

EPIMUL, a simulator of foci and epidemics in  
mixtures of resistant and susceptible plants,  
mosaics and multilines

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P. Kampmeijer and J.C. Zadoks

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# 1 Introduction

In epidemiology aspects of time and space are of utmost importance. A theory on the progress of disease with time has been developed by van der Plank (1963) which resulted in Equation (1.1). Several aspects of space have been described by Gregory (1945, 1968), van der Plank (1963, 1975), and Waggoner (1962). But a theory combining aspects of time and space does not exist. With computer simulation an integrated approach is possible. Computer programs which describe the influence of both time and space on the epidemic, have been written by Kiyosawa (1972, 1976) and Shrum (1975). A model is presented here, EPIMUL76, with which the combined influence of time and space is further studied.

Simulation models can be classified according to the extent they conform to reality. Waggoner (1969, 1972, 1974) adheres to models that are as realistic as possible. Such models are said to be a powerful tool in disease prediction; however many parameters have to be estimated for lack of experimental data. EPIMUL is a theoretical model that represents an abstraction of reality only. This model is designed so that we can learn something of the epidemiologic parameters themselves. Variation of parameters in consecutive runs leads to detection of those factors that are most important for the development of the epidemic. In EPIMUL emphasis is laid on spore dispersal and host plant resistance.

An epidemic is a multidimensional phenomenon. It has the three dimensions of space: length, width, and height, the dimension of time, and the dimension severity. Epidemiologic models, analytic as well as simulation models, usually plot severity against time in a two-dimensional space. In EPIMUL the severity is plotted against time, length, and width in a four-dimensional space; EPIMUL also plots severity against distance.

The calculation of the increase of the pathogen per unit of time is based on van der Plank's equation (1963):

$$\frac{dx_t}{dt} = R_c (x_{t-p} - x_{t-i-p}) (1 - x_t) \quad (1.1)$$

as adapted to dynamic simulation by means of a digital computer (Zadoks, 1971). The time unit or integration step is one day. Every day the disease severity is updated. In the present model this updating does not take into account the effects of weather and of host development. This omission is not a fundamental point here, but a matter of computing capacity.

The computer program, although first written in CSMP, is given here in FORTRAN. The change from CSMP to FORTRAN saved about 20 percent computing time. Moreover the advantages of CSMP with its special features, such as INTGRL and DELAY, could not be used because of the many arrays in this program. The reader is expected to have a general knowledge of FORTRAN.

The computer used is a DEC10, the main computer of the Agricultural University in Wageningen. It has a core size of 128 K of which 48 K is available for a single user.

## 2 Description of EPIMUL

### 2.1 Introduction

EPIMUL was initiated as a model to simulate epidemics in multilines. A multiline (Browning and Frey, 1969), shorthand for multiline variety or composite variety, is a mixture of plants that have nearly identical agricultural properties, but differ in the genetic basis of resistance against disease. In a multiline a part of the spores falls on resistant plants, fails to reproduce and thus is eliminated from the epidemic process. This elimination can be simulated by the reduction of the number of spores by a factor representing the proportion of susceptible plants. A computer program doing just this gave results confirming those of Zadoks (1967). Obviously this approach is a very crude one. A more fundamental approach is needed.

Field trials with yellow rust (*Puccinia striiformis*) on wheat in the Netherlands showed the suppression of focus formation to be a major effect of varietal mixtures (Zadoks, 1958, 1959) when inoculum was scarce but arrived early. The modalities under which mixtures of varieties and multiline varieties show best effects will be discussed later. As under Netherlands conditions the suppression of foci was so conspicuous, and this effect satisfactorily explained the favourable effect of variety mixtures on the control of yellow rust epidemics (Zadoks, 1972), efforts were concentrated on the modelling of the development of foci. According to a formal definition (Anonymus, 1953), 'a focus is the site of local concentrations of diseased plants or disease lesions, either about a primary source of infection or coinciding with an area originally favourable to establishment, and tending to influence the pattern of further transmission of disease'. The present interest is mainly in the primary source of infection.

Other simulation models (Rijdsdijk, 1975; Waggoner et al., 1969; Zadoks, 1971) were based on the assumption that inoculum is evenly distributed. However the development of a focus can be studied only if a spatial distribution of inoculum is taken into account. Then as many epidemics need to be simulated as there are points in space to be studied. Differentiation in

space is part of EPIDEMIC (Shrum, 1975), a program written for uniform crops, and part of the programs of Kiyosawa (1976).

The computer program consists of two sections. In the initial section the parameters are read. For some parameters default values have been set. From the basic parameters derived parameters are calculated. The dynamic section contains the simulation instructions. Some calculations are written as separate functions and subroutines.

## 2.2 Compartmentalization of space

In EPIMUL space is represented by a square block, divided into square compartments. In each compartment a separate epidemic is simulated. The compartments influence each other only by way of spore transport. This mutual influence is represented in the model by the dispersal formula. All spores leave the compartment and are dispersed along a certain gradient. The centre of dispersal is assumed to be in the middle of the compartment; while the whole area of the compartment can receive spores. The spores are then evenly divided over the compartment. During the dispersal calculations each compartment once is the centre of dispersal spreading spores over all compartments, including itself; and each compartment can receive spores from all compartments, again including itself.

Some spores are dispersed so far that they land outside the block. These spores are omitted from the calculations. Spore influx from outside the block is ignored here.

The time unit of the calculations is one day. Each day the progress of the epidemic is computed for each compartment, and each day the spore dispersal is determined. Though dispersal as a process takes time, this time is disregarded. The result as seen in actual spore catches is thought to be obtained in one instant.

The number of compartments determines the detail in which the epidemic is studied. The number of dispersal calculations increases quadratically with the number of compartments, while the number of memory locations has some effect on the time needed for the simulation. Here a number of 400 compartments is chosen (Fig. 1): the resulting detail is adequate for the present and the computing time over some 100 days is less than 15 minutes. This compromise allows many runs to be made, whereas increased detail reduces the number of runs possible. For example a block of 1600 compartments ( $= 40 \times 40$ ) necessitates



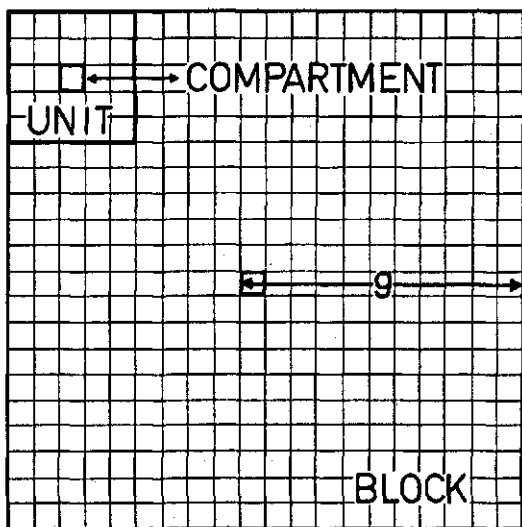


Fig. 1 | Lay-out of the simulator EPIMUL: block, unit, and compartment. For transect g the gradient is printed.

a computing time of more than three hours.

The multiline effect can no longer be calculated with a simple reduction factor (see Section 2.1). Each compartment belongs to one and only one line. The possibility of mutual infection is determined by a compatibility factor that has either a yes or no value. The distribution of compatibility values over the compartments is random. So far the model does not differentiate mathematically between a multiline and a mosaic; the physical size of a compartment compares with the multiline situation when the compartment is small and with a mosaic crop pattern when the compartment is large. The mosaic may consist of different cultivars covering an area (the block), or it may consist of different crops, one of which is studied.

Internal differentiation within a compartment cannot be calculated (by definition) so that for mosaics the internal differentiation for a small area is lost. A part of this differentiation is regained by assigning a 'unit'. A unit is an area of adjacent compartments with identical compatibility values. Introduction of the unit permits comparison of different aggregations of compartments.

## 2.3 Model parameters

The basic parameters of the model can be divided into three groups; the dimensions are given in square brackets. A dimensionless parameter is indicated by [1].

### 1. Parameters defining the crop:

- RIBB is the length in metres of the side of a square compartment. [L]
- M is the number of compartments in one row. As compartments are arranged in a square, the total number in a block is  $N = M^2$ . [1]
- LAI is the leaf area index or the leaf area in  $m^2$  per  $m^2$  of soil. [1]
- NVAR is the number of cultivars, lines or crops. [1]
- CMPU is the number of compartments per unit. The unit, the smallest area that can be covered by a cultivar, is defined by its number of compartments (see Section 2.6.2). [1]

### 2. Parameters defining the pathogen:

- NLPD is the latent period in days. [T]
- NIPD is the infectious period in days. [T]
- DMFR is the maximum number of daughter lesions produced per mother lesion per day. This number can be derived from the number of spores produced, but in EPIMUL it is a set value. The real number of daughter lesions per mother lesion per day depends also on the correction factor  $1-x_t$  in Equation (1.1) (Zadoks, 1971). [T<sup>-1</sup>]
- LESION is the area of one lesion in  $mm^2$ . [L<sup>2</sup>]
- HALF is a measure for the spore dispersal (see Section 2.4). [L]

### 3. Parameters organizing the program:

- FINTIM, the number of days simulated. [T]
- OUTPUT, the output frequency of the table and the graphs. [1]
- INFO, the array index indicating the compartment where the simulation is initialized. [1]

The derived parameters are:

- N, the number of compartments in the block. [1]
  - SITE, the number of available infection sites. [1]
- $SITE = LAI * RIBB^2 * N / (LESION * 1.E-6)$ .  
For example when  $LAI = 5$ ,  $RIBB = 5$ ,  $N = 400$ , and  $LESION = 10$  the number of infection sites, or units of infection is  $SITE = 5.10^9$ .

- FRSI, the number of sites in one compartment:

FRSI = SITE/N.

[1]

- NSUS, a correction factor for the number of compartments indicated as susceptible by means of COMPAT (see Section 2.6.1). The expectation value of NSUS is NVAR, but it can deviate from NVAR due to randomization procedures (see Section 2.6.1).

[1]

## 2.4 An epidemic in a compartment

This part of the model discusses computations performed for each compartment on each day. In EPIMUL this is N times per day (normally  $N = 400$ ). Apart from spore fluxes a compartment is a closed entity: given the spore influx (FLIN), the spore outflux (FLOF) is computed, using the daily multiplication factor (DMFR) and variables that are only available for this compartment. This part of the model is comparable to other simulation models for epidemics which are more comprehensive and more sophisticated. Here, however, all meteorological influences, such as temperature and rain, and also plant growth, are excluded, to keep the program concise and the computation time short.

The principles of computation as described by Zadoks (1971) are followed here (Fig. 2). The course of the epidemic is described by a flow of sites through four levels indicated by the following integrals: vacant sites (VAC), latent lesions (LAT), infectious lesions (INF), and removed lesions (REM). The amount of host material available in this compartment, expressed in sites, is FRSI.

The transitions between levels are described as rates. The rate of occupation (ROCC) is computed from the vacant fraction  $VAC/FRSI$  and from the amount of incoming spores (FLIN), with a limiting condition  $0 \leq ROCC \leq VAC$ . The rate of infection (RINF) and the rate of removal (RREM) are computed from ROCC by means of the function PERIOD (see Section 2.7), using only the latent period (NLPD) and the infectious period (NIPD). Because there is no meteorological variation the periods NLPD and NIPD are kept constant during the whole run.

As an epidemiologic model needs to work with discrete spores and discrete lesions, all rates and integrals are kept at discrete values in contrast with those used by Zadoks (1971) but in accordance with EPIDEM (Waggoner & Horsfall, 1969).

```

DO 80 I=1,N
VAC=LIMIT(0.,FRSI,FRSI-LAT(I)-INF(I)-REM(I))
ROCC=LIMIT(0.,VAC,FLIN(I)*VAC/FRSI)
RINF=PERIOD(1,I,NLPD,ROCC)
RREM=PERIOD(13,I,NIPD,RINF)
LAT(I)=LAT(I)+ROCC-RINF
INF(I)=INF(I)+RINF-RREM
REM(I)=REM(I)+RREM
FLOF(I)=DMFR*INF(I)
80 CONTINUE

```

- \*) On the third line VAC/FRSI stands for the correction factor  $1-x_c$  in Equation (1.1). The upper limit of ROCC is reached when  $FLIN(I) > FRSI$ . The maximum of NLPD + NIPD is 20 days, as is the reserved space in the function PERIOD.

