Chapter 8. Modeling Yield Losses Due to Pests - The GENEPEST Structure

The analysis and modeling of crop losses is central to plant protection in general, and to plant pathology, in particular: no plant protection scientific reasoning could possibly exist without a measure of crop losses (Chiarappa, 1971; Rabbinge et al., 1989; Savary et al., 2006; Teng, 1987; Zadoks, 1985; Zadoks and Schein, 1979). In many ways, the applied side of phytopathology, as a science, would thus not exist if crop losses to diseases did not occur. Ironically, the information on crop loss is scarce for a number of reasons we shall not elaborate here. Simulation modeling is one approach to complement the existing data, upscale them, and project ourselves in future environmental, social, and technical scenarios. However, this is only possible if reliable field data are available in sufficient number to assess the outputs of models — simulation outputs cannot be seen *per se* as substitute for measured realities. This latter point cannot be addressed here despite its critical importance.

This chapter introduces the effects of pests (pathogens, but also animal pests, and weeds) on crop growth and how they can be incorporated into crop growth simulation models in order to model yield losses. Crop losses, or more specifically yield losses, occur because the physiology of the growing crop is negatively affected by pests in a dynamic way over time as crop both grows (i.e., increases in biomass) and develops (i.e., passes through the different stages of its physiological development). As a necessary first step to achieve the modeling of yield losses we therefore need to introduce concepts that are related to yield levels and damage mechanisms because they represent the conceptual basis of modeling yield losses. The effects of pests on crop growth using the so called "radiation interception - radiation use" (RI-RUE) framework discussed in the previous chapter will then be addressed again. Lastly, the implementation of damage mechanisms into a crop growth model will be presented and illustrated.

Developing simulation models that integrate the dynamic effects of damage mechanisms of injuries caused by pests, and their translation into yield reduction can provide several types of outcomes, both scientific and practical. Note that, because we deal with (physiological) damage mechanisms on the growing crop, the focus is not on the pathogens (pests) themselves, but the injuries each pathogen (pest) may cause: one pathogen (pest) may cause one or several (and quite different) injuries.

Such models enable, for instance, one to gain:

- A better understanding of processes involved in the attrition of crop growth and yield caused by pest injuries; this is the heuristic value of (simple) simulation models. In that case, the system's

behavior is analyzed and allows pinpointing knowledge gaps and deriving hypotheses on the system's functioning.

- A better view of the respective importance of pests, with respect to the yield losses they can cause: simulation of yield losses caused by one injury in isolation, versus a combination of injuries, and their respective contribution to yield loss, allows ranking of individual diseases (pests) according to the yield losses they cause (or might cause under pre-set scenarios).
- A prospective view of yet-to-achieve progress in disease (pest) management. Simulation of yield gained from improved management tools or strategies provides a formal and quantitative basis for strategic decisions in pest management, including setting research priorities. This applies, in particular, but not solely, to plant breeding, where research efforts are both long and expensive. One can also think of policy applications for better natural resource use and conservation, improvement of production situations, or landscape management.

Important note: crop loss models as presented here therefore ***do not simulate the dynamics of epidemics*** (or of pests, in general), but the dynamics of yield build-up (with or without injuries). As you will see in this chapter, modeling of damage mechanisms and yield losses entails processes (and therefore involves model structures) that are directly connected to the growing crop. As a result, the emphasis in modeling yield losses presented here is completely different from the standpoint used in addressing the modeling of epidemics (described in Chapters 4, 5 and 6 of this module). The crop loss modeling approach in this chapter is instead a direct application of Chapter 7.

This chapter describes concepts used for yield loss modeling, and illustrates how these concepts can be implemented when developing a simulation model for yield loss. Such an approach has been applied to a number of crop pests, for example in the case of groundnut rust and leaf spots (Savary et al., 1990; Savary and Zadoks, 1992), multiple pests of potato (Johnson, 1992), rice leaf blast (Bastiaans, 1993), virus diseases (Madden et al., 2000), multiple pests of rice (Willocquet et al., 2000) and multiple pests of wheat (Willocquet et al., 2008).

Concepts and definitions related to yield levels, production situations and injuries

The concept of yield levels (potential, attainable, actual) and the factors which determine them (Chiarappa, 1971; Zadoks and Schein, 1979; Rabbinge et al., 1989; Rabbinge, 1993) provides a framework which has been, and still is, largely used to address the performances of agrosystems from

the biophysical and socio-economical points of view (van Ittersum and Rabbinge, 1997). Fig. 8.1 provides an overview of yield levels and the factors which determine them.

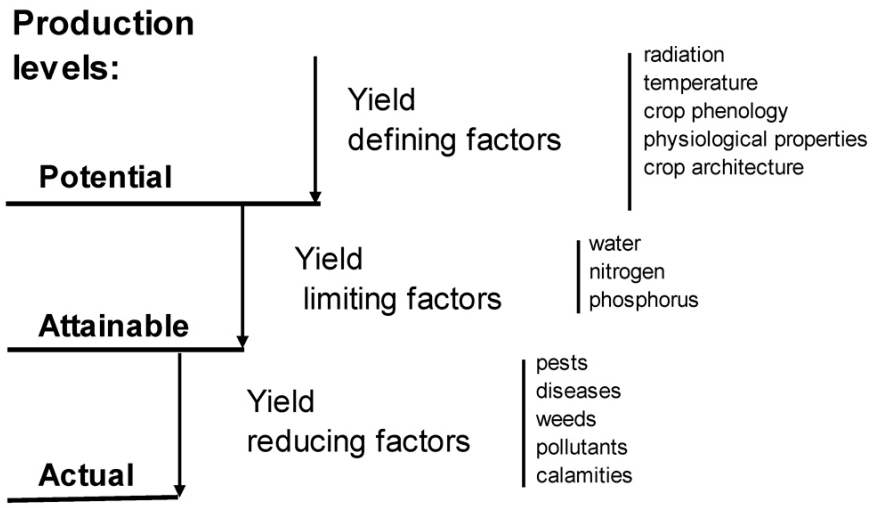

Figure 8.1. Relationships among potential, attainable and actual yields and growth-defining, growth limiting and growth-reducing factors (Rabbinge, 1993; van Ittersum and Rabbinge, 1997).

