Symposium: Estimating Yield Reduction of Major Food Crops of the World

Coupling Pests to Crop Growth Simulators to Predict Yield Reductions

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CLASSIFYING PEST EFFECTS ON CROP CARBON FLOW PROCESSES

There are several levels at which functions for pest populations and their effects on plants can be coupled to carbon flow processes in crop growth simulators. Pests may reduce inputs such as light, CO_2, and water; they may affect rates of metabolic and growth processes directly; or they may remove or consume previously produced assimilate or crop structural material (Fig. 1). Differentiation between direct and indirect effects of pests on carbon balance processes may be of value in the proper coupling of it to a crop growth simulator. Pests can be classified in the following categories based on the types of damage they do: stand reducers, photosynthetic rate reducers, leaf senescence accelerators, light stealers, assimilate sappers, tissue users, and turgor reducers. Of course, a single pest or pathogen can fall into more than one category.

Stand reducers. The effect of stand reducers such as damping-off fungi is to reduce plant biomass and number (ie, state variables). To simulate damage from stand reducers we need to know the number of plants lost, the time of loss, the approximate field distribution, and the capacity of the crop to compensate for missing plants in the stand.

Photosynthetic rate reducers. In contrast to foliage feeders or stand reducers, the photosynthetic rate reducers directly affect the rate of carbon uptake in the remaining host tissue. Mechanisms by which pathogens affect photosynthesis have been summarized by Buchanan et al (10). Viruses may reduce numbers of chloroplasts per unit of leaf area or alter chloroplast ultrastructure, electron transport, and partial reactions of photosynthesis. Fungi may alter chloroplast ultrastructure and certain components of the electron transport chain (10,28,29). Bacteria may also cause structural damage to chloroplasts (27).

In spite of subtly different mechanisms of damage by different pathogens, the end results are similar: CO_2 fixation per unit area is reduced. Depending on the type of crop growth model, it may be sufficient to enter the pathogenic effect on the photosynthetic light response curve of intact leaves or entire canopies. The light response curve can be defined by the parameters P_{max} and K_m, which are entered in the model as shown in Fig. 2. P_{max} and K_m are constants in the Michaelis-Menten photosynthetic light response curve defined as $P = P_{max} * PPFD/(K_m + PPFD)$, in which P = photosynthesis and $PPFD$ = photosynthetic photon flux density.

Leaf senescence accelerators. Some pathogens, such as *Cercospora* spp. on peanut (*Arachis hypogaea* L.), induce or accelerate senescence and abscission of leaves (8,23,32). Senescence accelerators remove leaf and petiole mass and thereby reduce light interception (see Fig. 1). To simulate this effect, the rates of senescence induced by pathogens at various levels of infection must be quantified and added to rates of senescence in response to N deficiency, leaf shading, and drought stress.

Light stealers. Weeds and some leaf pathogens have a "light stealing" effect on crops by absorbing photosynthetically active radiation. Pathogens that induce necrotic lesions not only stop carbon uptake in the affected spots, but also interfere with photosynthesis in other leaves by intercepting light before it reaches those leaves. Necrotic lesions constitute a loss of photosynthesizing tissue (a state variable change); when the necrotic tissue remains and affects light interception, that also must be considered in the crop simulator. Rabbinge and Rijsdijk (34) incorporated leaf coverage (shading) as one factor in the reduction of wheat growth and yield by powdery mildew.

Assimilate sappers. Plant pathogens, nematodes, and sucking

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insects may remove soluble assimilates from host cells. Haustoria produced by biotrophic fungi invade host cells and actively transport nutrients from the host cytoplasm across the haustorial plasma membranes (16). The plant is left with a reduced amount of assimilate to translocate and convert to growth. The carbon removal rates, which are a function of pest density and activity, must be quantified to simulate this effect.

Tissue consumers. Tissue consumers differ from assimilate sappers by acting on host material after the assimilate has been converted to host tissue. This difference is important because the plant must expend up to half of its originally produced carbohydates to produce plant tissue (31). The "tissue consumers," which include chewing insects and necrotrophic pathogens, could be further subdivided into foliage feeders, root feeders, and reproductive tissue feeders. Foliage-feeding chewing insects remove photosynthetic tissue without leaving the "light stealing" necrotic spots typical of foliar pathogens and without altering the effectiveness of the remaining leaf tissue (21,33). The minimum information needed to simulate defoliation effects is the amount of leaf mass consumed per unit land area and the approximate timing of leaf removal (35,43).

Turgor reducers. Root feeders such as nematodes, root-feeding insects, and root-rot pathogens can be referred to as "turgor reducers" because of their effects on plant water balance (Fig. 2). They also affect crop nutrient balance by disrupting phloem transport to roots which reduces the energy supply for active uptake of nutrients such as K and by disrupting passive flow of water and nutrients in the xylem by the eventual decay of that tissue (2,20,42). Vascular wilt pathogens such as species of *Pseudomonas, Fusarium*, and *Verticillium* are "turgor reducers" that act directly in the xylem. *Verticillium dahliae* on cotton caused effects characteristic of turgor reduction: reduced internode elongation, reduced dry matter accumulation in leaves and bolls, and defoliation (14). To model the effects of turgor-reducing pests requires not only the crop carbon flow, but also the crop water balance (44) and a root growth simulator similar to that in RHIZOS (25). Information is needed on reductions in root length density and root-stem conductivities caused by various levels of root feeders or wilt pathogens. We also need to quantify the degree to which increased carbon translocation to root growth can compensate for such pest damage or whether potential root length growing sites are also limited by the pests.

Ayers (2) reviewed another group of pathogens that affect gas phase resistance to leaf transpiration. Rusts, powdery mildews, and some virus diseases cause guard cells to malfunction resulting in greater resistance to CO_2 uptake by well-watered plants, but insufficient stomatal closure to restrict water loss when plants are drought-stressed. Rabbinge and Rijsdijk (34) simulated one aspect of stripe rust (*Puccinia striiformis*) damage to winter wheat (*Triticum aestivum*) as a "hole-making" effect which increased water loss.

