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Modeling Crop Response to Growth Reducing Factors

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Summary

Pests, diseases, and weeds are growth reducing factors which affect the physiology of the crop in several ways. The final effect on yield can be quantitatively understood by evaluating the effects of the respective injury components and their interactions by means of simulations with comprehensive crop growth models. This approach elucidates the integrative physiology of the "stressed" crop. Moreover, it provides new insights that suggest ways for developing simple descriptive models which can be applied in crop loss assessment and warning systems. Examples are drawn from studies on fungal leaf diseases, the viral leaf disease beet yellows virus, and aphids.

Introduction

Yields of agricultural crops vary among different agricultural regions of the world and, within those regions, from year to year and location to location. To structure thinking about this variation in yield, three yield levels can be distinguished (*Figure 1;* de Wit & Penning de Vries, 1982; Rabbinge & de Wit, 1989): (1) the ***potential*** level, (2) the ***attainable*** level, and (3) the ***actual*** level.

Potential yields are attained with ample supply of water, nutrients, and other resources in the absence of weeds, pests, diseases, or other injurious factors. This situation is rare and may only be obtained in protected cultivation. The potential yield depends on site-specific abiotic conditions and crop physiological characteristics. Site parameters are sunshine profile over the year and over the day, temperature, humidity, CO_2 concentration, and physical soil properties. Major crop characteristics are phenology and architecture, assimilate allocation, and physiological mechanism of CO_2 binding (C3, C4, or CAM). Together these factors can be regarded as yield *defining* factors. Potential growth rates are in the order of magnitude of 25 g DM (dry matter) m^{-2} d^{-1} (= 250 kg DM ha^{-1} d^{-1}). Methods for simulating potential yields on the basis of defining factors are discussed by de Wit *et al.* (1978), Penning de Vries and van Laar (1982), and Penning de Vries *et al.* (1989). As radiation is often the dominant limiting resource for growth under optimal conditions, the growth rate also can be roughly estimated as 3.0 (μg DM J^{-1}) times the intercepted photosynthetically active radiation, expressed in J m^{-2} d^{-1} (Biscoe & Gallagher, 1977; Monteith, 1977; Russell *et al.,* 1989; Haverkort *et al.,* 1990).

The yield level decreases to the ***attainable*** level when one or more resources are not provided *ad libitum.* De Wit and Penning de Vries (1982) distinguish production situations with yield limitation by shortage of water, shortage of water + nitrogen, and shortage of water + nitrogen + phosphorus. Water loss through the stomata is an inevitable consequence of the uptake of the CO_2 needed for photosynthesis, and depends on incoming radiation, vapour pressure deficit of the air, and stomatal aperture. For each gram of dry matter assimilated in photosynthesis, about 150 to 300 g of water is evaporated. Such transpiration coefficients indicate that crops transpire 4 to 8 mm water per day to attain the potential growth rate. When less water is available, the stomata close such that the rate of photosynthesis is reduced. The high rates of leaf photosynthesis needed for potential yields can only be attained at elevated nitrogen concentrations in the leaf dry matter of ±6% (van Keulen *et al.,* 1989). Assuming an LAI of 4 m^2 (leaf) m^{-2} (ground) and a specific leaf area of 20 m^2 (leaf) kg^{-1} (leaf DM), 12 g m^{-2} of nitrogen would be needed only for the leaves to reach the potential growth rate. Such resource quantities of nitrogen are often not available. Shortages of resources *limits* growth rate. Of course, these calculations provide only "first" estimates. Methods for estimating crop growth under limiting availability of resources more precisely are discussed

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by van Keulen and Wolf (1986) and by Penning de Vries *et al.* (1989).

Due to pests, diseases, weeds, adverse conditions and pollutants, the attainable growth rate is seldom realized in practical agriculture. The rate of growth is *reduced* to the ***actual*** level. The size of the reduction depends on the ways in which the growth reducing factor affects the plant. Often a growth reducing factor has more than one effect on the plant, and the effect on final yield (damage) is the outcome of all different types of injury (injury components) and their interactions. Crop growth models can be of great help in understanding and quantifying the interactions between different injury components. This approach provides insight into the way in which and the amount by which growth is reduced in connection with growth defining and growth limiting factors and may lay a basis for simple management models (Rabbinge & Rijsdijk, 1981; Boote *et al.*, 1983; Rabbinge, 1986, 1988a,b; Rabbinge & Rossing, 1987, 1988; Rabbinge *et al.*, 1989, 1990). In this paper, some simulation studies of crop response to growth reducing factors from the Wageningen school of theoretical production ecology will be presented to illustrate the underlying principles and possible applications.

Injury Components

Damage is defined as any reduction in the quantity and/or quality of yield (Zadoks, 1985). *Injury* is any visible or measurable changes in the plant caused by a growth reducing factor. Damage can be split into components in the same way as yield. For instance, cereal aphid attack may decrease the *number* of kernels, the *weight* of kernels, and their *quality* (protein concentration). Damage is the final result of the effects of the injuries during a growing season on the physiology of the crop and the rate of growth. A growth reducing factor has seldom only one effect on the host. Often different types of injury are caused. Therefore, we can speak of *injury components.* Many effects from the molecular to the crop level can be found in the literature *(Table 1).* To understand the consequences of a growth reducing factor on crop growth, the effects on the physiology of the whole plant as part of the crop must be studied. On this integration level, four major functional systems can be distinguished: the economies of carbon, water, nutrients, and morphogenesis *(Table 2).* These systems are, of course, highly interdependent. Growth reducing factors interfere with all these systems, but most research has been conducted on the effects on the source term of the carbon balance — photosynthesis.

Fungal Leaf Pathogens

Introduction

Many important fungal leaf diseases are initiated by air-borne spores which land on the leaf surface and cause infection. A colony of mycelium develops locally in or on the leaf, disrupting normal leaf functioning and resulting in the development of visible symptoms. The effect on gas exchange by single leaves can be described with a simple equation which can be built into a crop growth simulation model to calculate effects of different diseases on the crop. In this approach, the physiological relations between the fungus and the host plant are neglected. Details on biochemical and leaf physiological details are given by Farrar and Lewis (1987). Here, we focus on the description of the effects on the integration level of the leaf, as outlined by Bastiaans (1990).

The Relation Between Disease Severity and Leaf Photosynthesis: A Model

The percentage of leaf area covered with lesions, the severity, is a function of the number of lesions and their size according to

$$s = 1 - (1 - \alpha)^N \approx 1 - e^{-N \cdot \alpha} \quad (1)$$

Here, s is severity (proportion) and α, a small number, is lesion size expressed as a proportion of leaf size. N is the number of lesions. The equation is valid when lesions are randomly distributed over the leaf (Justesen & Tammes, 1960). The effect of the disease on photosynthesis can be described with

$$\text{fraction photosynthesis reduction} = 1 - e^{-N.\alpha.\beta} \quad (2)$$

where the product $\alpha.\beta$ denotes the "influence area" of a lesion. In this influence area, the rate of photosynthesis is assumed to be 0 (Bastiaans, 1990). It follows that the relation between leaf photosynthesis and disease severity is given by

$$P_s = P_0 \times (1 - s)^{\beta} \quad (3)$$

or, establishing a linear relationship by taking logarithms

$$\ln (P_s) = \ln (P_0) + \beta \times \ln (1 - s) \quad (4)$$

where P_s is photosynthesis at severity s, P_0 is photosynthesis of a healthy leaf, and β is the ratio of the

influence area of a lesion and its visible surface. Though this description is developed from the idea of a lesion with surrounding leaf area with inhibited photosynthesis, its descriptive power also can be used in cases where such localized physiological lesions do not exist. To stress this fact, the influence area is called "virtual."