## 2.5 Spore dispersal

### 2.5.1 The dispersal formula

The fungal generation cycle is considered to begin when a propagule infects a suitable host. Propagules are dispersed by rain splashing, turbulence, wind, insects, etc.

Spore dispersal is the process from take off to deposition; every spore goes through an act of dispersal (van der Plank, 1975). Spread is the collective result of many acts of dispersal. Spread cannot be seen, but it results in a spatial distribution of disease which can be assessed. Spatial distribution of secondary infections can be visualized as a concave curve, representing severity against the distance of the source. There are many secondary infections close to the origin; from the centre of the focus outwards the number of secondary infections decreases fast initially and then approaches slowly to zero. Spore dispersal is the action resulting in spread. There are many functions depicting this type of dispersal, which seem equally suitable: hyperbolic functions, logarithmic functions, exponential functions, the last including normal distributions.

The dispersal formula to be used in the present model must satisfy the following conditions:

- Integration of the function must be possible: the boundary conditions of this function must be such that infinite values are not found.
- Negative infection densities may not occur.

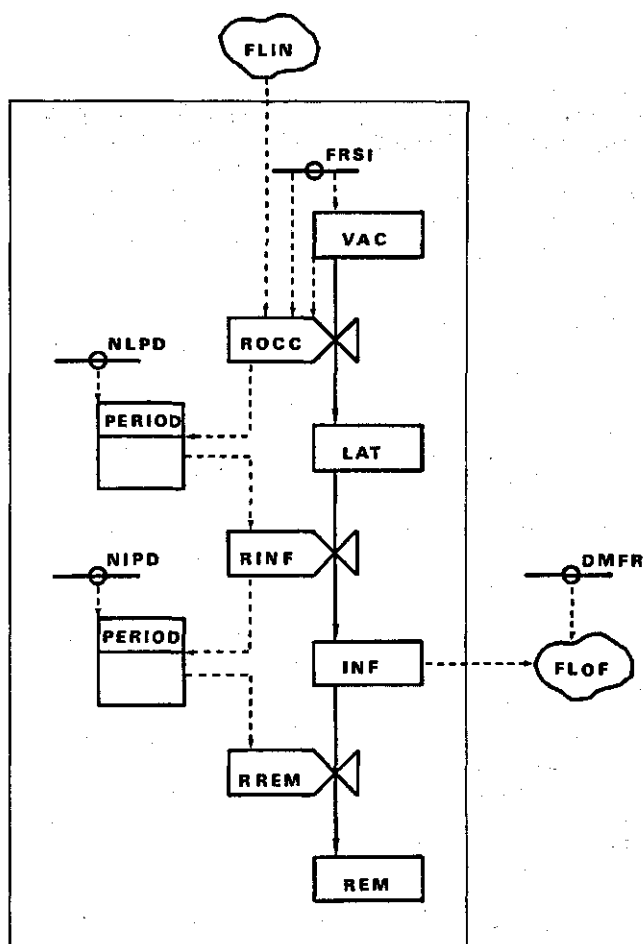


Fig. 2 | Flow diagram of an epidemic in a compartment, showing the complete picture of the flow channel, the information network, and the constants. The amount of incoming spores (FLIN) is a result of the spore dispersal routine. For each compartment the calculations give the number of spores entering again into the spore dispersal (FLOF).

- The formula must take into account dispersal in the two dimensions of the horizontal plane.
- The volume under the two-dimensional curve has to be a measure for the total number of spores dispersed.

To define the dispersal formula, the model of turbulent diffusion was adopted. In a set of eddies, which each can be subdivided into smaller eddies, and so on, the movement of an eddy provides for the spore transport, while spore transport between the eddies and the surrounding air provides for dilution of the spore cloud. Turbulence consists of two components: mechanical turbulence caused by the wind, and thermal turbulence caused by a negative temperature gradient in the lower atmosphere. Both forms of turbulence are effective in enlarging the spore cloud. Here only their combined effect is considered. Wind causes horizontal displacement of the whole spore cloud. Crosswind the spore concentration always follows a Gaussian distribution (Pasquill, 1962), in the direction of the wind the distribution depends on the kind of spore source.

Gregory (1945, 1968) assumed the source to be continuous. A continuous source is comparable to a chimney. During a certain period spores are emitted at constant rate. Gregory proved that a hyperbole is a good description for the spore concentration downwind. However, it is impossible to apply his formula to the source itself, because the hyperbole reaches infinity at the source.

A momentaneous source expels all spores at once. For such a source Pasquill stated that a Gaussian distribution applies in the direction of the wind, the place of the maximum concentration depending on wind velocity. Roberts et al. (1970) constructed a continuous source from a time series of momentaneous sources, using the model of Pasquill.

Schrödter (1960) computed the probable flight line, from which the average travelling distance of the spores can be computed. Shrum (1975) used a Gamma-function with this distance as the median. He chose a Gamma-function, because it is very easy to change the form of the function from a negative exponential to a nearly normal distribution by changing the parameters  $\alpha$  and  $\beta$ . His article suggests to compute for about 500 classes of spores where they will come for every source compartment: a very time-consuming method. Shrum's 1975 paper gives, however, no evidence for the actual use of the method advocated.

In EPIMUL the formula of Pasquill is applied. The spore source is considered to be momentaneous, and the whole dispersal pro-

cess takes place within one day. Along the x coordinate and along the y coordinate there is a Gaussian distribution, both distributions with the same variance. The vertical component has not been considered. The wind effect is not included in the computation as it would cost too much computing time. Therefore every formula remains an approximation, but it answers the present purpose.

For any compartment serving as a source, the concentration  $\chi$  of the spores produced is a function of the distance to the compartment

$$\chi = \frac{1}{2\pi\sigma^2} \cdot \exp\left(-\frac{x^2+y^2}{2\sigma^2}\right) \quad (2.1)$$

Integration over the area of the receiving compartment will produce the fraction of the spores entering this compartment (see Fig. 3).

$$\int_{x_1}^{x_2} \int_{y_1}^{y_2} \frac{1}{2\pi\sigma^2} \cdot \exp\left(-\frac{x^2+y^2}{2\sigma^2}\right) dx dy \quad (2.2)$$

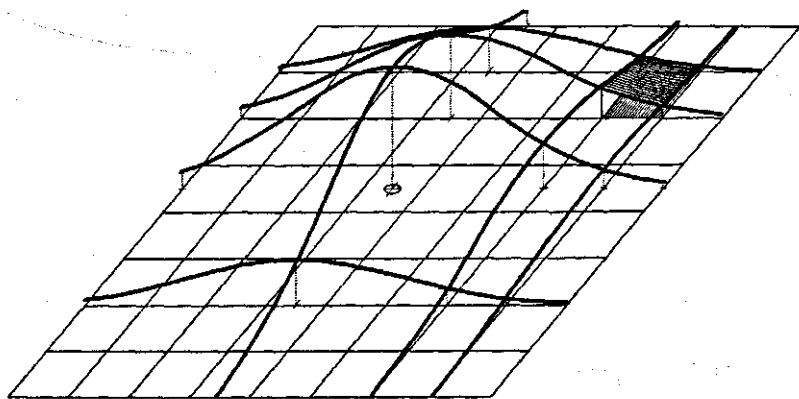


Fig. 3 | Spore concentration in a two-dimensional space. The maximum concentration is in the middle of the source compartment. Integration over the area of the receiving compartment produces the fraction of spores entering this compartment. The figure shows one source compartment (circle), and a receiving compartment (shaded). The curves illustrate the two-dimensional normal spore density distribution. Each compartment in turn serves as a source.

This may be rewritten as

$$\int_{x_1}^{x_2} \frac{1}{\sigma\sqrt{2\pi}} \cdot \exp\left(-\frac{x^2}{2\sigma^2}\right) dx \cdot \int_{y_1}^{y_2} \frac{1}{\sigma\sqrt{2\pi}} \cdot \exp\left(-\frac{y^2}{2\sigma^2}\right) dy \quad (2.3)$$

The single integrals are computed once, then stored in an array (ARRY). Integral (2.2) is obtained by multiplying two single integrals. In this way the computation of the distance between compartments is circumvented. The array-index of ARRY is determined by the position of the receiving compartment (I, II) relative to the sporulating compartment (J, JJ). Of course the positions of both the sporulating and the receiving compartments are indicated by array-indexes. The number of spores (SPORE) entering the receiving compartment is a result of the number of spores (FLOF2) leaving the producing compartment and the value of the integral at the receiving compartment.

$$\text{SPORE} = \text{FLOF2}(J, JJ) * \text{ARRY}(I-J) * \text{ARRY}(II-JJ)$$

## 2.5.2 Initialization of the dispersal

In EPIMUL the dispersal is measured by the parameter HALF. HALF is the distance in metres between the spore source and the place where the concentration is half of the concentration at the source. According to Equation (2.1) the concentration  $Q$  at the centre of dispersal is

$$Q = \frac{1}{2\pi\sigma^2} \quad (2.4)$$

$$\frac{1}{2}Q = \frac{1}{2\pi\sigma^2} \cdot \exp\left(-\frac{\text{HALF}^2}{2\sigma^2}\right) \quad (2.5)$$

$$\frac{1}{2} = \exp\left(-\frac{\text{HALF}^2}{2\sigma^2}\right) \quad (2.6)$$

From Equation (2.6)  $\sigma$  can be computed. In the model not  $\sigma$  but  $\frac{1}{\sigma\sqrt{2}}$  (= DIVE) is calculated.

$$\ln \frac{1}{2} = -\frac{\text{HALF}^2}{2\sigma^2} \quad (\text{note: } \ln = \log_e)$$



$$\ln 2 = \frac{\text{HALF}^2}{2\sigma^2}$$

$$\frac{1}{2\sigma^2} = \frac{\ln 2}{\text{HALF}^2}$$

$$\frac{1}{\sigma\sqrt{2}} = \frac{\sqrt{\ln 2}}{\text{HALF}} \quad (2.7)$$

DIVE = SQRT(ALOG(2.))/HALF.

In mathematics the error function  $\text{erf}(a)$  is defined as

$$\text{erf}(a) = \frac{2}{\sqrt{\pi}} \int_0^a e^{-t^2} dt \quad \text{with the complement}$$

$\text{erfc}(a) = 1 - \text{erf}(a)$  where  $t$  is an arbitrarily chosen symbol. Equation (2.8) applies for the borderlines of the compartments.

$$x \int_0^\infty \frac{1}{\sigma\sqrt{2\pi}} \cdot \exp\left(-\frac{x^2}{2\sigma^2}\right) dx = \frac{1}{2} \text{erfc}\left(\frac{x}{\sigma\sqrt{2}}\right) \quad (2.8)$$

An approximation of the error function, suitable for computers (Hastings, 1955), is used in the FUNCTION DISP. With this the values of ARRY can be obtained for the given value of HALF.

```

C
C   DIMENSION ARRY(-19,20)
C
  DO 50 I=1,20
    AI=I
    DIST=RIBB*(AI-.5)
    ARRY(I)=DISP(DIVE,DIST)
50  CONTINUE
C
  ARRY(0)=2.*(.5-ARRY(1))
C
  DO 60 I=1,19
    ARRY(I)=ARRY(I)-ARRY(I+1)
    ARRY(-I)=ARRY(I)
60  CONTINUE

```

### 2.5.3 The spore-sweeping routine

SPORE has been defined as a continuous variable. However, the compartmental calculations call for a discrete value of ROCC (see Section 2.4). For that reason the first infection of a compartment must consist of at least one whole spore. But after the first few cycles of dispersal most compartments have received only fractions of spores by means of SPORE. As fractions are rounded off to the next lower integer, ROCC remains zero in these compartments, and the compartmental epidemics cannot start. To overcome this handicap we have two options:

- the use of the spore influx per compartment as input in a probabilistic calculation.
- the use of a spore-sweeping routine which adds up spore fractions until a whole spore is obtained.