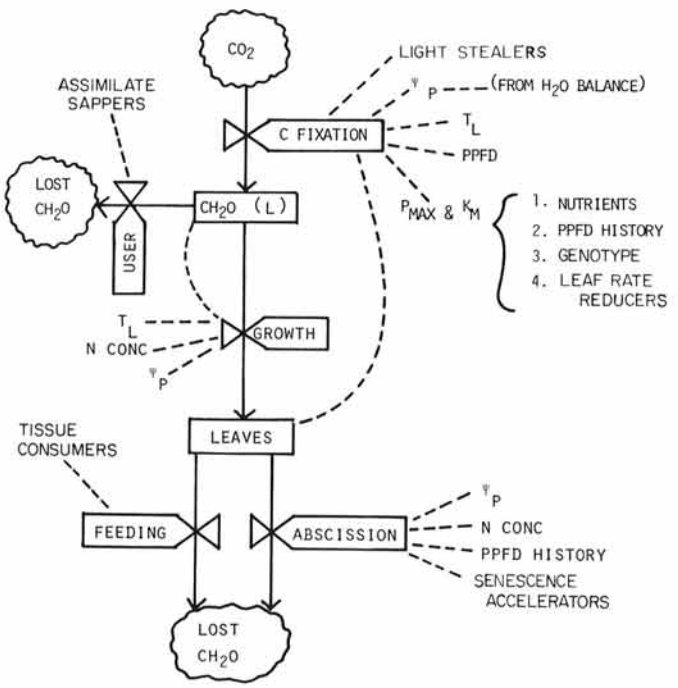

Fig. 1. A conceptual model of crop carbon fixation, leaf growth, and leaf abscission processes as affected by various pest categories and abiotic environment. T_L = leaf temperature, ψ_p = leaf turgor potential, $PPFD$ = photosynthetic photon flux density, P_{max} and K_m are Michaelis-Menten parameters defining leaf photosynthetic response to light ($PPFD$).

CROP MODEL FEATURES NEEDED TO COUPLE PEST DAMAGE

This section briefly describes features that should be incorporated in process-level crop simulators to include effects of pests on yield. For a more detailed discussion, see Boote (5).

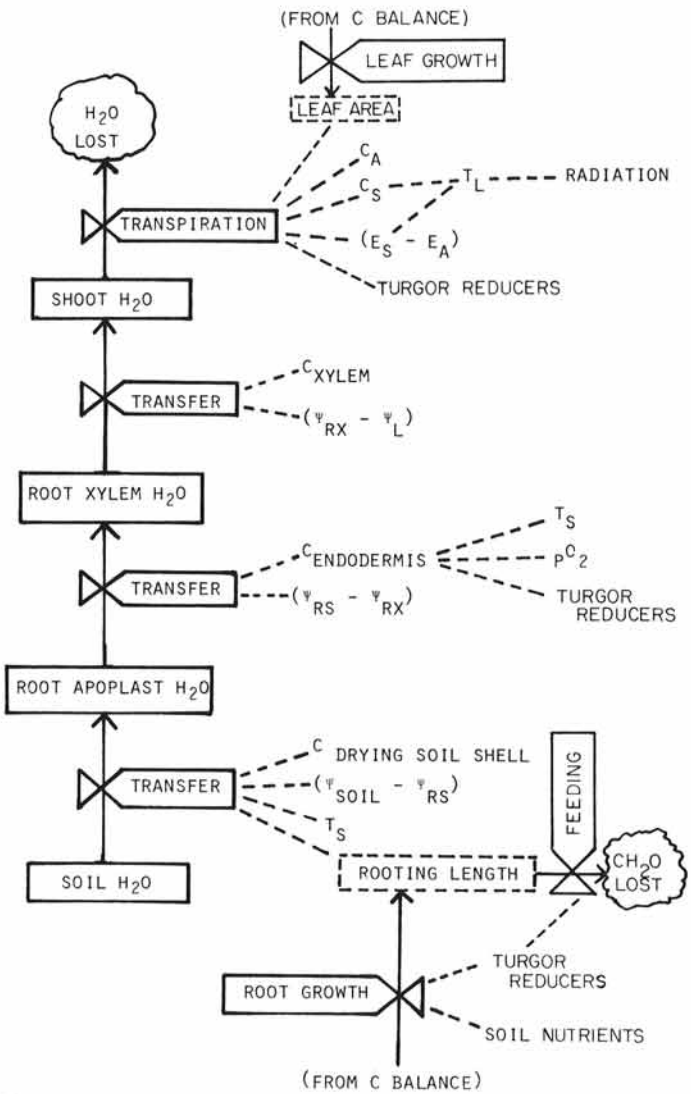

Fig. 2. A conceptual model of water flow processes in the soil-root-plant-atmosphere system as affected by turgor-reducing pests and abiotic environment. C_A and C_S are boundary and stomatal conductances, respectively, to water vapor efflux. C_{xylem}, $C_{endodermis}$, and $C_{drying\ soil\ shell}$ are the respective conductances of xylem, root endodermis, and soil shell about the root to liquid water flux. T_L = leaf temperature, E_S = saturation water vapor pressure in leaf at leaf temperature, E_A = ambient water vapor pressure of air around leaf, T_S = soil temperature, ψ_L, ψ_{RX}, ψ_{RS}, ψ_{soil} are the respective total water potentials of leaf, root xylem, root epidermis, and soil about the root.

Crop development versus growth. How rapidly the plant progresses through its life cycle is termed crop development rate. This differs from crop growth rate, which is the rate of mass accumulation. Crop development rate is governed primarily by temperature and daylength, but is relatively insensitive to absolute crop dry matter accumulation or growth processes (26). Crop development determines the duration of vegetative growth, the onset and duration of reproductive growth, and thus, the partitioning to various tissues which differ in respiratory costs for synthesis.