Leaf photosynthesis depends on many factors, of which incident light, ambient CO_2, and leaf water status are among the most important. The relation between photosynthesis and light is conveniently described with a negative exponential equation with three parameters (Goudriaan, 1982; *Figure 2*):

$$P_n = -R_d + (P_m + R_d) \cdot \left(1 - \exp\left(-\frac{\varepsilon \cdot H}{P_m + R_d}\right)\right) \quad (5)$$

where

- P_n = net rate of photosynthesis (= gross assimilation minus respiration)
- P_m = the asymptotic maximum net photosynthesis rate at light saturation
- ε = the initial slope of the photosynthesis-light response curve
- R_d = the rate of respiration (growth respiration + maintenance respiration)
- H = light intensity

Application of the β-Model

The β-model is now used to explore some published leaf injury-photosynthesis relationships. An extended version of Equation 3 *(see Appendix)* is used to describe relationships in which gas exchange at 100% severity deviates significantly from zero. The models were fitted to the data using the nonlinear least squares regression algorithm DUD (Ralston & Jennrich, 1979) implemented in the NLIN procedure of the SAS statistical software package.

Literature Data and Results

Rabbinge *et al.* (1985) determined the effect of mildew infection on carbon exchange parameters in winter wheat. The results with the fitted models are given in *Figure 3.* Parameter values are given in *Table 3.* In the two-parameter model, the effect on P_m is characterized by a β value of 5.8 ± 0.56 (SEM), indicating that the effect on leaf photosynthesis is significantly greater than can be explained by a loss of photosynthesis in the mildew-covered area alone. The effect of mildew on the initial slope of the photosynthesis light response curve is characterized by a β of 1.5 ± 0.37, not significantly different from the value of 1 that would reflect the absence of photosynthesis only in mildew-covered spots. Effects on R_d cannot be described with the two-parameter model because the function value at 100% severity deviates too much from zero.

Application of the three-parameter model to the wheat mildew data gives slightly better fits to the data. However, the accuracy of the parameter estimates, especially β, is strongly reduced. β estimates are much higher than in the two-parameter model, which indicates that the effect of the disease at low severities is greater than the two-parameter model suggests *(Figure 3).*

Other diseases for which leaf photosynthesis-severity relationships have been measured are wheat glume blotch *(Septoria nodorum)*, wheat brown rust *(Puccinia recondita)*, barley leaf blotch *(Rhynchosporium secalis)*, and peanut leafspot (caused by *Cercospora* spp.). For the three cereal diseases, the effect on P_m was established (Rooney, 1989; Spitters *et al.,* 1990; Martin, 1986). For *Cercospora* leafspot disease in peanut (Boote *et al.,* 1980), photosynthesis was measured at an arbitrary (undefined) light intensity. Data and fitted models are given in *Figure 4.* Parameter estimates are given in *Table 3.* The three cereal diseases appear to have β values that do not differ significantly from 1. This implies that their impact on leaf functioning may simply be regarded as a reduction of green area and a waste of the light that is intercepted by the lesions. For wheat brown rust, the non-deviation from 1 of β is confirmed in the three-parameter model, that can allow for the occurrence of a residual respiration in leaves that are 100% rusted. *Cercospora* leaf spot in peanut has a β value of 11 ± 3.5, indicating an effect on leaf photosynthesis that exceeds the proportion of leaf area occupied by lesions.

Sances *et al.* (1982) give an example of an injury-photosynthesis relationship for an invertebrate pest, avocado brown mite *(Figure 5).* As shown by the three-parameter model, the impact of mite injury is characterized by a β value not different from 1. Thus, the loss in photosynthetic rate is proportional to the leaf area injured. Injured leaves exhibit residual photosynthesis rates amounting to approximately 50% of those in healthy leaves. It is unclear whether the low photosynthesis rates given by Sances *et al.* are typical for avocado or consequences of sub-optimal conditions.

Discussion

The different β values for the effects on P_m and ε allow some speculations to be made on the physiological basis of the effect of mildew on wheat

photosynthesis. The large β values for the effect on P_m and the low value for the effect on ε are consistent with the hypothesis that closure of stomata (either homogeneously over the leaf or patchwise; Terashima *et al.,* 1988; Downton *et al.,* 1988) is a major physiological response of wheat to mildew infection. Such stomatal closure would limit photosynthesis at light saturation by decelerating the diffusion of CO_2 into the leaf. It might not have a significant effect on the photosynthesis at low light levels, as diffusion through partially closed stomata (in the case of homogeneous closure) or lateral diffusion (in the case of patchwise closure) might be sufficient to maintain the internal CO_2-concentration at the same level as in healthy leaves.

In the different examples, the three-parameter model generally gave the best fit. The two-parameter model provided more accurate parameter estimates, however, and it has the advantage of a simpler physiological interpretation.

Beet Yellows Virus: A Viral Leaf Pathogen

Unlike most fungal leaf diseases, viruses are generally systemic, as they are transported in the phloem (Matthews, 1981). Thus, the symptoms often cover whole leaf blades, rendering the equations described in the previous section unsuitable for the description of effects on photosynthesis. A well-studied example of the effects of a viral leaf disease on photosynthesis is beet yellows virus, one of the causal agents of virus yellows, a major disease of sugarbeet worldwide.

Beet yellows virus belongs to the closterovirus group and is transmitted by aphids in the semi-persistent manner (Bar-Joseph *et al.,* 1979). The principal vector is *Myzus persicae,* the green peach aphid. Upon transmission of the virus to a plant, the virus is multiplied in the inoculated leaf. After one or a few days, the virus is transported to the growing tissues, leaves, and roots (Bennett, 1960). Symptoms develop on the inoculated leaf and on the systemically infected leaves. Mature non-inoculated leaves do not become systemically infected (van der Werf *et al.,* 1989a), presumably because there is no or negligible phloem transport of virus to these assimilate-exporting leaves. Yellowing symptoms develop after an infected leaf is fullgrown (van der Werf *et al.,* 1989b). Until these symptoms appear, the rate of photosynthesis is not markedly affected. Thus, BYV-infected plants in the Dutch climate may have three distinct whorls of leaves: (1) an inner whorl of young leaves which are systemically infected but still green and photosynthetically active, (2) an outer whorl of healthy mature and old leaves that appeared before the plant became infected and which are photosynthetically active, and (3) an intermediate whorl of mature systemically infected leaves which are yellow and (almost) photosynthetically inactive (Hall & Loomis, 1972a,b; van der Werf, 1988).

Other viruses causing leaf yellowing symptoms in sugarbeet are beet mild yellowing virus and beet western yellows virus. Both viruses belong to the luteovirus group and are persistently transmitted by *Myzus persicae.* The effects of these viruses on the beet plant resemble those of beet yellows virus. Symptoms take somewhat longer to develop.