The second option has been chosen. Some target compartments are cleared of incoming fractions of spores that are transferred to a compartment, which then receives one unfractionated spore. The sweeping starts at one corner and spirals inwards according to Fig. 4. The result is practically non-directional.

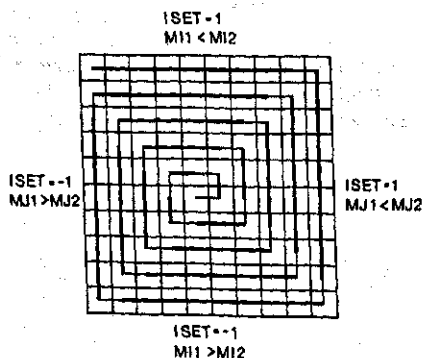


Fig. 4 | Rules for the spiral-like movement through a two-dimensional array. The spiral is a set of concentric squares. At one corner the jump is made inwards to the next square. The variable ISET determines the direction of array-scanning; MJ1, MJ2, MJ1, and MJ2 are the corners of the actual sides of the squares.

```

C
C   ISET = variable for direction of array-scanning
C   MI1, MI2 = array-indexes indicating begin and end of the
               computation in the horizontal direction
C   MJ1, MJ2 = idem (vertical direction)
C   LOOP = order number of a loop of the spiral
C   LOOPA , LOOPB = subdivisions of LOOP
C   I, J = actual array-indexes
C
  MI1=0
  MI2=20
  MJ1=1
  MJ2=19
  ISET=1
  J=1
  DO 180 LOOP=1,10
    MI1=MI1+1
    MI2=MI2-1
    DO 180 LOOPA=1,2
      DO 170 LOOPB=1,2
        GO TO (130, 150)LOOPB
170  I=MI2+ISET
      MI2=MI1+ISET
      MI1=I
      GO TO 170
150  J=MJ2+ISET
      MJ2=MJ1+ISET*2
      MJ1=J
170  CONTINUE
      ISET=ISET * -1
180  CONTINUE

```

The sweeping itself is obtained by the following set of statements:

```

C   (horizontal sweeping)
C
  IF (FLIN2(I,J).GE.1.)GO TO 140
  IF (FLIN2(I+ISET,J).GE.1.) GO TO 140
  FLIN2 (I+ISET,J)=FLIN2 (I+ISET,J)+FLIN2 (I,J)
  FLIN2 (I,J)=0.
140 CONTINUE

```

In the spiral I+ISET,J is the index of the compartment following that with I,J. The vertical sweeping follows the same principles as the horizontal sweeping.

## 2.6 Host resistance

### 2.6.1 Compatibility

Within limits one can use EPIMUL to simulate an epidemic in a multiline or in a crop with a mosaical pattern of lines. For that purpose a compatibility value is assigned to each compartment. The 400 compatibility values are stored in an array (COMPAT). Two compartments with the same compatibility value can infect each other. If their compatibility values differ, the crop in the receiving compartment is fully resistant: no infections occur.

In the model each line or cultivar has its own compatibility value. So the number of different compatibility values is equal to the number of lines (NVAR). The compatibility values are assigned to the compartment by a random generator with uniform distribution.

```
COMPAT(I) = IFIX(NVAR * RAN(DUMMY))
```

Because of the randomization, the number of susceptible compartments, which is  $N/NVAR$  on the average, varies somewhat. The program counts the number of susceptible compartments by comparing the compatibility values with the value of the initial focus.

```
JN=N  
COMPAR=COMPAT(INFO)  
DO 30 I=1,N  
  IF (COMPAT(I).EQ.COMPAR) GO TO 30  
  JN=JN-1  
30 CONTINUE
```

At two places in the program the compatibility is checked: first during the calculation of the course of the epidemic in a compartment, and second for the spore transport to or from a compartment. In both cases further calculations are useless when infection is impossible, and consequently, these parts of the program are skipped for the compartments in question.

### 2.6.2 Unit size

The relation between multiline and mosaic is somewhat difficult to grasp. The reader can imagine a mosaic pattern as a grouping of large fields with different compatibility values. He can see the multiline pattern as a grouping of miniature fields (with  $RIBB = 0.1$  m) with different compatibility values. There is no

essential difference in pattern between multiline and mosaic, but a large difference in the scale of events. EPIMUL operates on the hypothesis that there are no qualitative differences between processes on a large scale and on a miniature scale, so that the same equations and algorithms can be applied to both. The quantitative differences between the two scales of events are determined by the parameters RIBB and HALF.

From the epidemiologic point of view there is one serious objection to the hypothesis mentioned above. When the fields are large, plants within one field can infect each other, thereby essentially changing the epidemiologic pattern. When the fields are of miniature size (think of single-stem wheat fields;  $RIBB = 0.1$  m), mutual infection of plants is excluded; self-infection of plants can occur in the vertical direction but this (relatively small) effect is neglected.

The problem of the mutual infection of plants within a field is solved by creating units. A unit is a square section of the block consisting of adjacent compartments with identical compatibility values. The number of compartments per unit is given by the parameter CMPU.

## 2.7 The delay function PERIOD

In Section 2.4 it was stated that the number of successful inoculations determines the number of newly appearing infections after a delay which is called latent period (NLPD). This and other delays are handled by a function PERIOD. PERIOD is a delay function adapted to indexed variables (= the compartment numbers). A delay function stores its input as many time steps as indicated by the program, and retrieves the input when the indicated number of steps has passed. In PERIOD the variable (XIN) is stored in an array  $XX(I,II)$ , where the index I indicates the number of the compartment, and II indicates the place where the input has been stored one delay period before.

It is possible to use PERIOD for more than one input variable at the same moment. In the model the function is used for the delays caused by the latent period and by the infectious period. The different parts of the storage array are indicated by the location parameter L. The exact storage location of an input is found with a MOD function (the remainder when the time DAY is divided by the delay time DELA):

$$II = L + \text{MOD}(\text{DAY}, \text{DELA})$$

With this way of programming, the sum of NLPD and NIPD should

not exceed the 20 positions specified in the DIMENSION statement of XX.

```

FUNCTION PERIOD(L,I,DELA,XIN)
DIMENSION XX(400,20)
INTEGER DAY, DELA
COMMON DAY

```

C

```

II = L+MOD(DAY,DELA)
PERIOD=XX(I,II)
IF (DAY.LT.DE LA) PERIOD=0.
XX(I,II)=XIN
RETURN
END

```

## 2.8 Output of the model

### 2.8.1 Computation of the output variables

The output consists of the following data sets:

- a table with spore losses and disease severities.
- disease progress curves plotted on linear-linear, on log-linear, and on logit-linear scales.
- disease profiles and disease intensity maps showing severity on different days.

From these data sets the relevant information can be derived. For the completion of these data sets the following additional output data are calculated:

SEV	severity of the block expressed in number of sites; SEV is here the total of the infectants and the removals	[N] [1]
XSEV	log transformation of SEV	
RAT	relative visible attack, or the relative severity (this is the value x of van der Plank)	[1] [1]
LOGIT	logit transformation of RAT	[1] [1]
SPOT(I)	relative disease severity of a compartment	[1]
CENTR	relative severity of the initial focus	
SINK	sum of the fractions of the spores lost because they fall outside the block and those lost because of host plant resistance; spore losses due to saturation of sites are not considered by SINK	[1]

```

SUM=0.
TINF=0.
TREM=0.
DO 110 I=1,N
TINF=TINF+INF(I)

```

```

TREM=TREM+REM(I)
SPOT(I)=(INF(I)+REM(I))/FRSI
110 SUM=SUM+FLIN(I)
SEV=TINF+TREM
XSEV=ALOG10(SEV)
RAT=SEV/SITE
LOGIT=ALOG(RAT/(1.-RAT))
CENTR=SPOT(INFO)
SINK=(TINF*DMFR-SUM)/(TINF*DMFR)

```

RAT, SEV, XSEV, and LOGIT are different ways to represent the disease severity of the block. The disease severity per compartment (SPOT(I)) is used as input for two subroutines: PROFIL and DISPLA, representing aspects of focus formation. SINK and RAT are tabulated. CENTR, SEV, XSEV, and LOGIT are presented in printplots.

The interval for the printing of output data for the tables and for the graphs is determined by the parameter OUTPUT; the output frequency of PROFIL and DISPLA is set separately.

### 2.8.2 Preparing the printplots

PRTPLT is the subroutine that generates the graphs on the line printer, using subroutine GRAFIC. PRTPLT is used three times in succession:

```
- PRTPLT('NAME',VALUE,AMIN,AMAX)
```

This is the call for the graphs to be prepared. In this call the variable NAME is the name of the heading of the graph; VALUE is the value of the variable; and the minimum and maximum AMIN and AMAX determine the scaling factor so that the ordinate of the graph extends from AMIN to AMAX. When AMIN equals AMAX the scaling factor is computed in another way. A maximum of ten graphs can thus be made. At this stage the intermediate values still are in an array.

```
- PRTPLT('TIME')
```

The intermediate values for the graphs that have been stored in an array (ROW) are written on disk. With this way of handling data graphs can be of indeterminate length.

```
- PRTPLT('PLOT')
```

This call stands at the end of the simulation. It generates the graphs using the data on disk.

To accomplish these tasks PRTPLT is divided into three sections. The first section is used at the start of the simulation. The names of the variables to be plotted are noted, and the number of graphs (NC) is counted to organize disk area.

The second section of PRTPLT is of interest during the simulation. The VALUES from the input are stored in array ROW. Also the real minimum XMIN and the real maximum XMAX are computed.

In the third section, where the graphs are made, the data are read from disk. The heading of the graph is now printed, including the minimum and maximum computed in the second section. The scaling factor modifies all inputs for the subroutine GRAFIC into a scale with 50 points. When AMIN is equal to AMAX the scaling factor is determined by XMIN and XMAX.

```

DO 89 N=1,NC
DIFF=AMAX-AMIN
SCALE=50./DIFF
READ(20) (INP(I),I=1,NC)
WRITE (3,1010) INP(N),XMIN(N),XMAX(N)
80 READ (20,END=89) (ROW(I),I=1,NC),IDAY
VALUE =(ROW(N)-AMIN) * SCALE
CALL GRAFIC(IDAY,ROW(N),VALUE)
GO TO 80
89 CONTINUE

```

### 2.8.3 Line-printer graphs

The subroutine GRAFIC produces one line of a graph on the line-printer. First the order number of the variable on the abscissa is printed: the time step (usually the day) or the compartment number. Then the input variable VAL is printed in E-FORMAT. VAR determines one point of the graph. VAR is a continuous variable, but for the purpose of graph-making the points must be distributed over the ordinate consisting of 50 discrete printing positions. VAR is computed from VAL in the subroutine PROFIL and PRTPLT with a scaling factor. On the abscissa an asterisk is printed, on the other points hyphens, a plus, or blanks, depending on VAR. Sometimes VAR is greater than 50 or less than zero. When the limits of VAR are exceeded, a warning is given by printing the variables MIN and MAX. If VAR is less than zero MIN = '#', and if VAR is more than fifty MAX = '#'.  
 MAX = '#',

### 2.8.4 The disease profile

Subroutine PROFIL produces a graph, representing the disease profile around the initial focus. For this purpose the relative severities of the compartments 210-220 are used to compute values to be included in GRAFIC. In all runs the epidemic



starts in compartment 210. Compartment 220 is on the outside of the block. The row 210-220 is chosen for the ease of programming.

In most runs a profile on log-linear scales is made. With a chosen minimum AMIN and a scaling factor SCALE the logarithmic values of relative severities are transformed for use as an input into GRAFIC. For AMIN and SCALE the values -5 and 10 are chosen; so points of the graph are found for relative severities between  $1.E-5$  and 1.

