3.1 Population models

R. Rabbinge, J.C. Zadoks and L. Bastiaans

3.1.1 Introduction

This Section presents some widely-used population models and elaborates on a numerical model that can be used to simulate epidemics in relation to time. First, a summary of models is presented, describing plant disease epidemics (van der Plank, 1963). After demonstrating the limitations of these simple models the pathosystem, powdery mildew-wheat is used to illustrate the development of a simulation model. Various important features will be added to improve the accuracy.

3.1.2 Exponential, logistic and paralogistic growth

Consider a population of size Y, in which each individual has a constant rate of reproduction, λ (in units of t^{-1}) and a constant rate (probability) of dying, μ. The rate of change in the population is then

$$\frac{dY}{dt} = (\lambda - \mu) \cdot Y \qquad \qquad \text{Equation 35}$$

The size of the population at time t is thus

$$Y_t = Y_0 e^{r \cdot t} \qquad \qquad \text{Equation 36}$$

where $r = \lambda - \mu$, the intrinsic per capita rate of increase, or the population's relative growth rate (RGR).

This exponential growth model assumes that the relative reproduction rate and relative mortality rate of the population are independent of the number of individuals. In many species, however, this is simply not true. For example, as the number of fungal lesions increases, the number of sites available for new lesions declines; and pest insects often compete for food. In these cases, reproduction will decline or mortality will increase with density. This is introduced into the rate equation by using a density-dependent reduction factor. The simplest assumption is that the relative growth rate declines linearly with the population size:

$$\frac{dY}{dt} = r \cdot Y \cdot (1 - Y/Y_m) \qquad \qquad \text{Equation 37}$$

where the actual growth rate (dY/dt) equals the growth rate under non-limiting conditions (r · Y) times redution factor (1 − Y/Y$_m$).

Y_m is the maximum possible population size, r is the relative growth rate of the population under non-limiting conditions; the net result of birth and mortality. The growth of a population in time can now be described by a logistic growth equation:

$$Y_t = \frac{Y_m}{1 + K \cdot e^{-r \cdot t}}$$

Equation 38

Exercise 31
K (Equation 38) is a scaling factor. Express this factor in Y_m and Y_0, the initial value of Y at time t = 0.

When Y_t is plotted against time, a typical S-shaped curve appears. In phytopathology, such S-shaped curves are found in epidemic diseases; logit transformation yields straight lines, representing the relation between the logit value

$$\text{logit } Y_t = \ln(Y_t/(Y_m - Y_t))$$

Equation 39

and time. This logit value is a way of representing disease intensities that takes into account an upper limit for the amount of disease.

However, it is a serious mistake to think that all S-shaped curves found in phytopathology are produced by logistic growth. The logistic growth process assumes that the effect of population density on population growth is instantaneous, i.e. newly-born individuals reproduce immediately. This is true only for processes like the growth of yeast, where, when cell division is completed, the two newly formed cells can begin to divide again immediately, without a substantial lag time. For populations of plant pests and diseases, however, considerable waiting times exist: e.g. latency periods, p, in fungi, and non-reproductive periods in insects – such as the egg, larval and pupal stages in holometabola. Furthermore, the length of the reproductive period is finite: e.g. infectious periods, i, in fungi and reproductive periods in insects. Therefore, a more accurate description of the growth of a population may be expected when the relative growth rate is related to the reproductive part of the population only.

For a fungal disease – where an individual can be defined as a site with the area of a lesion – this means that the total number of infected sites (Y in Equation 37) should be replaced by the number of infectious sites:

$$\frac{dY_t}{dt} = R_c \cdot (Y_{t-p} - Y_{t-p-i}) \cdot \left(1 - \frac{Y_t}{Y_m}\right)$$

Equation 40

The three components of the right hand side of the equation represent:

R_c , the multiplication factor or relative growth rate;

$Y_{t-p} - Y_{t-p-i}$, an activator representing the number of sporulating lesions;

$1 - \dfrac{Y_t}{Y_m}$, a correction factor to prevent double infections and to introduce an upper limit for the lesion density.

The growth model represented here is called paralogistic (Zadoks & Kampmeyer, 1977). In Table 4, the various descriptive formulae for population development in relation to time, from the exponential growth model to the paralogistic growth model, are summarized.

In several fungal diseases, the definition of an individual is rather arbitrary and, therefore, it is more convenient to work with the fraction diseased-leaf tissue. After replacing the number of infected sites by the fraction diseased-leaf tissue, we obtain the equation produced by van der Plank (1963):

$$\frac{dy_t}{dt} = R_c \cdot (y_{t-p} - y_{t-p-i}) \cdot (1 - y_t) \qquad \text{Equation 41}$$

where y is the fraction diseased-plant tissue.