Models of the yield impact of these viruses are developed along two lines: (1) comprehensive, and (2) descriptive. The comprehensive modeling approach on the basis of injury components makes use of the SUCROS model, as described by Spitters *et al.* (1989a). Four injury components are quantified to calculate the effect of virus infection on the plant: (1) reduced leaf expansion, (2) increased scattering of incident light by yellow leaves, (3) reduced photosynthesis rate in yellow leaves at high and low light intensities, and (4) increased respiration in yellow leaves. These injury components were initially observed in beet yellows virus-infected plants, but are now also applied for simulating the growth of plants infected with beet mild yellowing virus.

The first injury component is presently built into the model by introducing measured leaf area indices as a forcing function *(Figure 6).* Injury component 2 is introduced by calculating a weighed average scattering coefficient for the whole leaf canopy on the basis of the observed proportions of green and yellow leaf area and measured scattering coefficients of 0.12 and 0.40, respectively (van der Werf, 1988). In the model, only two types of leaves are distinguished—green and yellow. For the green leaves, the leaf photosynthesis parameters of Spitters *et al.* (1989a) are used: P_m = 1.25 mg CO_2 m^{-2} s^{-1}; ε = 12.5 μg CO_2 J^{-1}. For yellow leaves (injury component 3), the parameter values are: P_m = 0.28 mg CO_2 m^{-2} s^{-1}; ε = 9.7 μg CO_2 J^{-1}. Increased respiration (injury component 4) is taken into account by assuming that yellow leaves exhibit 2.5 times higher maintenance respiration than normal healthy leaves. Details are given in van der Werf (1988).

Model predictions are compared with results of a field experiment in 1989 in which sugarbeet plants were infected with either beet yellows virus or beet

mild yellowing virus *(Figure 7)*. The infection was made in the cotyledon stage. The model slightly underestimates the real production figures. Differences in yield among the three treatments are, however, fairly well described by the model. An earlier sensitivity analysis of the model (Rabbinge *et al.*, 1990; *Table 4*) showed that the reduction of photosynthetic capability of the yellow leaves is the principal injury component, explaining 70% of the yield reduction.

The descriptive model is based on the overruling importance of the reduction of photosynthesis in the yellow leaves as demonstrated by the comprehensive model. Photosynthesis in the yellow leaves is neglected. By weekly field observations with a grid, the percentage soil cover by green leaves was determined *(Figure 8)*, and the integral of photosynthetically active radiation (PAR) intercepted on green leaves was calculated from these data and meteorological figures from a nearby weather station. In *Figure 9*, total biomass during the season is plotted against the cumulated PAR interception on the green leaves. Data points for the three treatments are described by production efficiencies of 1.4 to 1.7 μg (DM) J^{-1} (intercepted PAR), indicating that this simplifying approach gives a fair first estimate of yield loss due to virus yellows, caused by beet yellows virus or beet mild yellowing virus. However, production efficiencies for beet yellows virus-infected plots seem to be lower than for control or beet mild yellowing virus-infected plants. Thus, not all the variation is explained.

Perspective

Modeling with injury components provides a tool for calculating the implications of measurements at leaf level for the crop as a whole. Thus, the gap between plant physiology and crop science is bridged. Sensitivity analyses may indicate principal injury mechanisms and the model may be used for assessing the effects of crop husbandry and virus control measures. The light interception approach seems to provide a tool for determining yield reduction at the field level.

Aphids

Aphids injure their host plants through several mechanisms (Miles, 1989a,b). The three most important are: (1) consumption of sugars, amino acids, and other phloem constituents; (2) leaf coverage with honeydew; and (3) injection of physiologically active substances, toxins, or growth regulators.

Groenendijk *et al.* (1990) simulate the effect of the black bean aphid, *Aphis fabae,* on growth of young sugarbeet plants by quantifying the withdrawal of sugars by the aphid population *(Figure 10)*. The model assumes an assimilate requirement of aphids of 1.6 mg (sugar) mg^{-1} (aphid dry weight) d^{-1}. Photosynthesis is calculated on the basis of simulated leaf area, incident radiation, and measured photosynthesis parameters. Injury component 2 (honeydew) was not included in the model because measurements by Hurej and van der Werf (unpublished) did not demonstrate photosynthesis inhibition by honeydew. Injury component 3 was also neglected because no data demonstrating toxic effects have been published. Simulation results correspond well with actual yield data *(Figure 11)*. This result supports the hypothesis that assimilate consumption is the most important injury component.

Sensitivity analysis of the model (unpublished) shows that the timing of infestation is crucial for the effect of the aphids on the plant. When aphid infestation is late, the sugar drain due to aphid feeding is insignificant as compared to the assimilation rate, such that the effect on daily production and leaf growth is negligible. When aphid infestation begins in an early growth phase of the plant, assimilate withdrawal constitutes a significant drain and causes a marked decrease in the daily relative growth of the plant. Thus, in an experiment in which the aphids were introduced when sugarbeet seedlings were in the two-leaf stafe, the relative growth rate decreased from .018 d^{-1} in control plants to 0.14 d^{-1} in aphid-infested plants (Groenendijk *et al.*, 1990). Due to the positive feedback between growth and light interception, these daily growth reductions culminate in a reduction in weight from 30 to 9 g after four weeks. Thus, insight is obtained that may help in establishing economic thresholds for this aphid.

Rossing (1991a,b) simulates the effects of the cereal aphid, *Sitobion avenae,* in winter wheat, incorporating injury components 1 and 2 according to measurements of Rossing and van de Wiel (1990). He quantifies both the consumption of sugars, *ca* 1.6 mg (sugar) mg^{-1} (aphid dry weight) d^{-1}, and the consumption of nitrogen via the amino acids in the phloem sap: *ca* 30 μg (N) mg^{-1} (aphid dry weight) d^{-1}. Consumption of sugars affects the growth of the kernels directly through reduction of the amount of assimilates available for growth. Two alternative possibilities are evaluated for the quantification of the effects of consumption of nitrogen:

(1) The aphids have priority over the kernels in the acquisition of the available nitrogen, the amount

of which does not change as compared to the aphid-free situation. Thus, the rate of kernel growth is reduced by lack of sugars and nitrogen from the onset of the aphid infestation.

(2) The nitrogen demand by the aphids accelerates the redistribution of nitrogen from leaves. Thus, kernel growth is initially not hampered; but later on, rates of growth fall below those of the aphid-free simulation because leaf senescence has been accelerated.

It is not known which of these hypotheses best reflects the real situation. Fortunately, the different hypotheses have only slight consequences for the ultimate effect of the aphids on the yield. Measured effects of honeydew on leaf photosynthesis parameters are also incorporated in the model. Based on the simulation model, a descriptive model for yield loss as a function of aphid density is derived which is as accurate as the best empirical model published in the literature. Moreover, the modeling study provides dynamic relationships between aphid load and effects on crop growth that can fine-tune existing supervised control decision rules for cereal aphid control (EPIPRE; Drenth *et al.*, 1989). Such fine-tuning is only possible with detailed dynamic simulation models.

Conclusion

This paper gives an arbitrary selection of simulation studies on pests and diseases as yield reducing factors. Methodologically similar studies were made on the air pollutant SO_2 (Kropff & Goudriaan, 1989; Kropff, 1990), groundnut rust (Savary *et al.*, 1990), and weeds (Kropff, 1988; Spitters, 1989; Spitters *et al.*, 1989b; Kropff & Spitters, 1990). Other studies are in progress. The aim of this type of work is to provide better quantitative insight concerning the effects of growth reducing factors on the physiology and production of crops in interplay with growth defining and growth limiting factors. Such insight is needed for rationalizing pesticide usage against growth reducing factors of biotic origin, such that the productivity of crops can be maintained with a minimum of side effects on the environment.