## 2.8.5 Display of the focus

Subroutine DISPLA produces a map of the severity in the 400 compartments on the line-printer. Each compartment is represented by a square with a certain gradation of gray, which is an indication of the amount of disease in this compartment. In Fig. 5 eleven severity classes are given.

0.0 - 0.001	:	:
0.001 - 0.101	: : : : :	: : : : :
0.101 - 0.201	: : : : :	: : : : :
0.201 - 0.301	: : HHHHH	: : HHHHH
0.301 - 0.401	: : : : : //	: : : : : //
0.401 - 0.501	: : : : : + + + + +	: : : : : + + + + +
0.501 - 0.601	: : NNNNN ZZZZZ	: : NNNNN ZZZZZ
0.601 - 0.701	: : : : : TTTTT	: : : : : TTTTT
0.701 - 0.801	: : HHHHH TTTTT	: : HHHHH TTTTT
0.801 - 0.901	: : + + + + + NNNNN ZZZZZ	: : + + + + + NNNNN ZZZZZ
0.901 - 1.	: : HHHHH TTTTT : : : : :	: : HHHHH TTTTT : : : : :

Fig. 5 | Severity classes and the construction of gradations of gray from normal printing characters.

With DISPLA it is possible to visualize the focus development and to discover daughter foci, if any.

### 3 Results

#### 3.1 Model performance and distance parameters

The model has two independent parameters of distance. One is RIBB, measuring the length in metres of the side of a compartment. The other is HALF, indicating the distance in metres from the spore source to the place where the spore density reaches the value 0.5 (see Section 2.5.2). RIBB defines the crop, HALF determines the behaviour of the pathogen. Both parameters have the dimension length. The complexity of the model does not allow a simple description of the relation between its performance and the two distance parameters. To study this relation some results have been plotted against the quotient of HALF and RIBB, a dimensionless ratio called HALRIB.

As a characteristic speedometer (van der Plank, 1975) we chose the logistic infection rate. The growth rate of the epidemic depends on HALRIB and is measured by the logistic infection rate ( $r$ ) at half-time  $t(0.5)$  (this is the time at which the epidemic reaches a relative severity of  $x = 0.5$ ). The value  $r$  can be calculated for the central compartment or for the block as a whole (Fig. 6.a, b). The absolute values of RIBB and HALF have some but not much effect; it is their ratio that matters. The logistic infection rate of the central compartment ( $r_c$ ) is largest when HALRIB is smallest, that is when most spores remain contained in the central compartment. With increasing HALRIB,  $r_c$  shows a decreasing trend because of spore loss. The logistic infection rate of the block ( $r_b$ ) has a maximum at HALRIB = about 10. Apparently the balance between the spore loss and the spore influx is such at HALRIB = 10 that the average compartment has the highest  $r$ .

Fig. 6 a | The relation between the logistic infection rate  $r_c$  of the central compartment at half-time  $t_c(0.5)$  and HALRIB, plotted on linear-log paper. High values of  $r_c$  are found at low values of HALRIB; it is difficult to see why the decrease of  $r_c$  is not monotonous. Abscissa: HALRIB = HALF/RIBB. Ordinate: logistic infection rate of the central compartment  $r_c$  at half time of the compartment  $t_c(0.5)$ . Entries: RIBB = 5. (—), RIBB = 0.5 (---), RIBB = 0.05 (.....). Parameters: NLPD = 8, NIPD = 8, DMFR = 10., NVAR = 1., CMFU = 1.

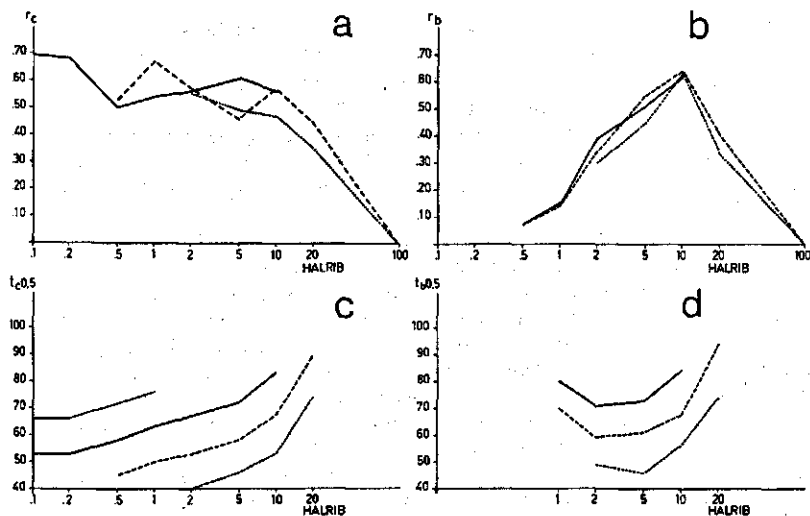


Fig. 6 b | The relation between the logistic infection rate  $r_b$  of the block at half-time  $t_b(0.5)$  and HALRIB, plotted on linear-log paper. The optimum value of HALRIB under the conditions of the experiment is about 10. Abscissa: as in Fig. 6 a. Ordinate: logistic infection rate of the block  $r_b$  at half-time of the block  $t_b(0.5)$ . Entries: as in Fig. 6 a. Parameters: as in Fig. 6 a.

Fig. 6 c | The relation between the time in days at which the severity of the central compartment reaches the value 0.5 (half-time of central compartment,  $t_c(0.5)$ ) and HALRIB, plotted on linear-log paper. The minimum value of  $t_c(0.5)$  is obtained when all spores remain contained within the central compartment. Abscissa: HALRIB = HALF/RIBB. Ordinate: Number of days from start to  $t_c(0.5)$ . Entries: RIBB = 50. (thin line), other lines as in Fig. 6 a. Parameters: as in Fig. 6 a.

Fig. 6 d | The relation between the time in days at which the average severity of the block reaches the value 0.5 (half-time of the block,  $t_b(0.5)$ ) and HALRIB, plotted on linear-log paper. The optimum value of HALRIB under the conditions of the experiment is between 2 and 5. Abscissa: as in Fig. 6 a. Ordinate: number of days from start to  $t_b(0.5)$ . Entries: as in Fig. 6 a. Parameters: as in Fig. 6 a.

Another measure of the growth rate of the epidemic is half-time which depends both on HALRIB and RIBB. In the central compartment  $t_c(0.5)$  decreases to a minimum when HALRIB decreases (Fig. 6.c). The smaller the RIBB, the smaller the minimum value. For the block as a whole  $t_b(0.5)$  has a minimum ranging between the HALRIB values 2 and 5. Again, the smaller the RIBB, the smaller the minimum value of  $t_b(0.5)$  (Fig. 6.d).

When the epidemiologic parameters (NLPD, NIPD, DMFR) have been set, and the epidemic has entered its compound phase, it should develop in accordance with van der Plank's (1963) equations, provided that a correction for spore loss is not needed. During the course of an epidemic either the logistic infection rate  $r$ , or the basic infection rate  $R$  can be constant. When  $R$  is taken to be constant, as in the present model, the disease progress curve plotted on logit-linear scales bends upwards between  $x = 0.5$  and  $x = 1.$ , as van der Plank explained. This observation is confirmed by the curves in Fig. Some curves show a hump, which is due to saturation of the focus. The effect of saturation of the focus was not treated by van der Plank, who based his equations on the assumption

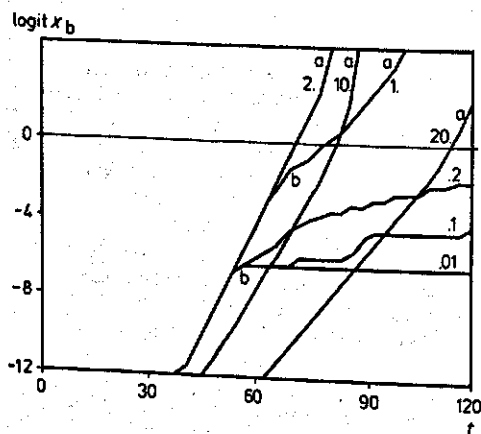


Fig. 7 | Para-logistic growth of epidemics within fields. Upward bends a are due to the constancy of  $R$ , humps b are due to disease saturation in the focus. Ordinate:  $\text{logit}(x_b)$ , average disease severity of the block expressed in logit units. Entries: HALRIB, a constant size. Parameters: NLPD = 8, NIPD = 8, DMFR = 10., NVAR = 1., CMU = 1, RIBB = 5.

that infection is evenly distributed over the field. Focus formation also causes the optima in Figs 6.b and 6.d. Low values of HALRIB show the effect of the correction factor  $1-x$  in the central compartments. At high values of HALRIB the spores are dispersed too much. In the optimum there is a balance between the two factors, spore dispersal and spore retention. At what level of HALRIB the balance is struck, depends on the parameter under consideration  $t_b(0.5)$  or  $r$ . For an explanation see Section 4.1.

### 3.2 Severity profiles and focus extension

During an epidemic, the disease is severe in one place, mild in another place, and possibly absent at a distant location. In the field, there are gradients of disease from severe through mild to absent. At any place the 'gradient' is measured as the derivative of severity to distance,  $dx/dd$ . Each scale of distance has its own gradient. A gradient on a large scale of distance is not necessarily constant, because there are gradients and counter-gradients on smaller scales of distance. During the yellow rust (*Puccinia striiformis*) epidemic on wheat cv. Alba in Belgium and the Netherlands in 1957, there was a gradient over 200 km in the frequency of fields affected, the severity in these fields, and the frequency of the loci, all on a macro-scale of distance (Zadoks, 1961). On smaller scales of distance each infected field and every focus presented a counter-gradient.

The word 'profile' has been used in different ways; here it is used for the curve of severity against distance from the source. The program plots the severity profiles from which the gradients can be computed. The focus intensifies and extends until the centre is saturated (Fig. 8). From then onwards the focus extends radially at constant speed. The graphs on log-linear scales convincingly show that severity curves on successive days are parallel, even when the centre is not saturated. This point will be discussed again in Section 4.2.

### 3.3 Multilines and mosaic crop patterns

EPIMUL has been used to calculate the course of epidemics in situations with one susceptible line or crop from 1, 2, 4 or 8 lines or crops. With the model one can calculate the logistic infection rate, the half-time, and the disease gradients. It is evident that an increase of diversity causes a decrease in the rate of intensification and the rate of spread. These aspects are shown in Figs 9, 10 and 11.

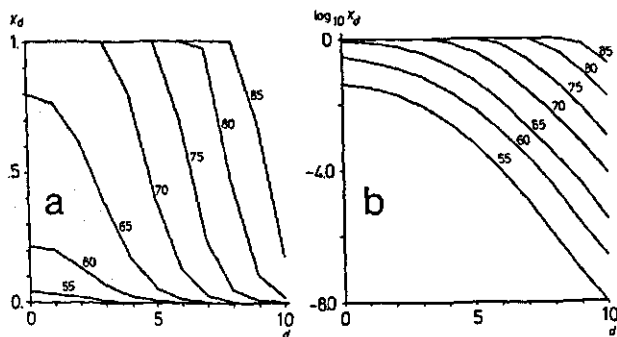


Fig. 8 | Disease gradients of a growing focus, plotted at various days. The focus was started by a single effective spore in the central compartment. There were 400 compartments per block. Abscissa: distance  $d$  from centre measured in compartments. Ordinate: Fig. 8 a - severity  $x_d$  per compartment. Fig. 8 b -  $\log_{10} x_d$  per compartment. Entries: number of days from start. Parameters: HALF = 5, HALRIB = 1., others as in Fig. 7.