The **potential yield (*Yp*)** of a crop is determined by ***defining factors***: radiation, temperature, and morphological and physiological attributes determined by the genotype of the crop. The potential yield thus corresponds to the yield that would be produced by a crop grown under optimum conditions.

The **attainable yield (*Ya*)** is determined by the defining factors in combination with ***limiting factors***: water and nutrients. The attainable yield corresponds also to the yield that would be produced by a crop when free of ***injuries***.

The combination of yield defining and yield limiting factors can be embedded in the concept of **production situation** (de Wit and Penning de Vries, 1982; Savary and Zadoks, 1992; Rabbinge *et al.*, 1993). ***The attainable yield of a given crop thus corresponds to the production situation under which this crop is grown.***

The attainable yield can be reduced by the effect of **reducing factors** such as *pest* (disease, insects, weeds) *injuries*. An **injury** is a visible, measurable symptom caused by a harmful organism (Zadoks, 1985).

The resulting yield, obtained in a crop that has been injured by one or several pests, is defined as the ***actual yield***, ***Y*** (Rabbinge, 1993): the actual yield, therefore, is the crop yield actually harvested in a farmer's field.

Yield loss (***YL***), or ***damage*** (Zadoks, 1985), represents the difference between the attainable and the actual yield; that is, the yield lost from pests' injuries. Yield loss can be associated to individual pests as well as to multiple pests. The functional relationships between production situation, attainable and actual yields, yield losses and injuries are summarized in Fig. 8.2.

Yield loss is frequently expressed as the fraction (percentage) of the attainable yield lost to pest injuries. It is then called ***relative yield loss*** (***RYL***), and is computed as: $RYL = 100 \times [(Ya - Y) / Ya]$.

The relationship between injury levels and the yield loss they cause is one important quantitative component analyzed when addressing yield losses. This relationship is called a ***damage function***.

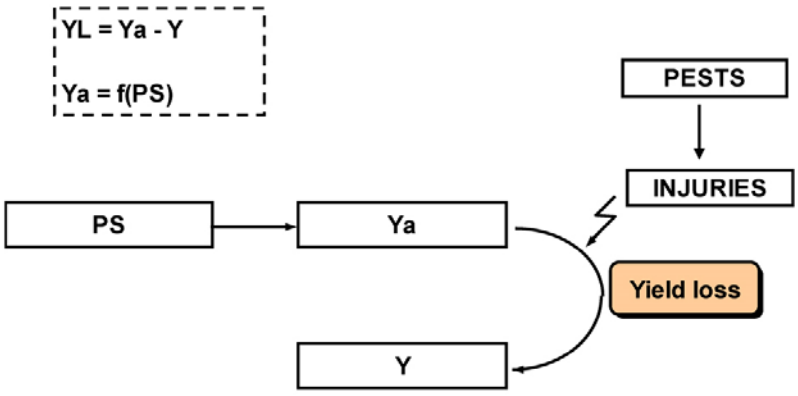

Figure 8.2. Relationships between production situation (PS), attainable yield (Ya), and actual yield (Y); yield loss (YL) (Willocquet et al., 1998).

Production situations may correspond to varying levels of attainable and actual yields, as illustrated in Fig. 8.3. For example:

- two different production situations (i.e., combinations of yield-defining and yield-limiting factors) may correspond to the same level of *Ya*, but to different levels of yield losses (i.e., different combinations of pest injuries), and therefore to different actual yields (PS1 and PS2);
- two production situations may correspond to different levels of *Ya*, but to the same level of actual yield (because yield losses are different: PS2 and PS3);

- two production situations may correspond to different levels of *Ya* and actual yield (PS3 and PS4), the ranking of yield levels between the two production situations being the same ($Ya_{PS3} > Ya_{PS4}$ and $Y_{PS3} > Y_{PS4}$);
- two production situations may correspond to different levels of *Ya* and actual yield (PS4 and PS5), the ranking of yield levels between the two production situations being opposite ($Ya_{PS4} > Ya_{PS5}$ and $Y_{PS4} < Y_{PS5}$).

This diversity of possibilities implies that the quantification of the relative role of the different factors determining the actual yield is a first step when aiming at improving agrosystems' performance.

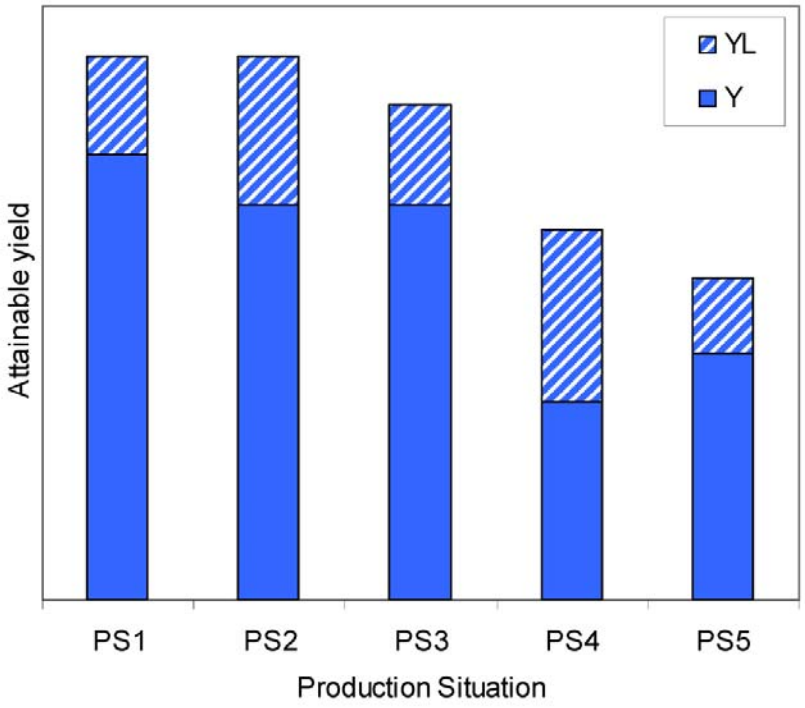

Figure 8.3. An illustration of yield levels in a range of production situations.The concept of damage mechanisms

The concept of damage mechanisms

Damage functions, which quantify the relationships between injuries and yield losses (Zadoks, 1985), can be determined experimentally. They can also be determined from crop loss simulation models, because, as processes, the damage functions represent processes that are underpinned by sub-processes: ***damage mechanisms*** (**DM**). In these models, the processes involved in plant growth are represented, as well as DMs. Damage mechanisms refer to the processes involved in crop growth that

are affected by a harmful agent. Different mechanisms can be described (Rabbinge and Rijsdijk, 1981; Boote et al., 1983). The main categories of DMs are listed in Table 8.1.

