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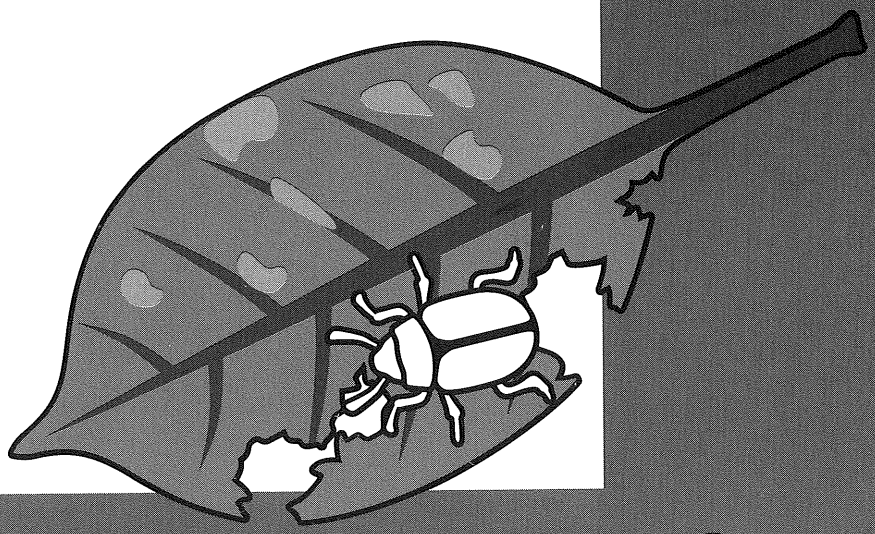
Pests and Pathogens

plant responses to foliar attack



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11

Modelling the effects of foliar pests and pathogens on light interception, photosynthesis, growth rate and yield of field crops

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11.1 Introduction

Differences in crop growth rate between years or locations may be explained by differences in the amount of light (solar radiation, 400–700 nm) received by the crop. However, variation in crop growth rate and yield may also be caused by other factors. According to resource levels and the presence or absence of injurious factors, three yield levels can be defined: the potential yield level, the attainable level and the actual level (Figure 11.1; Zadoks and Schein, 1979).

Potential yields are attained when crops grow with an ample supply of water and nutrients, while injurious biotic and abiotic agents are absent. Such situations are rare and may be obtained only in protected cultivation. Under such conditions, yields depend on site-specific abiotic conditions and crop physiological characteristics. Site factors include the courses of solar radiation, temperature and humidity over the year and over the day, the CO_2 concentration in the air and the physical soil characteristics. Major crop-dependent factors comprise phenology, three-dimensional structure of the canopy, temporal assimilate allocation pattern and the biochemical pathway of CO_2 fixation (C_3, C_4 or Crassulacean acid metabolism (CAM)). Together these factors constitute the growth- and yield-defining factors. Potential growth rates in a temperate, humid climate, e.g. in The Netherlands, appear to be in the order of 25 g dry

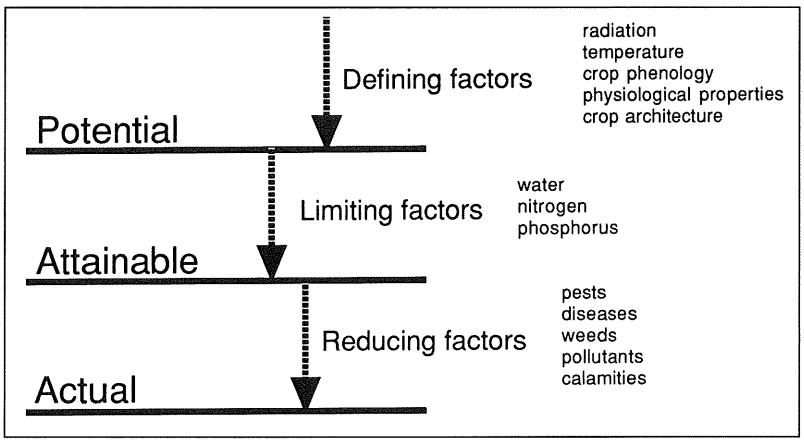

Figure 11.1. *Levels of crop production.*

matter (DM) m^{-2} day^{-1}. Expressed per unit of light, by definition the only limiting resource under optimal conditions, the growth rate is approximately 3 μg (DM) J^{-1} (light).

Shortage of water, nutrients, or both, limits yield to the attainable level. In addition to uptake of CO_2, which is used in photosynthesis, transpiration of water takes place through the stomata. The rate of transpiration depends on radiation, the drying power of the air (vapour pressure deficit) and stomatal aperture. The ratio of transpiration and CO_2 assimilation, the transpiration coefficient, is about 150–300 g (water) g^{-1} (DM) for crops in temperate climates. Thus, to maintain potential growth rates, 4000–8000 g (water) m^{-2}, or 4–8 mm, must be available for transpiration each day. Rates of CO_2 assimilation needed for potential crop growth rates can only be maintained at nitrogen concentrations of approximately 6% of the leaf dry matter (e.g. van Keulen and Seligman, 1987). The amount of nitrogen needed to support potential growth of a crop with a leaf area index of 4 m^2 (of leaf) m^{-2} (of ground) and a specific leaf area of 20 m^2 (of leaf) kg^{-1} (leaf DM) is 12 g m^{-2}. When less nitrogen is available in the leaf, the rate of photosynthesis is reduced. The figures presented are rules of thumb. Methods for estimating water- or nutrient-limited growth rates are described in more detail by van Keulen and Wolf (1986).

Pests, diseases, weeds, extreme weather conditions or pollutants reduce yield to the level which is actually realized in the field (Figure 11.1). The size of the yield reduction, i.e. damage (Zadoks, 1985), depends on the plant processes affected by a growth-reducing factor, i.e. injury components (after Zadoks, 1985), the growth rate of the healthy crop, and the timing and the intensity of growth reduction. The timing and the intensity of growth reduction due to biotic causes depends on the life cycle and population dynamics of the particular agent. Dynamic crop growth models offer a valuable framework within which to structure thinking about the interactions between growth-reducing factors and crop growth processes under variable environmental conditions.

