the subterranean habit and obligate parasitism of the fungus and the absence of visible lesions, necessitating microscopic inspection of roots for quantification of population growth.

In this paper, we develop and analyze a model for the buildup of *P. betae* that integrates the dynamics of root growth, infection, and inoculum within seasons. The model is designed to form a coherent, quantitative, and rigorous summary of the dynamics of *P. betae*. The model serves to identify key parameters in the population dynamics of *P. betae* upon which future experimental programs can be efficiently designed and tested. We propose that the model can be used to analyze the dynamics of the fungus and to refine hypotheses prior to experimentation, thus enabling optimal use of experimental resources.

The model incorporates two sources of infection. One source arises from primary resting spores present in the soil at the start of the season. The second source of infection comprises secondary zoospores produced by sporangial plasmodia in infected roots. The model draws on recent work in epidemiology (16–19) using a compartmental structure for root growth with free-living inoculum; but here the compartments comprise sections of fibrous root that pass through different classes of susceptibility and infectiousness, and there are two types of free-living inoculum. The model is used to investigate (i) the relative magnitudes of primary and secondary infection; (ii) the relationship between the initial inoculum density, the dynamics of infection, and the production of new resting spores; and (iii) whether or not a saturation effect exists, whereby increasing the initial inoculum density of resting spores has little effect on the final amount of inoculum. The incorporation of a simple model for host growth also enables us to examine the effect fibrous root growth (in particular, the rate of supply of

susceptible tissue), the latency between infection and production of secondary inoculum, and the rate at which roots become resistant to infection has on the dynamics of infection.

In the first section of the paper, we summarize the essential features of the life cycle of *P. betae* in relation to the population dynamics of the fungus in the sugar beet crop. We then describe the compartments of the model and the model assumptions. The model consists of seven compartments, five of which describe the dynamics of the host in relation to infection by primary and secondary inoculum. Estimates of 7 out of the 12 model parameters are obtained from independently collated experimental data. A sensitivity analysis is used to investigate the response of the model to small changes in the parameter values. The output of the model is compared qualitatively with data on inoculum buildup over seasons. Analytical solution of the model using asymptotic analysis is possible if we assume certain parameters are very small and is discussed further in a more mathematical paper (29).

THE LIFE CYCLE OF *P. BETAE*

The essential features of the life cycle of *P. betae* are summarized below in relation to the population dynamics of the fungus in the sugar beet crop. More detailed accounts of the life cycle are given by Keskin (23), Keskin and Fuchs (24), and Barr (5).

Infection is initiated by primary zoospores that germinate from resting spores resident in the soil. The zoospores infect the epidermal cells of the fibrous roots. Within the infected cell, a sporangial plasmodium develops, which differentiates to produce secondary zoospores. The zoospores are released into the soil where they may infect adjacent roots, giving rise to repeated cycles of secondary infection. At some stage, possibly correlated with the age of infected material (7), there is a switch from the development of sporangial to cystogenous plasmodia, which differentiate to produce new resting spores. These resting spores are released into the soil either when roots die during the season or when the roots are sloughed off at harvest.

Resting spores occur in the form of cystosori, comprising between 4 and 300 cysts 4 to 7 μm in diameter, each of which contain a single (primary) zoospore (4). Germination of the cysts depends on a combination of soil temperature, moisture, and pH (1, 8, 28) and probably on root exudates from the host and an endogenous dormancy period for the fungus (31). Microscopic examination of resting spores indicates that only a small proportion of the cysts within each cystosorus germinate during a single season (M. J. C. Asher, unpublished data). It follows that an individual resting spore may release primary zoospores from different cysts over successive seasons in the presence of susceptible plants until the supply of primary zoospores is exhausted or the cystosorus is otherwise rendered ineffective. The release of primary zoospores in the absence of the crop is likely to be minimal (25), because soil inoculum is not rapidly depleted.

In principle, the density of the fungus in soil can be expressed quantitatively in terms of the number of zoospores and resting

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spores. In practice, however, estimates of fungal density can only be made using the most probable number (MPN) technique based on infective units, in which one infective unit corresponds to 50 to 100 resting spore clusters. Each resting spore cluster has been estimated to contain an average of approximately 37 resting spores (31).

