A Model of Verticillium Wilt in Relation to Cotton Growth and Development

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ABSTRACT

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Based on systems analyses, the phenology and the growth and development of healthy cotton plants (*Gossypium hirsutum* 'Acala SJ-2') were compared with those of cotton plants showing foliar symptoms of Verticillium wilt. A submodel for Verticillium wilt was developed and coupled to an existing simulation model for the growth and development of a population of cotton plants. In the Verticillium wilt submodel, the effects of inoculum density, pathotypes of *Verticillium dahliae* (defoliating and nondefoliating), and the timing of foliar symptom appearance in relation to physiological time (degree days) were the main variables considered in respect to cotton phenology, dry matter accumulation in plant parts, and lint yields. The model also simulates the influence of shading and canopy compensation on photosynthate production in cohorts of diseased and healthy plants. Simulations of cotton crops grown in soils containing 0, 5, 30, and 60 propagules per gram of soil (nondefoliating pathotype) indicated decreasing trends in yields of lint with increasing inoculum density. With an increasing incidence of wilt, the fraction of total yield contributed by diseased plants increased; however, the earliness of foliar symptom development and the percentage of plants with foliar symptoms were the most significant factors associated with reductions in lint yields.

Additional key words: epidemiology, soilborne plant pathogens.

Models describing the physiology and growth of single plants have been developed for several crop plants, including cotton (6). Generally these models have been in the form of complex algorithms requiring substantial computer time. Although having good predictive values, such models are often as difficult to understand as the biological systems they mimic. The reason for this is that many modelers tend to strive for a one-to-one simulation of nature rather than a reduced description that captures the essential features of the process. In this paper, we expand the simple (yet realistic) mathematical population model for cotton growth and development (29) to include density-dependent interactions between plants (ie, compensation effects) and couple a model for Verticillium wilt to it. In prior work, the cotton model has been linked to models of cotton insect defoliators (16). The data used to develop this model are contained in several references (1,8,9,21–23); these will not be reproduced here, except to clarify a point or to test the model. The model was tested by comparing simulated data to field observations of plant phenology, dry matter accumulation in plant parts, the number and pattern of large green fruit (bolls) production, and foliar symptom occurrence in relation to the concentration of soilborne propagules of *Verticillium dahliae* Kleb.

Verticillium wilt, one of the most important diseases of cotton, probably causes greater economic losses in that crop in the San Joaquin Valley of California than any other disease or pest (including arthropods). The causal fungus, *V. dahliae*, is soilborne and it infects plants throughout the growing season. Pathogenesis and resultant losses of lint yields involve the occlusion of the vascular system in diseased plants (20) and a reduction in the photosynthetic capabilities (19). Pathogenesis is further complicated by the existence of at least two major strains of *V. dahliae*, one of which, in addition to causing common symptoms of Verticillium wilt, can cause complete defoliation of infected plants by altering their ethylene metabolism (26). Infection of cotton involves direct penetration of the roots, mainly in the areas of differentiation and the root hair zone (11), and probably in epidermal tissues ruptured by the emergence of lateral roots. Propagules of *V. dahliae* in natural soils consist of free microsclerotia, pieces of plant debris containing bound microsclerotia, hyphae, and conidia; however, in soils that have been air-dried for 6 wk or longer, free and bound microsclerotia are the principal surviving propagules (4). Important factors that influence the development of Verticillium wilt include the strains and concentrations of propagules of *V. dahliae* in soil (1,3,5,21,22,26), the species and cultivar of cotton (1,25), air and soil temperatures (12,23), cropping history (24,27), mineral nutrients (17,24), and moisture (24). The stage of cotton plant development when foliar symptoms of Verticillium wilt appear, however, is the main factor in lint losses caused by Verticillium wilt (2,23).

It is important to emphasize at the outset that low concentrations of propagules of *V. dahliae* (1) can cause up to 100% infection resulting in vascular discoloration of cotton plants under field conditions; however, plants with vascular discoloration may not show foliar symptoms if the average air temperature is high (>28 C) or the concentrations of soilborne propagules of *V. dahliae* are low (4). Extensive invasion of the leaf vascular system by *V. dahliae* is considered necessary for development of foliar symptoms (20). Thus, in the model of Verticillium wilt described here, the word disease involves a degree of leaf infection that results in foliar symptoms.

COTTON PLANT GROWTH AND DEVELOPMENT

Biology of cotton plant growth and development. Figure 1 shows the generalized development of pest- and disease-free cotton in the San Joaquin Valley. The peak of square formation, the beginning of boll production, and the cessation of leaf, stem, and root growth all coincide with the onset of rapid accumulation of dry matter in fruits (15,16,29). This point, labeled t_s, is the time when the demand for carbohydrates by the growing plant parts becomes much greater than the supply. At this stage of development, the cotton

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plants shed a large proportion of their excess fruit consisting mostly of squares and bolls less than 10 days of age. Gutierrez et al (16) described the above biological details, while Wang et al (29) presented a mathematical description of the population dynamics of cotton growth and development.

The cotton growth and development model. A population of cotton plants (ρ_p) consists of individual plants, and each plant has a population of leaves (L), stems (S), roots (R), and fruits (number = F, mass = M) of varying age. The discrete plant population model proposed by Gutierrez et al (16), Gutierrez (14), and a continuous version by Wang et al (29) provide the bases for modeling the effects of Verticillium wilt on cotton growth, development, and yield (equation 1). The population model for cotton growth and development is composed of separate models for the population of individual plants ($\rho_p(t,a)$) per meter of row and for L, S, R, M, and F within plants at time (t) and age (a). Because the crop is planted within a brief span of time, $\rho_p(t,a)$ reduces to $\rho_p(t)$. These models are linked as follows:

$$\text{numbers}\quad \frac{d\rho_p}{dt} = -\mu_p(e)\,\rho_p(t) \qquad [1i]$$

$$\left\{\begin{array}{ll} \dfrac{\partial L}{\partial t} + \dfrac{\partial L}{\partial a} = -\mu_L(e)\,L(t,a) & [1ii] \\ \dfrac{\partial R}{\partial t} + \dfrac{\partial R}{\partial a} = -\mu_R(e)\,R(t,a) & [1iii] \\ \dfrac{\partial S}{\partial t} + \dfrac{\partial S}{\partial a} = -\mu_S(e)\,S(t,a) & [1iv] \\ \dfrac{\partial M}{\partial t} + \dfrac{\partial M}{\partial a} = -\mu_M(e)\,M(t,a) & [1v] \\ \text{numbers}\quad \dfrac{\partial F}{\partial t} + \dfrac{\partial F}{\partial a} = -\mu_F(e)\,F(t,a) & [1vi] \end{array}\right.$$