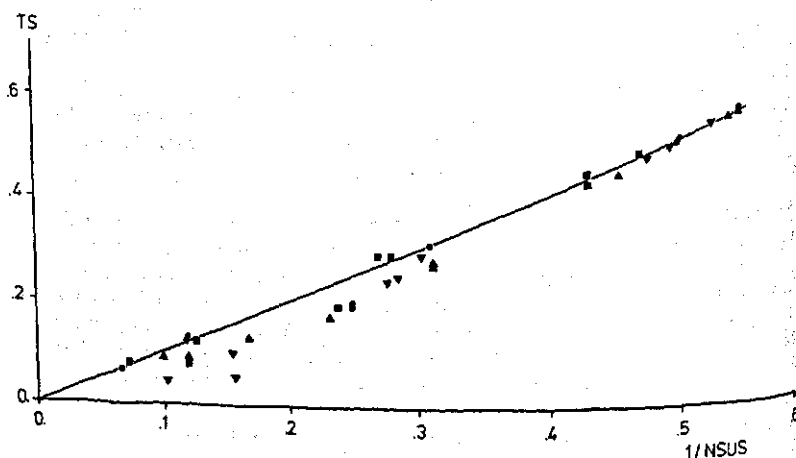


Fig. 9 | Terminal severity TS of epidemics in multiline situations, determined at the end of runs of 120 days. Because of the random distribution of units over the block, three runs per CPU have been made to indicate scatter. Note that at lower CPU values, meaning a better mixture, terminal severities fall below expectations. Abscissa: number of susceptibles expressed as a fraction of the total number,  $1/NSUS$ . Ordinate: average terminal severity of blocks, TS. Entries: expected values (drawn line) CPU = 1 -  $\nabla$ , 4 -  $\triangle$ , 16 -  $\blacksquare$ , 25 -  $\bullet$ . Parameters: as in Fig. 8.

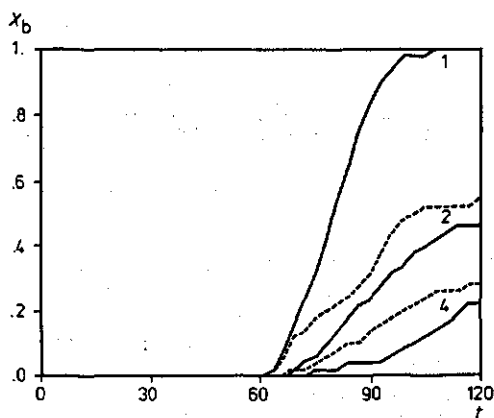


Fig. 10 | Disease progress curves for multiline situations. Abscissa: time in days. Ordinate: average disease severity of the block  $x_b$ . Entries: NVAR, number of lines involved. CMU = 1 (—) many small fields). CMU = 4 (---) few large fields. Parameters: NLPD = 8, NIPD = 8, DMFR = 10., RIBB = 5., HALF = 5., HALRIB = 1.

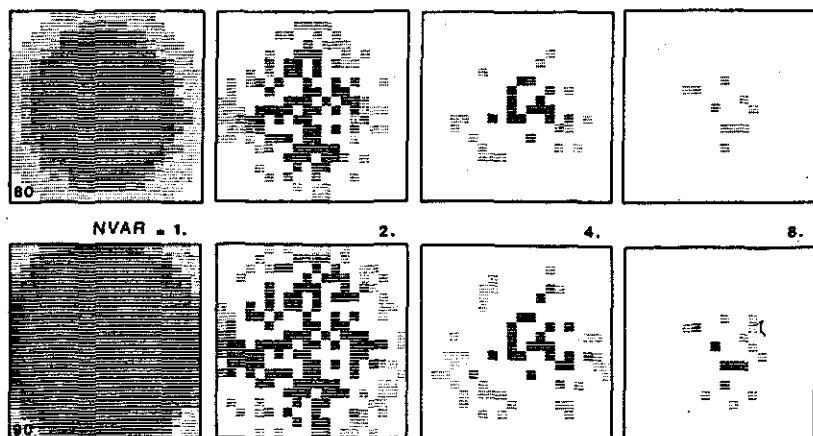


Fig. 11 | The multiline effect on focus formation. Horizontal: NVAR, number of lines in the multiline. Upper row: day 80 from start. Lower row: day 90 from start. Parameters: NLPD = 8, NIPD = 8, DMFR = 10., RIBB = 5., HALF = 5., HALRIB = 1.

In Fig. 9 the line indicates the expected values of terminal severity at the end of a run of 120 days, for different values of NSUS. The distance from the dots to this line represents the effect of the multiline. The scatter of the dots is due to the randomization of compatibility values (see Section 2.6.1). With increasing NVAR the infection rate is smaller (Fig. 10), because the rate of intensification is lower and because the spread of the disease is diminished (Fig. 11).

The effect of increasing the size of the unit is especially discernable in the epidemic (Fig. 10). An increase in the size of the unit can be seen as a forward shift of the epidemic curve along the abscissa without a conspicuous change in the slope of the severity curve. For one value of NVAR the two curves with different unit size are approximately parallel, at least in the second half of the epidemic. The differences between the two curves per NVAR values are due to spore transport, which is more hampered at low CMPU than at high CMPU. An increase in unit size reduces the vertical distance between dots and expected values (line) in Fig. 9.

### 3.4 *The effect of the number of sources*

So far all epidemics have been initiated with one spore in the centre of the block. Frequently many spores arrive in a single field to initiate several simultaneous foci. What is the effect on the rate of infection  $r_b$  when the epidemic is started by a number of scattered spores leading to multiple foci?

Simulation runs have been made with 400 spores as the initial inoculum, arriving at the central compartment (A), or scattered as one spore at each of the 400 compartments (C), or 25 spores at each of 16 compartments placed in a regular grid (B). The result (Fig. 12) shows that with equal amounts of initial inoculum per block, a distribution of the inoculum over more compartments gives a higher  $r_b$  than placement of all spores in one compartment only. The effect is manifest in the range of one to 16 inoculated compartments per 400, and negligible above.



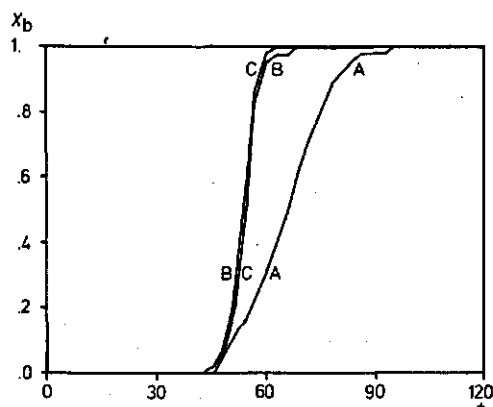


Fig. 12 | Disease intensity curves showing the effect of inoculation pattern. Abscissa: time in days. Ordinate: average disease severity of the block  $x_b$ . Entries: A - 1 focus à 400 spores, B - 16 foci à 25 spores, C - 400 foci à 1 spore. Parameters: NLPD = 8, NIPD = 8, DMFR = 10., NVAR = 1., CMPI = 1, RIBB = 5., HALF = 5., HALRIB = 1.

## 4 Interpretation

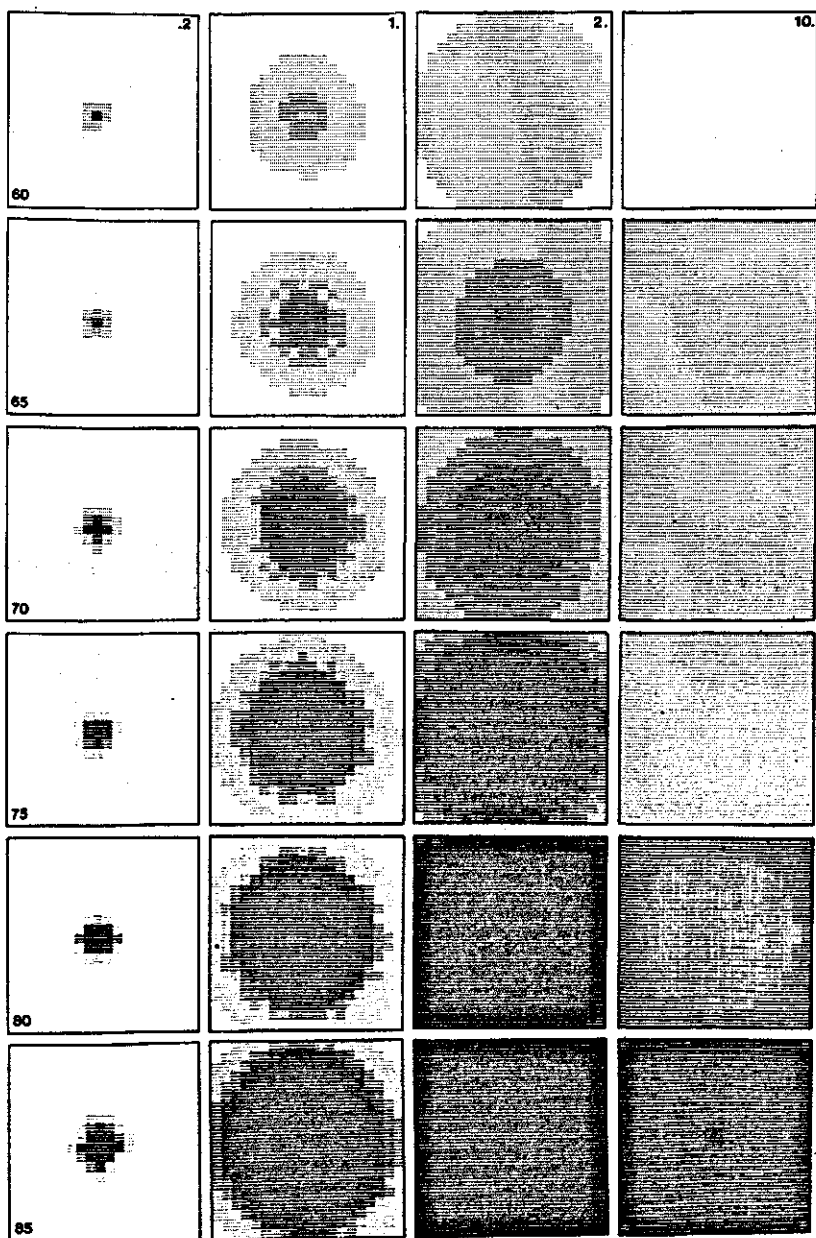
### 4.1 Intensification of the epidemic

No epidemic of foliar pathogens proceeds without dispersal. The model shows that the dispersal, represented by HALF, needs to conform to certain criteria to be optimal. When the dispersal distance is too short, all spores remain within a small area, and no epidemic develops (Fig. 13). When the dispersal distance is too long, most spores are blown away and the epidemic is severely retarded. With optimal dispersal distance all spores fall on the right spot, and the results of the model practically equal the results of models without spore dispersal feature, as far as intensification is concerned.

The intensification of the epidemic can be characterized by the logistic growth rate  $r$ . The logistic growth rate is a relative rate of increase. It provides a measure of intensity. In the model measures of intensity are not particularly sensitive to the absolute values of the distance parameters RIBB and HALF. The measure of intensity does, however, respond to the ratio of the distance parameters, to HALRIB.

Half-time reflects growth not only as intensification but also as spread. Spores are effective either in intensification or in spread. Spread consumes time because it is a polycyclic phenomenon. Hence in the  $t(0.5)$  versus HALRIB graphs (Figs 6.c and 6.d), the curves for the various RIBB values are similar and nearly parallel, but not identical. Polycyclic spread over a large area takes more time than spread over a small area, because the distance must be covered by a number of successive 'acts of dispersal', the actual number depending on HALF. The average number of 'acts of dispersal', needed to cover a distance  $D$ , equals  $D/HALF$ . As a result  $t_c(0.5)$  is always less than  $t_p(0.5)$  for a given HALRIB.

Fig. 13 | The development of focal epidemics, shown at various days (vertical) and various values of HALRIB (horizontal). Parameters: NLPD = 8, NIPD = 8, DMFR = 10., NVAR = 1., CMU = 1, RIBB = 5.



Time is an extensive measure, and half-time reflects extensity rather than intensity. The contrast between intensity and extensity is reflected in Fig. 6, where the optimum for  $r_b$  has a HALRIB value of 10 and the optimum for  $t_b(0.5)$  a value <5. The shape of the severity curve (Fig. 7) indicates on which side of the optimum we are: when the hump b is present, HALRIB is smaller than the optimum of Fig. 6.d, whereas too high values of HALRIB slow down the epidemic in the early stages.