The root system of sugar beet consists of a long tap root that expands during the season and many fibrous lateral roots. Infection by the fungus occurs only on the fibrous roots. Some spread of the zoospores occurs within roots, but this is minimal compared with infection via the epidermal cells (6). Evidence for the effect of the fungus on root growth is not consistent, with some authors reporting retardation of root growth and others reporting no retardation. Tamada et al. (27) found no evidence of an effect on root weight by virus-free *P. betae* when plants were grown for 40 days in a climate room followed by 3 months in a greenhouse. Blunt et al. (8), however, reported that virus-free *P. betae* reduced dry weight of tap roots on young plants at high soil temperatures, while Gerik and Duffus (15) found that three out of six isolates of the fungus reduced lateral root weight, leading them to suggest that isolates of *P. betae* may differ in aggressiveness. In general, it seems that root growth is unlikely to be much affected by fungal infection at the soil temperatures prevailing in the field during the early part of the growing season (8). At the same time, fibrous roots appear to develop physiological resistance to infection with age (9).

Fig. 1. Flow diagram for the dynamics of *Polymyxa betae* in relation to the fibrous root system of sugar beet. Flow of fibrous root through different compartments is indicated by solid lines; primary and secondary infection are indicated by dotted lines.

THE MODEL

Model outline. The model consists of two components: one for the dynamics of the host in relation to infection by the fungus and the second for the dynamics of primary and secondary inoculum (Fig. 1). The principal variables and parameters used in the model are summarized in Tables 1 and 2.

Host dynamics. The host population is described as root length per unit of ground area. This metric was chosen over root density, since it provides an accurate and simple summary of the amount of host available for infection without the additional complexity of modeling the variation in root density through the soil layers. Total root length, N , follows a logistic growth curve. The total root length is divided into five classes that describe the state of the host in relation to fungal infection: susceptible, S ; exposed (infected but not yet infectious), E ; infected with sporangial plasmodium (infectious), I_1 ; infected with resting spores (infected), I_2 ; and resistant, R . Within the season, host cells follow one of three paths: $S \rightarrow R$, $S \rightarrow E \rightarrow I_1 \rightarrow R$, or $S \rightarrow E \rightarrow I_2$ (Fig. 1).

Fungal dynamics. Two classes are used to describe the fungal population; germinable resting spores, P (the source of primary infection), and secondary zoospores, Z . Decay of resting spores occurs only in the presence of susceptible host material.

Model assumptions. The assumptions of the model can be summarized as follows. (i) Root growth is not affected by the presence of the fungus. (ii) Susceptible host material becomes resistant to infection at a rate proportional to the amount of existing susceptible host material. (iii) Infected host material cannot be reinfected. (iv) There is a latent period between infection and release of secondary zoospores. (v) Secondary zoospores decay at a constant rate in the absence of susceptible host material. (vi) Resting spores do not decay in the absence of susceptible host material. (vii) Resting spores created within the season remain dormant until the next season. (viii) There is homogeneous mixing of roots and fungi. (ix) There is no spatial

TABLE 2. Summary of model variables and initial values used in simulations^a

Variable	Definition	Initial value
S	Susceptible root length	0.5 cm U_a^{-1}
E	Exposed root length	0 cm U_a^{-1}
I_1	Length of root infected with sporangial plasmodium	0 cm U_a^{-1}
I_2	Length of root infected with resting spores	0 cm U_a^{-1}
R	Length of root resistant to infection	0 cm U_a^{-1}
N	Total root length	0.5 cm U_a^{-1}
Z	Number of secondary zoospores	0 zoospores U_a^{-1}
P	Number of viable resting spores in the soil	P_i spores U_a^{-1}
P_i	Inoculum density at the start of the season	Varied
P_f	Inoculum density at the end of the season	Not applicable

^a Dimensions are centimeters (cm) and the unit of ground area occupied by a single sugar beet (U_a).