Pest damage and nutrient stresses have very minimal effects on rate of reproductive development, but can affect absolute growth rates and reduce such state variables as plant numbers and leaf, stem, and pod weights. The well-known sensitivity of certain crop growth stages to pest damage may result from the way the plant is partitioning carbon at the time of pest damage. For instance, the effect of a pest on seed yield will depend on whether it affects the carbon input rate going to vegetative growth or to reproductive yield. For these reasons, we agree with Rabbinge and Rijsdijk (34) that development rate is one of the most critical variables in a crop-growth simulator.

Carbon allocation, organogenesis, and fruit addition as controlled by crop development. We know little about the timing, extent, and causes of partitioning of assimilate for utilization at new growing sites. Changes in partitioning appear to follow visual changes in crop growth stage, which in turn can be predicted from crop development rate as a function of temperature and daylength. In our soybean modeling work, we (43) developed calendars of crop partitioning versus growth stage based on field data on partitioning relative to the crop's vegetative and reproductive stages.

During early growth, assimilate is partitioned only to roots, stems, and leaves. After given accumulations of crop development units, reproductive sites are initiated and vegetative node production slows or stops. The partitioning of assimilate to reproductive growth increases steadily from zero up to a given genetic limit during the transition from vegetative to reproductive growth. During this transition, fruits are added steadily to the limit of assimilate partitioned to them until a full fruit load is set when partitioning reaches its genetic potential. The time from zero to maximum partitioning is determined by crop development rate. The potential number of fruits carried at full fruit load can be estimated from total assimilate partitioned to them each day divided by assimilate required per fruit per day. Carbohydrate requirement per fruit per day is a cultivar characteristic as modified by temperature. Carbon costs for maintenance respiration and N assimilation are subtracted prior to determination of partitioning.

Respiratory costs for synthesizing plant or pathogen tissue. The conversion of simple sugars to complex tissue requires both a biosynthesis respiration cost and a carbon condensation cost depending on protein, lipid, and other biochemical constituents. Penning de Vries et al (31) calculated production values (grams of tissue produced per gram of glucose) for several types of tissue. The production value of an oilseed can be as low as 0.5 g/g. Assimilation of N is another important cost to include. In addition to biosynthesis cost, the crop also undergoes maintenance respiration just to keep existing tissues, proteins, membranes, etcetera in good repair (30).

The amount of crop production lost to a given pest depends on whether the pathogen uses recent assimilates (biotrophs) or structural plant tissues killed in advance of pathogen spread (necrotrophs). Secondly, the pathogen incurs respiratory costs to synthesize and to maintain its tissue regardless of its food source. Thirdly, the host plant respiration is increased in response to the infection (11). The increased portion of plant respiration may be associated with production of secondary metabolites, isoflavonoids, and lignin (24). Friend (15) suggested that some of these compounds are produced merely in response to any pathogen or wound, whereas others are truly phytoalexins which have fungitoxic effects. These phenolic and lignin compounds are expensive for the plants to make, requiring 1 g of glucose to make 0.465 g of lignin (31). The net effect is that for a given assimilate supply, diseased tissue may truly show a higher respiration rate from these two sources even though the total phytomass (crop plus pathogen) is less than in healthy plants.

Canopy light interception and photosynthesis. A light-interception photosynthesis submodel is needed to handle damage from several types of pests: stand reducers, foliage feeders, leaf photosynthetic rate reducers, leaf senescence accelerators, light stealers, and turgor reducers. An ideal light interception simulator would account for hedge rows, row skips, and several leaf layers to allow the use of different photosynthetic light response functions as affected by disease, age, nutrients, shade, turgor, or sudden increase in light to lower leaves (caused by foliage feeders on upper leaves). As shown in Fig. 1, the light response curves for leaf photosynthesis depend on leaf nutrients (7), approximate leaf age (41), previous light history (40), disease history (8), and water stress history (P. R. Harris, K. J. Boote, J. W. Mishoe, and J. M. Bennett, *unpublished*).

During early growth, crop plants have incomplete cover and form hedge rows having characteristically expanding heights and widths. Row spacing, plant population, row skips, and stand reductions from pests are important at this time. Various simulators of canopy light interception have considered hedge row effects (1,18). Since early vegetative growth is somewhat preprogrammed, one could simulate the development of plant height, plant width, and leaf area from which a simple hedge row envelope can be derived.

During early growth, plants have branching and tillering capacity which allows compensation for "thin" stands, but the capability to initiate branches is delayed until after a certain number of nodes have developed; it ends when all reproductive growth sites are initiated. This branching capability allows plants the flexibility to respond to plant population density, to stand reducers, and to foliage feeders.

An alternative to a detailed light interception simulator is needed because of the lack of experimental data on spatial patterns of pest damage and on the complexities involved with canopy geometry. Direct measurements of pest populations, canopy damage, and canopy photosynthesis can be used to develop empirical relationships between canopy disease rating, leaf area index, and canopy photosynthesis to be used in simulating the effects of varying disease levels on crop growth and yield. We have used such an empirical process description approach in the examples presented in this paper.

Leaf senescence. Leaf senescence should be included in a process-level simulator because senescence is increased by deficits of N, light, and water as well as by pathogens (Fig. 1). All of these four senescence accelerators reduce rate of leaf carbon uptake. One might simulate leaf abscission to occur when daily integrated leaf carbon exchange is negative over a sufficiently long period that leaf carbon reserves are depleted. Leaf abscission can also result from other factors such as ethylene produced by pathogens, as in leaf spot of peanut (23). Leaf senescence induced by a given pathogen can be coupled in the simulator by either entering percentage defoliation or coupling a given disease level to produce a given rate of leaf senescence.

Soil-root-plant water balance submodel. An adequate crop-pest simulator needs a good water balance submodel for several reasons. First, in most seasons drought causes more yield reduction than any given pest. Second, the turgor-reducing pests influence plant water balance and plant turgor as well as the nutrient balance which is related to rooting and water uptake (Fig. 2). Third, plant-absorbed radiation must be partitioned to latent heat loss and sensible heat loss as affected by water absorption by roots.