(density-dependent interactions; supply demand; age)

in which $\rho_p(t)$ and $F(t,a)$ are number density functions and $L(t,a)$, $R(t,a)$, $S(t,a)$, and $M(t,a)$ are mass density functions and require initial conditions (eg, $L(t,0)$ and $L(0,a)$) to guarantee the uniqueness of the solution. Because all of the plants are of nearly the same age, age structure for the plant population can be ignored. The various $\mu_k(e)$ are complicated functions containing elements of population birth and death rates (production and removal or shedding rates), and other variables (e) as outlined in this text. The brackets in the array above show the various levels of linkage. Conceptually, the model (16,28) is the continuous form of the well-known Leslie Matrix model (18), which can be written in discrete form as

$$\vec{N}_{k,t+\Delta t} = \boldsymbol{A}_k \cdot \vec{N}_{k,t} \qquad [2]$$

in which N_k is the vector of plant parts (k) of n cohorts of age (j), t is time, and $\boldsymbol{A}_k$ is the appropriate time varying (as opposed to constant) matrix of age-dependent production and survivorship rates. The allocation of photosynthate ($P(e)$) to production of new plant parts is included in birth rates per Δt, where $P(e) = P(\text{age, leaf area index, solar radiation, water, } \cdots)$. The plant part models are linked via a metabolic pool model described in the next section.

EFFECTS OF *Verticillium dahliae* ON COTTON PLANT INFECTION, FOLIAR SYMPTOM DEVELOPMENT, AND GROWTH

Virulence of *V. dahliae*. Pathotypes (v) of *V. dahliae* vary greatly in virulence. The pathogen may occlude the vascular system, inhibit the rate of nutrient flow to growing plant parts, and also reduce photosynthetic productivity in leaves (19,20) (Fig. 2). In the model, the effects of virulence are incorporated via the scalar Z ($0 \leqslant Z(v) \leqslant 1$) in which $Z = 1$ implies no foliar symptoms and $Z = 0$ implies maximum disease effects and no plant growth or development. Values for the parameter Z were fitted to data on pathotypes T9 (defoliating, $Z = 0.4$) and SS4 (nondefoliating, $Z = 0.5$) by using computer simulations.

Phenology of infection. The population of plants consists of healthy plants, plants with vascular discoloration, and plants that exhibit both vascular discoloration and foliar wilt symptoms at various times during the growing season. Plants diseased early in the season are stunted and set and ripen few or no bolls, while those diseased progressively later in the season are correspondingly less stunted, have greater numbers of bolls, and produce higher yields.

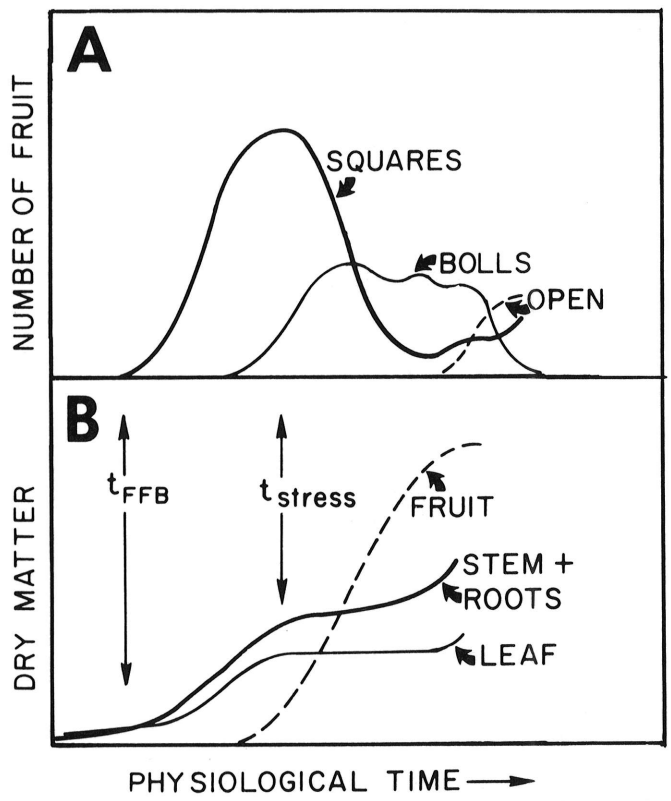

Fig. 1. The patterns of **A,** fruit (squares, bolls, and open bolls) and **B,** dry matter accumulation in cotton plant parts in disease-free fields. The time of the first fruiting branch (t_{FFB}) and the time of crop carbohydrate stress (t_s) are noted.

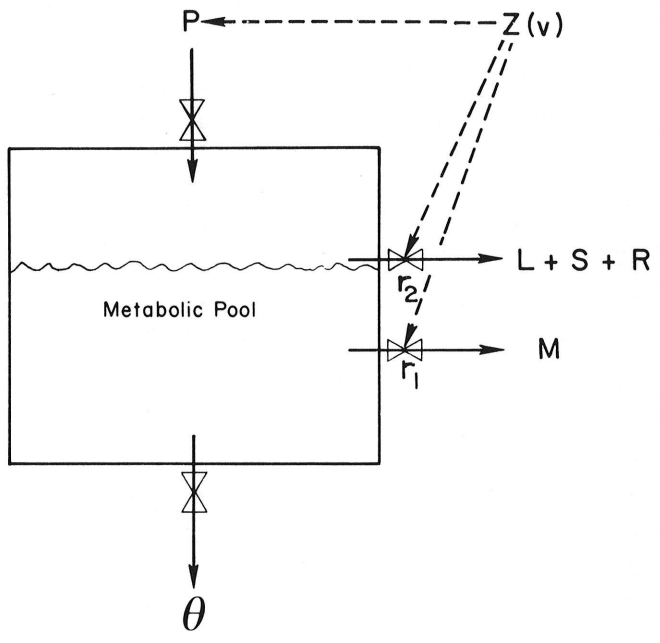

Fig. 2. The modified metabolic pool model showing the effects of Verticillium wilt (Z) on photosynthate production (P) and on its allocation to metabolic costs (θ), leaf (L), stem (S), root (R), and fruit (M). A complete discussion of this model is in the text.