Table 11.1. *Principal processes in plants which can be affected by growth-reducing factors*

Processes	Growth-reducing factors
Carbon and energy economy	
Photosynthesis	
Light interception and distribution	Weeds, necrotrophic fungi, leaf-eating insects
CO_2 diffusion CO_2 fixation other chloroplast processes	Fungal and viral pathogens, phloem-feeding insects and mites, SO_2
Respiration	
Maintenance	Many fungal and viral pathogens, mites, phloem-feeders
New syntheses (growth)	
Photorespiration	Leaf diseases
Allocation	Biotrophic fungi, phloem-feeders, SO_2, viruses
Transport	Phloem-feeding insects
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.)

Crop growth models can include process descriptions at many different levels of organization, e.g. molecular, cellular, tissue, organ, plant, crop or even agro-ecosystem. In studies of crop injury and damage, the levels of interest are those of the crop, the plant and its organs. When lower levels are included in models, problems arise due to lack of knowledge and large differences in the temporal and spatial scales of the processes (de Wit and Penning de Vries, 1982). At the same time, the explanatory power of the model may not increase substantially, or even diminish due to loss of model lucidity. For these reasons, many of the mechanisms of injury studied by physiological plant pathologists, e.g. differences in photosynthesis rate or turgor relations within areas of an infected leaf (reviews in Ayres, 1981; Pegg and Ayres, 1987; Scholes, Chapter 7), are often not used in crop growth models. In crop growth models, processes are more often defined at the plant level, a crop consisting of a population of plants. Here, four major functional subsystems can be distinguished; the economies of carbon, water and nutrients, and morphogenesis. Injury at the plant level can be classified according to the subsystem affected and the processes affected within each subsystem (Table 11.1). A phenomenological classification of injury components was given by Boote *et al.* (1983) who distinguished tissue consumers, leaf senescence accelerators, stand reducers, light stealers, photosynthetic rate reducers, assimilate sappers, and turgor reducers.

Explanatory crop growth models represent powerful tools to gain more insight into the consequences of different injury components for damage, thus allowing simple management rules to be derived. In recent years, two types of crop growth model have frequently been put forward for analysing the consequences of injury for crop growth and yield. These models differ mainly in the degree of detail to which light interception and crop photosynthesis are represented. These models are described here and applied to examples from two studies at Wageningen. The purpose of the comparison is to highlight the merits and limitations of each approach.

11.2 Two approaches to analysing damage with crop growth models

11.2.1 *Summary approach: light interception and utilization by the injured crop*

In many crops growing at the potential production level, growth rate is approximately linearly related to light intercepted by green foliage (Monteith, 1977). Thus, when total biomass is plotted against cumulative intercepted light a straight line is obtained. The slope of this line represents the average crop light-use efficiency (CLUE). The CLUE has been shown to be fairly constant over the growing period (Biscoe and Gallagher, 1977), provided that the average is calculated over periods of at least 14 days (Spitters, 1990). This relation between light interception by green area (LI) and biomass can be used for analytic and integrative purposes.

When analysing experimental results, differences between treatments can be attributed to LI or CLUE, thus distinguishing between effects on photosynthetic area and activity per unit green leaf area, respectively. When used for integrative purposes, LI and CLUE form the basis for a summary model of crop growth in which daily growth rate is calculated as the product of daily LI and CLUE. Total dry matter is obtained by integrating the daily growth rate over time in the growing season. Partitioning factors can be applied to account for the fractions of total biomass allocated to the various plant organs (Spitters, 1987; Russell *et al.*, 1989).

Damage by foliar pests and diseases may be due to reduction of LI, reduction of CLUE, or both (Figure 11.2). When CLUE is unaffected by injury, the photosynthetic capacity of the remaining green area is likely to be unaltered. Injury then simply causes a reduction of the amount of energy available for dry matter formation and damage is proportional to the amount of energy foregone. However, when CLUE is decreased, production capacity per unit of green area is affected and the relation between damage and cumulative intercepted light is more complex.

Examples of studies in which the LI/CLUE concept has been used to analyse injury and damage are given by Haverkort and Bicamumpaca (1986), Johnson (1987), Waggoner and Berger (1987) and van Oijen (1991b). Waggoner (1990) gives formulae to account for vertical and horizontal gradients of pests and pathogens in a crop when calculating LI.