TABLE 1. Model parameters, definitions, and values used in simulations^a

Parameter	Definition	Estimated value
r	Growth rate of the fibrous root system	0.14 cm day ⁻¹
k	Maximum length of the fibrous root system	50,000 cm U_a^{-1}
λ_1	Rate of primary infection	0.02 U_a day ⁻¹
λ_2	Rate of secondary infection	0.002 U_a day ⁻¹
μ^{-1}	Average period of root susceptibility	20 days
β_1^{-1}	Average latent period prior to generation of secondary zoospores	2.5 days
β_2^{-1}	Average latent period prior to generation of new resting spores	10 days
σ^{-1}	Average period over which roots remain infectious	1.25 days
γ	Rate of release of secondary zoospores	80 zoospores cm ⁻¹ day ⁻¹
α^{-1}	Average survival time of secondary zoospores in the absence of infection	1.25 days
η	Rate of loss of secondary zoospores through infection	0.1 U_a cm ⁻¹ day ⁻¹
ϵ	Density-dependent rate of decay of viable resting spores	0.1 U_a cm ⁻¹ day ⁻¹
j	Average number of new resting spores per unit length of infected root	80 spores cm ⁻¹
l	Proportion of initial inoculum that remains viable in a succeeding growing season	0.8

^a Dimensions are days (day), centimeters (cm), and the unit of ground area occupied by a single sugar beet (U_a).

spread of the resting spores within seasons. (x) All environmental variables are constant.

The assumption of homogeneous mixing of roots and fungi can be made since the resting spore population consists not only of those from senescent roots, which are distributed through the soil layers by the extensive root system of sugar beet, but also from roots that are broken off as the storage root is harvested.

Model equations. Using the schematic outline of the model (Fig. 1) and the assumptions above, we constructed a compartmental model for the change in status of roots from susceptible through

infected to resistant, with concomitant dynamics for free-living inoculum (P and Z) as typified by Anderson and May (2) and Gubbins and Gilligan (18). We obtained the following system of equations to describe the dynamics of the host and fungus.

$$\text{Susceptible} \quad \frac{dS}{dt} = rN \left(1 - \frac{N}{k} \right) - \lambda_1 SP - \lambda_2 SZ - \mu S$$

$$\text{Exposed} \quad \frac{dE}{dt} = \lambda_1 SP + \lambda_2 SZ - \beta_1 E - \beta_2 E$$

Fig. 2. Temporal dynamics of **A**, susceptible root length, S ; **B**, exposed root length, E ; **C**, infectious root length, I_1 ; **D**, infected root length, I_2 ; **E**, resistant root length, R ; **F**, total root length, N ; **G**, primary resting spores, P ; and **H**, secondary zoospores, Z ; for initial resting spore densities of $P_i = 10$ spores U_a^{-1} (solid line), $P_i = 50$ spores U_a^{-1} (dotted line), and $P_i = 200$ spores U_a^{-1} (dashed line) using the parameter values given in Table 1 and the initial values from Table 2.

Infectious	$\frac{dI_1}{dt} = \beta_1 E - \sigma I_1$
Infected	$\frac{dI_2}{dt} = \beta_2 E$
Resistant	$\frac{dR}{dt} = \mu S + \sigma I_1$
Secondary zoospores	$\frac{dZ}{dt} = \gamma I_1 - \eta SZ - \alpha Z$
Resting spores	$\frac{dP}{dt} = -\epsilon PS$

in which the total root length is $N = S + E + I_1 + I_2 + R$. Tables 1 and 2 contain definitions of the model parameters and variables, respectively, and t = time in days and d = derivative.

Stability of the model. The model equilibria are not uniquely defined; stationary values depend on the initial conditions. The stationary states of the model lie on the lines $R + I_2 = k$, in which k is the maximum total root length, and $P \leq P(t = 0)$. Linear stability analysis revealed the points on these lines to be neutrally stable. A more detailed analysis of the model, considering the transient behavior of the system, is described by Truscott et al. (29).

PARAMETER ESTIMATES

Approximate values of seven of the model parameters were obtained from published data, the remaining parameter values were arbitrarily assigned within biologically plausible bounds. It is shown in the sensitivity analysis that the default values of the parameters chosen here are sufficient to reveal the qualitative dynamic behavior of the model.

Root growth (k , r , μ). Root lengths are expressed per unit of ground area, U_a , defined as the product of the distance between rows (typically 50 cm) and the distance between plants (15 to 20 cm). Measurements of fibrous root length of the sugar beet crop have been calculated per plant for seedlings (7), per unit of cropped area, and per unit of volume (10), all of which can be converted to root length per unit of ground area (13). The fibrous root system of a typical sugar beet plant grown in the field is about 0.5 km long (12). Experimentally, the root length per unit of ground area has been shown to reach an equilibrium, with growth matching senescence, between 100 and 150 days after sowing. By this time, the rooting density is approximately uniform throughout the cropped area (11). Using the above observations and limited sequential data (7,10), we obtained the carrying capacity, $k \approx 50,000 \text{ cm } U_a^{-1}$, and the growth rate, $r \approx 0.14 \text{ cm day}^{-1}$.