Root growth should be included in a process-level crop model because rooting density and rooting depth play important roles in water and nutrient uptake. Root and shoot growth establish a functional equilibrium based on the ability of each tissue to supply something that the other tissue needs (4). Shoots supply carbon to root growth, and roots, in turn, supply water and nutrients to shoots. The "equilibrium" root length density needed to support a given transpirational demand depends on temperature-limited root conductivity to water, soil water available in the root zone, and soil conductivity to water. If pests disturb this "equilibrium" by

reducing root length density or conductivity, then shoot turgor is reduced and shoot growth declines while more carbon "may" be allocated to compensatory root growth. The effect of nematode or pathogen damage on rooting extent vertically and horizontally away from the plant may require a two-dimensional soil profile model similar to that in RHIZOS (25). To simulate crop response, we need to quantify the effects of given levels of soilborne pests on root length density or root conductivities and the degree to which increased assimilate input to roots can overcome such pest damage. In this regard, the potential interaction between soil water supply and pest activity must be considered.

The expansion of plant leaves, stems, and pods is more sensitive to plant turgor than is leaf photosynthesis (9). Therefore, soilborne pests may reduce canopy expansion more than they reduce leaf photosynthesis. This subtle effect is important because of a feed-forward effect whereby reduced canopy expansion is reflected in reduced leaf area index, less light interception, and less photosynthesis.

Nutrient balance and remobilization. Nutrient balance is important to most seed-producing annuals because half or more of the N, P, and K accumulating in the seeds may have been taken up during vegetative growth and then remobilized from the vegetative parts during seed growth (19). Remobilization from vegetative parts is important during seed fill because seed demand for nutrients often exceeds the rate of uptake, especially for N (39). Foliage-feeding insects and leaf pathogens remove nutrients as they consume leaf mass, resulting in loss of remobilizable nutrients as well as reduction in photosynthesis.

Limited sink and feedback inhibition. Feedback inhibition on canopy photosynthesis may be a necessary feature in crop growth simulators to limit excessive, simulated leaf and stem growth under pest or environmental conditions that limit the number of fruit and seeds set. Reduced fruit or seed set may result from pests that prevent fertilization (eg, silk removal on corn) or that feed on pods or damage seeds. To simulate feedback inhibition, one would need to know the degree and duration of compensatory fruit addition that can occur and the amount of additional assimilate that leaf, stem, and root tissue can accept without inhibiting photosynthesis.

TABLE 1. Simulated soybean yield response[a] to nematode root pruning under drought or irrigation

Water treatment	Percent of "steady state" root length	Simulated yield (kg/ha)
Rainfed	100	1,412
	75	1,353
	50	1,179
Irrigated	100	3,908
	75	3,713
	50	2,766

[a] Simulations based on phenology of Cobb soybean and weather for 1981 at Gainesville, FL.

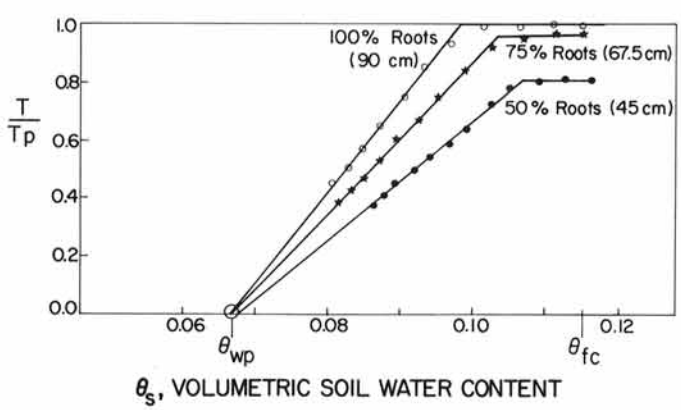

Fig. 3. Ratio of transpiration to potential transpiration (T/TP) as a function of volumetric soil water content (θ) of a sandy soil for three simulated root length densities at a common leaf area index. θ_{fc} and θ_{wp} are the soil water contents at field capacity and at the permanent wilting point, respectively.

SIMULATING NEMATODE REDUCTION OF SOYBEAN YIELD

We used two simulators (43,44) to determine the yield response to hypothetical reductions of 25 and 50% in the "steady state" ratio of root length to leaf area index caused by nematode feeding. With little definitive evidence, we assumed that healthy plants have a constant ratio of root length density to leaf area index (*LAI*). Increased carbon allocation to roots damaged by nematode feeding was considered insufficient to replace enough roots to offset the damage. The result would be a reduction in water uptake at a given soil water content, thereby reducing transpiration, photosynthesis, and growth. This does not assume any particular nematode population density; however, such information is needed to couple nematode and crop growth models.

We used the diurnal simulator of Zur and Jones (44) to simulate the photosynthetic and transpirational response to soil water given a "steady state" reduction in the ratio of root length density to *LAI*. The healthy crop situation was based on root length density, rooting depth, soil characteristics, and *LAI* of Williams soybean as reported in Jones et al (22). Fig. 3 shows the ratio of transpiration to potential transpiration (T/TP) as a function of soil water content for three root length situations (90 cm/cm^2 of soil area =