The differential equation for paralogistic growth (Equation 40) cannot be solved analytically, as i and p are not constant and depend on various environmental factors. Therefore, numerical integration techniques must be used. As a first step, the principle of paralogistic growth is translated into a simple relational diagram (Figure 24). The host is represented by a number of sites, vacant (NI) or infected. Infected sites are divided into three classes, latent (LAT), infectious (INF) and no longer infectious sites or removed sites (NLINF). The total number of sites is computed from the leaf area and the space needed for one lesion. For powdery mildew on wheat, with a site size of 3.5 mm^2, each hectare of leaf area contains $10^{10}/3.5 = 2.9 \times 10^9$ sites (NMAXHA). Thus, a field of wheat with a leaf area index of 3 (LAI, ha ha^{-1}) contains 8.6×10^9 sites (NMAX). This maximum number, of course, is never reached as this would mean that all leaf area is completely covered by lesions. R is the relative growth rate of the population, and LP and IP the latency and infectious periods, respectively.

Exercise 32
Write a CSMP model for paralogistic growth, without using the subroutine BOXCAR, Section 2.2. Use one state variable per class and test the model for sensitivity to R, LP and IP.

Table 4. A family of logistic equations; differential form, integral form (when available) and rate equations.

| No	Differential equations (T^{-1})	Integral equations (1)	Rate equations (T^{-1})
1	exponential growth $\dfrac{dx_t}{dt} = r \cdot x_t$	$x_t = x_0 \cdot e^{r \cdot t}$	$r = \dfrac{\ln(x_2) - \ln(x_1)}{t_2 - t_1}$
2	logistic growth $\dfrac{dx_t}{dt} = r \cdot x_t \cdot (1 - x_t)$	$f(x_t) = \ln x_t = \ln x_0 + r \cdot t$ $x_t = \dfrac{x_0 \cdot e^{r \cdot t}}{1 - x_0(1 - e^{r \cdot t})}$ $f(x_t) = \ln \dfrac{x_t}{1 - x_t} = r \cdot t + c = logit(x_t)$	$r = \dfrac{logit(x_2) - logit(x_1)}{t_2 - t_1}$
3	introduction of latency period p (i = ∞) $\dfrac{dx_t}{dt} = R \cdot x_{t-p} \cdot (1 - x_t)$		
4	paralogistic growth: introduction of p and a limited infectious period i $\dfrac{dx_t}{dt} = R_c \cdot (x_{t-p} - x_{t-i-p}) \cdot (1 - x_t)$		
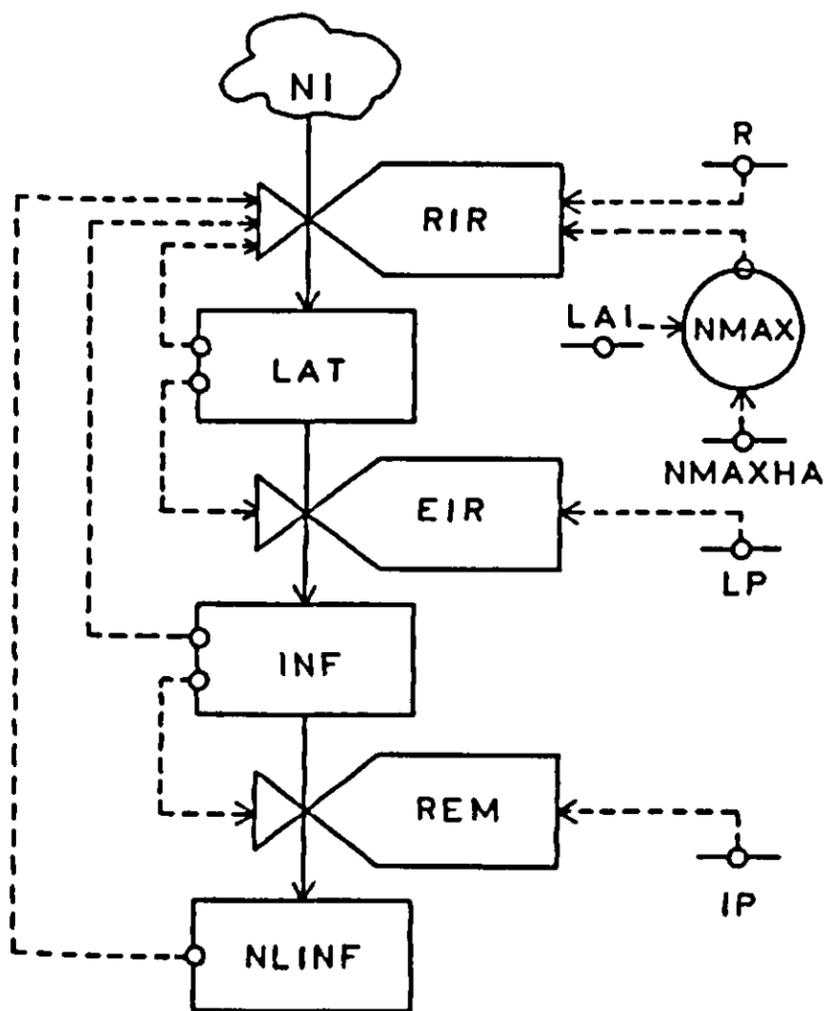
Figure 24. Relational diagram for the life cycle of a fungal disease such as *Erysiphe graminis*. NI = vacant sites, LAT = latent lesions, INF = infectious lesions, NLINF = no longer infectious lesions, RIR = real infection rate, EIR = effective infection rate, REM = rate of removal of infectious lesions, R = relative growth rate, NMAX = maximum number of infections, LP = latency period, IP = infectious period, LAI = leaf area index, NMAXHA = maximum number of lesions per ha of leaf area.