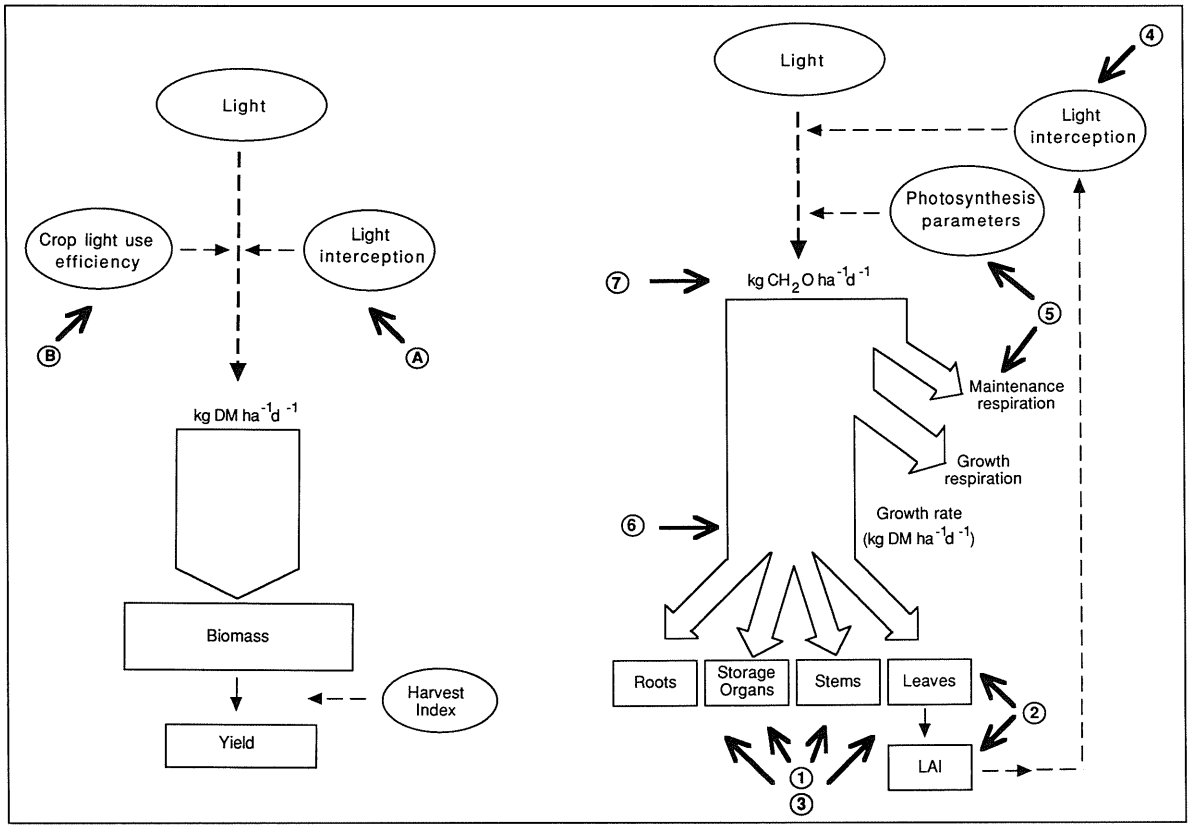

Figure 11.2. *Schematic representation of two explanatory approaches to modelling crop growth and injury (after Spitters, 1990). On the left the summary model of crop growth based on light interception, crop light-use efficiency and harvest index. On the right the comprehensive approach utilizing the light profile within the canopy, photosynthesis characteristics of individual leaves, respiration and dry-matter partitioning factors. Arrows indicate potential components of injury. In the summary model these are: effects on light interception (A) and utilization (B) by the crop. In the comprehensive model these are: tissue consumption (1), leaf senescence acceleration (2), stand reduction (3), light theft (4), (net) photosynthetic rate reduction (5), assimilate sapping (6), and turgor reduction (7).*

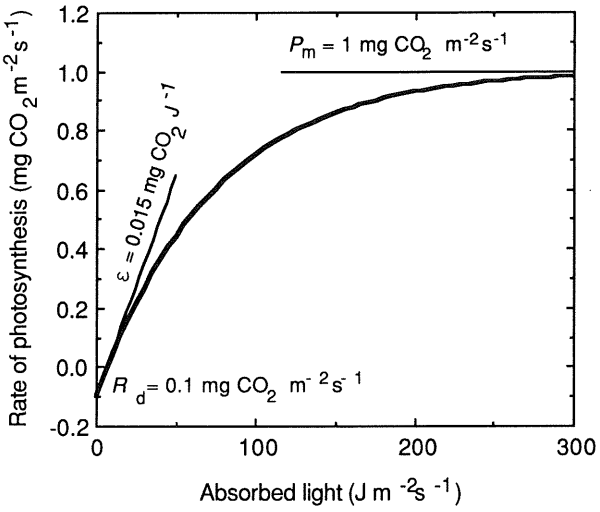

Figure 11.3. *Illustration of a photosynthesis* versus *light response curve, characterized by the parameters* P_m, ε *and* R_d, *see text. The curve is described by a negative exponential equation (Goudriaan, 1982).*

11.2.2 *Comprehensive approach: light interception and utilization by the injured plant*

A comprehensive approach to crop growth starts at the whole plant level. Light utilization by individual leaves, measured in detailed experiments, is combined with the light profile within the crop to arrive at estimates of daily crop growth rates. Light utilization is described by the light response curve, characterized by an initial slope, the initial light use efficiency of single leaves, ε, an asymptote at high light intensities, P_m, and an intercept representing the rate of dark respiration, R_d (Figure 11.3). Experimental analysis of injury components affecting light utilization is in terms of effects on the parameters describing the light response curve of individual leaves. The consequences of altered light utilization by leaves for crop growth are evaluated with a model. In the model (e.g. SUCROS, Spitters *et al.*, 1989) the vertical light profile is calculated using, basically, Beer's law for the penetration of three distinct light fluxes in the canopy: direct, diffuse and scattered light. At selected depths within the canopy and at selected times of the day, the rate of gross CO_2 assimilation is calculated using the response of individual leaves to light. The rate of gross CO_2 assimilation by the crop during 1 day is found by integration over the layers within the canopy and over time within the day. The rate of daily dry matter production is found by subtracting the rate of maintenance respiration from the calculated gross assimilation rate and accounting for the costs of allocation of the assimilates to various organs, and conversion into structural biomass. Integration over the days in the growing season results in total dry matter production.