Most studies on the physiology of plant-pathogen interactions are made on the molecular and biochemical integration level (reviews in Pegg & Ayres, 1987). On the higher integration level of the whole plant, the research effort is considerably smaller. Little attention has been paid, for instance, to the relation between disease severity and leaf CO_2 exchange parameters. In only one publication (Rabbinge *et al.*, 1985) are all three parameters describing the photosynthesis response on light quantified. The other publications study the effect on only one of these parameters and therefore give insufficient background for making photosynthesis calculations for field conditions. This lack of knowledge is one of the reasons for the current practice of risk-avoiding insurance-sprays.

Simulation studies based on quantified injury components provide a crop-physiological basis for the development of practical models such as linear relationships between intercepted radiation and yield (Waggoner & Berger, 1987; Waggoner, 1990; Spitters, 1990). The β-model (§3) may describe disease-host relationships on the leaf level and provide a useful extra disease-specific parameter in these production efficiency models. β values of 1 suggest that the effect of a disease may be described with the simplest type of model, neglecting radiation intercepted on lesions, provided that the vertical disease profile is more or less homogeneous.

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Appendix: Basis of the Three-Parameter Model

Assume a leaf with N randomly distributed lesions with visible relative size α and virtual influence area $\alpha.\beta$. Photosynthesis (or another gas exchange parameter) of the healthy leaf area is P_0 and the value for the influence area is P_a. In the two-parameter model, P_a is 0. Severity is again $s = 1 - e^{-N \cdot \alpha}$ and the area in which gas exchange is affected by the disease covers again a proportion, $1 - e^{-N \cdot \alpha \cdot \beta} = 1 - (1 - s)^{\beta}$, of total leaf area while the healthy leaf area covers a proportion $(1 - s)^{\beta}$. It follows that the gas exchange, averaged over the leaf area is now:

$$P_s = P_0 \cdot (1 - s)^{\beta} + P_a \cdot \{1 - (1 - s)^{\beta}\}.$$

Thus, $P_s = P_a + (P_0 - P_a) \cdot (1 - s)^{\beta}$.

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TABLE 1. Effects of growth reducing organisms on plants.

Organisms	Effects
Fungi	Withdrawal of materials from the host, creation of holes in the leaf surface, increasing water loss, reduction of light absorption through myselium development on the leaf surface, disruption of host tissue integrity by excretion of lytic enzymes, excretion of physiologically active substances acting as toxins or hormones (Williams, 1979).
Bacteria	Tissue degradation, alteration of membrane permeability, obstruction of water movement in xylem, excretion of toxins (Kelman, 1979).
Viruses	Disruption of hormonal balances, reduction of rate of photosynthesis, interference with phloem translocation of sugars, increase of rate of respiration, inhibition of leaf expansion (Matthews, 1981; van der Werf, 1988).
Leaf-eating insects	Reduction of leaf area, creation of entry ports for leaf pathogens.
Aphids	Withdrawal of sugars and amino acids, injection of physiologically active compounds with the saliva, excretion of sugary honeydew on leaf surface resulting in pathogen stimulation, sealing of stomata and coverage of leaf surface with light intercepting black moulds, reduction of rate of photosynthesis (Wood *et al.*, 1988; Miles 1989a,b; Rossing & van de Wiel, 1990).
Mites	Mechanical damage due to punctures, removal of cell contents, closure of stomata due to disfunctioning of guard cells, injection of physiologically active substances with the saliva, reduction of rate of photosynthesis (Tomczyk & Kropczynska, 1985).
Nematodes	Removal of cell contents, injection of physiologically active excretions, induction of giant cells, cell wall dissolution, reduction of rate of photosynthesis, disruption of hormone production in root tips, tissue destruction, hampering uptake of water and nutrients by roots, creation of entry ports for root pathogens (Dropkin, 1979; Wallace, 1987; Melakeberhan *et al.*, 1988).

TABLE 2. Principal processes in plants which can be affected by growth reducing factors.

PROCESSES	GROWTH REDUCING FACTORS
CARBON AND ENERGY ECONOMY	
Photosynthesis	
Light interception & distribution	Weeds, necrotrophic fungi
CO_2 diffusion	
CO_2 binding	Many fungal & viral leaf pathogens
Other chloroplast processes	
Respiration	
Maintenance	Many fungal & viral leaf pathogens
New syntheses (growth)	
Photorespiration	Leaf diseases
Allocation	
Transport	
WATER ECONOMY	
Uptake	Root pathogens, nematodes
Transport	Vascular wilt diseases *(Verticillium, Fusarium)*
Transpiration, stomatal regulation	Leaf diseases
NUTRIENT ECONOMY	
Uptake	Root pathogens, nematodes
Transport	Vascular wilt diseases *(Verticillium, Fusarium)*
Redistribution	Leaf diseases
MORPHOGENESIS	
Organ initiation	Mycoplasmas, galling aphids
Organ growth	Most growth reducing agents (per def.)

TABLE 3. Characterization of published relations between diseased (injured) leaf area & leaf photosynthesis rate w/the β-model.

Leaf area disease	Parameter	Two- (one-) parameter model			Three-parameter model				n
		P_0*	β	R^2	P_0*	P_a**	β	R^2	
Wheat powdery mildew[1]	P_m (mg CO_2 $m^{-2}s^{-1}$)	1.11 ± 0.021	5.8 ± 0.56	0.63	1.17 ± 0.022	0.56 ± 0.045	25.0 ± 4.8	0.73	95
	ε (μg CO_2 J^{-1})	7.4 ± 0.14	1.5 ± 0.37	0.23	7.6 ± 0.19	6.0 ± 0.48	19.0 ± 13.1	0.28	64
	R_d (mg CO_2 $m^{-2}s^{-1}$)	not applied			0.036 ± 0.0013	0.049 ± 0.0026	25.0 ± 12.4	0.34	76
Wheat glume blotch[2]	P_m (proportion)	1 (fixed)	1.66 ± 0.34	0.55	not applied				14
Wheat brown rust[3]	P_m (mg CO_2 $m^{-2}s^{-1}$)	0.51 ± 0.020	1.26 ± 0.16	0.77	0.51 ± 0.020	-0.08 ± 0.066	0.95 ± 0.24	0.78	68
Barley leaf blotch[4]	P_m (proportion)	1 (fixed)	2.1 ± 0.61	0.68	not applied				12
Peanut leafspot[5]	P_n (proportion)	0.92 ± 0.10	11.0 ± 3.5	0.88	0.99 ± 0.058	0.25 ± 0.056	27.0 ± 8.3	0.98	5
Avocado brown mite[6]	P_n (mg CO_2 $m^{-2}s^{-1}$)	not applied			0.21 ± 0.020	0.10 ± 0.014	1.3 ± 0.7	0.96	4

*P_0 is the photosynthesis parameter at severity 0; **P_a is the parameter value at 100% severity.
[1]*Erysiphe graminis* (Rabbinge *et al.*, 1985); [2]*Septoria nodorum* (Rooney, 1989); [3]*Puccinia recondita* (Spitters *et al.*, 1990);
[4]*Rhynchosporium secalis* (Martin, 1986); [5]*Cercospora* spp. (Boote *et al.*, 1980); [6]*Oligonychus punicae* (Sances *et al.*, 1982).