The results of Exercise 32 are given in Figure 25 as log number of infections versus time. The increase on a logarithmic scale is linear, and flattening occurs only when NMAX is limited (Figure 25b).

The model in its present form is useful for demonstration purposes only. It shows that the latency period is of major importance for the growth rate of an epidemic.

Exercise 33

Express LAT, INF, and NLINF in terms of the elements of Equation 40.

The paralogistic growth of an epidemic (Equation 40) is governed by four parameters: the latency period p (days), the infectious period i (days), the maximum number of sites Y_m (number) and the relative growth rate of the epidemic R (day^{-1}). In real epidemics, these four parameters are not constant. They depend on environmental conditions such as temperature and leaf wetness, on
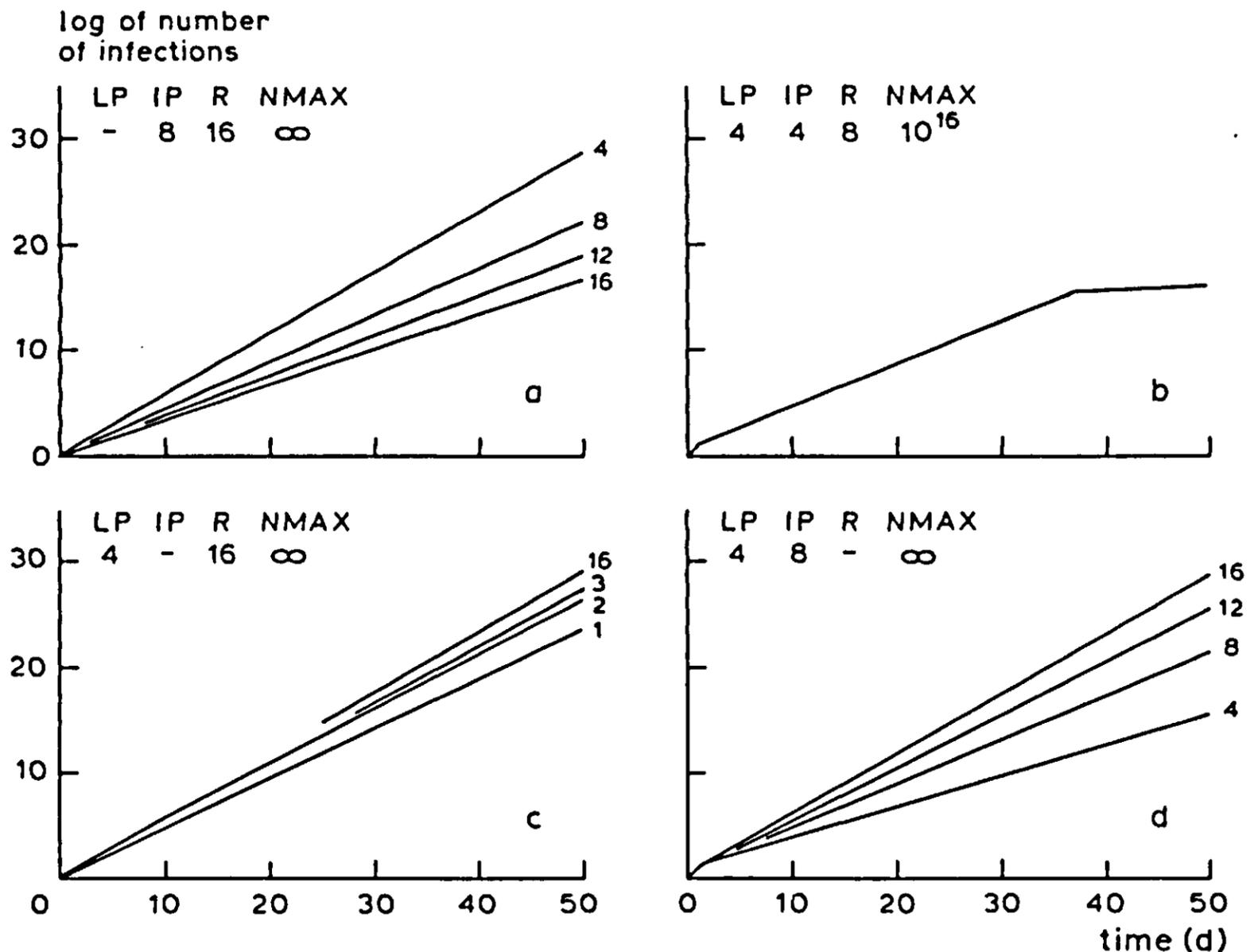
Figure 25. The increase of an epidemic over time, vertical axis: log (number of infections); horizontal axis: time. a) Effect of length of latency period (LP), in days, on upsurge of the epidemic. b) Effect of a finite number of possible infections (NMAX). c) Effect of length of infectious period (IP). d) Effect of daily reproduction rate or relative growth rate (R).