Work on the effect of sowing date on the infection of the crop by *P. betae* showed that the fibrous roots of early sown crops, established when soil temperature was not conducive to infection, showed lower levels of infection through the season. This implies that the roots develop resistance with age (7). A specific period is not known and an arbitrary value for μ^{-1} of 20 days was chosen.

Transmission and decay parameters (λ_1 , λ_2 , β_1 , β_2 , γ , α , ϵ , η , σ). The estimated values of the parameters driving the dynamics of the fungus are based on experiments in which the mean day-time soil temperature lay in the range 20 to 25°C, the optimum temperature range for infection (8).

Using the MPN method on a known quantity of new resting spore clusters results in a recovery rate of only 1 to 2% (31). The process of extracting new resting spores from fibrous roots before reintroducing them to soil may have a detrimental effect on the recovery rate. Some endogenous dormancy period may exist, and it is possible that clustering of resting spores is an artifact of the extraction process. In field conditions, a higher rate of infection might be expected. Here, we assumed that, on average, 50 resting spores are required to obtain a single infection, hence the rate of infection by primary resting spores, $\lambda_1 \approx 0.02 U_a \text{ day}^{-1}$.

Microscopic examination of resting spores recovered from soil suggests that only a small proportion of the cysts in a resting spore germinate in a single season. Assuming that the infection rates of primary and secondary zoospores are the same and that each resting spore releases a mean of 10 primary zoospores per season, we obtained the rate of infection by secondary zoospores, $\lambda_2 \approx 0.002 U_a \text{ day}^{-1}$.

The latent period between infection and production of either zoospores or resting spores is highly dependent on temperature (7). At 25°C, the average latent period prior to production of secondary zoospores, β_1^{-1} , is approximately 2.5 days, and the average latent period prior to the production of resting spores, β_2^{-1} , is approximately 10 days (22).

The number of zoospores released per infected cell is variable, lying in the range of 50 to 200 (estimated from micrographs in Barr [5]). Here, we assume that the number of zoospores released per unit length of infected root includes only the number of viable zoospores and choose $\gamma = 80 \text{ spores cm}^{-1} \text{ day}^{-1}$. Insufficient experimental evidence was available to estimate the parameters α , ϵ , η , and σ , and their values were arbitrarily assigned (Table 1).

Model simulations. The model was solved in Matlab (3) using a variable-order numerical method suitable for stiff equations with marked differences in the rates at which compartments change. The model was used to simulate the dynamics of infection and root growth for 200 days after sowing, using the parameter values and initial conditions given in Tables 1 and 2. The temporal dynamics of each of the model variables were examined for initial inoculum values ranging from 1 to 100 resting spores per unit of ground area. These densities are represen-

Fig. 3. Image-density plots of **A**, susceptible root length, S ; **B**, secondary zoospores, Z ; and **C**, primary resting spores, P , over a range of initial resting spore densities using the parameter values given in Table 1 and initial values from Table 2. The bar besides **C** gives the relationship between shading and the value of each variable. The threshold for darker shading is very low with all values **A**, above $60 \text{ cm } U_a^{-1}$ and **B** and **C**, $60 \text{ cm } U_a^{-1}$ shaded black.

tative of the amounts of inoculum introduced by transfer of machinery or on infected plants to a field.

The density of resting spores at the end of a season, P_f , is calculated as a weighted sum of I_2 (length of roots containing new resting spores) and initial inoculum, P_i ;

$$P_f = jI_2 + lP_i$$

in which j is the average number of resting spores per centimeter of infected root and l is the proportion of initial inoculum that is viable in the next growing season, recalling that resting spores that are uninfected in the current season may retain some potentially infective zoospores that can be carried through to later seasons. In the absence of experimental evidence, we used an arbitrary weighting of $j = 80$ spores cm^{-1} and $l = 0.8$. We defined the multiplication ratio, MR, as

$$\text{MR} = \frac{P_f}{P_i}$$

The relationship between the MR and initial inoculum density is compared qualitatively with data presented by Tuitert and Hofmeester (32).