TABLE 4. Simulated relative contribution of components of injury by beet yellows virus in sugarbeet field experiment, 1986.

Damage components	Early infection		Late infection	
	Yield (%)	% damage*	Yield (%)	% damage*
No disease	100.0		100.0	
		7.1		1.5
1 (reduced leaf area index)	92.9		98.5	
		5.2		0.2
1+2 (reduced light absorption)	88.0		98.2	
		36.4		1.7
1+2+3 (reduced photosyn., ε, Am)	56.0		96.6	
		11.7		0.5
1+2+3+4 (increased respiration)	49.4		96.1	
Measured	48.2±2.5		93.4±5.1	

*% damage is the damage (%) calculated by incorporating the injury components one-by-one in the model:

Early infection: June 5, 1986 7 leaves LAI = 0.1
Late infection: July 17, 1986 21 leaves LAI = 5.1

More details are given in van der Werf (1988) and in Rabbinge *et al.* (1990).

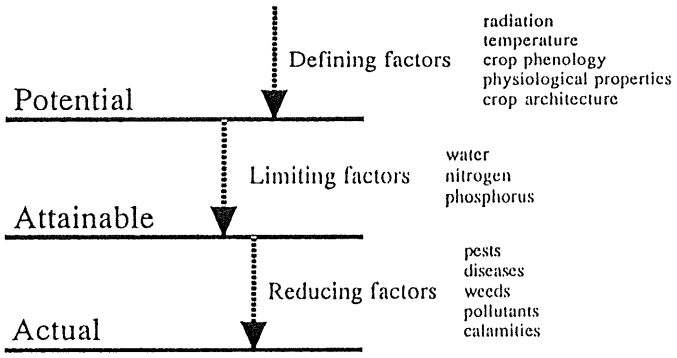


FIGURE 1. Crop production levels.

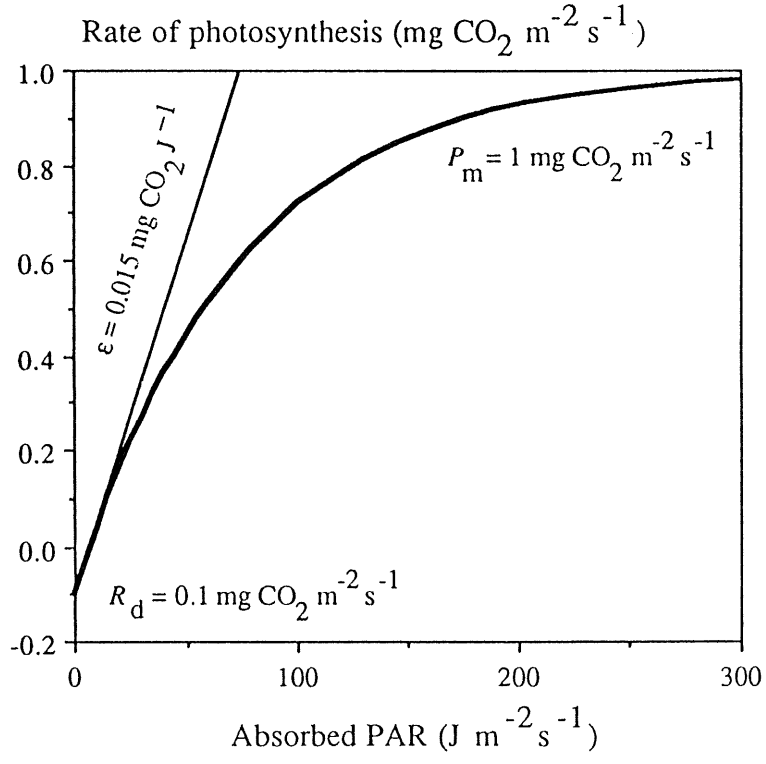
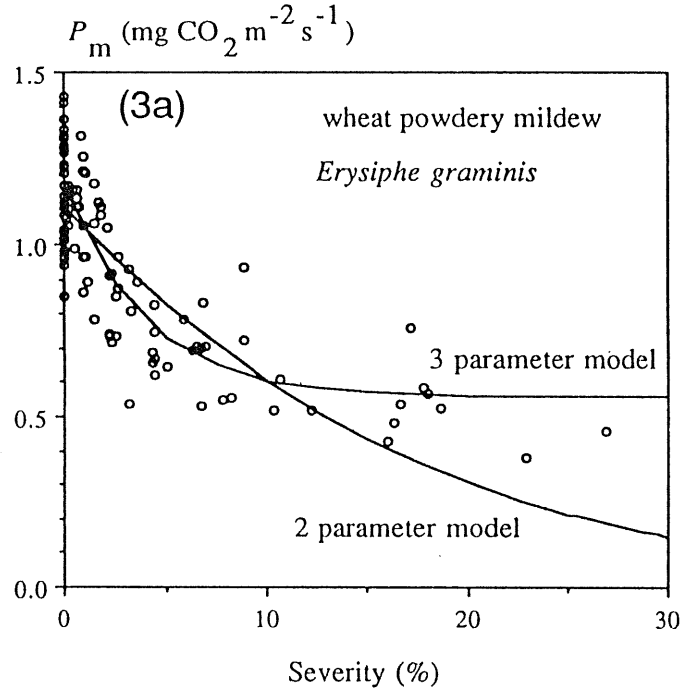
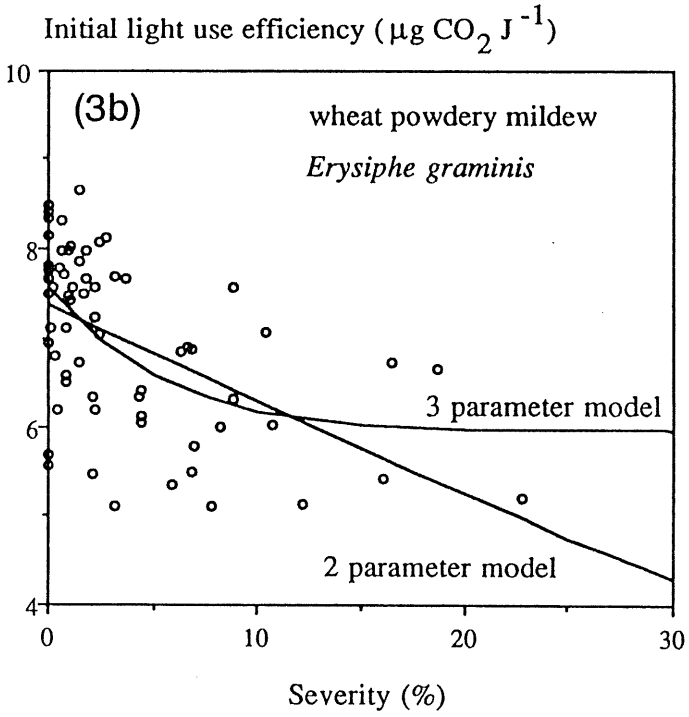


FIGURE 2. Negative exponential equation for describing the photosynthesis light response curve of leaves (Equation 5).