crop growth and on the condition of the various parts of the crop. In the next Subsections, these factors will be considered and incorporated step by step in the model of Exercise 32, finally resulting in a more accurate simulation model of a powdery mildew epidemic.

3.1.3 Computation of the latency period (LP) and the infectious period (IP)

In many fungal pathogens, the latency period and the infectious period depend on temperature, humidity and crop condition. If humidity does not limit development and the crop condition is optimal, both periods are computed from functions which describe their relations with temperature (TEMP):

LP = f(TEMP)
IP = f(TEMP)

These relations may be used, but they are often replaced by their inverses, since, at suboptimal temperatures, there is often an approximate linear relation between the rate of development and temperature. The model implicitly assumes that

88

development reacts instantaneously to changes in temperature. If this assumption is not true, considerable errors may result.

Exercise 34

Table 5 shows the duration of the latency period of powdery mildew, *Erysiphe graminis*, on winter wheat at different temperatures. At 5 °C the development rate equals zero, the temperature threshold is 7 °C. Express the development during latency in units from 0 to 1.

a. Compute the development stage of the fungus after 3 days at a constant temperature of 15 °C.

b. Compute the development stage after the same period when 12 h at 12 °C is alternated with 12 h at 18 °C.

c. Perform the same calculation when the temperature varies between 2 °C and 18 °C and compare this with the results for a constant temperature of 10 °C. Explain the results of these comparisons of development at constant and fluctuating temperatures.

The nitrogen status of the crop (or nitrogen content of the leaves) may affect development considerably (Aust, 1981). To incorporate this plant effect, another state variable should be introduced by coupling a crop growth simulator to the epidemiological model. For the sake of simplicity, this nitrogen effect is neglected here. This simplification seems reasonable when crop husbandry practices are such that potential yield levels are reached. In this case, nitrogen is always abundantly available and nitrogen levels in the leaves are quite high. In Section 4.4 of this textbook, an approach will be described in which the nitrogen status of the crop is considered.

Table 5. Duration of the latency period (LP), infectious period (IP) and their standard deviations (SD) for powdery mildew, *Erysiphe graminis*, on winter wheat grown with abundant nitrogen at various temperatures.

Temperature (°C)	Length latency period (days)		Length infectious period (days)	
	X	SD	X	SD
10	16	3	10	3
15	8	2	4	2
20	4	1.5	3	1
25	2.5	1.	2	0.5
30	2.2	1.	1	0.25

The latency period and the infectious period were both simulated with one boxcar, thus assuming that in both stages relative dispersion equals unity. This is not realistic, as dispersion in time is not constant. Controlled dispersion is now introduced into the program by means of a BOXCAR subroutine (see Section 2.2). The inverse of the average latency and infectious periods and their relative dispersions should now be introduced explicitly into the model and used in these BOXCAR subroutines, which mimic dispersion in time. Using an AFGEN function, the inverse of the latency and infectious periods and their relative dispersions are read from tables DRLATT, DRINFT, RDLATT and RDINFT, respectively.

3.1.4 Computation of the maximum number of infectious sites (NMAX)

In the model of the paralogistic growth, the maximum number of sites was introduced as a parameter and held constant throughout the growing season. This is an unrealistic simplification, since the leaf area increases and decreases with time. To take this into account, leaf area can be introduced in a table that gives the actual leaf area values at different times. Another method would be the coupling of the epidemic simulator to a crop growth model (see Section 4.3).

In the present model, total leaf biomass (TLBM, kg ha^{-1}) is introduced by way of a forcing function (Table 6). This total leaf mass is converted to leaf area by multiplying the mass by a constant specific leaf area (SLA, ha kg^{-1}). The number of sites is found directly from the leaf area (LAI, ha ha^{-1}) and is used in the model of paralogistic growth:

$$\text{NMAX} = \text{LAI/SLES}$$

Table 6. Leaf mass (TLBM) of a wheat crop in relation to the start of the epidemic.