The injury components of a growth-reducing factor can be introduced into SUCROS at the level of the whole plant (Figure 11.2). Vertical gradients of pests and pathogens can be taken into account by distinguishing different leaf layers and, within each layer, the categories 'healthy', 'injured' and 'dead' leaf area. Within the category 'injured', the intensity of injury, expressed for example as severity, i.e. the fraction of

leaf area covered by lesions, is related to leaf photosynthesis. Recently, a universal approach to describe the effect of pests and pathogens on CO_2 assimilation by single leaves has been put forward (Bastiaans, 1990). The approach was developed for lesions of the leaf pathogen *Pyricularia oryzae* in rice, which decreases both the initial light use efficiency (ε) and the maximum rate of CO_2 assimilation (P_m) beyond the visual borders of a lesion. To relate the photosynthesis parameters to disease severity, 'virtual lesions' are introduced in which CO_2 assimilation is absent while in the remaining leaf area it is unaffected. This idea results in a standard function to describe the relation between disease severity and photosynthesis which is characterized by a single parameter, β, the ratio of virtual and visual leaf area. Values of β exceeding 1.0 imply that the rate of leaf photosynthesis is reduced more than can be explained by a reduction of green area and a loss of light intercepted by the visual lesion. Apparently, the 'zone of influence' of the pathogen extends beyond the borders of the visual lesion, possibly due to the presence of a mobile toxin. Biochemical studies complementary to photosynthesis measurements at the whole leaf level are needed to elucidate the causes of the existence of virtual lesions. The β-model can be used in pathosystems where injury causes clearly confined symptoms. van der Werf *et al.* (1990) compiled severity–photosynthesis relations from the literature and found for cereal leaf diseases β-values ranging from 1.26 ± 0.16 (brown rust) to 5.8 ± 0.6 (mildew), for *Cercospora* leafspot in peanut a β-value of 11.0 ± 3.5, and for brown mite on avocado a value of 1.3 ± 0.7.

Introducing the quantitative effects of the injury components into the crop growth model allows analysis of the consequences on crop growth and yield. Thus, damage is explained using the economies of carbon, water and nutrients, and plant morphogenesis as explanatory levels. Examples of analysis of damage using information on injury components at the whole plant level are given by Rabbinge and Rijsdijk (1981), Boote *et al.* (1983), Kropff (1988), van der Werf (1988), Kropff and Goudriaan (1989), Bastiaans (1990), Daamen and Jorritsma (1990), van Roermund and Spitters (1990) and Rossing (1991).

11.3 Analysis of injury and damage caused by foliar attack – three examples

11.3.1 *Introduction*

The two concepts of crop growth are now used to analyse experiments with three pathosystems. Potato late blight, *Phytophthora infestans*, may reduce yield directly when sporangia are rain-washed into the soil from sporulating leaf lesions and infect the tubers. However, yield loss is usually the result of the rapid destruction of the foliage, which results in shortening of the growing season.

Beet yellows virus (BYV) belongs to the closterovirus group and is transmitted by aphids, especially *Myzus persicae*, in a semi-persistent manner (Bar-Joseph *et al.*, 1979). Following transmission, the virus multiplies in the inoculated leaf. After some time, the virus is transported to the growing tissues, leaves and roots. 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 fully grown (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 often 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 intermediate whorl of mature systemically infected leaves which are yellow and photosynthetically impaired (Hall and Loomis, 1972a,b; Van der Werf, 1988); (3) an outer whorl of healthy mature and senescing leaves that appeared before the plant became infected and which are photosynthetically unimpaired.

Beet mild yellowing virus (BMYV) belongs to the luteovirus group and is persistently transmitted by *M. persicae*. The effects of this virus on sugarbeet have much in common with those of BYV. Leaves turn yellow, exhibit decreased rates of photosynthesis (van der Werf, unpublished results) and occur in whorls similar to those in BYV-infected plants. However, the symptoms take somewhat longer to develop (van der Werf, 1988).

11.3.2 *Application of the summary approach to crop growth*

The potato–late blight pathosystem: data and results. The effect of *P. infestans* on light interception and tuber yield was evaluated in a field experiment in 1988 with three cultivars, the early, susceptible cultivar Bintje, the early, resistant cultivar Surprise, and the late, resistant cultivar Pimpernel. Four different combinations of inoculum and contact fungicide were applied to obtain different levels of disease. In the inoculated treatment, a suspension of sporangia of *P. infestans* was applied about 2 months after planting and no fungicide was applied. In the control treatment no inoculum was applied and the fungicide maneb/fentin acetate was sprayed weekly, from leaf appearance until harvesting. In the treatment 'unsprayed-A', the weekly fungicide application was stopped at the same time as inoculum was applied in the inoculated treatment, while in the 'unsprayed-B' treatment, fungicide application was stopped 3 weeks later. Further details are given by van Oijen (1991b).

Infection led to yield losses in the inoculated, unsprayed-A and unsprayed-B treatments (Table 11.2). Percentage yield loss was always least in cv. Surprise and usually greatest in cv. Bintje.

Infection accelerated the onset of ground cover decline by up to 5 weeks, but had no effect on the rate of decline (Figure 11.4). Light interception accumulated over the growing season (LI) was calculated for each plot, assuming ground cover to be equivalent to light interception (Burstall and Harris, 1983). Relations between tuber yield and cumulative light interception are described by straight lines with a common slope (CLUE) of 2.06 g (tuber DM) MJ^{-1} (light) (Figure 11.5). No differences in slope were detected between cultivars or treatments.

Table 11.3 lists estimates of CLUE for potato crops made by different authors and from different experiments. In no experiment did *P. infestans* significantly affect CLUE. All experiments were conducted under optimum potential growth conditions for the

Table 11.2. *Final yield of tuber dry matter (t ha^{-1})*

Cultivar	Treatment			
	Inoculated	Unsprayed-A	Unsprayed-B	Control
Bintje	2.0	5.7	5.6	7.8
Surprise	4.2	6.4	7.4	7.7
Pimpernel	3.2	6.0	7.1	8.7

Least significant difference (LSD, $P = 0.05$) for pairwise comparison of cultivar means within treatment levels is 1.5 t ha^{-1}. Source: van Oijen (1991b).

healthy crop. Nevertheless, values for crop light-use efficiency varied between 1.8 and 3.2 g (tuber DM) MJ^{-1} (light).