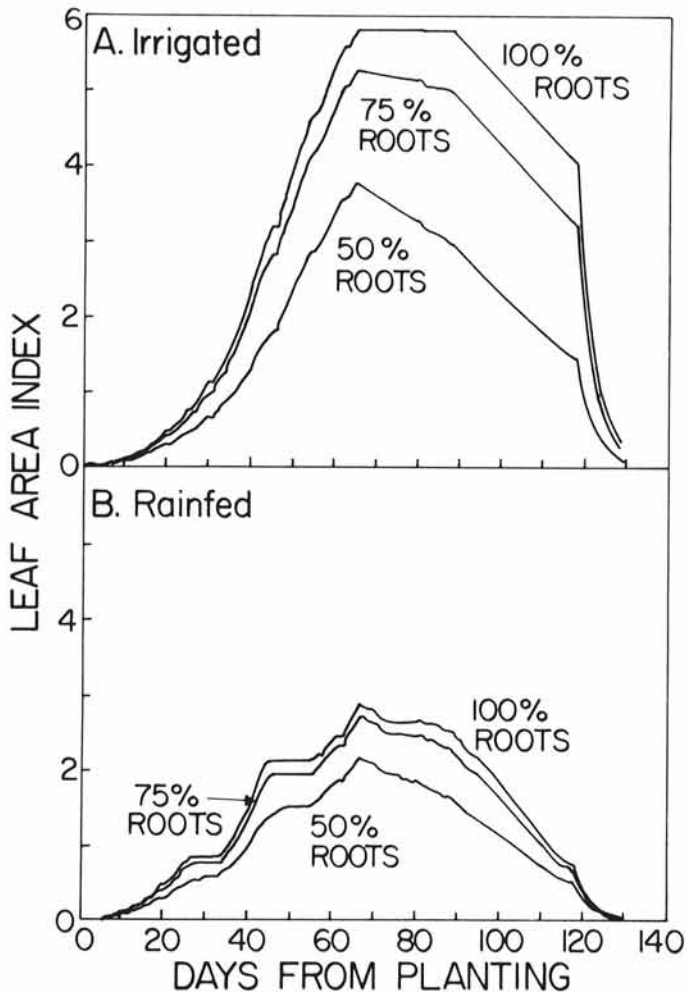

Fig. 4. Simulated leaf area growth of soybean in response to three "steady state" ratios of root length to leaf area index under: **A,** Irrigated or **B,** rainfed conditions. Simulations based on phenology of Cobb soybean and weather for 1981 at Gainesville, FL.

healthy; 67.5 cm/cm^2 = 25% reduction; 45 cm/cm^2 = 50% reduction). Photosynthesis was reduced proportionally to reductions in T/TP. Reducing root length density caused a lower T/TP at any given soil water content. The model predicted that for a 50% reduction in root length, even full irrigation would not eliminate effects of reduced root length.

The relationship shown in Fig. 3 represents a soil water factor (0 to 1.0 T/TP) that was entered in SOYGRO Version 4.2 (43) and multiplied times daily photosynthesis. SOYGRO is a process-level carbon balance model of season-long soybean growth operating on a daily time step (43). The daily photosynthetic function was developed from canopy photosynthetic light response data and has a presumed response to *LAI* similar to the relationship of dry matter increase versus *LAI* estimated by Shibles and Weber (37).

In Fig. 4, the effect of hypothetical nematode damage on leaf area growth is simulated in irrigated and rainfed situations. The rainfed situation represented a fairly severe drought year in Florida. The reduction in steady state rooting length decreased *LAI* in both irrigated and drought situations, especially for 50% lower root length density. In Table 1, the simulated yield response for three steady state reductions in rooting length is given for irrigated or rainfed situations. Surprisingly, irrigation could not offset the effects of simulated reductions in root length, especially at the 50% decrease in root length.

SIMULATING LEAF SPOT DAMAGE ON PEANUT PHOTOSYNTHESIS AND YIELD

We converted SOYGRO for use as a peanut model by changing phenology, partitioning, pod addition, tissue synthesis costs, and shell and seed growth rates and sizes. The conversion was fairly simple because peanut is also a leguminous oilseed crop that has growth stages similar to those of soybean (6). We also reevaluated the results from previous studies of effects of Cercospora leaf spot on canopy photosynthesis of peanut (8). Fig. 5 shows daily total photosynthesis versus daily moles of photosynthetic photon flux for three levels of infection by *Cercospora* on 91-day-old Florunner peanut canopies (developed from equations of Boote et al [8]). We hypothesized that disease effects on photosynthesis were mediated through: loss of *LAI* (senescence acceleration), self-shading of healthy leaf area by leaf spots (light stealing), and a toxic effect of leaf spot disease on the photosynthetic mechanism of the remaining leaves. Boote et al (8) did not measure *LAI*, but assumed it to be equal to 5.0 for control plants with low disease. This, with disease and defoliation ratings, allowed us to estimate healthy *LAI*, diseased *LAI*, and *LAI* lost to defoliation for the three leaf spot treatments (Table 2). First, we simulated the effect of reduction in healthy *LAI* by entering healthy *LAI* into the *LAI* equation of SOYGRO. The resulting value (0 to 1.0) was multiplied by the light response equation for the low leaf spot treatment. Only 33% of the photosynthetic reduction due to the severe leaf spot treatment could be accounted for by loss of healthy *LAI* per se.

Next we assumed that photosynthesis was also reduced because healthy *LAI* was randomly shaded by diseased nonphotosynthetic *LAI*. To account for this we calculated the *LAI* function twice: once with healthy *LAI* only and again with healthy plus diseased *LAI*. The value obtained for healthy *LAI* only was multiplied by the ratio of light extinction by healthy leaves to that of healthy plus diseased leaves. This effect accounted for another 17% of the total difference between the low and high leaf spot treatments. The remaining 51% of the reduction in photosynthesis was attributed to altered capacity (P_{max} and K_m) of remaining healthy leaf area in response to toxin effects and disruption of electron transport by *Cercospora* spp. The degree of reduction due to toxic effect is consistent with the mode of action of cercosporin toxin (12), and the failure of attempts to simulate leaf spot damage by artificial defoliation of a disease-free peanut canopy (17). Disease parameters (*K1* and *K2*) for the toxic effect versus fraction visible leaf spot (*DIS*) were derived by nonlinear regression on the data of Boote et al (8) in which:

$$PG=[(1.0-DIS*K1)\,P_{max}*PPFD]/[(1.0-DIS*K2)K_m+\mathrm{PPFD}]$$

Before solving for disease parameters, we corrected for the effects of *LAI* and self-shading. *PG* is apparent canopy carbon exchange rate in the light plus the absolute value of CO_2 efflux from the crop-soil-root system in darkness (8). This was done to estimate true photosynthetic uptake of CO_2.