TIME (Julian days)	TLBM (kg ha^{-1})
70	75
90	100
115	200
130	1250
150	2200
180	2250
200	2000
220	1300
240	0

in which SLES expresses the Surface occupied by one LESion, for mildew 3.5 mm^2.

Since leaf area is no longer a constant, the rate of dying leaf area is required to compute the rate of dying lesions. For simplicity, it is assumed that the probability of death due to leaf senescence is equal for all four categories of sites, i.e. NI, LAT, INF and NLINF. The death rate of the canopy can be computed from the forcing function by calculating the derivative of biomass with respect to time: RDYING = $-$DERIV(0., TLBM). To prevent an increase in the number of lesions due to the growth of leaf mass, a provision is needed that sets a lower limit of zero, so:

$$RDYING = -AMIN1(0., DERIV(0., TLBM))$$

The relative mortality rate of both leaf area and number of different categories of sites is now RMRLA = RDYING/TLBM. The area of dead leaves is accumulated using the CSMP integral function:
DEADLA = INTGRL(0., RDYING * SLA).

Further, RMRLA is introduced into the boxcar trains for latent and infectious sites. The number of sites that are no longer infectious is now calculated as:

$$NLINF = INTGRL(0., REM - RMRLA * NLINF)$$

3.1.5 Computation of the relative growth rate

Each infectious site produces spores, but these must disperse, land, germinate and infect before new lesions can be produced. Although some models of dispersion and germination have been developed (Chamberlain, 1972; Legg & Powell, 1979; Waggoner in de Wit & Goudriaan, 1978; see Section 3.2), the data available on most fungal epidemics are insufficient for such a detailed, realistic approach. Unfortunately, therefore, it is often necessary to compact all these processes into a single variable: the 'effective spore' – the number of daughter lesions per mother lesion. In the previous model of Subsection 3.1.2, this single variable is expressed as the relative growth rate of the epidemic (R, number of daughter infections per mother lesion per day). In this way, a substantial amount of biological information is lumped together, which may decrease the value of the model as an explanatory tool. Some attempts have, however, been made to model the process of spore formation, spore emergence, spore dispersal, spore landing and spore germination. Attempts in cereal rusts have resulted in a submodel on spores. In these cereal rusts, the majority of spores die during dispersal or fail to germinate, and no more than 0.1 % of the spores produce lesions. The proportion of 'effective spores' depends on environmental conditions and on the condition and characteristics of the crop. For example, leaf wetness has a considerable effect on spore germination in wheat rusts, and therefore greatly influences the number of daughter lesions per mother lesion per day. This effect of leaf wetness may be a 'yes/no' reaction; for example, in potato

late blight, *Phytophthora infestans*, germination occurs only if there is free water on the leaves. In many cases, the reaction of the fungus is less abrupt, and the number of effective spores gradually declines as leaf wetness decreases.

Another important factor is temperature. Temperature affects the germination period, spore mortality and rate of infection. These processes are incorporated into the one variable that comprises all the sporal processes: the relative growth rate. In our simple simulator of a mildew epidemic, a preliminary model of spore production, spore death and spore germination is incorporated (Figure 26). Spores are produced at a particular rate. After dispersal, a fraction of these spores is caught. The number caught depends on the leaf area index, LAI and the fraction of intercepted spores per leaf layer FINTLL. Only the spores landing on a site that has not yet, been infected have a chance to establish an infection. The number of caught spores on a vacant site (INTSPO) increases with:

$$IRPSP = R * INFTOT * FINTLL * LAI * (1 - OCC/NMAX)$$

in which IRPSP is the interception rate of produced spores, R the spore production rate per infectious lesion, INFTOT the number of infectious lesions and OCC the number of occupied sites. The spores that have been caught begin to germinate, at a rate that is dependent on the average germination period and the number of spores available:

$$GRISP = RGRISP * INTSPO$$

in which GRISP is the germination rate of intercepted spores, RGRISP the inverse of the germination period (Table 7) and INTSPO the number of intercepted spores. Not all spores germinate, some die during development. The relative spore mortality rate per day is often calculated from the experimentally determined fraction of germinated spores as:

$$RMRISP = (1 - GF)/GP$$

in which GF is the germinated fraction and GP the germination period.