Data from Butterfield and DeVay (2), Pullman and DeVay (23), and Friebertshauser (8,9) describe the growth patterns of plants that develop foliar wilt symptoms at various times in the season.

Fungal infection of plants often occurs via the root tip (11). The percentages of plants showing foliar symptoms (ϕ_s) from a nondefoliating Verticillium wilt pathotype at two inoculum densities (ρ_v per gram of soil) are seen in Fig. 3. Plants having two or more leaves with foliar wilt symptoms were classed as diseased (22). Vascular discoloration (infection) of a plant occurs earlier than foliar symptom expression (22), the exact time being a function of the number of root tips penetrating the soil, plant size, inoculum density (ρ_v), and pathotype severity ($Z(v)$) (8,22).

Equation 3 estimates the rate of change ($d\phi_s/dt$) in the proportion of plants with foliar symptoms from pathotype v after the time (t^*) in degree days (D°) of first symptom appearance,

$$0 \leqslant d\phi_s/dt = [(-1.3 \times 10^{-5}\rho_v^2 + 1.6 \times 10^{-3}\rho_v - 4.5 \times 10^{-4})z]/100 \qquad [3]$$

in which $t \geqslant t^* = (1{,}440\ D° - 6\rho_v)z^{-1}$, and $z = Z(v)/Z(SS4)$. Note that z increases the rate of symptom expression of $Z(v)$ relative to the standard pathotype (SS4), as well as decreasing the time to first appearance of foliar symptoms. The time of plant infection is approximately 200 D° earlier than that for foliar symptom expression (2,8,22), but the slopes of plant infection and foliar symptom development over time are expected to be equal. Hence the proportion of plants showing foliar symptoms is $0 \leqslant \phi_s = (d\phi_s/dt)\cdot\Delta t \leqslant 1$, in which $\Delta t = t - t^*$. The above relationship is approximate because newly infected plants do not develop foliar symptoms when the maximum air temperature is ≥38 C, but do develop symptoms quickly when temperature conditions are lower (21,22). This phenomena is seen as a discontinuity in field data during periods of high air temperatures.

Planting density and row spacing also influence the rate of disease development. Leaf symptoms are reduced as the number of plants per unit area are increased (10). This relationship is not addressed in the current model.

Effects of Verticillium wilt on plant growth and development. In the model, growth and development of the plant(s) are controlled by priority allocation of photosynthate (P) from a metabolite pool $Q_{max} = 0.2\int (L+S+R)da$, first to respiration ($\theta$), then fruit growth ($M$), and lastly vegetative growth (L, S, R, and F). The value 0.2 estimates the maximum carbohydrate reserves in cotton. All physiological costs are summarized in $\theta = \theta(e)$ (28). The *first priority* of plants (infected = I, and healthy = H) is to meet basic respiratory cost, hence:

If $Q(t) = Q(t-\Delta t) + Z(t)\ P_I(t) - \theta\int(L+R+S+M)da \leqslant 0$, the plant dies. The constant θ is the respiration per unit of plant tissue per Δt; L, R, S, and M are the mass of leaf, root, stem, and fruit alive at time (t), and age (a), respectively.

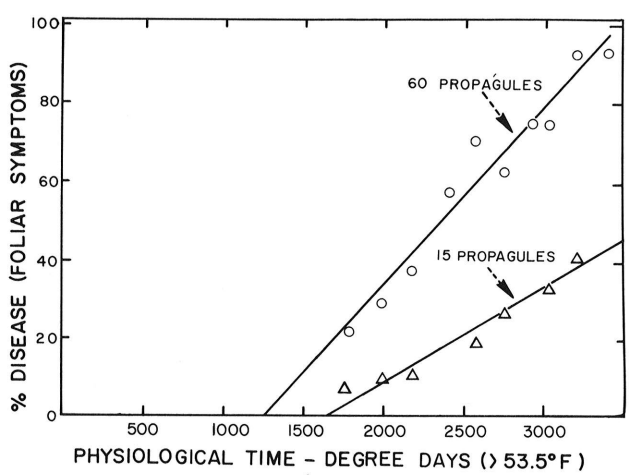

Fig. 3. The percentage of cotton plants showing foliar symptoms of Verticillium wilt at 60 and 15 propagules of *Verticillium dahliae* per gram of soil (data from Pullman et al [22]).

In the last two priority levels, the remaining photosynthate is allocated as follows:

If $Q = Q(t) \geqslant 0$

(a) Q is allocated first to fruit if $t \geqslant t_{FFB}$ when t_{FFB} is the time of first fruit (see Fig. 1B).

(b) and then proportionally to leaf, stem, and root if $Q - \Delta M^*(t) \geqslant 0$, in which ΔM^* is the maximum (*) demand for fruit population growth.