Next, we simulated the seasonal peanut leaf area growth, vegetative growth, and fruit growth in response to a hypothetical moderate epidemic of Cercospora leaf spot. A moderate rate of leaf spot development and leaf defoliation due to *Cercospora* spp. was mimicked by using a Gompertz function:

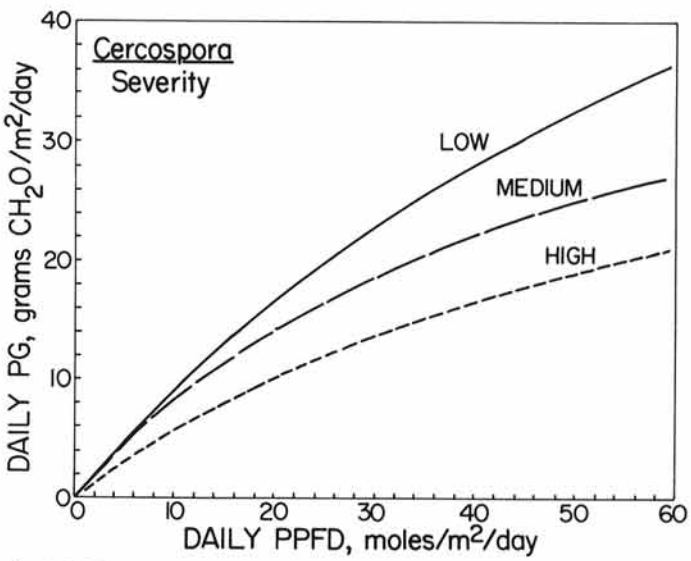

Fig. 5. Daily total photosynthesis (*PG*) versus daily photosynthetic photon flux density (*PPFD*) for low, medium, and high Cercospora leaf spot infection on Florunner peanuts at 91 days of age.

TABLE 2. Allocation of the effects of Cercospora leaf spot on canopy photosynthesis of peanut

Leaf spot	Leaf area index (*LAI*)[a]			Measured daily PG[b]	Simulated PG (grams $CH_2O \cdot m^{-2} \cdot day^{-1}$) corrected for:		
	Healthy	Diseased	Total		Healthy[c] *LAI*	*LAI* and self-shading[d]	*LAI*, self-shading and toxin effect[e]
Low	4.99	0.01	5.00	26.1	26.1	26.1	26.1
Medium	4.55	0.26	4.81	22.5	25.9	25.6	22.5
High	2.53	0.61	3.14	16.8	22.8	21.4	17.6

[a] Healthy and diseased *LAI* were calculated for the three treatments from percent disease and percent defoliation ratings, assuming an *LAI* = 5.0 for the low disease plants.

[b] The three canopy photosynthetic light response equations of Boote et al (8) were used with hourly radiation values measured over a 22-day period of summer weather. Canopy photosynthesis was integrated over each day and is presented as average daily photosynthesis.

[c] Total photosynthesis reduced only because of reduction in healthy *LAI*.

[d] Total photosynthesis resulting from reduction in healthy *LAI* plus light-stealing by leaf spots.

[e] Total photosynthesis resulting from reduction in healthy *LAI*, light-stealing by leaf spots, plus a toxic effect of *Cercospora* spp. on photosynthesis rate by healthy leaf area.

$$y = \exp(-B * \exp(-k(t - N)))$$

as defined by Berger (3). The development rate, k, was 0.10 with visible leaf spot (*DIS*) beginning on day 50 and visible defoliation on day 60. The defoliation rating was allowed to drive a leaf senescence routine in the crop simulator. Canopy photosynthesis was allowed to be affected, as previously described, by: loss of *LAI*, self-shading by leaf spots (*DIS*), and a toxic effect of leaf spot (*DIS*) on light response of healthy leaves.

Figs. 6 and 7 show that the simulated Cercospora leaf spot effect on *LAI*, vegetative growth, and reproductive yield were reasonable, although the effect is strictly hypothetical and needs to be tested against growth analyses and leaf spot assessment (similar to data of Elston et al [13]). The growth curve for a nondiseased peanut crop is realistic because the model was calibrated to fit data for disease-free peanuts (K. J. Boote, J. M. Bennett, and L. C. Hammond, [*unpublished*]). Validation of the peanut growth simulator will require further data from another year or location. The end-of-season simulated yield loss was 36% which, while substantial, is probably a conservative estimate of potential field losses from leaf spot on peanut (36,38).

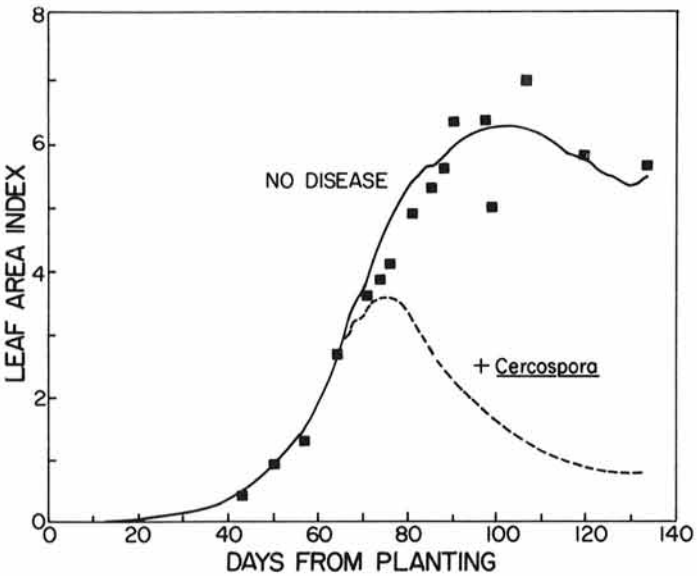

Fig. 6. Simulated leaf area index of Florunner peanut as affected by Cercospora leaf spot. The Cercospora epidemic was simulated by the following equation: $TOT\ DIS = \exp(-6.9 * \exp(-0.10 * (N - 50)))$ which caused visible spots to appear at 50 days. Percent defoliation due to leaf spot was derived from the same equation but keyed to start at 60 days. Symbols represent actual data points for a nondiseased crop in 1981 to which the nondisease simulation was calibrated.