Model calculations. Late blight reduces the cumulative light interception in two ways: first, by covering leaves with lesions, and, second, by accelerating leaf senescence in the remaining, green area. Because late blight occurs mainly on the lower leaves, damage due to shading of healthy leaf area by dead leaf area is unimportant. To evaluate the contribution of these two injury components to damage, the LI/CLUE crop growth model was parameterized for potato (Spitters and Schapendonk, 1990) and coupled to a model of *P. infestans* population dynamics (van Oijen, 1989). Acceleration of leaf senescence by the disease was included in the model. At any time during the epidemic, disease severity, expressed as the percentage of leaf area covered by lesions, induced an equal percentage of leaf senescence in the remaining, lesion-free leaf area (van Oijen, 1991a). In addition, leaf area was lost as a result of normal, physiological ageing. The performance of the model was tested by simulating the course of ground cover in the four treatments, using measured values of average daily

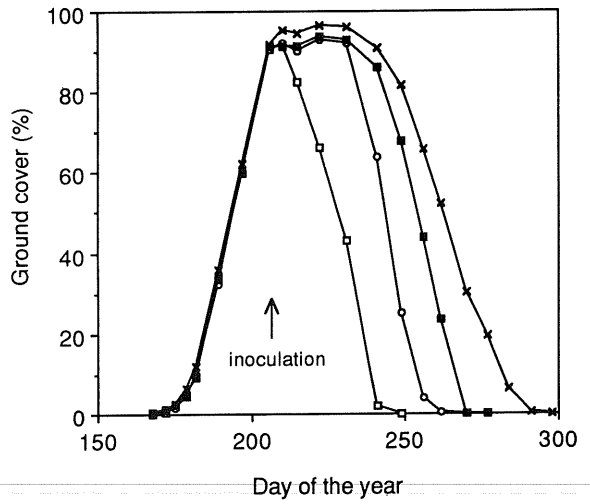

Figure 11.4. *Time course of ground cover of potato. Points represent means over cultivars within treatments. Inoculated (——□——), unsprayed-A (——○——), unsprayed-B (——■——) and control (——×——). Reproduced from van Oijen (1991b) with permission from the European Association for Potato Research.*

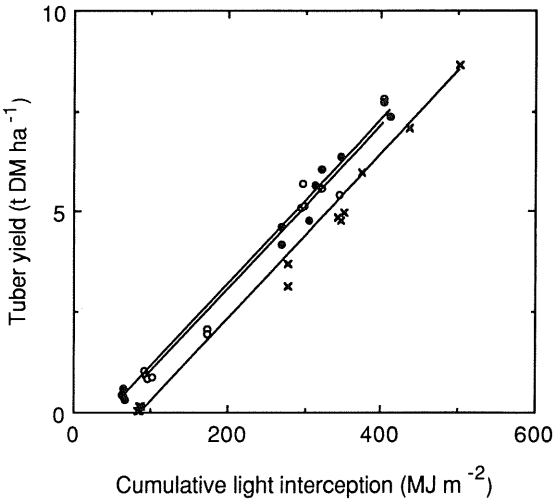

Figure 11.5. *Relation between yield of tuber dry matter and cumulative light interception by green area of three cultivars. For each cultivar results of four treatments at three harvest dates are shown. Points represent averages in four replicates, regression lines are based on individual measurements. Cultivars are Bintje (○), Surprise (●) and Pimpernel (×). Reproduced from van Oijen (1991b) with permission from the European Association for Potato Research.*

temperature and light, and a CLUE of 2.35 g MJ^{-1} for dry matter production as measured in the experiment. The agreement between simulations and measurements was excellent for most cultivars and treatments (Table 11.4). The only exception was the overestimation of green leaf area duration of the late cv. Pimpernel in the absence of disease. Yield was estimated well in all cases.

The contribution of disease-induced leaf senescence to yield loss was assessed by re-running the model with normal, physiological ageing as the only cause of leaf senescence. The results showed that disease-induced leaf senescence accounted for 4–15% of yield loss by *P. infestans*. Thus, the major fraction of yield loss was caused by loss of green leaf area due to lesion extension.

Discussion. Light-use efficiency of the potato crop, averaged over the growing season, was similar for different cultivars and was not affected by the different levels of infection by *P. infestans*. Thus, differences in yield between treatments were caused mainly by differences in the amount of light intercepted.

In experiments similar to the one described here, but at different locations and with

Table 11.3. *Estimates of light use efficiency of potato crops in different experiments*

Crop light use efficiency (g (tuber DM) MJ^{-1} (light))	Authors	Location
3.17	van Oijen, 1991b	The Netherlands
2.06	van Oijen, 1991b	The Netherlands
1.81	van Oijen, 1991b	The Netherlands
3.06	Haverkort and Bicamumpaka, 1986	Rwanda
~2.60	Haverkort and Bicamumpaka, 1986	Rwanda
3.20	Waggoner and Berger, 1987	Israel

Table 11.4. *Comparison of cumulative light interception (LI) and yield of tuber dry matter in field experiments and simulations*

Cultivar	Treatment	LI (MJ m^{-2})		Yield (t ha^{-1})	
		Measured	Simulated	Measured	Simulated
Bintje	Control	403	448	7.8	7.9
	Inoculated	174	212	2.0	2.4
Surprise	Control	404	448	7.7	7.9
	Inoculated	270	298	4.2	4.4
Pimpernel	Control	502	554	8.7	9.0
	Inoculated	279	299	3.2	3.0
r^2		0.99		0.99	
$\sqrt{MSE}$		39		0.3	

Two measures of the degree of agreement between simulated and measured values are shown: the coefficient of determination, r^2, and the square root of the mean squared error, $\sqrt{MSE}$. Source: van Oijen (1991b).

20 potato cultivars, van Oijen (1990) never found a significant change in light-use efficiency due to late blight. These results corroborate the findings of Haverkort and Bicamumpaka (1986) in experiments with tropical highland potato crops and late blight in Rwanda. Similarly, Waggoner and Berger (1987), re-analysing data of Rotem *et al.* (1983), concluded that CLUE of potato was unaffected by disease.