Sensitivity analysis. The sensitivity of the length of roots containing new resting spores, I_2 , to changes in parameters over a range of biologically plausible values for host and parasite behavior was examined for initial inoculum values varying between 1 and 150 resting spores per unit of ground area.

RESULTS

Temporal dynamics of the model components. Temporal profiles for components of the host and parasite populations are shown in Figure 2 for a range of initial inoculum densities. The dynamics were dominated by a single large flush of activity that was governed by secondary infection (Fig. 2B, C, and H).

Fig. 4. Detailed plots of temporal dynamics to show the two phases of zoospore production for A, $P_i = 10$ spores U_a^{-1} and B, $P_i = 50,000$ spores U_a^{-1} .

In practice, there are two flushes of secondary zoospores. The initial flush of activity is driven by primary infection and is so small that it is obscured by the scales used in Figure 2. The dynamic sequence of the flushes is more clearly illustrated by image-density plots for the time course of infection over a range of initial inoculum densities (Fig. 3). Figure 3B clearly shows the initial phase of zoospore production derived from infection by the primary inoculum source. This preliminary phase is completed before substantial growth of the susceptible roots occurs (cf. Figs. 2A and 3A). As the source of primary inoculum is exhausted (Fig. 3C), the production of zoospores decreases until production of susceptible root (Fig. 3A) is sufficient to fuel the principal phase of secondary zoospore production (Fig. 3B). This phase subsides as roots become resistant to infection (Fig. 2A and E) and the supply of susceptible roots diminishes. The initial inoculum density determines the relative magnitudes of the phases and the timing of the second phase of secondary infection (Fig. 4A and B). Lower levels of initial inoculum yield correspondingly low levels of secondary zoospores at the end of the first phase (Fig. 4A). This delays the onset of the second phase of secondary zoospore activity (Fig. 2H).

Relationship between initial and final inoculum. The relationship between initial inoculum, P_i , and the length of root per unit of ground area infected with new resting spores, I_2 , is monomolecular with the asymptotic value of $I_2 \approx 1 \times 10^4 \text{ cm U}_a^{-1}$. The asymptote is approached to within 5% when $P_i \geq 100$ spores U_a^{-1} (Fig. 5A). This leveling-off occurs once there are sufficient primary and secondary zoospores to saturate the available susceptible roots throughout the season.

The amount of inoculum in the soil at the end of the season including that trapped in decaying roots, P_f , is calculated by taking a weighted sum of I_2 and P_i (defined in Model simulations) (Fig. 5B). The weights, j and l , were arbitrarily defined, with j representing the production of resting spores in a unit length of infected root and l being the proportion of initial inoculum that survives from one season to the next. The MR is very high at low P_i (Fig. 5C) because of the high potential for infection due to the secondary multiplication cycle (Fig. 2H). The MR decreases exponentially with the logarithm of the initial inoculum density (Fig. 5C). The relationship between MR and P_i is similar in shape to data presented by Tuitert and Hofmeester (32) for *P. betae* inoculum carrying BNYVV (Fig. 5D).

Sensitivity analysis of I_2 to small parameter changes for different initial inoculum densities. The response of I_2 , the length of infected root, at $t = 200$ days to varying each parameter value at different initial inoculum levels is shown in Figure 6. The qualitative behavior of the model is unaffected by small changes in the parameter values, i.e., two, usually asymmetric, phases of infection corresponding with the decay of primary inoculum and a simple (monomolecular) saturation of infected tissue with increasing initial inoculum. We considered the analysis in relation to primary infection, secondary infection, susceptible root growth, and latency (Table 3). Decreasing ϵ and increasing λ_1 prolongs the primary infection period, resulting in higher I_2 (Fig. 6A and B). High values of I_2 are obtained when there is a large multiplication of inoculum through the secondary infection cycle. Thus, increasing the rate of release of secondary zoospores, γ , or the rate of secondary infection, λ_2 , results in an increase in I_2 (Fig. 6C and D). Conversely, increasing the decay of secondary zoospores in the absence of infection, α , the rate of decay of infectious roots, σ , and the rate of loss of secondary zoospores through infection, η , results in a decrease in I_2 for low primary inoculum levels since they all have an adverse effect on the amount of inoculum available (Fig. 6E, F, and G). Similarly, increasing the rate at which roots develop resistance to infection, μ , results in a decrease in the number of susceptible sites and hence a decrease in I_2 (Fig. 6H).