Table 8.1. Damage mechanisms of crop pest injuries[a]

Damage mechanism	**Physiological effect**	**Effect in a crop growth model**	**Examples of pests**
Light stealer	Reduces the intercepted radiation	Reduces the green LAI	Pathogens producing lesions on leaves
Leaf senescence accelerator	Increases leaf senescence, causes defoliation	Reduces the biomass of leaves by increasing the rate of leaf senescence	Foliar pathogens such as leaf-spotting pathogens, downy mildews
Tissue consumer	Reduces the tissue biomass	Outflows from biomasses of the injured organs	Defoliating insects
Stand reducer	Reduces the number and biomass of plants	Reduces biomass of all organs	Damping-off fungi
Photosynthetic rate reducer	Reduces the rate of carbon uptake	Reduces the RUE	Viruses, root-infecting pests, stem-infecting pests, some foliar pathogens
Turgor reducer	Disrupts xylem and phloem transport	Reduces the RUE, accelerates leaf senescence	Vascular, wilt pathogens
Assimilate sapper	Removes soluble assimilates from host	Outflows assimilates from the pool of assimilates	Sucking insects, e.g. aphids, some planthoppers, biotrophic fungi exporting assimilates from host cells

[a] Derived from Rabbinge and Vereyken (1980), Rabbinge and Rijsdijk (1981) and Boote et al. (1983).

Damage mechanisms have been experimentally measured for many pests, for example on groundnut rust (Savary et al., 1990), rice leaf blast (Bastiaans, 1991), bean diseases (Bassanezi et al., 2001; Lopes et al., 2001), and wheat *Septoria tritici* blotch (Robert et al., 2006). Such quantification allows a better understanding of the underlying mechanisms of the effects of pests on crop growth.

The use of DM parameters can serve at least three purposes:

- DMs can be incorporated into models simulating components of crop growth, e.g., canopy photosynthesis (Bastiaans and Kropff, 1993), and assimilate partitioning (Bancal et al., 2012).
- DMs can be incorporated in crop growth simulation models in order to simulate their effect of crop growth and yield. How to implement this will be described in section 8.4, and examples from the literature are given in the introduction of this chapter.
- parameters for DMs can also be used to compare host plant resistance levels amongst genotypes of a given crop (e.g., Bastiaans and Roumen, 1993).

The use of damage mechanism parameters illustrates again one important characteristic of mechanistic simulation modeling, that is, the mobilization of parameters that have been acquired ***experimentally***. Therefore, there is no disconnection, but, to the contrary, ***a complete loop from experimental data to model*** (parameters) ***and from model to experiments*** (experimentally measured system's response).

The effects of pests on crop growth using the RI-RUE framework

The damage mechanisms described above can be linked to the RI-RUE concepts described in the previous chapter. Johnson (1987) grouped damage mechanisms in two broad categories, according to their major effect on RI (the first four damage mechanisms: light stealers, leaf senescence accelerators, tissue consumers, and stand reducers) and RUE (the last three damage mechanisms: photosynthetic rate reducer, turgor reducers, and assimilate sappers).

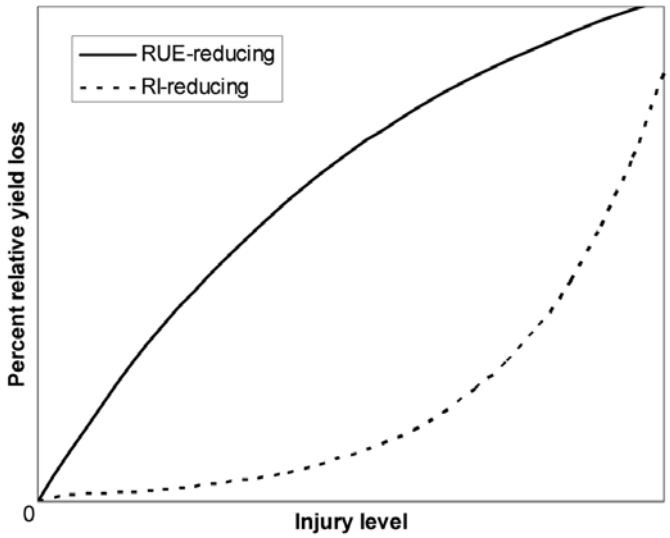

Figure 8.4. Types of damage functions corresponding to RI-reducing and RUE-reducing pests (derived from Johnson, 1987).

Using a potato crop growth simulation model including damage mechanisms for several pests, Johnson (1987) exemplified the effects of injuries on yield losses according to their yield-reducing effects (through a reduction of RI or of RUE; Fig. 8.4).

Because of Beer's law relationship between LAI and RI, a pest reducing the LAI will have a small reducing effect on yield at low pest intensity. On the other hand, RUE-reducing pests will have a large effect even at low pest intensity, and this effect will decrease (relatively) as pest intensity (and injury) increases.

Grouping pests according to their effects on RI and RUE may be useful for crop loss assessment and disease (pest) management. Analyzing these relationships (damage function, damage mechanism, RI-RUE-reducing effect) allows one to:

(1) address this type of research question in a synthetic way, while

(2) still accounting for the underlying biological mechanisms.

These underlying mechanisms involve questions pertaining to (1) the impact of pests on yield losses, (2) the injury thresholds for pest management, and (3) multiple-pest systems (Johnson, 1987). This approach has been used to analyze many, diverse, pathosystems. It remains very appealing when analyzing interactions between pests, yield, and production situations (Savary et al., 2006). The simplicity of the framework may provide an appealing way for analyses incorporating other factors, e.g., decision making or incorporating other species such as antagonists.

A simple crop growth simulation model for actual growth and yield, and yield losses – GENEPEST

Stages to simulate yield losses, and possible outcomes

Simulation of crop growth and yield affected by pest injuries can be made by incorporating into a crop growth model (such as GENECROP) the damage mechanisms corresponding to the injuries addressed. We shall call this new model GENEPEST. A complete listing of the program can be found in Appendix 8.1.