The estimates of CLUE obtained in different experiments show a large variation. In all experiments, crops were well supplied with water and nutrients. However, variation in growth rates may have been caused by factors such as late planting time and destruction of leaves by frost (van Oijen, 1991b), or water shortage at mid-day (Haverkort, personal communication, 1992).

Assessment of the relative importance of the injury components causing reduced light interception in infected crops was possible only by using the model. Knowing that lesion growth is the major injury component enabled van Oijen (1991a) to suggest that breeding strategies for late blight-tolerant potato varieties might benefit from putting greater emphasis on lesion growth rate as a selection criterion.

The BYV and BMYV pathosystems: data and results. In 1989 field plots of sugarbeet were either infected with BYV or BMYV, or treated regularly with an aphicide to produce uninfected controls. Infection was carried out by transferring 5–10 infectious *M. persicae* onto each plant at the cotyledon stage. At 2-week intervals from emergence to harvest, ground cover by green leaf area was estimated using a grid. As with potato, the leaf orientation of young sugarbeet plants tends to be planophile, such that the proportion of ground covered is approximately equivalent to the fraction of incident light intercepted (van der Werf, unpublished). In older plants the inner leaves are more erect.

Total biomass, root biomass and sugar yield were reduced by infection with BYV or BMYV (Table 11.5), BYV causing the greater effects. In spite of the early time of

Table 11.5. *Total dry matter, root dry matter and sugar yield in the 1989 experiment*

Treatment	Yield ($t\,ha^{-1}$)		
	Total biomass	Root biomass	Sugar yield
Control	16.0	7.8	5.1
BMYV-infected	14.0	6.0	4.0
BYV-infected	9.0	3.3	2.1
LSD[1]	1.6	0.7	0.4

[1] Least significant difference (LSD, $P=0.05$) for pairwise comparison of yield means between treatment levels.

infection, the first symptoms did not appear until after the exponential growth phase (Figure 11.6). This was due to the long incubation period of the viruses (approximately 3 weeks for BYV and 5 weeks for BMYV at early crop development stages) (van der Werf *et al.*, 1989b). As infection was at the cotyledon stage, there were no healthy uninfected leaves on these plants (see Section 11.3.1). The green leaf area of plants consisted entirely of young systemically infected leaves which had not yet developed symptoms. The fraction of yellow leaves increased during the growing season due to the decreasing rates of leaf appearance and expansion, and due to symptom development on the older, fully grown leaves. Ground cover by yellow leaves after BMYV infection was lower than after BYV infection at all times.

CLUE of the BMYV-infected treatment (1.68 g (DM) $MJ^{-1} \pm 0.07$ (s.e.)) did not differ significantly from the control (1.62 g (DM) $MJ^{-1} \pm 0.04$ (s.e.)). Infection with BYV significantly reduced the slope of the relations (CLUE was 1.46 g (DM) $MJ^{-1} \pm 0.04$ (s.e.)).

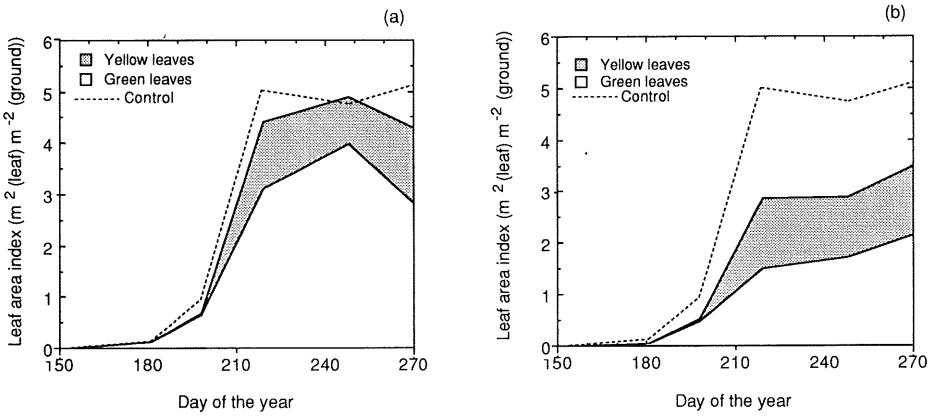

Figure 11.6. *Time course of ground cover of sugarbeet infected with BMYV (a) or BYV (b), and controls in the 1989 field experiment.*

Discussion. Although both BYV and BMYV reduced total biomass, root biomass and sugar yield, the nature of their interference with crop growth processes appears to be different. BMYV did not affect the light use efficiency of the crop. Therefore, damage due to BMYV appears attributable only to a reduction in the amount of light intercepted by green leaves. BYV, in contrast, reduced both the amount of intercepted light and the efficiency of light use.

11.3.3 *Application of the comprehensive approach to crop growth*

The potato–late blight pathosystem: data and results. Cultivars Bintje and Surprise were grown outdoors in pots, inoculated in a greenhouse 5 weeks after planting, and placed in a climate chamber after a further 2 weeks. CO_2 exchange was measured on relatively young, uninoculated leaves at different irradiances (details are given by van Oijen, 1990). Disease severity, expressed as the percentage of leaf or stem area below the measured leaf covered by lesions, varied between 10 and 20% in the inoculated plants and was 0% in the controls. In all inoculated plants at least one lesion completely encircled the stem at some point below the measured leaf. Nevertheless, infection did not significantly affect P_m, ε or R_d of either cv. Bintje or cv. Surprise.

Discussion. The photosynthetic capacity of green leaves was not affected by *P. infestans*. Thus, the virtual lesion area of *P. infestans* appears to be equal to the visual lesion area. These findings support the results of the analysis with the summary model: *P. infestans* reduces light interception, but does not affect light utilization within the green leaf area. Further analysis with the comprehensive crop growth model is unnecessary.