A low growth rate, r , in root length results in high levels of I_2 , since more susceptible sites are available late in the season enabling a longer period of secondary infection (Fig. 6I). If the growth

rate is very small, the total fibrous root length fails to reach the carrying capacity within the season, thus limiting the buildup of infected roots. Increasing the carrying capacity of the roots, k , resulted in a linear increase in I_2 at $t = 200$ days for the range of values examined (Fig. 6J).

The effects of changing the latent periods depend on the relative magnitude of β_1 and β_2 (Figs. 6K and L and 7). If $\beta_2 \geq \beta_1$, then there is very little secondary infection and I_2 is very small (Fig. 7). As β_2 becomes smaller, for a given β_1 , there is also a decrease in I_2 as inoculum is trapped in the secondary infection cycle.

DISCUSSION

P. betae is a ubiquitous parasite of sugar beet that typifies an important class of plant parasites that have multiple infection cycles that are autonomously driven by parasite and host dynamics. The model presented here for the temporal buildup of *P. betae* on sugar beet synthesizes these dynamics. The model integrates cycles of primary infection initiated by resting spores and cycles of secondary infection driven by secondary zoospores with the growth and turnover of fibrous roots on the host. We used the model to analyze the dynamics of host, resting spores, and zoospores and to predict the MR of initial to final resting spores during a season.

The model is an elaboration of a SEIR description of host growth with free-living infective stages (2) originally used to describe the dynamics of microparasitic infections in animal diseases with a single source of infection. Gubbins and Gilligan (17–19) previously generalized this type of model to include dual sources of infection, host response to infection, and disturbance. Here, we

expand the model to five host compartments: susceptible, S ; exposed, E ; infectious, I_1 ; infected, I_2 ; and resistant, R , root material. We also expanded the free-living infective stage to two compartments for ephemeral zoospores, Z , and long-lived resting spores, P . This leads to a model with 12 parameters and two nontrivial initial conditions for the initial densities of susceptible roots and resting spores. Seven parameters were approximated from independent experiments, and the remainder were arbitrarily assigned within biologically plausible bounds. We examined several methods for model reduction including removal of the latent class; replacement of the primary inoculum class, P , with initial values for secondary inoculum, Z ; together with removal of host-density dependent decay of resting spores and zoospores (C. R. Webb, *unpublished data*). The full model is retained here, however, for generality in order to analyze the underlying dynamics of *P. betae* infection as a precursor for later analysis of the effects of environmental driving variables and the transmission of BNYVV. We discuss elsewhere methods for model reduction using asymptotic analysis (29).

The dynamics of *P. betae* infection are characterized by two phases of infection that differ in magnitude and duration according to the initial inoculum density (Figs. 2 to 4). The first phase is initiated by primary zoospores. Roots become infected, pass through the latent period, and produce secondary zoospores that give rise to secondary infection. This initial phase is constrained by the availability of susceptible roots and the decay of viable primary inoculum. The second phase follows the onset of rapid root production, but the duration and magnitude of this phase depends on the amount of inoculum that survives the first phase and, hence, on the amount of initial inoculum (Figs. 2F, 3B, and 4). This corresponds with

Fig. 5. Relationship between initial inoculum density and **A**, the length of infected root 200 days after sowing; and **B**, the amount of inoculum that is viable at the start of the next season (assuming no spatial spread). **C**, Relationship between initial inoculum density and the multiplication ratio calculated from the model. **D**, Experimental data for log initial density of beet necrotic yellow vein virus-infected *Polymyxa betae* against the multiplication ratio. **C**, The model was run over inoculum densities ranging from 0.84 to 8.4×10^7 spores U_a^{-1} . This was converted to most probable number (MPN) per 100 g of soil by assuming an average soil volume of $128 \text{ kg } U_a^{-1}$ and that 100 resting spores are equivalent to a MPN of one.

the experimental observations of Blunt (7) that initial inoculum levels determine both the intensity and rate of development of later stages in the life cycle. For very low densities of initial inoculum, the second phase is delayed and there is a sharp spike of secondary zoospores produced by the abundance of susceptible root available.