A three-stage approach then allows the simulation of yield losses:

1. Simulation of non-injured growth, enabling one to model the attainable growth and attainable yield (*Ya*) of a crop under a given production situation. By definition, all injury levels are then set to zero.
2. Simulation of growth under specified levels of injuries in order to model the actual growth and actual yield (*Y*).
3. Computation of yield losses, that is, the difference between simulated attainable and actual yields.

Note 1. Simulating growth and yield with levels of injuries corresponding to improved pest management (*Ym*) allows estimating yield that would be gained on the actual yield (*Y*) from this improvement in pest management (*Ym-Y*), thus providing a basis to guide strategic decisions such as research priorities in pest management.

Note 2. Yield losses can be simulated for a range of production situations, by setting the crop drivers (i.e., parameters and interpolation functions for crop growth) to values corresponding to each production situation, and proceeding to the three stages described above.

Note 3. Yield losses can be simulated for injuries considered individually and for combinations of injuries (i.e., grouped as pre-defined injury profiles), thus allowing ranking injuries according to their importance in terms of the yield losses they cause. Such results can help in ranking crop health problems and, again, help for guiding research priorities in pest management.

Incorporating damage mechanisms into a crop growth model

The damage mechanisms given in Table 8.1 can be incorporated in the crop growth model, GENECROP described in the previous chapter, leading to the GENEPEST model (Fig. 8.5).

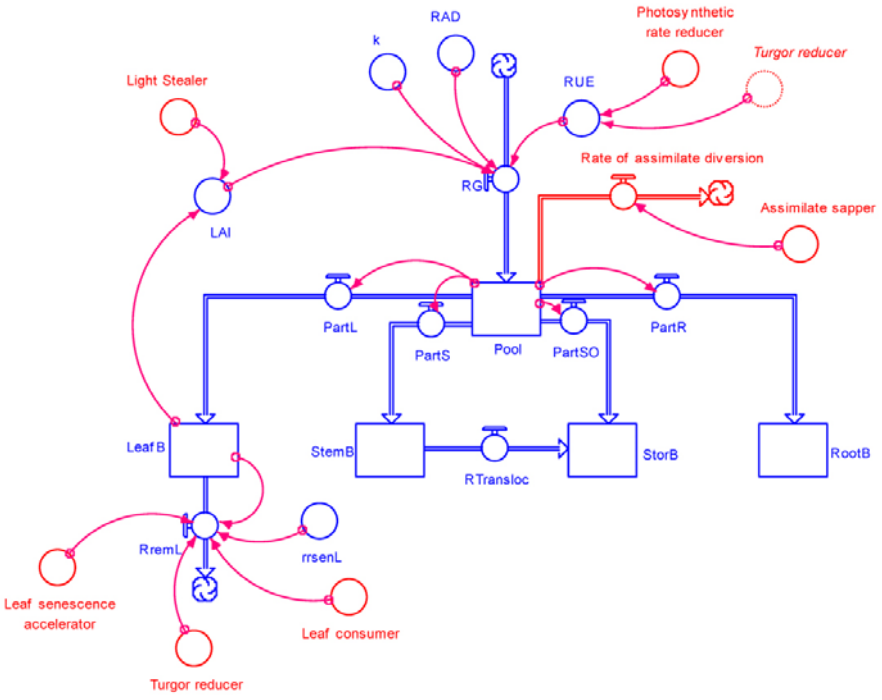

Figure 8.5. GENEPEST: general structure of a crop growth model incorporating damage mechanisms from pest injuries.

Stand reducers are not included in Fig 8.5 in order to avoid crowding the diagram. Stand reducers would affect the biomass of all organs, and would be reflected by rates of reduction of biomass for all for organs.

Fig. 8.5 indicates that:

(1) all damage mechanisms can be accounted for in GENEPEST,

(2) the different damage mechanisms correspond in general to effects on different processes (rates) or on different variables,

(3) different damage mechanisms however can affect the same process (i.e., leaf consumers and leaf senescence accelerators cause a reduction in [green] leaf biomass, and

(4) a damage mechanism can affect more than one process or variable, as in the case of turgor reducers.

Damage mechanisms are now considered with examples from varying pests in order to illustrate how damage mechanisms can be coupled to a crop growth model.

Light stealers

Light stealers decrease the area of green LAI. This typically corresponds to leaf diseases. Thus, equation (7.6) in Chapter 7:

$$LAI_t = SLA_t \times LEAFB_t \tag{7.6}$$

becomes, for one leaf disease, LD1:

$$LAI_t = SLA_t \times LEAFB_t \times RF_{LD1t} \tag{8.1}$$

where *RF* stands for the 'Reduction Factor' associated to the injury caused by leaf disease 1.
Note that this reduction factor is dynamic, as indicated by the *t* index. In the case of a foliar disease, which produces lesions that decrease the green LAI, equation (8.1) can be simply written as:

$$LAI_t = SLA_t \times LEAFB_t \times \left(1 - x_{LD1t}\right) \tag{8.2}$$

where x_{LD1t} is the disease severity of LD1 (i.e., the fraction of leaves covered by lesions varying between 0 and 1) at time *t*. Equation (8.1) reflects the decrease in (green) LAI caused by disease, which corresponds to the leaf area covered by lesions and not photosynthesizing any more. Again, the reduction in LAI is dynamic, as disease severity can be made to vary over the course of an epidemic.

If we consider three leaf diseases LD1, LD2, LD3, equation (8.2) becomes:

$$LAI_t = SLA_t \times LEAFB_t \times (1 - x_{LD1t}) \times (1 - x_{LD2t}) \times (1 - x_{LD3t}). \quad (8.3)$$

The underlying hypotheses of this equation are that (1) decreases in LAI can be due to one disease only (overlapping of lesions from two different diseases will reduce the LAI only once), and (2) the three diseases are randomly distributed in the crop canopy.

Leaf senescence accelerators and tissue (leaf) consumers

Leaf senescence accelerators and leaf consumers generally refer to different pests, the former typically corresponding to pathogens, and the second to insect defoliators. From a modeling point of view, they are however handled together and in the same manner here, because they correspond to the same effect on crop growth, i.e., a reduction in leaf dry biomass. The incorporation of these effects into the model is first described in the case of leaf senescence accelerators and then in the case of leaf consumers.