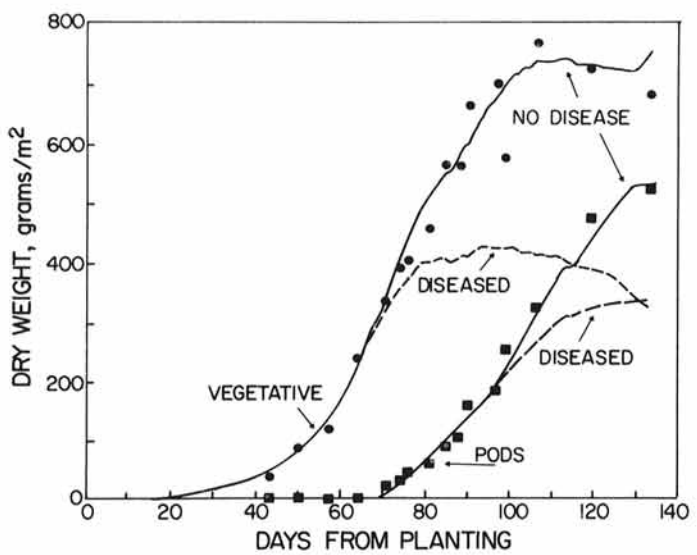

Fig. 7. Simulated vegetative and reproductive growth of Florunner peanut in response to Cercospora disease. Disease effects were mediated by loss of leaf area index, self-shading by leaf spots, and toxin effects on photosynthetic capability of remaining healthy leaf area. Symbols represent actual data points for a nondiseased crop in 1981 to which the nondisease simulation was calibrated.

CONCLUSION

Crop pests, including pathogens, can be presumed to affect crop carbon balance in ways that reduce yield. Our challenge is to discover the mechanisms by which this happens. In this paper we categorized pests on the basis of influence on carbon flow as light stealers, leaf rate reducers, leaf senescers, stand reducers, assimilate sappers, tissue users, and turgor reducers; some pests fall into more than one category.

The simulations in this paper demonstrate the utility of categorizing pest damage and emphasize the need for research to quantify the damaging effects on the crop. Process-oriented crop growth models provide a framework for integrating multiple effects of pests on crop growth and yield. As we learn more about the ways that pests affect crop processes, we will better understand how to predict yield reductions from pests and to design optimum pest management strategies.

LITERATURE CITED

1. Allen, L. H., Jr. 1974. Model of light penetration into a wide row crop. Agron. J. 66:41-47.
2. Ayres, P. G. 1981. Effects of disease on plant water relations. Pages 131-148 in: Effects of Disease on the Physiology of the Growing Plant. P. G. Ayres, ed. Cambridge University Press, Cambridge, England.
3. Berger, R. D. 1981. Comparison of the Gompertz and logistic equations to describe plant disease progress. Phytopathology 71:716-719.
4. Boote, K. J. 1976. Root:shoot relationships. Proc. Soil and Crop Sci. Soc. Fla. 36:15-23.
5. Boote, K. J. 1981. Concepts for modeling crop response to pest damage. Paper 81-4007. ASAE Summer Meeting, Orlando, FL. 24 pp.
6. Boote, K. J. 1982. Growth stages of peanut (*Arachis hypogaea* L.). Peanut Sci. 9:35-40.
7. Boote, K. J., Gallaher, R. N., Robertson, W. K., Hinson, K., and Hammond, L. C. 1978. Effect of foliar fertilization on photosynthesis, leaf nutrition, and yield of soybeans. Agron. J. 70:787-791.
8. Boote, K. J., Jones, J. W., Smerage, G. H., Barfield, C. S., and Berger, R. D. 1980. Photosynthesis of peanut canopies as affected by leaf spot and artificial defoliation. Agron. J. 72:247-252.
9. Boyer, J. S. 1970. Leaf enlargement and metabolic rates in corn, soybean, and sunflower at various leaf water potentials. Plant Physiol. 46:233-235.
10. Buchanan, B. B., Hutcheson, S. W., Magyarosy, A. C., and Montalbini, P. 1981. Photosynthesis in healthy and diseased plants. Pages 13-28 in: Effects of Disease on the Physiology of the Growing Plant. P. G. Ayres, ed. Cambridge University Press, Cambridge, England.
11. Daly, J. M. 1976. The carbon balance of diseased plants: changes in respiration, photosynthesis, and translocation. Pages 450-479 in: Encyclopedia of Plant Physiology, New Series, Vol. 4. Physiol. Plant Pathol. R. Heitefuss and P. H. Williams, eds. Springer-Verlag, Berlin.
12. Daub, M. E. 1982. Cercosporin, a photosensitizing toxin from *Cercospora* species. Phytopathology 72:370-374.
13. Elston, J., Harkness, C., and McDonald, D. 1976. The effects of Cercospora leaf disease on the growth of groundnuts (*Arachis hypogaea*) in Nigeria. Ann. Appl. Biol. 83:39-51.
14. Friebertshauser, G. E., and DeVay, J. E. 1982. Differential effects of the defoliating and nondefoliating pathotypes of *Verticillium dahliae* upon the growth and development of *Gossypium hirsutum*. Phytopathology 72:872-877.
15. Friend, J. 1981. Alterations in secondary metabolism. Pages 179-200 in: Effects of Disease on the Physiology of the Growing Plant. P. G. Ayres, ed. Cambridge University Press, Cambridge, England.
16. Gay, J. L., and Manners, J. M. 1981. Transport of host assimilates to the pathogen. Pages 85-100 in: Effects of Disease on the Physiology of the Growing Plant. P. G. Ayres, ed. Cambridge University Press, Cambridge, England.
17. Gerlagh, M., and Bokdam, M. 1973. La production de l'arachide en