The effects of $Q - \Delta M^* \geqslant 0$, and Verticillium wilt ($Z = Z(v)$) on growth and production rates of plant parts (ie, the birth rate) are modelled as follows (see Fig. 2):

$$\Delta \text{ fruit} \begin{cases} \text{mass} & \Delta M_t = \dfrac{dM^*(a)}{dt}\ r_1 \cdot \Delta t \cdot Z, \\ & \text{in which } 0 \leqslant r_1 = \dfrac{Q}{\Delta M^*} \leqslant 1 \qquad [4i] \\ \text{numbers} & \Delta F_t = \dfrac{dF^*(\rho_p)}{dt} \cdot r_2 \cdot \Delta t \cdot Z, \\ & \text{in which } 0 \leqslant r_2 = \dfrac{Q - \Delta M^*}{\Delta W^*} \leqslant 1 \qquad [4ii] \end{cases}$$

$$\left.\begin{matrix} \Delta \text{ leaf} \\ + \\ \Delta \text{ stem} \\ + \\ \Delta \text{ root} \end{matrix}\right\} \text{ mass} \quad \Delta W = \left(\frac{dL^*(\rho_p)}{dt} + \frac{dS^*(\rho_p)}{dt} + \frac{dR^*(\rho_p)}{dt}\right) \cdot r_2 \cdot \Delta t \cdot Z \qquad [5]$$

in which r_1 and r_2 are supply/demand allocation ratios for photosynthate allocation to fruit and vegetative growth, respectively. ρ_p is plant density (ie, plants per meter of row) and ΔW

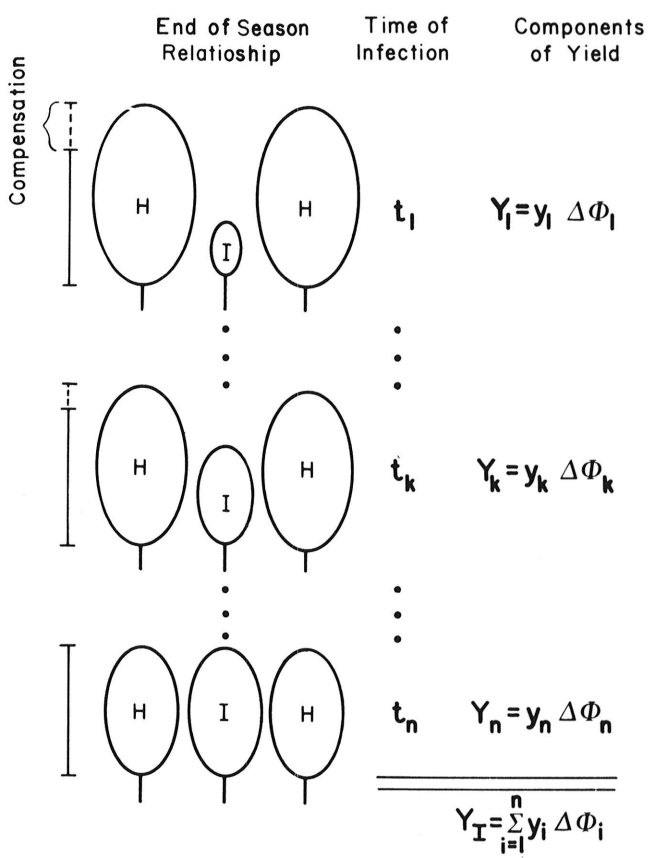

Fig. 4. The end-of-season relationship between healthy (H) and Verticillium wilt-affected (I) cotton plants, the degree of compensation and the computations for computing yield (y) per cohort of plants diseased ($\Delta\phi$) at times $t_1 \ldots, t_k, \ldots, t_n$. Note that the model treats populations of plants diseased at various times and not individual plants.

is the change in vegetative mass. Mainstem node production (N) is regulated by r_2 and is modeled as follows:

$$\text{Main stem nodes} = \Delta N_t = \frac{dN(\rho_p)}{dt} \cdot r_2 \cdot \Delta t \cdot Z \qquad [6]$$

The maximum growth rates for all plant parts except that for mass accumulation by fruits [f(age)] are functions of plant density [$g(\rho_p)$].

Excess immature fruit are shed when $r_1 \leqslant 1$ (ie, t_s in Fig. 1) at rates proportional to the deficit (ie, death rates); see Wang et al (29) for details. Thus, we postulate that Verticillium wilt affects not only the flow rate of photosynthates within the plant, but also the photosynthate supply, which, interacting with demand, controls all aspects of plant growth and development.

INTERACTION AMONG PLANTS

Effects of shading and pathotype on photosynthate production. Plants with foliar wilt symptoms are often stunted, overgrown, and shaded by their healthy neighbors (Fig. 4); the degree of stunting being most severe on plants diseased early in the season and progressively less as symptoms develop later. The degree of shading ($0 \leqslant \gamma(t) \leqslant 1$) may be estimated by comparing the ratio of the leaf area (αL) of diseased plants (subscript I) to that of healthy (H) neighbors, in which α relates leaf mass to leaf area. The amount of photosynthate ($P_I(t)$) produced by the diseased plant at time t is scaled by $\gamma(t)$.

$$0 \leqslant \gamma(t) = L_I(t)/L_H(t) \leqslant 1 \qquad [7]$$

In addition to shading effects, the flow of nutrients in the vascular systems of plants with foliar symptoms is slowed; the degree of reduction is dependent on virulence $Z(v)$. Consequently, photosynthesis is inhibited because photosynthate, water, and/or inorganic nutrients cannot be transported. The complete model for photosynthesis then becomes $P_I = P_I(t,e) \cdot \gamma(t) \cdot Z(t,v)$, in which the supply ($P_I(t)$) of photosynthate produced for growth by diseased plants is reduced. The mechanisms contributing to the reduced transport and reduction of photosynthate are only partially understood (19,20,30).