FIGURE 3. Photosynthesis parameters of winter wheat leaves as affected by infection with powdery mildew, *Erysiphe graminis*. (Data from Rabbinge *et al.*, 1985.)





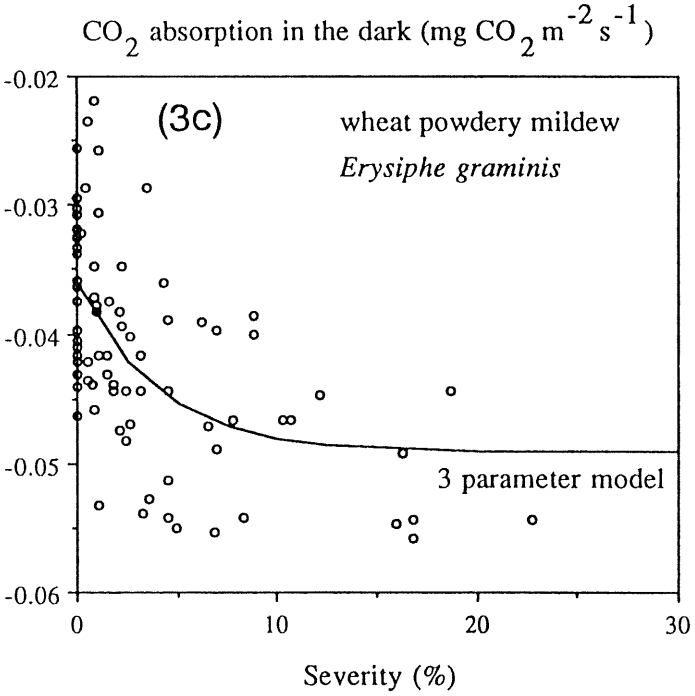
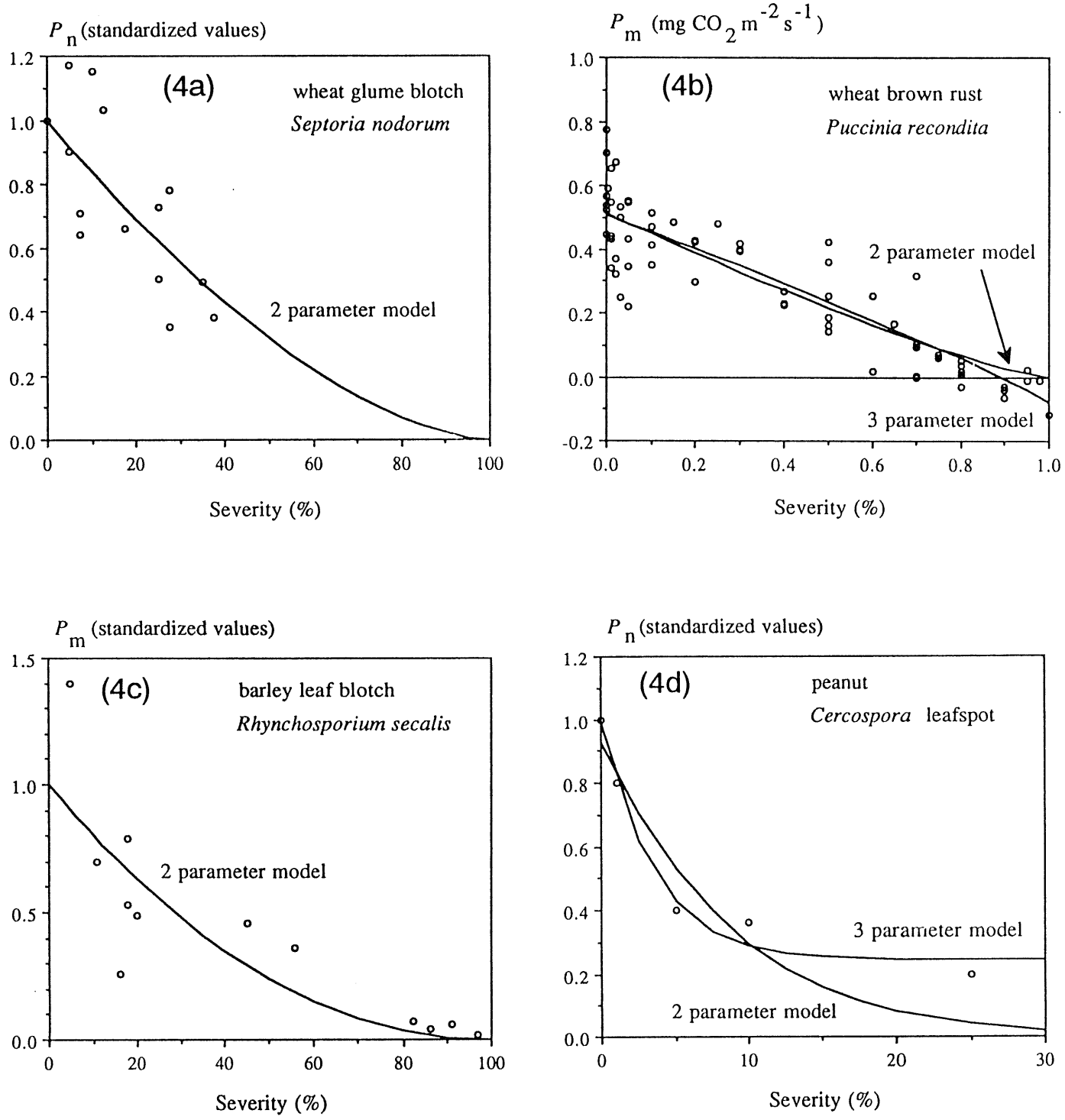


FIGURE 4. Leaf photosynthesis as affected by infection with foliar pathogens in four pathosystems: wheat glume blotch, *Septoria nodorum* (data from Rooney, 1989); wheat brown rust, *Puccinia recondita* (data from Spitters *et al.*, 1990); barley leaf blotch, *Rhynchosporium secalis* (data from Martin, 1986); and peanut leafspot, *Cercospora* spp. (data from Boote *et al.*, 1980).



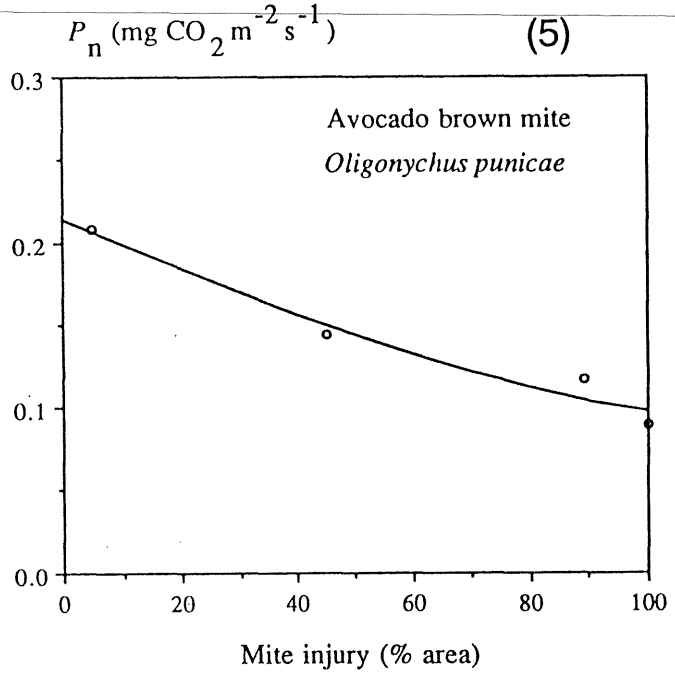


FIGURE 5. Leaf photosynthesis in avocado as affected by feeding injury by avocado brown mite, *Oligonychus punicae*. (Data from Sances *et al.*, 1982.)