Not all spores produced result in new infections. A fraction dies during transport, or, even when healthy tissue is reached, it fails to infect the plant. Such spore losses are incorporated in the daily multiplication factor (DMFR). In addition spores are lost because they travel and fall on incompatible compartments, which represent other crops or resistant plants. Spatial distribution of resistant plants is a factor not studied in earlier simulations. Two classes of these spore losses are shown in Table 1.

#### 4.2 The front of an epidemic

The extensity of an epidemic can be measured as the area in hectares affected by the disease. The 'rate of spread' is a measure for the rate of extensity, discussed in detail by van der Plank (1975). He reasoned that 'the speed of an epidemic determines its compactness at any given level of disease at the centre'. The 'gearing' of  $R_c$  to  $r$  increases as  $pr$  increases. His reasoning is based on the idea that a diffuse epidemic with a fastly advancing front necessitates a high value of  $R_c$ . Apparently this idea should be valid in the pre-saturation phase at the centre of the epidemic. The term speed is ambiguous, as it indicates a high  $r$ -value or a high rate of displacement of the front of the epidemic. Unfortunately the term front remains undefined. In this part of his reasoning van der Plank did not consider any kind of dispersal mechanism.

Severity profiles can be used to locate the front of an epidemic. A 'front' is a line separating two areas with two distinct qualities; here the 'front' separates areas with disease from areas without disease. How much disease? The 'front' is a subjective concept because one observer may perceive a front where the other observer does not. A front is recognized only when there is a sharp disease profile; so the derivative of severity to distance  $dx/dd$  has to be used. What is an indistinct transition to the observer wandering amidst the crops, may be a sharp profile to the same observer looking at a map with severity data. The clarity of perception depends to some

Table 1 Spore losses due to spore dispersal.

1.a. Spore losses from the block expressed as fractions of the number of spores produced, for different values of HALF. Parameters: NLPD = 8, NIPD = 8, DMFR = 10., NVAR = 1., CMPU = 1, RIBB = 5.

	HALF	2.	5.	10.
	HALRIB	.4	1.	2.
DAY = 15		0.0	7.2E-8	3.4E-6
$t_c(0.5)$		8.9E-9	5.2E-6	2.3E-2
$t_b(0.5)$		2.9E-4	5.9E-3	4.6E-2
DAY = 90		1.8E-7	1.0E-1	*

\* no sporulating lesions available due to saturation of the epidemic.

1.b. Spores lost on resistant plants due to the multiline effect, expressed as fractions of the number of spores produced, after correction for spore losses across the border of the block. Parameters: NLPD = 8, NIPD = 8, DMFR = 10., CMPU = 1, RIBB = 5., HALF = 5., HALRIB = 1.

	NVAR	1.	2.	4.	8.
DAY = 15	-		.28	.49	.79
$t_c(0.5)$	-		.36	.54	.71
$t_b(0.5)$	-		.43	.57	.72
DAY = 90	-		.33	.54	.58

extent on the distance between observer and front. Subjective judgements cause a problem. The problem is solved by assuming an arbitrary perception threshold, which is placed here at a level of 0.05. If 5% of the foliage within an area is visibly infected, the area is placed in the class 'with disease'. A front is defined when on the spot where  $x_d = 0.05$  the gradient is  $dx/dd > 0.04$ . Table 2 shows gradients computed from the model for various values of HALRIB. These gradients can be used to calculate the rate of progress from disease maps (Zadoks & Kampmeijer, 1977).

The results of EPIMUL do not conform to van der Plank's ideas. Severity profiles around foci appear steeper with time until

Table 2 Gradients determined by EPIMUL at various values of HALF.

The value of the gradient  $\Delta x_d / \Delta r_d$  has been determined at  $x_d = 0.05$ . A + in Column S indicates that the gradient has reached a steady state. A + in Column F means that the front of the epidemic can be seen (for explanation see text). Parameters: NLPD = 8, NIPD = 8, DMFR = 10., NVAR = 1., CMPU = 1, RIBB = 5.

HALF	HALRIB	$\frac{\Delta x_d}{\Delta r_d}$	S	F
1.0	0.2	- 0.45	+	+
2.5	0.5	- 0.19	+	+
5.0	1.0	- 0.065	+	+
10.0	2.0	- 0.005	+	.
25.0	5.0	- 0.0	.	.

saturation (Fig. 8.a). After saturation of the centre the profile changes no more. The focus expands radially at constant speed, the stabilized front moves along steadily. The constant speed is not caused by the dispersal formula used; other formulas tested gave comparable results. EPIMUL describes the front of an epidemic as a wave that rolls on when the centre of the focus has reached saturation. In nature an epidemic usually expands without saturation so that the wave-like character of the front may not be apparent. If, however, we look from Fig. 8.a to Fig. 8.b, the picture changes; the profile is constant from the beginning, even with unsaturated sources. This constancy is in accordance with results from medical epidemiology (Kendall, 1965; Daniels, 1975; Diekmann, pers. commun., 1977).

The term 'rate of progress' has been used loosely. We can now define and calculate the 'rate of displacement of the front'. Test runs indicate that the daily multiplication factor (DMFR) the equivalent of  $R_0$ , has little effect on the rate of displacement of the front (Table 3), in contrast to van der Plank's opinion.

The simulator permits calculation of front displacement on the micro-scale. Front displacements on the macro-scale can be calculated, though with some guesswork, using dynamic disease maps (Zadoks & Kampmeijer, 1977). The model necessitates the assumption that the dispersal mechanism is constant over a fairly long period. Such a situation may occur when there is

Table 3 Displacement of the front of the epidemic in compartments per day, calculated by EPIMUL, at various values of HALF and DMFR. The DMFR has some, but relatively little effect on the rate of front displacement. Note that at the lowest line of the table the 'front' no longer satisfies the definition given in the text (figures within brackets). Parameters: NLPD = 8, NIPD = 8, NVAR = 1., CMPU = 1, RIBB = 5.

HALF	HALRIB	DMFR					
		2.	5.	10.	20.	50.	100.
.5	.1	.02	.02	.03	.03	.04	.04
1.	.2	.03	.04	.08	.08	.09	.10
2.	.4	.10	.14	.14	.14	.17	.20
5.	1.	.22	.25	.28	.26	.33	.37
10.	2.	.	(.67)	(.74)	(.80)	.	.

turbulent transport of propagules in the lower atmosphere. The model cannot be used when the propagules are transported by frontal systems at high altitudes and are washed down by rain after having travelled over a long distance.

#### 4.3 EPIMUL and reality

EPIMUL is a highly artificial model. There is hardly an epidemic during which circumstances always are favourable to the disease, day and night. In EPIMUL all calculations are made with a fixed latent period and a fixed infectious period. To reduce the computing time a short latent period was chosen. The results of the calculations apply only to idealized circumstances, continuously optimal for the disease.

The basic assumption underlying most temporal simulations is, that at all times inoculum is regularly distributed over the crop. The effect of this assumption can be visualized by EPIMUL, when an amount of initial inoculum is applied to one compartment only, to all compartments in an equal distribution, or when an intermediate situation is chosen. Fig. 12 shows that an initial inoculum of 400 effective spores distributed in a regular pattern produces a faster epidemic than the inoculum concentrated in one focus. The difference is not a delay of the epidemic, but a real difference in speed. There is no appreciable difference between 16 and 400 foci. Fig. 12 proves that in focal epidemics with relatively low (suboptimal) values of HALRIB the basic assumption is incorrect.

Many epidemics pass through an early focal phase before becoming general. Focus formation from a single infectious lesion is a regular process, described by van der Plank (1963), Zadoks (1961), and many others. Some epidemics show sharply delineated foci, in others the foci are indistinct. Other things being equal, the difference must be due to an inherent characteristic of the epidemic, the dispersal function (Fig. 13). Van der Plank (1975) reasoned that epidemics with discrete foci produce fewer daughter foci (and at shorter distances from the mother focus) than epidemics with diffuse foci. The model produces daughter foci. These can be recognized by a counter-gradient in the severity profile. But counter-gradients in the model are faint, and they are readily obscured by the main gradient. When in subroutine DISPLA the disease becomes 'discernable' ( $x = 0.01$ ), all counter-gradients have already disappeared.

EPIMUL has only one dispersal function, with only one dispersal gradient. A large HALF apparently is not sufficient for producing daughter foci. Therefore we must assume that more than one dispersal gradient exists concurrently; their interaction will produce daughter foci. Four different ranges of spore dispersal can be thought of:

- the dispersal gradient at the border zone of a focus (1 m)
- the density gradient of the new daughter foci (10 - 100 m)
- new infections in the same region, due to spore transport along the 'flight line' of Schrödter (1 km)
- long distance transport (hundreds of km)

Spore transport in turbulent air can account for all these gradients with as extremes the smallest eddies for short-distance transport, and frontal systems for long-distance transport. Possibly the addition of a wind effect to the model will change the pattern of secondary infections and enhance the phenomenon of daughter foci.

In an earlier publication (Zadoks & Kampmeijer, 1977) we attempted to apply EPIMUL to long-distance dispersal, but generally speaking the model is more suitable to simulation of short-distance dispersal. The modeller must make a choice among the above-mentioned ranges of spore dispersal, before applying the model to a specific problem.

Section 3.4 shows the effect of multiple foci; we know of no experiments or observations that study this point explicitly. Evidently such studies are needed, as the effect of multiple foci on the rate of epidemic increase can be considerable.



In natural situations the severity profile is steep at the beginning of the epidemic, and flattens in the course of time at an increasingly high level. Flattening around a point source must be ascribed to invasion of inoculum from natural outside sources into the experimental area (Cammack, 1958; Gregory, 1968). In most experimental situations this 'background infection' does exist and causes flattening. In EPIMUL all spores that travel beyond the boundaries of the block are lost, and spore influx from outside of the block is not considered. So it is not surprising that flattening does not occur normally in the model. The absence of flattening is not a defect of the dispersal function. Another dispersal function has been tried (not an exponential function) and again flattening was absent or, in other words, a constant rate of front displacement was obtained. A steady displacement of the front follows also from other epidemiologic considerations (Kendall, 1965). In a variant of the program a one-time influx from outside was inserted. This influx had all characteristics of background infection: a low spore density was evenly distributed over the area (= the block). The flattening due to this influx is evident (Fig. 14.a).

Reports on flattening of profiles or severity curves could be due to problems of scaling in experimentation, as measurements in or near to the source are reputedly difficult, and observations far from the source are often impossible because of the physical limitations of plot size. The result could be truncated profiles, as shown in Fig. 14.b, where only two fragments of profiles from Fig. 8.b are copied. These truncated profiles, indeed, suggest flattening of gradients, but the suggestion is misleading.

In summary, EPIMUL does not depict real situations, but it approaches some aspects of reality rather closely. It is a discrete solution of epidemiologic problems, which can also be studied by analytical mathematics (Kendall, 1965; Daniels, 1975). The way EPIMUL handles epidemiologic problems may appeal to epidemiologists, because a discrete representation of reality complies with their daily experience.

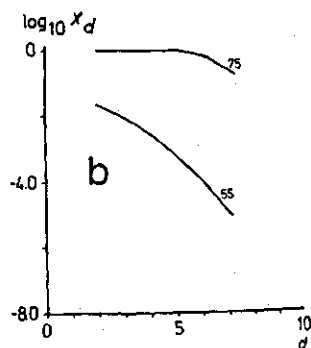
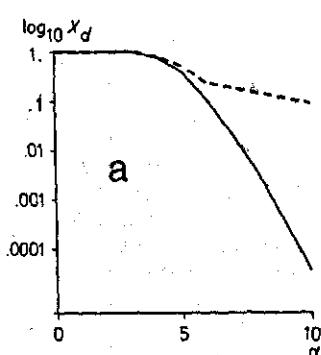


Fig. 14 a | Flattening of disease gradients. Influx of spores from outside sources on day 20, one effective spore per compartment, representing 'blanket infection'. Abscissa: distance from central compartment,  $d$ . Ordinate:  $\log_{10} X_d$ . Entries: without spore influx (—), with spore influx (---). Parameters: NLPD = 8, NIPD = 8, DMFR = 10., NVAR = 1., CMPI = 1, RIBB = 5., HALF = 5., HALRIB = 1.