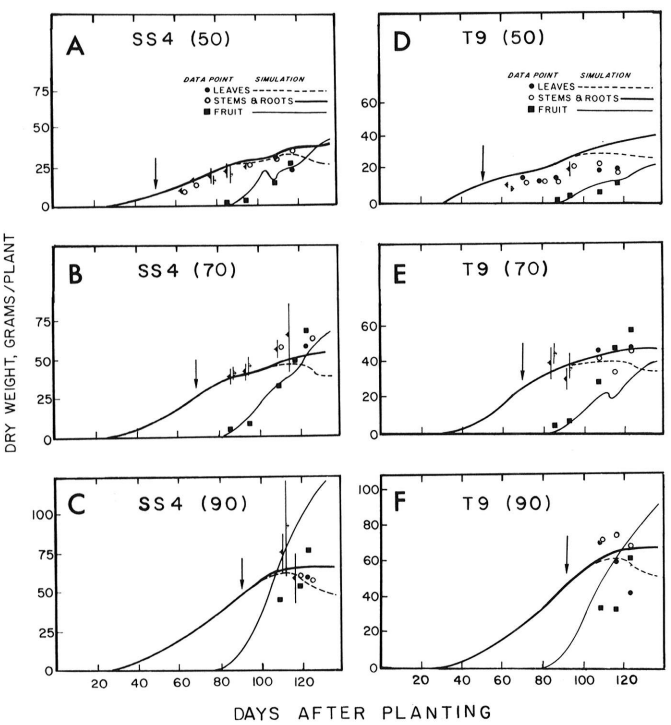

Fig. 5. Simulated and observed dry matter allocation in fruit, stem plus root, and leaves of cotton plants inoculated with two pathotypes of *Verticillium dahliae* (SS4 or T9) at 50, 70, and 90 (indicated by arrows) days after planting (4 May 1978). Note that petiole weights were included as leaf dry matter, hence the discrepancy of leaf stem ratio from previously reported results (15). Data points represent observed dry weights; broken and solid lines represent simulated dry weights (data from Friebertshauser et al [8]).

Compensation by healthy plants. The population of cotton plants includes plants with vascular discoloration (infected), those with vascular and foliar symptoms (diseased), and healthy plants. Since plants diseased late in the season are larger than those diseased earlier, the degrees to which such plants are shaded and their productivity affected are greatly influenced by the time of infection and foliar symptom appearance. Thus, disease development not only reduces the growth rates of infected plants, but also indirectly increases the growth rates of adjacent healthy plants as they avail themselves of the resources (space, sun, water, . . .) not used by the infected neighbors. Figure 4 illustrates the end-of-season differences in sizes of plants developing foliar wilt symptoms at various times during the season (t = 1,...,k, ...,n). Healthy plants compensate for diseased neighbors (until the crop canopy closes at LAI≃2.4) by growing into space which would have been occupied by diseased plants. Healthy plants therefore react to a relaxation of density-dependent growth constraints (see equations 1 and 4–6). In the model, healthy plants are used as the standard and their ability to compensate until they become diseased at t_I is computed as a scalar (C) (Gutierrez et al [15]) for $0 \leqslant \text{LAI} \leqslant 2.4$:

$$0 \leqslant C_t = 1 - \text{EXP}(-0.65\ \text{LAI}) \leqslant 1. \qquad [8]$$

The scalar (C) modifies the effective plant density (ρ_p) in the following manner:

$$\rho_p\,(t+\Delta t) = \rho_p(t) - \frac{d\phi_s}{dt} \cdot C(t) \cdot \rho_p(t_0) \cdot \Delta t \qquad [9]$$

in which $\rho_p(t_0)$ = the initial density of plants,
$d\phi_s/dt$ = the rate of disease development, and
Δt = time increment.

Note that $C(t)\ d\phi_s/dt$ equals μ_p in equation 1i.

Hence, plant compensation in the model occurs via the state variable ρ_p as it affects relaxations in density-dependent growth constraints in equations 4–6. However, crop compensation for diseased plants is always incomplete due to time lags in growth and maturation. In areas such as the San Joaquin Valley, where the growing season is short, this may be extremely important. Also, as inoculum density and/or virulence increase, the population of healthy plants able to compensate decreases. Thus, even though individual plant compensation may be maximum, the per-acre effect of compensation may be reduced by increased inoculum density or virulence.

SIMULATION RESULTS

Model validation. Field data (9,22,23) were used to validate the model. In one study (9), Acala SJ-2 cotton planted 5 May 1978 at the University of California West Side Field Station near Five Points, CA, were inoculated by stem puncture near the soil line at either 50, 70, or 90 days after planting, with concentrations of 2×10^4, 10^5, or 10^6 condia per milliliter, respectively. Conidial suspensions (300 μl) of two pathotypes, SS4($Z = 0.5$) and T9($Z = 0.4$) at the above concentrations, were injected into each plant with a small syringe. The planting density was approximately five plants per meter of row, and the weather data used were from the Fresno area (9).

Comparisons of simulated and observed dry matter in various plant parts as affected by the two pathotypes are shown in Fig. 5. The best fits to the data were obtained when variable time delays between inoculation and disease impact were used (Fig. 6), suggesting that older plants (70 and 90 days) are either less

susceptible than younger plants or larger dosages of inoculum, requiring time to develop within the plant, are necessary to produce foliar symptom expression. When time delays were incorporated, the best fits between observed and simulated data were found for plants inoculated on days 50 and 70. The results for plants inoculated on day 90 were not as good, possibly because our understanding of disease effects on the plants was inadequate. (Note that some of the data points do not have estimates of standard deviations). The simulated and observed patterns of boll production for plants diseased by the two pathotypes are illustrated in Fig. 7, again indicating good simulations for the day 50 and 70 inoculations, but more variable results for day 90.

Estimating yields. The model season is divided into n time periods, and the yields (y_i) in bales per acre of each of the n cohorts of diseased plants as well as healthy plants are estimated incorporating the biology described above (equations 1–9). Thus, for the entire season, the final yield (Y_T) for all n cohorts of diseased plants is computed as follows (see Fig. 4):

$$Y_T = Y_H(1-\sum_{i=1}^{n} \cdot \Delta\phi_i) + \sum_{i=1}^{n} y_i \Delta\phi_i \qquad [10]$$

in which $y_i\Delta\phi_i$ is the yield of the proportion ($\Delta\phi_i$) of plants becoming diseased during t-Δt to t and Y_H is the expected yield if all the plants were healthy.