The sugarbeet–BYV and sugarbeet–BMYV pathosystems: data and results. van der Werf (1988) measured the effect of BYV on CO_2 assimilation and respiration in the field. Leaves of infected plants were classified as 'uninfected' (whorl 3 as described in Section 11.3.1), 'infected but without symptoms yet' (whorl 1 as in Section 11.3.1), 'with cleared veins', 'greenish yellow', or 'bright yellow'. Using nylon gauze to create a range of shading allowed assessment of the photosynthesis–light response curve of individual leaves. The reflection and transmission of light by healthy and infected leaves was measured on one occasion (details are given by van der Werf, 1988).

Infection with BYV reduced P_m and ε, and increased R_d in leaves showing yellowing symptoms, but had no effect on photosynthesis in uninfected leaves on the same plants (Table 11.6). Later measurements showed similarly that photosynthesis was not impaired in infected, green heart leaves (the 1988 data were inconclusive; van der Werf, unpublished results). In leaves which showed symptoms on only one half of the blade, the rate of photosynthesis was unaffected in the other, green half.

Absorption of light in uninfected, green leaves (88%) was similar to absorption in

Table 11.6. *Photosynthesis parameters (±s.e.) of uninfected sugarbeet leaves and different categories of BYV-infected leaves on infected plants*

	P_m (mg (CO_2) m^{-2} s^{-1})	ε (μg (CO_2) J^{-1} (light))	R_d (mg (CO_2) m^{-2} s^{-1})	n
Healthy plant	1.09 (0.04)	10.9 (0.3)	0.06 (0.01)	43
Infected				
Uninfected	1.03 (0.08)	11.0 (1.0)	0.03 (0.01)	8
Vein clearing	0.62 (0.05)	9.9 (1.2)	0.11 (0.03)	5
Greenish yellow	0.32 (0.04)	8.8 (1.0)	0.15 (0.02)	8
Bright yellow	0.16 (0.04)	7.8 (1.3)	0.11 (0.02)	13

Source: van der Werf (1988).

infected, green leaves (85%), but greater than in leaves showing yellowing symptoms (63–78%) (van der Werf, 1988).

Model calculations. The comprehensive crop growth model was parameterized to simulate the growth of sugarbeet in the 1989 field experiment with a standard parameter set (Spitters *et al.*, 1989). Four injury components were 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. Although observed in BYV-infected plants, these injury components were assumed also to apply to the growth of plants infected with BMYV.

The first injury component was built into the model by introducing measured leaf area indices as a forcing function (Figure 11.7). The second injury component was introduced by calculating a weighted average scattering coefficient for the whole leaf canopy on the basis of the observed proportions of green and yellow leaf area (Figure

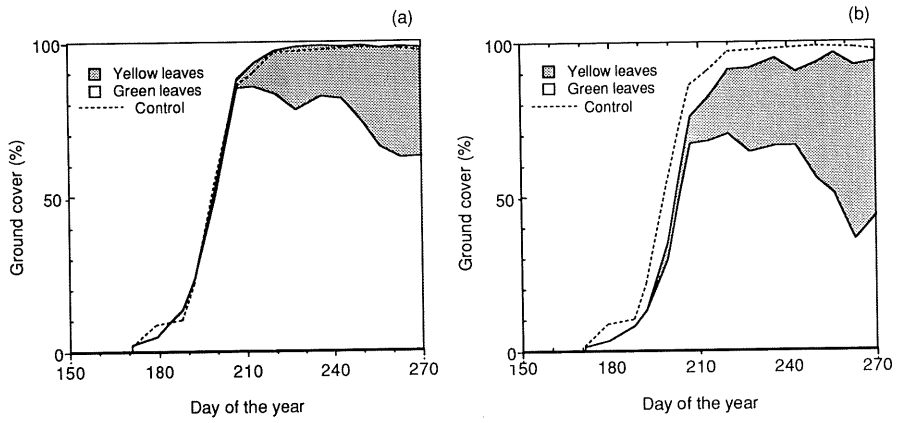

Figure 11.7. *Time course of leaf area index of sugarbeet infected with BMYV (a) and BYV (b), and control treatment in the 1989 field experiment.*

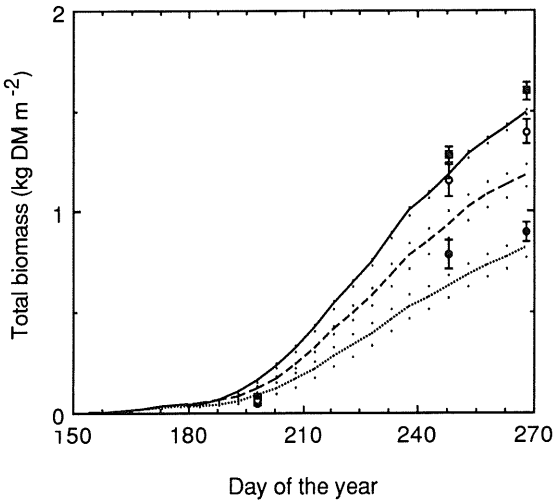

Figure 11.8. *Observed sugarbeet production in 1989 and simulation results obtained with the comprehensive crop growth model. The standard error of the simulation results (indicated by dots) was obtained by running the model with leaf area index data from individual plots. Field data: control (■), BMYV-infected (○) and BYV-infected (●). Simulation results: control (——), BMYV-infected (– – –) and BYV-infected (····).*

11.7) 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.* (1989) were 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 were: P_m = 0.28 mg CO_2 m^{-2} s^{-1}; ε = 9.7 μg CO_2 J^{-1}. Increased respiration (injury component 4) was taken into account by assuming that yellow leaves exhibit a rate of maintenance respiration 2.5 times higher than that of normal healthy leaves. Details are given in van der Werf (1988).