Fig. 14 b | Flattening of disease gradients. Apparent flattening caused by truncation of severity curves already shown in Fig. 8.b. Abscissa: distance from central compartment,  $d$ . Ordinate:  $\log$  severity. Entries: number of days from start.

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# Appendix A - List of symbols

1	*****		
2			
3	***	LIST OF SYMBOLS	***
4			
5			
6		ARRAY INDICES: I,II,J,JJ,IN,OUT,M11,M12,MJ1,MJ2	
7		AREA	[L**2]
8		ARRY	[L]
9		BLOC	[L**2]
10		CENTR	[N/T]
11		CMFU	[N]
12		CMFR	[N]
13		CMF2	[N/T]
14		COMPAR	[N]
15		COMPAT	[N]
16		DAY	[N]
17		DISP	[T]
18		DISPLA	[N]
19		DIST	[L]
20		DIVE	[L]
21		DMFR	[1/L]
22		FINTIM	[1/T]
23		FLIN	[T]
24		FLIN2	[N/T]
25		FLIN(INFO)	[N/T]
26		FLOF	[N/T]
27		FLOF2	[N/T]
28		FLOW	[N/T]
29		FRSI	[N/T]
30		HALP	[N]
31		INF	[L]
32		INFO	[N]
33		ISCT	[N]
34		LAI	[N]
35		LAT	[N]
36		LESION	[N]
37		LIMIT	[L**2]
38		LOGIT	[N]
39		LOOP	[N]
40		LOOFA	[N]
41		LOOPB	[N]
42		N	[N]
43		N	[N]
44		NIPD	[N]
45		NLPD	[T]
46		NVAR	[T]
47		NSUS	[N]
48		OUTPUT	[N]
49		PERIOD	[T]
50		PROFIL	[N/T]
51		RAT	[N]
52		RATABS	[N]
53		REM	[N]
54		RIBB	[N]
55		RINF	[L]
56		ROCC	[N/T]
57		RREM	[N/T]
58		SEV	[N/T]
59		SINK	[N]
60		SITE	[N]
61		SPORE	[N]
62		SPORES	[N]

63	*	SPOT	= DISEASE SEVERITY OF ONE COMPARTMENT	[=]
64	*	TINF	= TOTAL INFECTANTS (WHOLE FIELD)	[N]
65	*	TREM	= TOTAL REMOVALS (WHOLE FIELD)	[N]
66	*	TDT	= TOTAL DISEASE	[=]
67	*	UNIT	= GROUP OF ADJACENT COMP'S WITH SAME COMPAT	[=]
68	*	VAC	= NUMBER OF VACANTS (= AVAILABLE FOR INFECTION)	[N]
69	*	XSEV	= LOG10(SEV)	[=]
70	*			
71	*	*****		
72	*			
73	*			

## Appendix B - Listing of program

```

74 *      EPIMUL '76
75 *      PROGRAMMER: P.KAMPMEIJER
76 *      FORTRAN PROGRAM FOR A MULTILINE
77 *      SPATIAL VARIATION IN AN EPIDEMIC
78 *
79 *      DIMENSION FLOF(400),FLIN(400),ARRY(-19/20)
80 *      DIMENSION FLOF2(20,20),FLIN2(20,20)
81 *      INTEGER OUT,CMFU,CMFR
82 *      INTEGER DAY,FINTIM,OUTPUT
83 *      INTEGER CMP2(20,20),COMPAT(400),COMPAR
84 *      REAL LAT(400),INF(400),REM(400)
85 *      REAL NSUS,NVAR,LESION,LAI
86 *      REAL LIMIT,LOGIT
87 *      EQUIVALENCE (FLOF(1),FLOF2(1,1))
88 *      EQUIVALENCE (FLIN(1),FLIN2(1,1))
89 *      EQUIVALENCE (CMP2(1,1),COMPAT(1))
90 *      COMMON DAY,SPOT(400)
91 *      OPEN (UNIT= 1,ACCESS='SEQOUT')
92 *      OPEN (UNIT=25,ACCESS='SEQOUT',DEVICE='LPT')
93 *
94 *      *****
95 *      **** INITIAL SECTION ****
96 *      *****
97 *
98 *
99 *      *** PARAMETERS
100 *
101 *      CMFU=1
102 *      CMFR=10.
103 *      FINTIM=120
104 *      INFO=210
105 *      HALF=5.
106 *      LAI=5.
107 *      LESION=10.
108 *      M=20
109 *      NLPD=8
110 *      NIPD=8
111 *      NVAR=1.
112 *      OUTPUT=3
113 *      RIBB=5.
114 *
115 *      ACCEPT *,RIBB,HALF,NVAR,CMFU
116 *
117 *      PARAMETER PRINT
118 *
119 *      WRITE(3,9020) LESION,RIBB,LAI,HALF,CMFR,NLPD,NIPD,N,INFO,CMFU,
120 *      NVAR,FINTIM,OUTPUT
121 *
122 *      N=N/M
123 *      UNIT=N/CMFU
124 *      CMFU=SQRT(FLOAT(CMFU))
125 *      IF (MOD(M,CMFU).EQ.0) GO TO 5
126 *      WRITE (3,9090)
127 *      STOP
128 *      5 AREA=RIBB**2
129 *      BLOC=N*AREA
130 *      LESION=LESION*1.E-6
131 *      FRSI=LAI*AREA/LESION
132 *      SITE=FRSI/N
133 *      XSITE=ALOG10(SITE)
134 *      WRITE(3,9030) BLOC
135 *      WRITE (21,9050)
136 *
137 *      *** ZERO-SETTING
138 *

```



```

139      DAY=0
140      DO 10 I=1,N
141          LAT(I)=0.
142          INF(I)=0.
143          REN(I)=0.
144          FLIN(I)=0.
145          SPOT(I)=1.
146      10  CONTINUE
147          FLIN(INFO)=1.
148      *
149      ***
150      *
151      INITIALIZE RANDOM GENERATOR
152      *
153      CALL TIME(JN)
154      JN=JN.AND.777
155      CALL SETRAN(2*JN+1)
156      *
157      ***
158      *
159      HOST-PATHOGEN COMPATIBILITY SETTING
160      *
161      DO 20 I=1,(M/CMPU)
162      DO 20 J=1,(M/CMPU)
163      JN=IFIX(NVAR*CRAN(DUMMY))
164      DO 20 II=1,CMPU
165      DO 20 JJ=1,CMPU
166      CMP2( ((I-1)*CMPU+II) , ((J-1)*CMPU+JJ) )=JN
167      NSUS=1.
168      COMPAR=COMPAT(INFO)
169      IF (NVAR.EQ.1.) GO TO 40
170      JN=JN
171      DO 30 I=1,N
172      IF (COMPAT(I).EQ.COMPAR) GO TO 30
173      JN=JN-1
174      SPOT(I)=0.
175      30  CONTINUE
176      NSUS=FLOAT(N)/FLOAT(JN)
177      WRITE (3,9010) NSUS
178      WRITE (25,9070) NVAR
179      DO 40 I=1,N
180      WRITE (25,9080) (CMP2(J,I),J=1,N)
181      40  CONTINUE
182      CALL DISPLA
183      *
184      ***
185      *
186      INITIALIZE ARRAY FOR DISPERSAL FUNCTION
187      *
188      DIVE=SQRT(ALOG(2.))/HALF
189      DO 50 I=1,20
190      AI=I
191      DIST=RIBB*(AI-.5)
192      ARRAY(I)=DISP(DIVE,DIST)
193      50  CONTINUE
194      ARRAY(0)=2.*(5-ARRAY(1))
195      DO 60 I=1,19
196      ARRAY(I)=ARRAY(I)-ARRAY(I+1)
197      ARRAY(-I)=ARRAY(I)
198      60  CONTINUE
199      IF (ARRAY(0).GE..016) GO TO 70
200      WRITE (3,9095)
201      STOP
202      *
203      *****
204      ***** DYNAMIC SECTION *****
205      *****
206      *
207      70  CONTINUE
208      *
209      DEVELOPMENT OF THE EPIDEMIC ***
210      *
211      *
212      TINF=0.
213      TREH=0.
214      DO 80 I=1,N
215      IF (COMPAT(I).NE.COMPAR) GO TO 79
216      IN=I
217      *
218      VAC=LIMIT(0.,FRSI,FRSI-LAT(I)-INF(I)-REN(I))
219      CNFR=LIMIT(0.,VAC,VAC-FLIN(I)/FRSI)
220      ROCC=CNFR

```

```

215 RINF=PERIOD(1,IN,NLPD,ROCC)
216 RREM=PERIOD(1,IN,NIPD,RINF)
217 LAT(1)=LAT(1)+ROCC-RINF
218 INF(1)=INF(1)+RINF-RREM
219 REM(1)=REM(1)+RREM
220 FLOF(1)=DMFR+INF(1)
221 *
222 TINF=TINF+INF(1)
223 TREM=TREM+REM(1)
224 SPOT(1)=(INF(1)+REM(1))/FRSI
225 GO TO 80
226 79 SPOT(1)=0,
227 80 CONTINUE
228 CENTR=SPOT(INFO)
229 IF (DAY,LT,NLPD) GO TO 200
230 IF (RATABS,GT,.99) GO TO 200
231 OUT=MOD(DAY,5)
232 IF (OUT,NE,0) GO TO 90
233 IF (RATABS,GT,.8) GO TO 90
234 CALL PROFIL
235 IF (CENTR,LT,.05) GO TO 90
236 CALL DISPLA
237 90 CONTINUE
238 *
239 *** DISPERSAL OF SPORES ***
240 *
241 SINK=0,
242 DO 120 I=1,M
243 DO 120 II=1,M
244 IF (CMP2(I,II),NE,COMPAR) GO TO 120
245 FLOW=0,
246 DO 100 J=1,M
247 DO 100 JJ=1,M
248 IF (CMP2(J,JJ),NE,COMPAR) GO TO 100
249 SPORE=FLOF2(J,JJ)*ARRY(I-J)*ARRY(II-JJ)
250 FLOW=FLOW+SPORE
251 100 CONTINUE
252 110 SINK=SINK+FLOW
253 FLIN2(I,II)=FLOW
254 120 CONTINUE
255 IF (FLIN2(1,1),GE,1.) GO TO 200
256 MI1=0
257 MI2=20
258 MJ1=1
259 MJ2=19
260 ISET=1
261 J=1
262 DO 190 LOOP=1,10
263 MI1=MI1+1
264 MI2=MI2-1
265 DO 190 LOOPA=1,2
266 DO 170 LOOPB=1,2
267 GO TO (130,150) LOOPB
268 130 DO 140 I=MI1,MI2,ISET
269 IF (FLIN2(I,J),GE,1.) GO TO 140
270 IF (FLIN2(1+ISET,J),GE,1.) GO TO 140
271 FLIN2(I+ISET,J)=FLIN2(I+ISET,J)+FLIN2(I,J)
272 FLIN2(I,J)=0,
273 140 CONTINUE
274 I=MI2+ISET
275 MI2=MI1+ISET
276 MI1=1
277 GO TO 170
278 150 DO 160 J=MJ1,MJ2,ISET
279 IF (FLIN2(I,J),GE,1.) GO TO 160
280 IF (FLIN2(1,J+ISET),GE,1.) GO TO 160
281 FLIN2(1,J+ISET)=FLIN2(1,J+ISET)+FLIN2(I,J)
282 FLIN2(I,J)=0,
283 160 CONTINUE
284 J=MJ2+ISET
285 MJ2=MJ1+ISET+2
286 MJ1=J
287 170 CONTINUE
288 ISET=ISET+1
289 180 CONTINUE
290 IF (FLIN2(MI1,MJ1),GE,1.) GO TO 190