Figure 8 illustrates the simulated yields from each of the n(= 10) cohorts (time groups during the season) of diseased plants at 0, 5, 30, and 60 propagules per gram of soil for the nondefoliating pathotypes. The initial conditions and weather data used are as reported above. Three trends are apparent with increasing inoculum density: total yields decrease; the fraction of total yield accruing from diseased plants increases; and symptoms appear earlier. Increasing the virulence of the pathotype would affect the results in the same direction. A regression of normalized yield on ρ_v(SS4) indicates that there is a 0.5% reduction in yield per unit increase in p_v(SS4) (ie, $Y_T = Y_H(1-0.005p_v)$. These results are for a short season (4 May to 5 September 1978), but the results are qualitatively the same for a longer season. In general, the more severe the pathotype, the greater the reduction in yield per unit increase in pathogen density (propagules per gram of soil).

Figure 9 summarizes 95 simulated runs of cotton to varying ρ_p, ρ_v, and $Z(v)$ for a typical season in the Fresno area of the San Joaquin Valley, while equation 11 is a least squares fit to the data.

$$Y_T\,(\rho_p, \rho_v, Z(v)) = b_1 + b_2\rho_p + b_3\rho_p^2 + b_4\rho_v + b_5\rho_v^2 + b_6 Z(v)^3 \qquad [11]$$

$R^2 = 0.875, N = 95, b_1 = 0.742, b_2 = 0.253, b_3 = -0.011, b_4 = -0.040,$ $b_5 = 0.00036$, and $b_6 = 0.755$.

The results show a strong nonlinear yield response to ρ_p, and a negative response to ρ_v and to $Z(v)$. Note that virulence increases as $Z \rightarrow 0$.

Theoretical considerations. At a more theoretical level, the model suggests that weakly virulent pathotypes would produce little inoculum due to decreased infection and colonization of host plants, while pathotypes of high virulence would produce little inoculum due to early death of diseased plants. If the assumption is made that the number of microsclerotia produced by each pathotype is proportional to the final biomass of diseased plants, then over time some equilibrium of pathotypes should be reached in fields sown exclusively to one cultivar of cotton, due to interactions of cotton, the inoculum-producing potential of all strains of *V. dahliae* in the soil, and the changing populations of other soilborne microorganisms. As variability of species acts to maximize fitness (reproduction × survival over some effective time period-a season (7,13)), too-virulent or too-weak a pathogen is

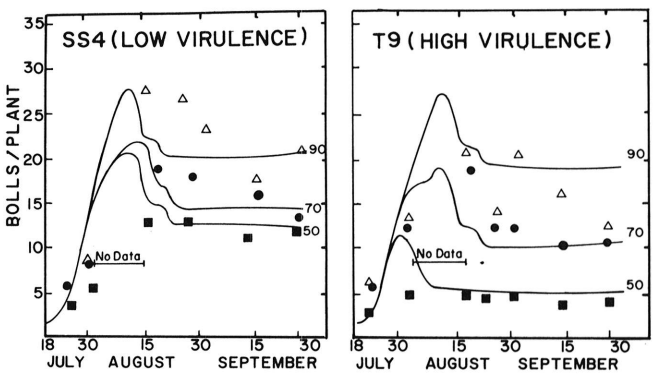

Fig. 7. Simulated and observed patterns of boll production for cotton plants inoculated at 50, 70, and 90 days with pathotypes of SS4 or T9 of *Verticillium dahliae* (data from Friebertshauser et al [8]). Data points are observed values; lines through data points are generated by the model.

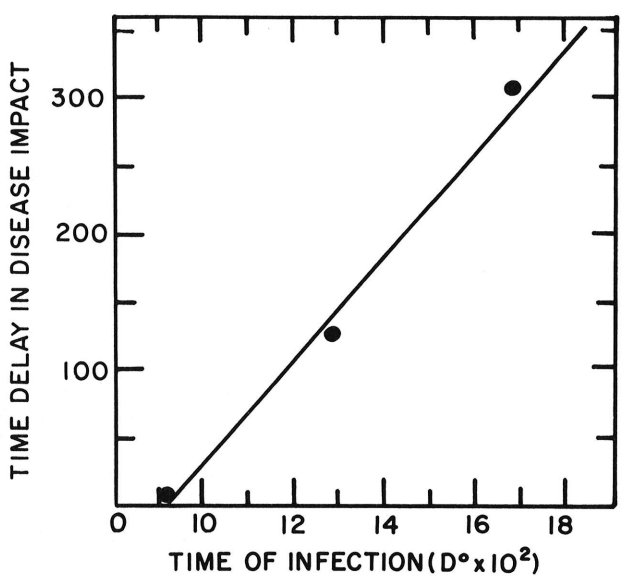

Fig. 6. Physiological time delay (degree days) after simulated infection of plants of cotton cultivar Acala SJ-2, when disease alters dry matter accumulation.

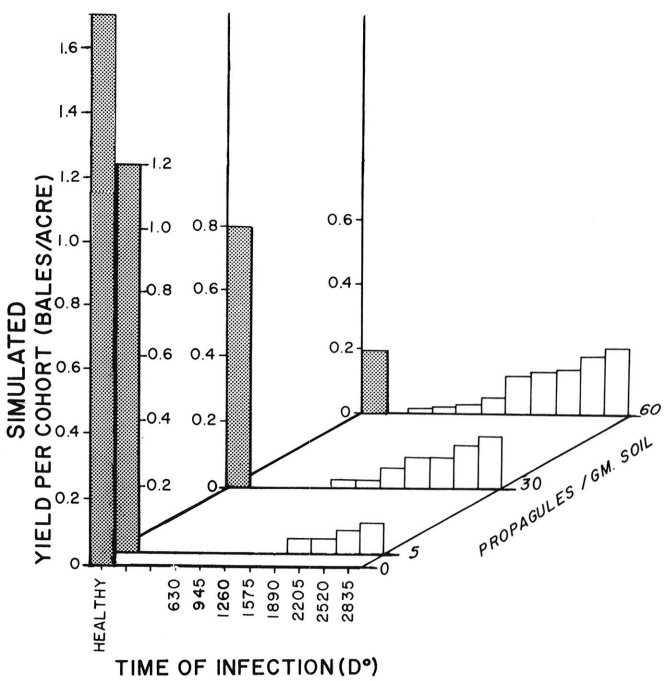

Fig. 8. Simulated end of season yields for cohorts of plants diseased at different times during the season in soils containing 0, 5, 30, and 60 propagules of *Verticillium dahliae* per gram of soil. These yields are compared to yields from uninfected plants in the crop.