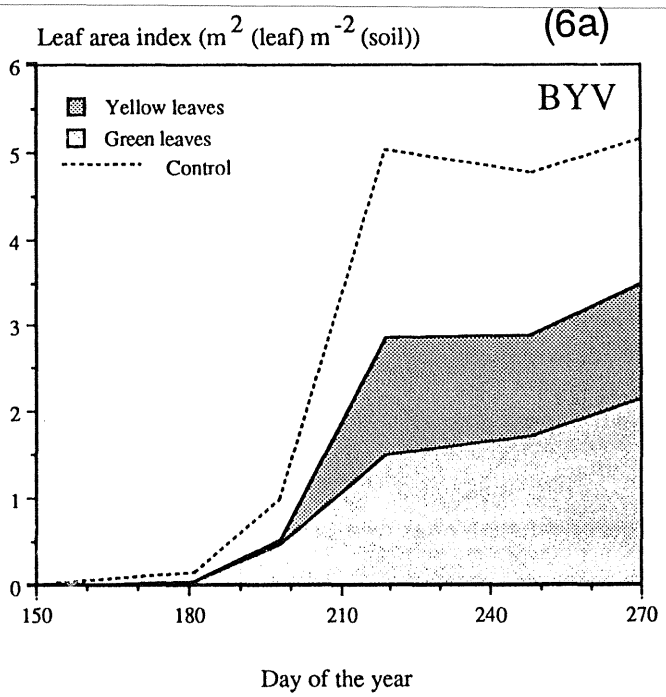
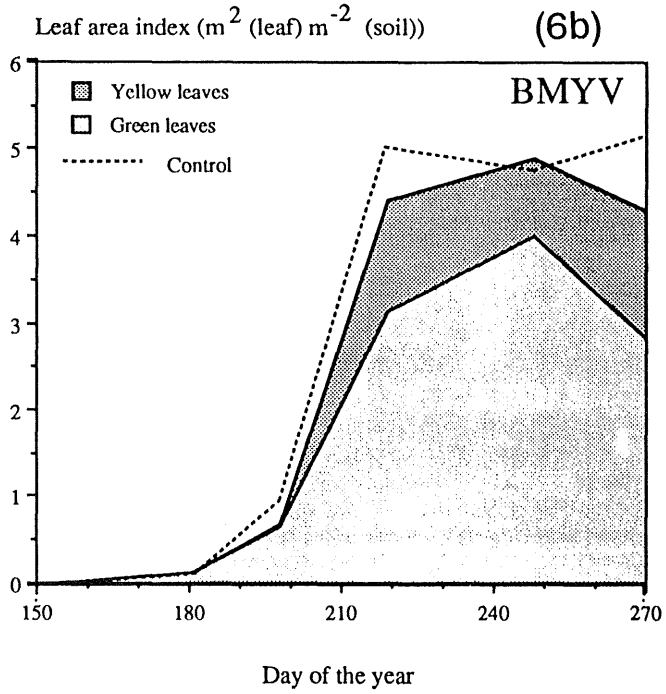


FIGURE 6. Observed course of leaf area index in sugarbeet field experiment, 1989.



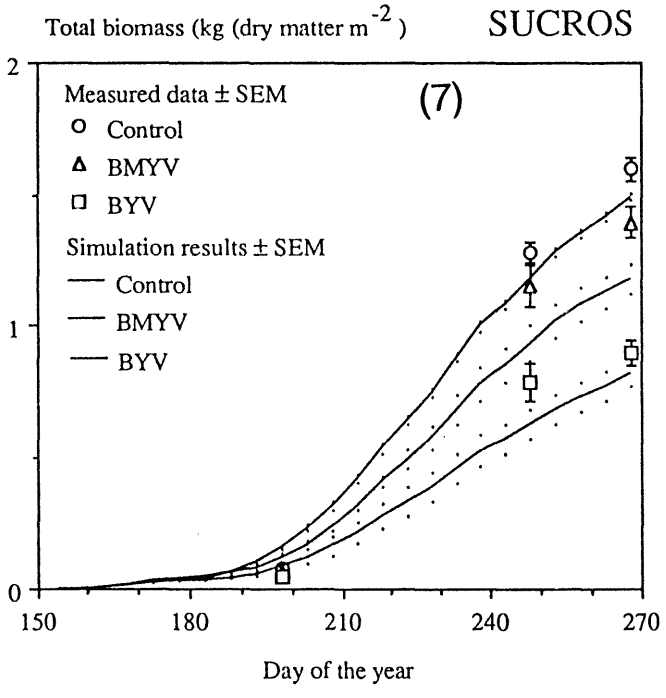
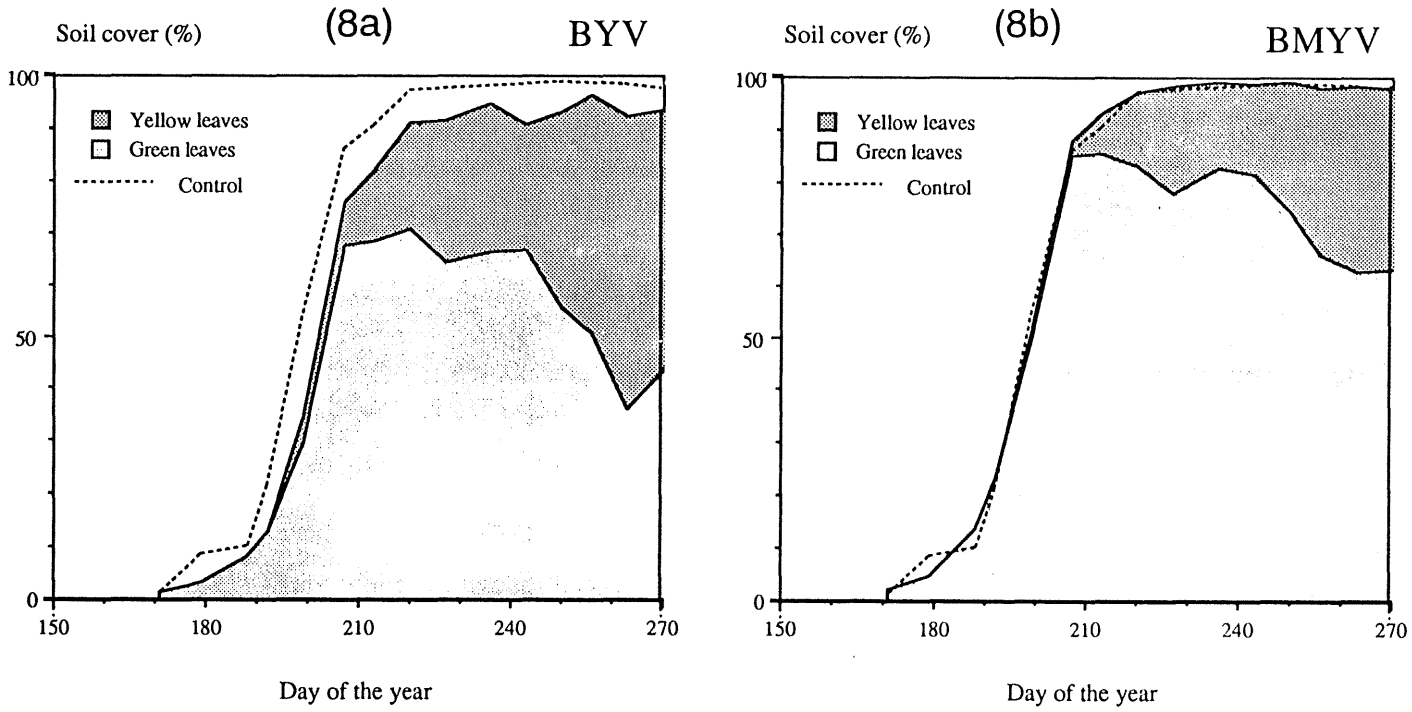