Figure 26. Relational diagram of a model of a fungal epidemic.
State variables: LATTOT = latent lesions, INFTOT = infectious lesions, NLINF = no longer infectious lesions, INTSPO = intercepted spores, GERSPO = germinated spores.
Rate variables: RIR = real infection rate, EIR = effective infection rate, REM = rate of no longer infectious lesions, MR... = mortality rates, IRPSP = interception rate of produced spores, GRISP = germination rate of intercepted spores.
Auxiliary variables: CP = colonization period, LP = latency period, IP = infectious period, GP = germination period, RMR... = relative mortality rates, R = spore production rate per lesion, FINTLL = fraction intercepted spores per leaf layer, TLBM = total leaf biomass, LAI = leaf area index, SLES = surface of a single lesion, NMAX = maximum number of sites, OCC = occupied sites, SLA = specific leaf area, TEMP = temperature.
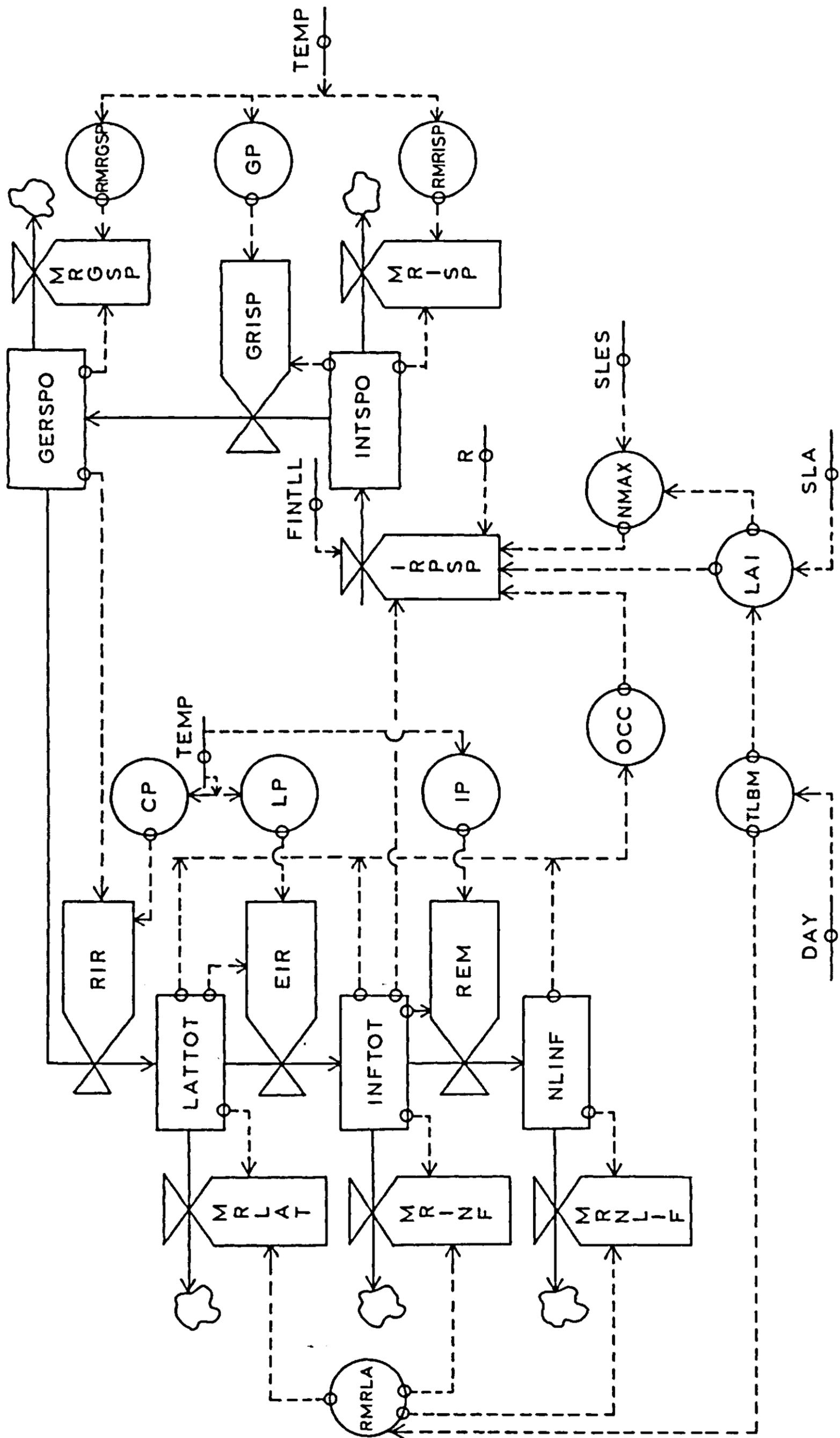
92

Table 7. Relation between tempera-
ture and germination period (days)
for mildew spores.

Temperature (°C)	Germination period (days)
10	30
15	10
20	8
25	4
30	3

Exercise 35

What is wrong with this method of computing relative mortality? Illustrate the consequences of this incorrect computation method and give the correct method.