Leaf senescence accelerators have the same physiological effect as physiological senescence, and are therefore accounted for in the crop growth model in the same way as physiological senescence. So, equation 7.18 in Chapter 7:

$$LEAFB_{t+\Delta t} = LEAFB_t + ((PARTL_t - RSENL_t) \times \Delta t) \quad (7.18)$$

becomes:

$$LEAFB_{t+\Delta t} = LEAFB_t + ((PARTL_t - RSENL_t - RSENIN1_t) \times \Delta t) \quad (8.4)$$

where $RSENIN1_t$ is the rate of leaf senescence caused by injury. It is convenient to establish a relationship between *RSENIN*1 and injury level by expressing this rate of senescence as the product of a relative rate of senescence by the leaf dry biomass:

$$RSENIN1_t = RRSENIN1_t \times LEAFB_t \tag{8.5}$$

with:

$$RRSENIN1_t = alpha \times IN1_t \tag{8.6}$$

Equations (8.5) and (8.6) simply mean that in the case of leaf senescence caused by an injury, the fraction of leaf senesced is proportional to the intensity of the injury. Injury can be expressed as disease severity (i.e., a fraction between 0 and 1). The magnitude of the effect of injury on senescence corresponds to the parameter *alpha*, which needs to be measured experimentally.

An important example of tissue consumers is the case of defoliating insects, which decrease the leaf biomass by eating leaves or fractions of leaves. This type of damage mechanism can be reflected in equation (7.18) by reducing the leaf biomass as a result of consumption by defoliating insects:

$$LEAFB_{t+\Delta t} = LEAFB_t + \left(\left(PARTL_t - RSENL_t - RDEF_t\right) \times \Delta t\right) \tag{8.7}$$

where $RDEF_t$ is the rate of defoliation. In the same way as for senescence accelerators, a relationship can be established between the rate of defoliation and the injury:

$$RDEF_t = RFDEF_t \times LEAFB_t \tag{8.8}$$

with $RFDEF_t$ is the rate of increase in fraction of leaf area damaged by defoliation. This rate can be derived from successive assessments of the fraction of leaf area defoliated.

When combining effects of senescence accelerators and leaf consumers, the following hypothesis is made: leaf consumers do not damage leaf tissues that are senesced, and leaf senescence cannot occur on defoliated parts of leaves. The combined effects of these two damages are therefore additive and can be written as:

$$LEAFB_{t+\Delta t} = LEAFB_t + \left(\left(PARTL_t - RSENL_t - RSENIN1_t - RDEF_t\right) \times \Delta t\right) \tag{8.9}$$

Photosynthetic rate reducers

Photosynthetic rate reducers can be incorporated in a crop growth model such as GENECROP (chapter 7) by decreasing the RUE. In crop growth models describing in more detail the photosynthesis processes, the effect of photosynthetic rate reducers would be reflected by a reduction in, for example, the initial light use efficiency of single leaves, and/or a reduction in the maximum rate of photosynthesis, and/or an increase in dark respiration (e.g., Rossing et al., 1992).

In GENECROP, equation 7.5 in Chapter 7:

$$RG_t = RUE_t \times RAD_t \times \left(1 - e^{-k \times LAI_t}\right) \tag{7.5}$$

becomes:

$$RG_t = RUE_t \times RAD_t \times \left(1 - e^{-k \times LAI_t}\right) \times RF_{PR1t} \tag{8.10}$$

In the case of light stealers such as (leaf-spotting) foliar diseases, the relationship between the reduction factor and the level of injury is straightforward: the reduction in green LAI corresponds to disease severity and $RF = 1 - x = 1$ - severity.

When addressing photosynthetic rate reducers, the relationship between the reduction factor and the level of injury is less straightforward, and often needs to be established experimentally. Two examples corresponding to pests which widely differ biologically (a viral disease and a root-infecting disease), but nevertheless cause similar damage mechanisms by reducing the *RUE*, are given below to illustrate how *RF* can be expressed.

Viral diseases are in general systemic and the virus particles are transported within the plant via its vascular system. Virus infection can reduce the rate of photosynthesis and this can be simply represented by the relationship between the proportion of disease plants and the reduction factor:

$$RF_{PR1t} = 1 - \left(delta \times VIR_t\right) \tag{8.11}$$

where *delta* is a parameter ranging between 0 and 1, which represents the magnitude in the effect of viral infection to reduce the *RUE*, and VIR_t is the proportion of diseased plants. The parameter *delta* needs to be measured with specific experiments.

Root-infecting diseases cause injuries, which directly affect the functioning of infected roots, and therefore the amount of water and nutrients absorbed by the roots. This in turn causes a reduction in *RUE*. A relationship between the disease level and *RF* can be written as:

$$RF_{PR2_t} = 1 - \left(gamma \times RDIS_t\right) \tag{8.12}$$

where, similarly to equation (8.11), *gamma* is a parameter ranging between zero and 1, which represents the magnitude in the effect of root infection to reduce the *RUE*, and $RDIS_t$ is the proportion of roots infected by the pathogen. The parameter *gamma* needs to be measured with specific experiments.

Accounting for the combined effects of the two above pests can be done by multiplying the reduction factors, which reflects (1) the interactions between both pests in their effect on RUE and (2) the hypothesis of random distribution of both pests. Equation (8.10) becomes:

$$RG_t = RUE_t \times RAD_t \times \left(1 - e^{-k \times LAI_t}\right) \times RF_{PR1t} \times RF_{PR2t} \tag{8.13}$$

Assimilate sappers

Assimilate sappers uptake assimilates produced from photosynthesis. Two important pest groups cause this type of damage mechanism: insects such as aphids or plant hoppers which are feeding from the phloem sap, and biotrophic fungi such as rusts which are diverting the assimilates to produce fungal organs, especially spores. One could also add a number of plant nematodes, at least those which do not cause tissue necrosis.