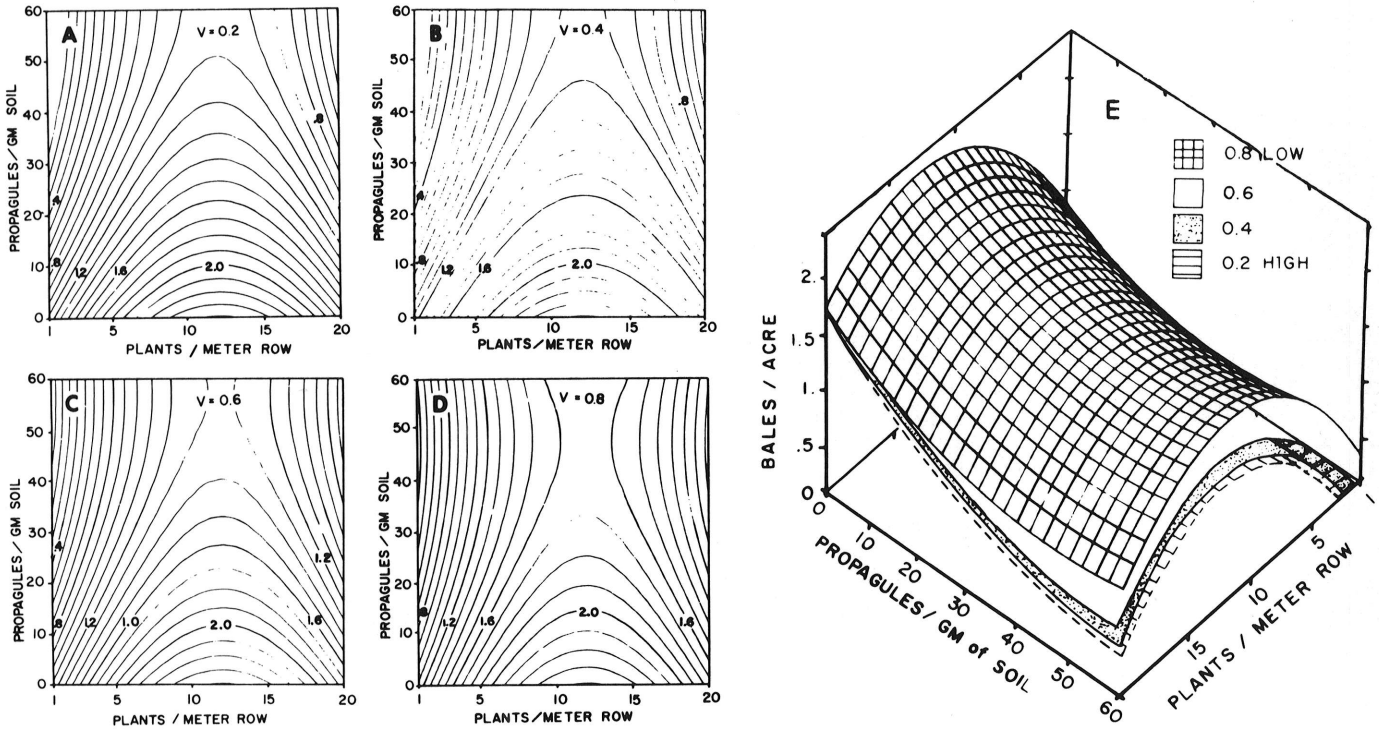

Fig. 9. The effects of inoculum density, planting density, and pathogen virulence on cotton lint yields per acre. **A–D** show the contours of cotton lint yields for various levels of inoculum density and planting density at four levels of relative pathogen virulence ($0 \leqslant Z \leqslant 1$). **E** shows a summary of the effects of inoculum density, planting density, and pathogen virulence.

suboptimal as it would tend to lower either plant and/or pathogen fitness. Arguments concerning fitness are usually made for natural systems, but within reasonable limits, the same arguments apply to agricultural crops. Also pertinent is the changed selection pressure of new cotton cultivars and the cross protection of cotton plants by the nondefoliating pathotype against the defoliating pathotype in field soils containing mixtures of these pathotypes (26).

DISCUSSION

The model is a concise summary of what is known about the interaction of cotton and Verticillium wilt as modified by weather, planting dates, density dependent effects, and other factors. Some parts of this model such as the reduction of virulence effects to a single Z factor may appear simplistic. However, Z is linked to photosynthesis, occlusion of the vascular system, rates of plant infection and disease development, growth and development of plant parts, and the production of cotton lint yields. More complicated relationships were tried, but these simple ones yielded the best simulation results. No method, our model included, can accurately predict yields because they cannot fully incorporate *all* of the influences of weather, plant compensation, soil type, plant water stress, and other factors; the results must be considered qualitative. Despite this apparent shortcoming, the model has many practical applications. For example, early season estimates of pathogen density (ρ_v) and virulence $Z(v)$ can be made for specific sites and the model can be used to estimate pathogen impact on yields. The model facilitates the development of economic decision rules for the integrated management of plant diseases, insect pests, and cultural practices which may in turn decrease production costs and increase yields. Perhaps, however, the most important aspects of the present research, aside from concerns of integrated pest management, are the time-course effects of Verticillium wilt that suggest physiological bases of disease development.

As our knowledge increases, the model will continually be updated and improved. For example, as new data become available on temperature interactions that inhibit disease development or re-initiate growth (heat therapy), degrees of symptom expression, or new cotton cultivars, they will be included within the model framework.