```

```

291 IF (FLIN2(MI+1,MJ1),GE,1.) GO TO 190
292 FLIN2(MI+1,MJ1)=FLIN2(MI+1,MJ1)+FLIN2(MI1,MJ1)
293 FLIN2(MI1,MJ1)=0.
294 * 190
295 * CONTINUE
296 * 200
297 * CONTINUE
298
299 SEV=TINF+TREM
300 XSEV=-1.
301 IF (SEV,GE,1.) XSEV=ALOG(0(SEV)
302 RAT=SEV/SITE
303 RATABS=RAT*NSUS
304 RATA=LIMIT(.6E-5,.991,RAT)
305 * LOGIT=ALOG(RATA/(1.-RATA))
306
307 IF(MOD(DAY,OUTPUT),NE,0) GO TO 210
308 SINC=DMFR*TINF
309 IF (SINC,NE,0.) SINK=(SINC-SINK)/SINC
310 * WRITE (21,9040) DAY,SINK,RAT
311
312 CALL PRTPLOT('CENTR',CENTR,0.,1.)
313 CALL PRTPLOT('SEV',A,SEV,1.,SITE)
314 CALL PRTPLOT('XSEV',XSEV,0.,XSITE)
315 CALL PRTPLOT('LOGIT',LOGIT,-12.,4.7)
316 * 210 CALL PRTPLOT('TIME',99.,0.,0.)
317 DAY=DAY+1
318 IF (DAY,GT,FINTIM) GO TO 220
319 * GO TO 70
320 *
321 * 220
322 CALL PRTPLOT('PLOT',0.,0.,0.)
323
324 9010 FORMAT(' CORRECTION FOR NUMBER OF SUSCEPTIBLES : ',F)
325 9020 FORMAT('1 EPIMUL **75**,' SIMULATION OF AN EPIDEMIC IN A MULT
326 1TLINEL,' SIMULATION PARAMETERS:', ' LEAF AREA INDEX',13X,F7.2
327 2,' MM2', ' *RIBB',24X,F7.2, ' M', ' LEAF AREA INDEX',13X,F7.2
328 3,2, ' *HALF',24X,F7.2, ' M', ' DAILY MULTIPLICATION FACTOR',
329 4F7.1, ' LATENT PERIOD',15X,14, ' DAYS', ' INFECTIOUS PER
330 5IOD',11X,14, ' DAYS', ' *M',27X,14, ' INITIAL FOCUS: CELL
331 6 NR, '5X,14, ' CELLS PER UNIT',14X,14, ' *MVAR',24X,F7.2, '
332 7 *FINTIM',22X,14, ' OUTPUT FREQUENCY',12X,14, ' DAYS')
333 9030 FORMAT(' TOTAL AREA INVESTIGATED:',1PE7.0, ' M2', ' ')
334 9040 FORMAT(' DAY',14,2(1PE12.1))
335 9050 FORMAT('1',12X,'SINK',7X,'SEV,R')
336 9060 FORMAT(3X,12)
337 9070 FORMAT('1 COMPATIBILITY-MATRIX NVAR= ',F3.0)
338 9080 FORMAT(' ',2014)
339 9090 FORMAT(' UNITS NOT FITTING IN THE BLOCK. CHOOSE A BETTER VALUE
340 10R CMPU,')
341 9095 FORMAT(' PARAMETER HALF TOO LARGE. THIS PROBLEM CAN NOT BE EXECU
342 ITED,')
343 * END
344
345 *****
346 *
347 FUNCTION DISP(Q,U)
348 *
349 * APPROXIMATION OF THE INTEGRAL FOR THE DISPERSAL FUNCTION
350 * THE INTEGRAL IS: Q/SORT(PJ) * INTGR(L EXP(-Q**2 * X**2) )
351 * INTEGRATED FROM U TO INFINITY.
352
353 DATA A1,A2,A3, A4/.278393, .230389, .000972, .078108/
354
355 FN=Q*U
356 IF (FN,GT,20.) GO TO 10
357 DENOM=1, A1*FN + A2*FN**2, + A3*FN**3, + A4*FN**4.
358 DENOM=1./DENOM**4.
359 DISP=.5*DENOM
360 RETURN
361 10 DISP=.0
362 RETURN
363 END
364
365 *****

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366      SUBROUTINE DISPLA
367      *
368      DIMENSION AA(20),AB(20),AC(20)
369      INTEGER OUT,DAY
370      COMMON DAY,SPOT(400)
371      DATA BA,BB,BC,BD,BE,BF,BG,BH,BI //      ",.....", "HHHHH", "===="
372      1,"////////", "+++++", "NNNNN", "ZZZZZ", "TTTTT"/
373      WRITE(25,9010)DAY
374      DO 130 I=1,20
375      II=(I-1)*20
376      DO 120 JJ=1,20
377      OUT=II+JJ
378      AC(JJ)=BA
379      IK=IFIX(SPOT(OUT)*10.*1.99)
380      GO TO (10,20,30,40,50,60,70,80,90,100,110,120)IK
381  10      AA(JJ)=BA
382      AB(JJ)=BA
383      GO TO 120
384  20      AA(JJ)=BB
385      AB(JJ)=BA
386      GO TO 120
387  30      AA(JJ)=BD
388      AB(JJ)=BA
389      GO TO 120
390  40      AA(JJ)=BA
391      AB(JJ)=BC
392      GO TO 120
393  50      AA(JJ)=BE
394      AB(JJ)=BD
395      GO TO 120
396  60      AA(JJ)=BF
397      AB(JJ)=BD
398      GO TO 120
399  70      AA(JJ)=BG
400      AB(JJ)=BH
401      GO TO 120
402  80      AA(JJ)=BI
403      AB(JJ)=BD
404      GO TO 120
405  90      AA(JJ)=BC
406      AB(JJ)=BI
407      GO TO 120
408  100     AA(JJ)=BG
409      AB(JJ)=BH
410      AC(JJ)=BF
411      GO TO 120
412  110     AA(JJ)=BC
413      AB(JJ)=BI
414      AC(JJ)=BD
415  120     CONTINUE
416      DO 130 JN=1,3
417      WRITE(25,9020)AC
418      WRITE(25,9030)AB
419      WRITE(25,9030)AA
420  130     CONTINUE
421      RETURN
422  9010     FORMAT('1',110K,'      DAY=',I4)
423  9020     FORMAT('0',20A5)
424  9030     FORMAT('0',20A5)
425      END
426      *
427      *****
428      *
429      *      FUNCTION PERIOD(L,I,DELA,XIN)
430      *
431      DIMENSION XX(400,20)
432      INTEGER DAY,DELA
433      COMMON DAY
434      *
435      *      DELA = DELAYTIJD
436      *      II = ARRAYWAARDE VAN DE DELAY
437      *      L = CORRECTIE WAARDE VOOR GERRUIKSNEMHALING
438      *      MOD = FORTRANFUNCTIE : RESTWAARDE VAN EEN DELING
439      *      XIN = INPUTWAARDE
440      *      XX = STORAGE ARRAY
441      *

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442      II=L+MOD(DAY,DELA)
443      PERIOD=XX(I,II)
444      IF (DAY,LI,DELA) PERIOD=0.
445      XX(I,II)=XIN
446      RETURN
447      END
448
449      *****
450
451      SUBROUTINE PROFIL
452
453      COMMON IDAY,SPOT(400)
454      DATA AMIN,SCALE/-5.,10./
455      WRITE(3,9010) IDAY
456      DO 10 I=210,220
457      IT=I
458      POINT=SPOT(I)
459      IF (POINT.EQ.0.) POINT=1.E-10
460      POINT=ALOG10(POINT)
461      POINT=(POINT-AMIN)*SCALE
462      CALL GRAFIC(IT,SPOT(I),POINT)
463      WRITE (3,9020)
464      10  CONTINUE
465      RETURN
466      9010  FORMAT(' ',10X,'SEVERITY-PROFILE THROUGH THE CENTRE OF THE EPIDE
467            1MIC ON DAY NR.:',I4, '/')
468      9020  FORMAT(' ')
469      END
470
471      FUNCTION KANS(P)
472
473      KANS=0
474      OUT=KAN(DUMNY)
475      IF (OUT.LE.P) KANS=1
476      RETURN
477      END
478
479
480      REAL FUNCTION LIMIT(P1,P2,X)
481
482      IF(X-P1)10,10,20
483      10  LIMIT=P1
484      RETURN
485      IF(X-P2)40,30,30
486      30  LIMIT=P2
487      RETURN
488      40  LIMIT=X
489      RETURN
490      END
491
492
493
494      SUBROUTINE PRTPLOT(NAME,VALUE,AMIN,AMAX)
495
496      DIMENSION INP(10),XMIN(10),XMAX(10),ROW(10)
497      DIMENSION YMIN(10),YMAX(10)
498      COMMON IDAY
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518 RETURN
519 50 IF (NAME.EQ."PLOT") GO TO 70
520 IF (NAME.EQ."TIME") GO TO 60
521 N=N+1
522 XMIN(N)=AMIN1(VALUE,XMIN(N))
523 XMAX(N)=AMAX1(VALUE,XMAX(N))
524 ROW(N)=VALUE
525 RETURN
526 60 N=0
527 WRITE (20) (ROW(I),I=1,NC),IDAY
528 RETURN
529 70 REWIND 21
530 71 READ (21,1040,END=72) ROW
531 WRITE (3,1040) ROW
532 GO TO 71
533 72 DO 90 N=1,NC
534 AMAX=XMAX(N)
535 AMIN=XMIN(N)
536 IF (YMAX(N).EQ.YMIN(N)) GO TO 73
537 AMAX=YMAX(N)
538 AMIN=YMIN(N)
539 73 SCALE=AMAX-AMIN
540 IF (SCALE.EQ.0.) SCALE=50.
541 SCALE=50./SCALE
542 REWIND 20
543 READ (20) (INP(I),I=1,NC)
544 WRITE(3,1010) INP(N),XMIN(N),XMAX(N)
545 WRITE(3,1020) INP(N)
546 80 READ (20,END=89) (ROW(I),I=1,NC),IDAY
547 VALUE=(ROW(N)-AMIN)*SCALE
548 CALL GRAFIC(IDAY,ROW(N),VALUE)
549 GO TO 80
550 89 WRITE(1,1030)
551 90 CONTINUE
552 CLOSE(UNIT=20,DISPOSE="DELETE")
553 CLOSE(UNIT=21,DISPOSE="DELETE")
554 1010 FORMAT('1',50X,"Y-VARIABLE: ",A5,/,/,34X,"MINIMUM",41X,"MAXIMUM"
555 1/,/,33X,1PE11.4,39X,E11.4,/,/,41X,"1".49X,"1")
556 1020 FORMAT(' ',17X,A5.9X,"TIME",5X,"00000000001111111111222222222233
557 833333333444444444445",/,41X,"01234567890123456789012345678901234
558 95678901234567890")
559 1030 FORMAT(' ',40X,"000000000011111111112222222222333333333344444444
560 445",/,41X,"012345678901234567890123456789012345678901234567890")
561 1040 FORMAT(10A5)
562 END
563 *
564 *
565 *
566 *
567 *
568 SUBROUTINE GRAFIC(NUM,VAL,VAR)
569 *
570 DIMENSION LINE(51)
571 MIN=" "
572 MAX=" "
573 IVAR=VAR
574 IF (IVAR) 10,20,20
575 MIN="1"
576 IVAR=0
577 GO TO 30
578 20 IF (IVAR.LT.51) GO TO 30
579 MAX="0"
580 IVAR=0
581 DO 40 I=2,51
582 LINE(I)=" "
583 DO 50 I=1,IVAR
584 LINE(I)="-"
585 LINE(IVAR+1)="+ "
586 LINE(1)="0"
587 WRITE(3,1010) VAL,NUM,MIN,LINE,MAX
588 RETURN
589 1010 FORMAT(' ',14X,1PE11.4,5X,14,5X,53A1)
590 END

```