The diversion of assimilates is accounted for in the simulation of the dynamics of the pool of assimilates. Equation (7.16) from Chapter 7:

$$POOL_{t+\Delta t} = POOL_t + \left[\left(RG_t - PARTL_t - PARTS_t - PARTR_t - PARTSO_t\right) \times \Delta t\right] \tag{7.16}$$

becomes:

$$POOL_{t+\Delta t} = POOL_t + \left[\left(RG_t - PARTL_t - PARTS_t - PARTR_t - PARTSO_t - DIV_t\right) \times \Delta t\right] \tag{8.14}$$

The amount of assimilates diverted by pests is retrieved from the amount of assimilates partitioned towards organs, and equations (7.12) to (7.15) in Chapter 7 become:

$$PARTL_t = (POOL_t - DIV_t) \times CPL_t \times (1 - CPR_t) \quad (8.15)$$

$$PARTS_t = (POOL_t - DIV_t) \times CPS_t \times (1 - CPR_t) \quad (8.16)$$

$$PARTR_t = (POOL_t - DIV_t) \times CPR_t \quad (8.17)$$

$$PARTSO_t = (POOL_t - DIV_t) \times CPSO_t \times (1 - CPR_t) \quad (8.18)$$

Again, a relationship needs to be established experimentally between the amount of assimilates diverted by the pest and the level of injury. In the case of insects, this corresponds to the sapping (or sucking) rate, and can depend on the crop development stage and the insect development stage (or weight). The diversion rate can be written as:

$$DIVINS_t = rrsap_t \times bmperins_t \times NBINS_t \quad (8.19)$$

where $DIVINS_t$ is the (daily) assimilate diversion rate, $rrsap_t$ is the relative rate of sapping (per biomass of insect and per day), $bmperins_t$ is the biomass of an individual insect, and $NBINS_t$ is the number of insects (per m^2), $rrspap_t$ and $bmperins_t$ need to be experimentally measured and may vary over time, and $NBINS_t$ is the insect pest driving function, which may vary over time, and represents the dynamics of insect density.

In the case of biotrophic fungi, the relationships between disease intensity and the diversion of assimilates can be done according to the carbohydrate uptake for spore production, the number of spores produced per lesion per day, and the lesion size:

$$DIVBIOT_t = rruptake \times NBLES_t \quad (8.20)$$

with:

$$NBLES_t = \frac{LAI_t \times SEVBIOT_t}{lesize} \quad (8.21)$$

where *rruptake* is the rate of carbohydrate uptake per lesion per day; and the number of lesions is derived from disease severity, $SEVBIOT_t$ (pest driver), lesion area (*lesize*), and LAI.

When combining the two above pests, a simple hypothesis corresponds to the independence between both pests and their injuries, leading equation (8.14) to become:

$$POOL_{t+\Delta t} = POOL_t + \left[\left(RG_t - PARTL_t - PARTS_t - PARTR_t - PARTSO_t - DIVINS_t - DIVBIOT_t\right) \times \Delta t\right] \quad (8.22)$$

and replacing $POOL_t$ - DIV_t by $POOL_t$ - $DIVINS_t$ - $DIVBIOT_t$ in equations (8.15) to (8.18).

Turgor reducer

Damage mechanisms associated with turgor reducers have been addressed when considering RUE reducers and leaf senescence accelerators. They will therefore not be illustrated specifically in this chapter. Accounting for turgor reducers will however be illustrated in the next chapter.

Important note: for the sake of simplicity, the incorporation of damage mechanisms into a crop growth simulation model has been described above for each damage mechanism, one at a time. ***Individual crop pests can, however, cause more than one type of damage.*** This will be illustrated in the GENEPEST model, and in the description of RICEPEST and WHEATPEST models in the next chapter.

Model parameters for damage mechanisms

The parameters needed to simulate damage mechanisms are derived from experiments. Two main types of parameters can be considered:

(1) parameters which represent the magnitude of the impact of pest injuries on the crop physiological processes:

alpha (leaf senescence accelerator);
delta (virus disease – effect on RUE);
gamma (root-infecting disease – effect on RUE);
rrsap (insect sapper – rate of sapping); and
rruptake (biotrophic pathogen – rate of assimilate uptake).

(2) parameters corresponding to ecological characteristics of the pests, which are needed to determine a relationship between the damage mechanism and the level of injury:

bmperins (biomass of an individual insect); and

lesize (area of a lesion).

The following values are set in GENEPEST (again, these have to be experimentally measured for a given pest).

alpha = 0.076 day^{-1} (case of rice sheath blight; Willocquet et al., 2000)

delta = 0.35 (case of wheat BYDV; Willocquet et al., 2008)

gamma = 1 (case of wheat take-all; Willocquet et al., 2008)

rrsap = 1 $mg \cdot mg^{-1} \cdot day^{-1}$ (arbitrarily chosen value)

rruptake = 4.62×10^{-6} day^{-1} (case of wheat brown or leaf rust; Willocquet et al., 2008)

bmperins = 0.5 mg (arbitrarily chosen value)

lesize = 10^{-6} m^2 (case of wheat brown or leaf rust; Willocquet et al., 2008)

Model drivers for pests injuries

The damage mechanisms described above have been implemented into GENEPEST by considering several pests, which provide a combination of the damage mechanisms described previously. The pests considered are described in Table 8.2.

Table 8.2. Examples of pests accounted for in GENEPEST[a]

Pest name	Pest type	Driving function	Damage mechanism 1	Damage mechanism 2
LD1	Foliar pathogen	Disease severity (fraction leaf area infected)	light stealer	
LD2	Foliar pathogen		light stealer	leaf senescence accelerator
LD3	Foliar pathogen		light stealer	assimilate sapper
VIR	Virus	Disease incidence (fraction plants infected)	photosynthetic rate reducer	
DEF	Defoliating insect	Daily fraction of leaf area defoliated	tissue (leaf) consumer	
RDIS	Root-infecting pathogen	Disease severity (fraction root infected)	photosynthetic rate reducer	
NBINS	Sucking insect	Nb of insects (per m^2)	assimilate sapper	

[a] Weeds can also accounted for in a simplified manner, see Chapter 9.