LITERATURE CITED

1. Ashworth, L. J., Jr., Huisman, O. C., Harper, D. M., Stromberg, L. K., and Bassett, D. M. 1979. Verticillium wilt disease of cotton: influence of inoculum density in the field. Phytopathology 69:483-489.
2. Butterfield, E. J., and DeVay, J. E. 1977. The effect of Verticillium wilt on the growth and development of cotton. (Abstr.) Proc. Am. Phytopathol. Soc. 4:173.
3. Butterfield, E. J., and DeVay, J. E. 1977. Reassessment of soil assays for *Verticillium dahliae*. Phytopathology 67:1073-1078.
4. DeVay, J. E., Forrester, L. L., Garber, R. H., and Butterfield, E. J. 1974. Characteristics and concentration of propagules of *Verticillium dahliae* in air-dried field soils in relation to the prevalence of Verticillium wilt in cotton. Phytopathology 64:22-29.
5. Erwin, D. C. 1977. Control of vascular pathogens. Pages 163–224 in: Antifungal Compounds. Vol. 1. M. R. Siegel and H. D. Sisler, eds. Marcel Dekker, Inc., New York. 400 pp.
6. Evans, L. T. 1975. Crop physiology: Some case histories. Cambridge Univ. Press, Cambridge, England. 374 pp.
7. Fisher, R. A. 1930. The Genetical Theory of Natural Selection. Clarendon Press, Oxford, England. 272 pp.
8. Friebertshauser, G. E. 1979. Differential effects of the defoliating and non-defoliating pathotypes of *Verticillium dahliae* Kleb. upon growth and development of *Gossypium hirsutum* L. M.S. thesis. University of California, Davis. 136 pp.
9. Friebertshauser, G. E., and DeVay, J. E. 1982. Differential effects of the defoliating and nondefoliating pathotype of *Verticillium dahliae* upon the growth and development of *Gossypium hirsutum*. Phytopathology 72:872-877.
10. Garber, R. H., DeVay, J. E., and Wakeman, R. J. 1981. Effect of plant spacing and symptom expression in Verticillium wilt of cotton. Page 30 in: Proc. Beltwide Cotton Prod. Res. Conf., National Cotton Council, Memphis, TN. 321 pp.
11. Garber, R. H., and Houston, B. R. 1966. Penetration and development of *Verticillium albo-atrum* in the cotton plant. Phytopathology 56:1121-1126.
12. Garber, R. H., and Presley, J. T. 1971. Relation of air temperature to development of Verticillium wilt of cotton in the field. Phytopathology 61:204-207.
13. Gilbert, N., Gutierrez, A. P., Fraser, B. D., and Jones, R. E. 1976. Ecological Relationships. Freeman and Co., New York. 156 pp.
14. Gutierrez, A. P. 1976. Applied population ecology: Models for crop production and pest management. Pages 255–280 in: Proc. Conf. Pest Management. G. P. Norton and C. S. Holling, eds. I.I.A.S.A.

(International Institute for Applied System Analysis), Luxenburg, Austria. 352 pp.
15. Gutierrez, A. P., DeMichele, D. W., Wang, Y. H., Curry, G. L., Skeith, R., and Brown, L. G. 1979. The system approach to research and decision making for cotton pest control. Pages 155–186 in: New Technology and Pest Control. C. B. Huffaker, ed. Wiley-Interscience, New York. 500 pp.
16. Gutierrez, A. P., Falcon, L. A., Loew, W., Leipzig, P. A. 1975. An analysis of cotton production in California: A model for Acala cotton and the effects of defoliators on its yield. Environ. Entomol. 4:125-136.
17. Hafez, A. A. R., Stout, P. R., and DeVay, J. E. 1975. Rubidium tracing of potassium fertilizer uptake by cotton plants in relation to Verticillium wilt. Agron. J. 67:359-361.
18. Leslie, P. H. 1945. On the use of matrices in certain population mathematics. Biometrika 35:213-245.
19. Mathre, D. E. 1968. Photosynthetic activities of cotton plants infected with *Verticillium albo-atrum*. Phytopathology 58:137-141.
20. Misaghi, I. J., DeVay, J. E., and Duniway, J. M. 1978. Relationship between occlusion of xylem elements and disease symptoms in leaves of cotton plants infected with *Verticillium dahliae*. Can. J. Bot. 56:339-342.
21. Pullman, G. S. 1979. Effectiveness of soil solarization and soil flooding for control of soil-borne diseases of *Gossypium hirsutum* L. in relation to population dynamics of pathogens and mechanisms of propagule death. Ph.D. dissertation. University of California, Davis. 95 pp.
22. Pullman, G. S., and DeVay, J. E. 1982. Epidemiology of Verticillium wilt of cotton: a relationship between inoculum density and disease progression. Phytopathology 72:549-554.
23. Pullman, G. S., and DeVay, J. E. 1982. Epidemiology of Verticillium wilt of cotton: Effects of disease development on plant phenology and lint yield. Phytopathology 72:554-559.
24. Ranney, C. D. (editor). 1971. Verticillium wilt of cotton. Proc. Work Conf. Nat. Cotton Pathology Research Lab., College Station, TX. USDA/ARS Publ. ARS-S-19. 134 pp.
25. Schnathorst, W. C., and Mathre, D. E. 1966. Host range and differentiation of a severe form of *Verticillium albo-atrum* on cotton. Phytopathology 56:1155-1161.
26. Schnathorst, W. C., and Mathre, D. E. 1966. Cross-protection in cotton with strains of *Verticillium albo-atrum*. Phytopathology 56:1204-1209.
27. Turner, J. H., Smith, E. G., Garber, R. H., Williams, W. A., Yamada, H. 1972. Influence of certain rotations upon cotton production in the San Joaquin Valley. Agron. J. 64:543-546.
28. von Forester, H. 1959. Some remarks on changing populations. Pages 382–407 in: The Kinetics of Cellular Proliferation. F. Stohlman, Jr., ed. Grune and Stratton, New York. 456 pp.
29. Wang, Y., Gutierrez, A. P., Oster, G., and Daxl, R. 1977. A population model for plant growth and development: Coupling cotton-herbivore interaction. Can. Entomol. 109:1356-1374.
30. Wiese, M. V., and DeVay, J. E. 1979. Growth regulator changes in cotton associated with defoliation caused by *Verticillium albo-atrum*. Plant Physiol. 45:304-309.