The real infection rate (RIR, number ha^{-1} day^{-1}) is now calculated from the number of germinated spores (GERSPO):

RIR = RIRGSP * GERSPO

in which RIRGSP is the inverse of the time it takes a germinated spore to colonize the host tissue.

Not all germinated spores are successful, some die. Like germinating spores, the relative mortality rate per day of germinated spores can be calculated from the fraction of successful spores and the time it takes a germinated spore to colonize the host tissue. The number of infected sites per germinated spore is called the colonization ratio.

A first concept for modelling spore germination has now been presented, although germination is not considered in detail in the model presented here. For instance, the germinating spores are lumped in one development class; but not all spores, produced at one moment, germinate at the same time: there are delays and dispersion in time. Again, the subroutine BOXCAR may be used to mimic this dispersion (see Section 2.2).

The model can now be used to simulate a polycyclic fungal epidemic. One important, implicit assumption may yet be incorrect: the leaves are all assumed to be equally sensitive to the fungus, but in many cases ageing leaves are less

sensitive than those that have just appeared. To include this effect, the epidemiological model should be coupled to an appropriate crop model. Various examples of coupling are presented in Section 4.3.

3.1.6 Another approach, leaf area instead of sites

The arbitrary unit of fungal density, number of lesions and the relation with the host plant is a considerable simplification. However, observations are seldom made in terms of the number of pustules or sites, but rather in the fraction-infected leaf area. To bring the model more in line with these observations, it is probably better to express disease intensity in leaf area infected instead of number of lesions.

The leaf area may contain four characteristics: sound leaf area, leaf area with latent lesions (TLATLA), leaf area with sporulating lesions (TINFLA) and leaf area with old, no longer sporulating lesions (NLIFLA). Spore production is still computed in a way similar to that in the 'site' model:

$$IRPSP = R * INFTOT * FINTLL * LAI * (1 - DISLA/LAI)$$

INFTOT has now to be computed from the leaf area with sporulating lesions:

$$INFTOT = TINFLA/SLES$$

The diseased leaf area (DISLA) is calculated as:

$$DISLA = TLATLA + TINFLA + NLIFLA$$

The real infection rate is multiplied by the leaf area of a single lesion (SLES) to change the growth of the number of infected sites into growth of infected leaf area:

$$RIRLA = RIR * SLES$$

A complete listing of the simulation program of this epidemic simulator is given in Figure 27.

Exercise 36
Study the listing of the epidemic simulator. If possible, run the program with the given values of the parameters, and with other selected values.

3.1.7 Insects and mites

The example given above was based on a fungal epidemic. A more detailed example of the simulation of a fungal epidemic, *Helminthosporium maydis*, is described by Waggoner, in de Wit & Goudriaan (1978). Simulation of the