Simulations

The STELLA model GENEPEST.STMX will allow you to:

- explore the model structure and equations,
- explore the model inputs, especially the driving functions of the different pests included
- explore the model outputs, and
- run the model with varying levels of injury, which will allow you to explore:
- the effects of individual injuries on crop growth and yield
- the effects of combined injuries on crop growth and yield

Summary

This chapter describes:

- Concepts and definitions related to yield levels, production situations and injuries
- The concept of damage mechanism
- The effects of pests on crop growth within the RI-RUE framework
- How damage mechanisms are captured in a quantitative and dynamic way into a generic simulation model, GENEPEST.
- Provides the equations, parameters, and flowchart of GENEPEST.
- Includes the STELLA file, which can be used to explore the model structure and the effect of injuries, individually or in combination, on the simulated dynamics of crop growth.

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Exercises and questions

1. Give examples of pests in wheat categorized by damage mechanism, following Table 8.1.

2. Indicate which of the following statement is (are) correct
   a. yield loss is the difference between attainable and actual yield
   b. yield loss is the difference between potential and attainable yield
   c. a yield reducing factor may be associated to different injury mechanisms
   d. a given damage mechanism can affect different physiological processes

3. A light stealer affects
   a. the RUE
   b. the partitioning towards organs
   c. the leaf biomass
   d. the LAI

4. Acceleration of leaf senescence affects
   a. the RUE
   b. the partitioning towards organs
   c. the leaf biomass
   d. the LAI

5. A possible unit for the relative rate of leaf senescence is
   a. $g \cdot g^{-1} \cdot day^{-1}$
   b. $g \cdot day^{-1}$
   c. $g \cdot g^{-1}$
   d. $g \cdot m^{-2} \cdot day^{-1}$

Answers to exercises and questions

1. pests by damage mechanism in wheat:
   b. light stealer: weeds, Septoria blotch;
   c. leaf senescence accelerator: Septoria blotch;
   d. tissue consumer: many defoliating insects (e.g., Lema spp.);

e. stand reducer: many soil pathogens: take-all pathogen (e.g., *Gaeumannomyces tritici*); weeds; barley yellow dwarf virus disease;
f. photosynthetic rate reducer: barley yellow dwarf virus disease; Septoria blotch;
g. turgor reducer: eyespot pathogen (*Rhizoctonia* spp.);
h. Assimilate sappers: rust pathogens (stripe [yellow], leaf [brown], and stem rust); aphids.

2. a: yield loss is the difference between attainable and actual yield, and c: a yield reducing factor may be associated to different injury mechanisms

3. a: the LAI.

4. c: the leaf area biomass

5. a: $g \cdot g^{-1} \cdot day^{-1}$.

Appendix 8.1. Program listing of GENEPEST

```
LeafB(t) = LeafB(t - dt) + (PartL - RSenL) * dt
INIT LeafB = 10
INFLOWS:
PartL = CPL*(Pool-rdiv)
OUTFLOWS:
RSenL = ((rrsen+(alpha*LD2)+RFDEF)*LeafB)
MaxStemb(t) = MaxStemb(t - dt) + (rmaxstemb) * dt
INIT MaxStemb = 6
INFLOWS:
rmaxstemb = PartS
Pool(t) = Pool(t - dt) + (RGrowth - PartS - PartL - PartO - PartR - rdiv) * dt
INIT Pool = 0
INFLOWS:
RGrowth = RAD*RUE*(1-EXP(-k*LAI))*(1-(delta*VIR))*(1-(gamma*RDIS))
OUTFLOWS:
PartS = CPS*(Pool-rdiv)
```

PartL = CPL*(Pool-rdiv)

PartO = CPO*(Pool-rdiv)

PartR = CPR*(Pool-rdiv)

rdiv = (rsrsap*bmperins*INS)+(rruptake*LAI*LD3/lesize)

REPTIL(t) = REPTIL(t - dt) + (Rmat - Rmortr) * dt

INIT REPTIL = 0

INFLOWS:

Rmat = if DVS<0.8 or DVS>1 then 0 else if VTIL<FST*Totil then 0 else RRMAT*VTIL

OUTFLOWS:

Rmortr = rrmort*REPTIL

RootB(t) = RootB(t - dt) + (PartR) * dt

INIT RootB = 5

INFLOWS:

PartR = CPR*(Pool-rdiv)

StemB(t) = StemB(t - dt) + (PartS - RTransloc) * dt

INIT StemB = 6

INFLOWS:

PartS = CPS*(Pool-rdiv)

OUTFLOWS:

RTransloc = IF(DVS>1) then ddist else 0

STEMP(t) = STEMP(t - dt) + (Dtemp) * dt

INIT STEMP = 320

INFLOWS:

Dtemp = ((TMAX+TMIN)/2)-TBASE

StorB(t) = StorB(t - dt) + (PartO + RTransloc) * dt

INIT StorB = 0

INFLOWS:

PartO = CPO*(Pool-rdiv)

RTransloc = IF(DVS>1) then ddist else 0

VTIL(t) = VTIL(t - dt) + (Rtil - Rmat - Rmrtv) * dt

INIT VTIL = 250

INFLOWS:

Rtil = PartLS*STW*(1-(VTIL/maxtil))*DVE

OUTFLOWS:

Rmat = if DVS<0.8 or DVS>1 then 0 else if VTIL<FST*Totil then 0 else RRMAT*VTIL

Rmrtv = (rrmort*VTIL)

alpha = 0.076

bmperins = 0.0005

CPL = CPPL*(1-CPR)

CPO = CPPO*(1-CPR)

CPS = (1-CPL-CPO)*(1-CPR)

DACE = TIME+14

ddist = 0.005*MaxStemb

delta = 0.35

DVS = if stemp<TFLOW then STEMP/TFLOW ELSE 1+((STEMP-TFLOW)/(TMAT-TFLOW))

FST = 0.05

gamma = 1

grain__yield = 0.85*StorB

INS = pINS*INSn

k = 0.6

LAI = LeafB*SLA*(1-LD1)*(1-LD2)*(1-LD3)

LD1 = pLD1*LD1n

LD2 = pLD2*LD2n

LD3 = pLD3*LD3n

lesize = 0.000001

maxtil = 900

PartLS = PartL+PartS

pINS = 0

pLD1 = 0

pLD2 = 0

pLD3 = 0

pRDIS = 0

pRFDEF = 0

pVIR = 0

RAD = 17

RDIS = pRDIS*RDISn

RFDEF = pRFDEF*RFDEFn

RRMAT = 0.3

rruptake = 0.00000462

rsrsap = 1

RUE = 1.2

STW = 20

TBASE = 8

TFLOW = 1500

TMAT = 2000

TMAX = 30

TMIN = 24

Totil = VTIL+REPTIL

VIR = pVIR*VIRn

CPPL = GRAPH(DVS)