The potential for buildup of inoculum during a single season is substantial. This, in turn, has important consequences for the probability of severe infection in second crops and for the risk of disease if *P. betae* becomes infested with BNYVV. Tuitert and Hofmeester (32) reported that initial inoculum densities of 0.14 to

0.58 infective units of *P. betae* per 100 g of soil resulted in mean MRs of 2.2×10^4 over two nonirrigated crops and 6.3×10^4 over two irrigated sugar beet crops. The relationship obtained using the model between the MR and initial inoculum density matches those of Tuitert and Hofmeester (32) in shape (Fig. 5C and D). Beyond this, the results are not strictly comparable since Tuitert and Hofmeester (32) measured only the *P. betae* infested with BNYVV. Additionally, specific field conditions cannot be simulated with the model because we assume constant and optimal conditions for buildup.

Fig. 6. A to L, Sensitivity of the amount of infected root, I_2 , 200 days after sowing (vertical axis) to each of the model parameters for initial inoculum densities ranging from 1 to 150 spores U_a^{-1} . Definitions of the parameters are given in Table 1. Note some of the y-axes directions for the model parameters are reversed for clarity.

The response of the model to small changes in the parameter values is consistent with intuitive expectations (Fig. 6, Table 3). For example, increasing the maximum total root length, k , results in an increase in the length of root infected with new resting spores, I_2 , 200 days after sowing; decreasing the values of the transmission parameters λ_1 and λ_2 resulted in a lower level of root infection. Both latent period parameters, β_1 and β_2 , show a nonmonotonic relationship between the values of the parameters and the value of I_2 after 200 days. High levels of infected root, I_2 , only occur when $\beta_2 < \beta_1$; however, as β_2 becomes small, relative to a given value of β_1 , there is also a decrease in I_2 . This occurs because most of the available susceptible sites become filled with secondary zoospores. Once infected, the sites become resistant to infection, reducing the number of sites available for new resting spores. The sensitivity analysis showed that the choice of parameter values did not critically affect the qualitative behavior of the model, i.e., two, usually asymmetric, phases of infection corresponding with the decay of primary inoculum and a simple (monomolecular) saturation of infected tissue with increasing initial inoculum. Alternative parameter values may delay the onset of the second phase of infection (Fig. 4) or reduce the threshold value of I_2 (Fig. 6), but overall, the relationship between initial inoculum density and the length of infected root at the end of the season is consistent with the behavior shown in Figure 5.

Fig. 7. Effect of varying the relative values of the latent period parameters, β_1 and β_2 , on the amount of infected root, I_2 , 200 days after sowing.

The model can be used to examine the effect of alternative control strategies on the development of the disease. For example, the rates of infection, λ_1 and λ_2 , and the multiplication of zoospores, γ , may be reduced using varieties resistant to the fungus (4), while soil fumigation or fungicidal control may affect the amount of primary inoculum, P , and the survival period for secondary zoospores, α . In practice, control of the disease by eradication of *P. betae* is not feasible (20). Partial eradication of the inoculum between seasons may limit symptom expression, but the control is temporary since inoculum continues to build up, thereby necessitating expensive repeated control. Precise guidance on the feasibility of the alternative strategies depends on improved information on the remaining parameters.

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TABLE 3. Summary of the sensitivity of the model in relation to infected root length, I_2 , for each of the model parameters

Compartment affected	Parameter	Effect of increasing the parameter value on I_2 at $t = 200$ days	Reason for effect
Primary infection	ϵ	Decrease	Primary inoculum source used up more rapidly.
Secondary infection	λ_1	Increase	Increases efficiency ^a of primary zoospores.
	γ	Increase	Increases Z.
	λ_2	Increase	Increases efficiency ^a of secondary zoospores.
	α	Decrease	Decreases Z.
	σ	Decrease	Decreases I_1 and hence Z.
Susceptible root growth	η	Decrease	Decreases Z.
	μ	Decrease	Increases the rate at which roots become resistant to infection.
	r	Nonmonotonic	For low values of r (≤ 0.1 cm day ⁻¹), the total root length fails to reach the carrying capacity within the growing season and hence I_2 is low. For high values of r (≥ 0.1 cm day ⁻¹), there are many susceptible sites available early in the season, resulting in rapid secondary infection, leaving fewer sites available for new resting spores.
Latent period	k	Increase	Increases total number of susceptibles produced.
	β_1	Depends on the relationship between β_1 and β_2 (described in Fig. 7)	
	β_2		

^a Efficiency: contact rate and probability of successful infection.

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