Figure 27. Listing of a program to simulate a powdery mildew epidemic. The same BOXCAR subroutine has to be used as in Figure 22, Section 2.2.

```
TITLE EPIDEMIC POWDERY MILDEW

STORAGE ILATLA(4),LATLA(4),IINFLA(3),INFLA(3)
FIXED N1,N2

INITIAL
*crop-parameters
PARAM SLA=20.E-4
*parameters connected with the fungus
PARAM STDAY=100.,N1=4,N2=3
TABLE ILATLA(1-4)=5.E-4,3*0., IINFLA(1-3)=3*0.
PARAM SLES=3.5E-10,R=1.E3,FINTLL=0.01

DYNAMIC
DAY    =STDAY + TIME
TEMP   =AFGEN(TEMPT,DAY)

*the crop
TLBM   =AFGEN(TLBMT,DAY)
LAI    =TLBM * SLA
*         TLBM:Total Leaf BioMass in kg(leaf)/ha(soil)
RDYING=-AMIN1(0.,DERIV(0.,TLBM))
RMRLA =RDYING/TLBM
*         RMRLA:Relative Mortality Rate Leaf Area in 1/day
DEADLA=INTGRL(0.,RDYING*SLA)
*         DEADLA:DEAD Leaf Area in ha(leaf)/ha(soil)

*the fungus
**spores
INTSPO=INTGRL(0.,IRPSP-MRISP-GRISP)
GERSPO=INTGRL(0.,GRISP-MRGSP-RIR)
*         INTSPO:INTercepted SPOres in number/ha
*         GERSPO:GERminated SPOres in number/ha
*         IRPSP:Interception Rate of Produced SPores in number/ha/day
*         MR-,GRISP:Mortality and Germination Rate of Intercepted
*         SPores in number/ha/day
*         MRGSP:Mortality Rate of Germinated SPores in number/ha/day
*         RIR:Real Infection Rate in number/ha/day
IRPSP =R * INFTOT * FINTLL*LAI * (1.-DISLA/LAI)
INFTOT=TINFLA/SLES
*         R:spore production rate in number/infectious lesion/day
*         TINFLA:Total INFectious Leaf Area in ha/ha
*         SLES:Surface of a single LESion in ha
*         INFTOT:TOTal number of INFectious lesions in number/ha(soil)
*         FINTLL:Fraction INTercepted spores per Leaf Layer
*         DISLA:DISeased Leaf Area in ha/ha
MRISP =RMRISP*INTSPO
RMRISP=AFGEN(RMRIST,TEMP)
*         RMRISP:Relative Mortality Rate Intercepted SPores in 1/day
GRISP =RGRISP*INTSPO
RGRISP=AFGEN(RGRIST,TEMP)
*         RGRISP:Relative Germination Rate Intercepted SPores in 1/day
MRGSP =RMRGSP*GERSPO
RMRGSP=AFGEN(RMRGST,TEMP)
*         RMRGSP:Relative Mortality Rate Germinated SPores in 1/day
RIR    =RIRGSP*GERSPO
RIRGSP=AFGEN(RIRGST,TEMP)
*         RIRGSP:Relative Infection Rate Germinated SPores in 1/day  .
```

```
**infected leaf area
RIRLA =RIR*SLES
*        RIRLA:Real Infection Rate expressed in Leaf Area in ha/ha(soil)/day

*Development Rate

DRLAT =AFGEN(DRLATT,TEMP)
DRINF =AFGEN(DRINFT,TEMP)
*Relative Dispersion
RDLAT =AFGEN(RDLATT,TEMP)
RDINF =AFGEN(RDINFT,TEMP)

NOSORT
LATO,LATLA,TLATLA,MRLAT,EIRLA,GAMMA1,GCYCL1=...
              BOXCAR(1,ILATLA,DRLAT,RDLAT,RMRLA,RIRLA,N1,DELT,TIME)

INFO,INFLA,TINFLA,MRINF,REMLA,GAMMA2,GCYCL2=...
              BOXCAR(2,IINFLA,DRINF,RDINF,RMRLA,EIRLA,N2,DELT,TIME)
SORT

NLIFLA=INTGRL(0.,REMLA - RMRLA*NLIFLA)
DISLA =TLATLA+TINFLA+NLIFLA

FUNCTION TLBMT =      0.,75.,   70.,   75.,   90., 100., 115., 200.,...
                 130.,1250., 150.,2200., 180.,2250., 200.,2000.,...
                 220.,1300., 240.,1., 300.,1.
FUNCTION RMRIST= 0.,0.12, 10.,0.12, 15.,0.36, 20.,0.46, 25.,0.92,...
                 30.,1., 35.,1.
FUNCTION RGRIST= 0.,0., 7.,0., 10.,0.0333, 15.,0.1000, 20.,0.1250,...
                 25.,0.2500, 30.,0.3333
FUNCTION RMRGST= 0.,20., 10.,16., 15.,18., 20.,20., 30.,24.
FUNCTION RIRGST= 0.,1., 10.,1.8, 15.,2., 20.,2.2, 30.,2.4
FUNCTION DRLATT= 0.,0., 7.,0., 10.,0.0625, 15.,0.1250, 20.,0.2500,...
                 25.,0.4000, 30.,0.4540
FUNCTION DRINFT= 0.,0., 7.,0., 10.,0.1000, 15.,0.2500, 20.,0.3333,...
                 25.,0.5000, 30.,1.0000
FUNCTION RDLATT= 0.,0., 7.,0., 10.,0.1875, 15.,0.2500, 20.,0.3750,...
                 25.,0.4000, 30.,0.4545
FUNCTION RDINFT= 0.,0., 7.,0., 10.,0.3000, 15.,0.5000, 20.,0.3333,...
                 25.,0.2500, 30.,0.2500
FUNCTION TEMPT = 0.,2., 60.,8., 120.,15., 180.,28., 240.,15., 300.,2.

METHOD RECT
TIMER FINTIM=120., DELT=0.05, PRDEL=5.
PRINT DAY,TLBM,LAI,DISLA,TLATLA,TINFLA,NLIFLA,DEADLA,RMRLA,...
      INTSPO,GERSPO,RIR,RIRLA
END
STOP

C********************************************************************C
      SUBROUTINE BOXCAR(COUNT,AI,DEVR,RD,RMR,INFL,N,DELT,TIME,
     S                  AO,A,ATOT,MORFL,OUTFL,GAMMA,GCYCL)
C********************************************************************C
```

population dynamics of a pest is roughly similar to that of a fungal epidemic, so this model can also be used to simulate the population growth of insects or mites. This will be illustrated in Section 3.4 where a detailed predator-prey model for an acarine system is discussed, and in Section 4.4 which considers a model for a pest epidemic (cereal aphids) combined with a crop growth model for wheat.