The model slightly underestimated the real growth figures (Figure 11.8). Differences in yield between the three treatments were, however, described fairly well. Earlier sensitivity analysis of the model (Rabbinge *et al.*, 1990; Table 11.7) had shown that the reduction of photosynthetic capability of the yellow leaves was the principal injury component, explaining 70% of the yield reduction.

Discussion. In view of the size of the reduction of net CO_2 assimilation of individual leaves, the assumption that yellow leaves do not contribute to growth would appear a reasonable simplification. Thus, the detailed analysis suggests the simpler crop growth model is valuable for summarizing injury. Virus infection is expected mainly to affect cumulative light interception, since net CO_2 assimilation, and therefore light utilization, is not impaired in the green (parts of) leaves. Infection is expected to increase slightly the light-use efficiency of the infected crop as yellow leaves contribute somewhat to crop production.

Table 11.7. *Simulated relative contribution of components of injury by BYV to sugarbeet in a field experiment in 1986*

Injury components	Early infection		Late infection	
	Yield (%)	%damage[1]	Yield (%)	%damage
No disease	100		100	
		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 photosynthesis, ε and P_m)	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	

Early infection: 5 June 1986, seven leaves, LAI = 0.1. Late infection: 17 July 1986, 21 leaves, LAI = 5.1. More details are in Rabbinge *et al.* (1990).

[1] %damage is the decrease in yield (%) due to incorporating the next injury component in the model, relative to the previously calculated yield. The % damage due to injury component $x + 1$ is calculated as

$$100 - 100 \cdot \frac{\text{yield with injury components 1 through } x + 1}{\text{yield with injury components 1 through } x}.$$

The results of application of the summary model to the 1989 field experiment (Section 11.3.2) showed that conclusions from the detailed analysis held good only for BMYV. For BYV-infected crops, in contrast, the decrease in CLUE found in the field experiment could not be explained on the basis of the injury mechanisms in the detailed analysis. Stated simply, it could be said that green, BYV-infected leaves contribute less to crop production than green, healthy leaves. A plausible explanation would be that infection with BYV increases respiration, not only in the yellow leaves, but also in other plant parts such as the petioles, crown and storage root. Such an increase in whole plant respiration was not taken into account in the comprehensive model because reliable data concerning increased respiration in tissues other than yellow leaves were not available. Further analysis of injury components of BYV is therefore necessary.

11.4 Evaluation

Crop growth models are potentially useful tools for estimating the effects of pests and pathogens on crop growth and for studying the interactions of underlying mechanisms of damage with each other and with the growing conditions. This chapter has discussed two related but still distinct approaches to modelling crop growth by way of example. The first, summary, approach is based on cumulative LI by the visibly uninjured portion of the leaf canopy of the crop. In this approach, yield is calculated as the product of LI and CLUE. Several pests and pathogens affect yield by lowering

LI, while leaving CLUE unaffected, suggesting that the uninjured portion of the plant functions normally. *P. infestans* and BMYV are examples of such pathogens. Other pests and pathogens not only decrease LI, but also affect CLUE, indicating that the apparently uninjured portion of the plant does not function normally. Beet yellows virus is an example of such a pathogen. The causes of the effect of a pest or pathogen on the CLUE can be analysed with a comprehensive crop growth model like SUCROS. Measurements and simulations, using this second approach, suggest that increased respiration in BYV-infected plants may explain the lowered CLUE.

Although in the examples attention has been focused on pathogens, both approaches are equally suitable for analysing the effects of other biotic and abiotic growth reducing factors, as shown in studies of potato–leafhopper (Johnson *et al.*, 1987), winter wheat–grain aphid (Rossing, 1991), sugarbeet–weeds (Kropff, 1988) and faba bean–SO_2 (Kropff and Goudriaan, 1989). Pathosystem-specific aspects comprise the major injury components, and the temporal and spatial dynamics of attack.

The LI/CLUE approach to analysing injury and damage is appealing because of its conceptual simplicity and the ease of empirical parameter estimation. However, the value of CLUE, even under conditions allowing potential production, may vary considerably (Table 11.3, Russell *et al.*, 1989). Seasonal temperature and radiation patterns, and management practices affect CLUE as they affect photosynthesis or canopy structure. The variability of CLUE precludes application of the LI/CLUE model for predictive purposes outside the agro-ecological environment in which the estimates were derived. Fortunately, the variability of CLUE does not hamper the usefulness of the LI/CLUE model as a diagnostic and summarizing tool in studies of injury and damage. The LI/CLUE model allows two broad categories of injury components to be distinguished: injury components affecting LI and injury components affecting CLUE. For more detailed analysis, especially of the latter category, a more comprehensive approach to light utilization is required.

The comprehensive SUCROS-type approach to analysing injury and damage is conceptually more involved and requires sophisticated equipment for estimating the parameters of the photosynthesis–light response curve. Consequences of effects measured at the plant level are evaluated at the crop level by applying the model. Such evaluation must take into account that the model outcome is uncertain as a result of uncertainty in the parameters of the model. Methods for objective, statistical estimation of parameters have been developed (e.g. Klepper and Rouse, 1991), so that such comprehensive crop growth models can also be 'tuned' to specific environmental conditions for the purpose of developing tools for prediction. As a tool for structuring thought, and assessing the relative importance of different injury components, the comprehensive crop model is very suitable, because of its 'bottom-up' approach, i.e. it starts at the whole plant level. The uncertainty in the model outcome renders the comprehensive approach less appropriate for starting the process of identifying injury components, as was shown in the beet–BYV study.

The approach chosen in a specific study will depend on the questions to be answered. The LI/CLUE approach seems a good starting point for analysis. However, studies using a comprehensive approach will remain necessary to draw conclusions about the components of injury operating at the whole plant or lower levels of

organization, as was shown in the beet–BMYV example. Studies at the plant level are also necessary to provide reasons for the variability of CLUE between studies, observed for instance in the work with potato crops.

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