FIGURE 7. Comparison of observed sugarbeet growth in 1989 with calculations by the comprehensive model SUCROS. The standard error of the simulation results (indicated by dots) was obtained by running the model with LAI data from single experimental plots.

FIGURE 8. Observed course of soil cover in sugarbeet field experiment, 1989.



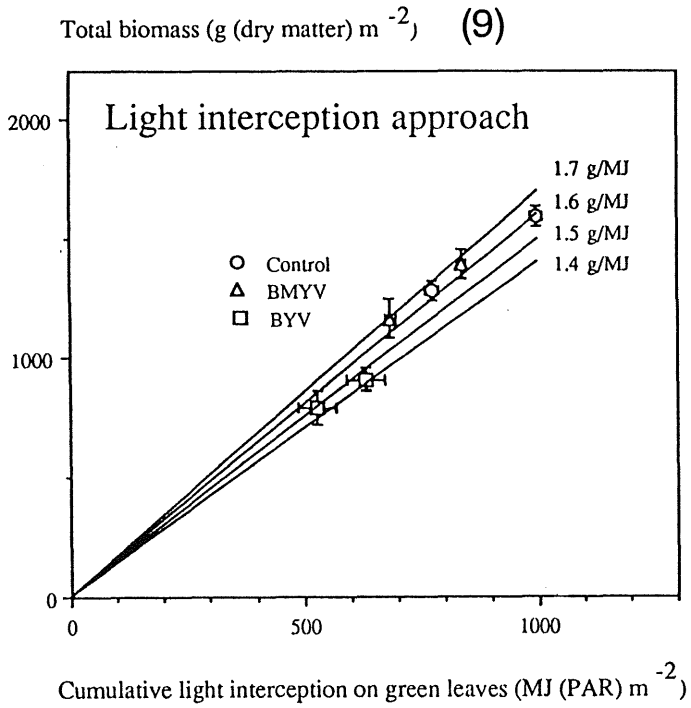


FIGURE 9. Observed relation between cumulative interception of photosynthetically active radiation (PAR) by green leaves and production in sugarbeet field experiment, 1989.

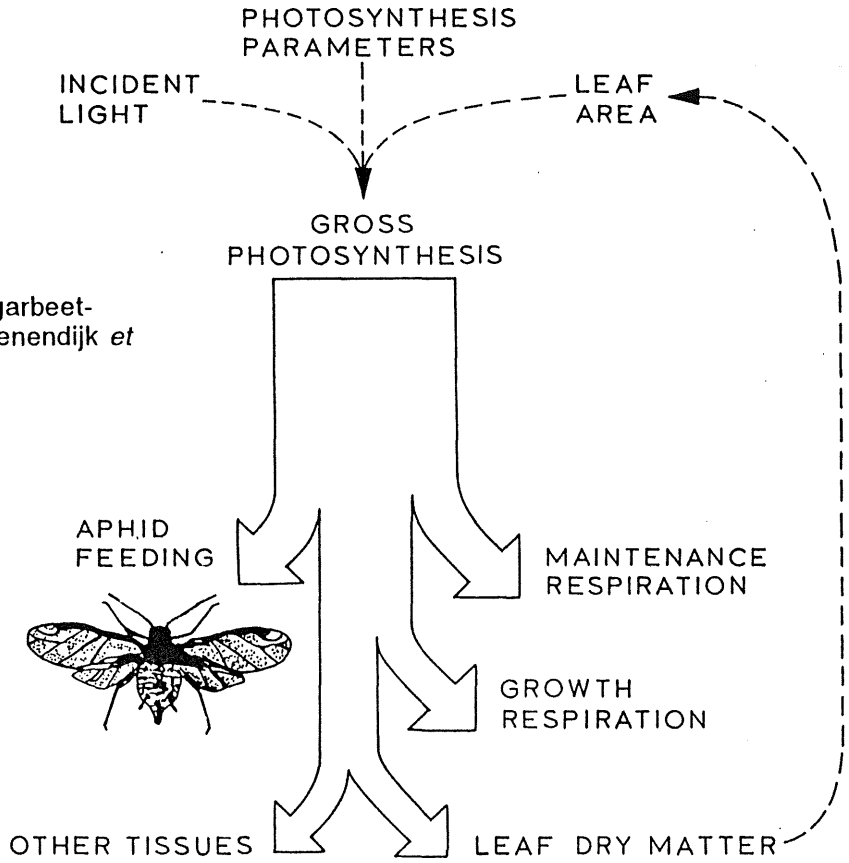


FIGURE 10. Structural diagram of sugarbeet-aphid damage-model. (Data from Groenendijk *et al.*, 1990.)

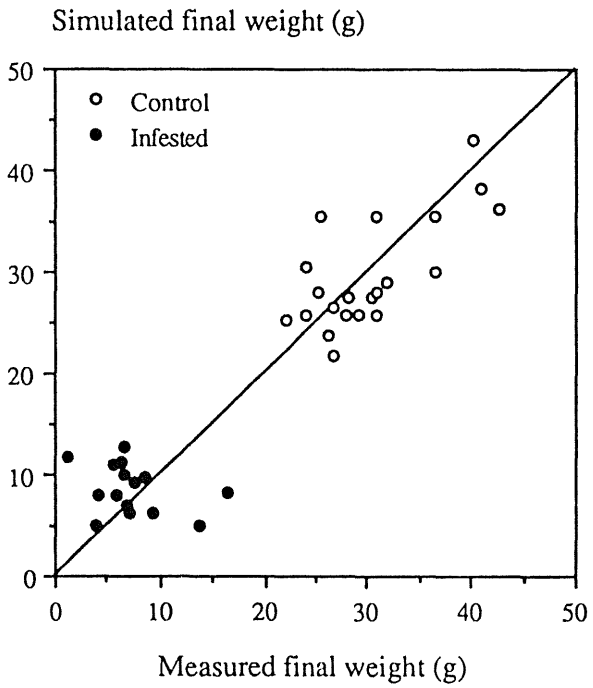


FIGURE 11. Simulated versus measured final dry weight of uninfested and aphid-infested sugarbeet plants, four weeks after infestation with *Aphis fabae* at the cotyledon growth stage. (Data from Groenendijk *et al.*, 1990.)