fonction de la réduction de la photosynthèse par la cercosporiose ou ses simulations. Oléagineaux 28:347-350.
18. Goudriaan, J. 1977. Crop micrometeorology: A simulation study. PUDOC, Centre for Agricultural Publishing and Documentation, Wageningen, Netherlands.
19. Hanway, J. J., and Weber, C. R. 1971. N, P, and K percentages in soybean (*Glycine max* (L.) Merrill) plant parts. Agron. J. 63:286-290.
20. Hornby, D., and Fitt, B. D. L. 1981. Effects of root-infecting fungi on structure and function of cereal roots. Pages 101-130 in: Effects of Disease on the Physiology of the Growing Plant. P. G. Ayres, ed. Cambridge University Press, Cambridge, England.
21. Ingram, K. T., Herzog, D. C., Boote, K. J., Jones, J. W., and Barfield, C. S. 1981. Effects of defoliating pests on soybean canopy CO_2 exchange and reproductive growth. Crop Sci. 21:961-968.
22. Jones, J. W., Zur, B., Boote, K. J., and Hammond, L. C. 1982. Plant resistance to water flow in field soybeans: I. Non-limiting soil moisture. Agron. J. 74:92-98.
23. Ketring, D. L., and Melouk, H. A. 1982. Ethylene production and leaflet abscission of three peanut genotypes infected with *Cercospora arachidicola* Hori. Plant Physiol. 69:789-792.
24. Kosuge, T., and Kimpel, J. A. 1981. Energy use and metabolic regulation in plant-pathogen interactions. Pages 29-45 in: Effects of Disease on the Physiology of the Growing Plant. P. G. Ayres, ed. Cambridge University Press, Cambridge, England.
25. Lambert, J. R., Baker, D. N., and Phene, C. J. 1975. Simulation of soil processes under growing row crops. Paper 75-2580. Winter meeting ASAE, Chicago, IL. 12 pp.
26. Landsberg, J. J. 1977. Effects of weather on plant development. Pages 289-307 in: Environmental Effects on Crop Physiology. J. J. Landsberg and C. V. Cutting, eds. Academic Press, London.
27. Magyarosy, A. C., and Buchanan, B. B. 1975. Effect of bacterial infiltration on photosynthesis of bean leaves. Phytopathology 65:777-780.
28. Magyarosy, A. C., and Malkin, R. 1978. Effect of powdery mildew infection of sugar beet on the content of electron carriers in chloroplasts. Physiol. Plant Pathol. 13:183-188.
29. Magyarosy, A. C., Schurman, P., and Buchanan, B. B. 1976. Effect of powdery mildew infection on photosynthesis by leaves and chloroplasts of sugar beets. Plant Physiol. 57:486-489.
30. Penning de Vries, F. W. T. 1975. The cost of maintenance processes in plant cells. Ann. Bot. 39:77-92.
31. Penning de Vries, F. W. T., Brunsting, H. M., and van Laar, H. H. 1974. Products, requirements, and efficiency of biosynthesis: A quantitative approach. J. Theor. Biol. 45:339-377.
32. Plaut, J. L., and Berger, R. D. 1980. Development of *Cercosporidium personatum* in three peanut canopy layers. Peanut Sci. 7:46-49.
33. Poston, F. L., Pedigo, L. P., Pearce, R. B., and Hammond, R. B. 1976. Effects of artificial and insect defoliation on soybean net photosynthesis. J. Econ. Entomol. 69:109-111.
34. Rabbinge, R., and Rijsdijk, F. H. 1981. Disease and crop physiology: a modeler's point of view. Pages 201-220 in: Effects of Disease on the Physiology of the Growing Plant. P. G. Ayres, ed. Cambridge University Press, Cambridge, England.
35. Rudd, W. G. 1980. Simulation of insect damage to soybeans. Pages 547-555 in: Proceedings, World Soybean Research Conference II, Raleigh, NC. F. T. Corbin, ed. Westview Press, Boulder, CO.
36. Sanden, G. E., Knutson, A. E., and Gorbet, D. W. 1975. Effect of various fungicides on yield and on development of *Cercospora* leaf spot on peanuts. Proc. Soil and Crop Sci. Soc. Fla. 34:179-180.
37. Shibles, R. M., and Weber, C. R. 1965. Leaf area, solar radiation interception and dry matter production on soybeans. Crop Sci. 5:575-577.
38. Shokes, F. M., Gorbet, D. W., and Sanden, G. E. 1982. Effect of planting date and date of spray initiation on control of peanut leaf spots in Florida. Plant Dis. 66:574-575.
39. Sinclair, T. R., and de Wit, C. T. 1976. Analysis of the carbon and nitrogen limitations to soybean yield. Agron. J. 68:319-324.
40. Singh, M., Ogren, W. L., and Widholm, J. M. 1974. Photosynthetic characteristics of several C_3 and C_4 plant species grown under different light intensities. Crop Sci. 14:563-566.
41. Trachtenberg, C. H., and McCloud, D. E. 1976. Net photosynthesis of peanut leaves at varying light intensities and leaf ages. Proc. Soil and Crop Sci. Soc. Fla. 35:54-55.
42. Trudgill, D. L., Evans, K., and Parrott, D. M. 1975. Effects of potato cyst nematodes on potato plants. II. Effects on haulm size, concentration of nutrients in haulm tissue and tuber yield of a nematode resistant and a nematode susceptible potato variety. Nematologia 21:183-191.
43. Wilkerson, G. G., Jones, J. W., Boote, K. J., Ingram, K. T., and Mishoe, J. W. 1983. Modeling soybean growth for crop management. Trans. of ASAE 26:63-73.
44. Zur, B., and Jones, J. W. 1981. A model for the water relations, photosynthesis, and expansive growth of crops. Water Resources Res. 17:311-320.