(0.00, 0.55), (0.1, 0.536), (0.2, 0.521), (0.3, 0.507), (0.4, 0.493), (0.5, 0.479), (0.6, 0.464), (0.7, 0.45), (0.8, 0.3), (0.9, 0.15), (1, 0.00), (1.10, 0.00), (1.20, 0.00), (1.30, 0.00), (1.40, 0.00), (1.50, 0.00), (1.60, 0.00), (1.70, 0.00), (1.80, 0.00), (1.90, 0.00), (2.00, 0.00)

CPPO = GRAPH(DVS)

(0.00, 0.00), (0.05, 0.00), (0.1, 0.00), (0.15, 0.00), (0.2, 0.00), (0.25, 0.00), (0.3, 0.00), (0.35, 0.00), (0.4, 0.00), (0.45, 0.00), (0.5, 0.00), (0.55, 0.00), (0.6, 0.00), (0.65, 0.00), (0.7, 0.00), (0.75, 0.00), (0.8, 0.143), (0.85, 0.286), (0.9, 0.429), (0.95, 0.571), (1.00, 0.714), (1.05, 0.857), (1.10, 1.00), (1.15, 1.00), (1.20, 1.00), (1.25, 1.00), (1.30, 1.00), (1.35, 1.00), (1.40, 1.00), (1.45, 1.00), (1.50, 1.00), (1.55, 1.00), (1.60, 1.00), (1.65, 1.00), (1.70, 1.00), (1.75, 1.00), (1.80, 1.00), (1.85, 1.00), (1.90, 1.00), (1.95, 1.00), (2.00, 1.00)

CPR = GRAPH(DVS)

(0.00, 0.3), (0.1, 0.263), (0.2, 0.225), (0.3, 0.188), (0.4, 0.15), (0.5, 0.112), (0.6, 0.075), (0.7, 0.038), (0.8, 0.00), (0.9, 0.00), (1, 0.00), (1.10, 0.00), (1.20, 0.00), (1.30, 0.00), (1.40, 0.00), (1.50, 0.00), (1.60, 0.00), (1.70, 0.00), (1.80, 0.00), (1.90, 0.00), (2.00, 0.00)

DVE = GRAPH(DVS)

(0.00, 1.00), (0.4, 1.00), (0.8, 0.00), (1.20, 0.00), (1.60, 0.00), (2.00, 0.00)

INSn = GRAPH(TIME)

(0.00, 0.00), (10.0, 0.00), (20.0, 0.00), (30.0, 50.0), (40.0, 100), (50.0, 150), (60.0, 200), (70.0, 150), (80.0, 100), (90.0, 50.0), (100, 5.00), (110, 5.00), (120, 5.00)

LD1n = GRAPH(TIME)

(0.00, 0.00), (10.0, 0.00), (20.0, 0.00), (30.0, 0.004), (40.0, 0.008), (50.0, 0.01), (60.0, 0.007), (70.0, 0.002), (80.0, 0.00), (90.0, 0.00), (100, 0.00), (110, 0.00), (120, 0.00)

LD2n = GRAPH(TIME)

(0.00, 0.00), (10.0, 0.00), (20.0, 0.00), (30.0, 0.002), (40.0, 0.005), (50.0, 0.008), (60.0, 0.01), (70.0, 0.008), (80.0, 0.007), (90.0, 0.006), (100, 0.005), (110, 0.004), (120, 0.004)

LD3n = GRAPH(TIME)

(0.00, 0.00), (10.0, 0.00), (20.0, 0.00), (30.0, 0.003), (40.0, 0.005), (50.0, 0.007), (60.0, 0.009), (70.0, 0.01), (80.0, 0.01), (90.0, 0.009), (100, 0.007), (110, 0.005), (120, 0.001)

RDISn = GRAPH(TIME)

(0.00, 0.00), (10.0, 0.00), (20.0, 0.00), (30.0, 0.001), (40.0, 0.002), (50.0, 0.01), (60.0, 0.01), (70.0, 0.01), (80.0, 0.01), (90.0, 0.01), (100, 0.01), (110, 0.01), (120, 0.01)

RFDEFn = GRAPH(TIME)

(0.00, 0.00), (10.0, 0.00), (20.0, 0.00), (30.0, 0.00), (40.0, 0.001), (50.0, 0.00), (60.0, 0.00), (70.0, 0.00), (80.0, 0.00), (90.0, 0.00), (100, 0.00), (110, 0.00), (120, 0.00)

rrmort = GRAPH(DVS)

(0.00, 0.00), (0.1, 0.00), (0.2, 0.00), (0.3, 0.00), (0.4, 0.00), (0.5, 0.02), (0.6, 0.02), (0.7, 0.02), (0.8, 0.02), (0.9, 0.02), (1, 0.00), (1.10, 0.00), (1.20, 0.00), (1.30, 0.00), (1.40, 0.00), (1.50, 0.00), (1.60, 0.00), (1.70, 0.00), (1.80, 0.00), (1.90, 0.00), (2.00, 0.00)

rrsen = GRAPH(DVS)

(0.00, 0.00), (0.1, 0.00), (0.2, 0.00), (0.3, 0.00), (0.4, 0.00), (0.5, 0.00), (0.6, 0.00), (0.7, 0.00), (0.8, 0.00), (0.9, 0.00), (1, 0.00), (1.10, 0.013), (1.20, 0.026), (1.30, 0.04), (1.40, 0.04), (1.50, 0.04), (1.60, 0.04), (1.70, 0.04), (1.80, 0.04), (1.90, 0.04), (2.00, 0.04)

SLA = GRAPH(DVS)

(0.00, 0.037), (1.00, 0.018), (2.00, 0.017)

VIRn = GRAPH(TIME)

(0.00, 0.00), (10.0, 0.00), (20.0, 0.00), (30.0, 0.002), (40.0, 0.01), (50.0, 0.01), (60.0, 0.01), (70.0, 0.01), (80.0, 0.01), (90.0, 0.01), (100, 0.01), (110, 0.01), (120, 0.01)