# APPROACHES TO MODELLING THE SPATIAL DYNAMICS OF PESTS AND DISEASES

W. Van Der Werf, W. A. H. Rossing & R. Rabbinge
*Department of Theoretical Production Ecology, Agricultural University-Wageningen, Netherlands*

M. D. De Jong
*Centre for Agrobiological Research-Wageningen, Netherlands*

P. J. M. Mols
*Department of Entomology, Agricultural University-Wageningen, Netherlands*

## SUMMARY

Spatial distribution patterns and dispersal process play an important role in the population dynamics of pests and the epidemiology of diseases. To account for this, different approaches can be taken. Three possible approaches are illustrated in this paper by means of case-studies. The first example concerns the flight of fungal spores in a forest. The problem is treated with diffusion and flux equations which describe the behaviour of spore masses but not that of individual spores. In the second example, predation and egg production by carabid beetles is simulated on the basis of a motivational and behavioural model which explains walking paths and attack rates in terms of the beetle's gut satiation. In the third example, the processes underlying the spread of viruses in sugarbeet are studied, using a simulation model which describes the walking, feeding and virus transmission activities of individual aphids as well as the progression of disease in individual plants. The degree of complexity simulated and the concomitant computing time increases from the first example to the third. Each of the approaches presented here has its merits and shortcomings. The choice of the approach depends upon the aim of the study and the available experimental data for model construction and validation.

## 1. INTRODUCTION

Biological populations vary in density over their habitat. This spatial heterogeneity results first of all from spatially variable abiotic conditions such as soil type, microclimate and availability of nutrients. Furthermore, individuals aggregate to find mates or protection. Limited dispersal of offspring results in aggregation as well. Finally, interactions with spatially distributed competitors, predators or food adds to spatial variability. Spatial distributions and spatial dynamics need to be studied and simulated to obtain a good insight into population density and its fluctuations in time and space.

The most appropriate approach to accounting for spatial heterogeneity depends on the purpose of the study. Statistical methods are adequate when determining minimum sample sizes for the estimation of disease severity or pest attack (DAAMEN, 1986a,b; WARD *et al.,*1985a,b, 1986). This approach deals with **dispersion**, i.e. the pattern of distribution of organisms over patches (SOUTHWOOD, 1978), but not with the process that leads to it, **dispersal**. Methods to describe dispersion have been adequately treated by SOUTHWOOD (1978) and by UPTON and FINGLETON (1985) while RABBINGE *et al.* (1989) describe methods to account for the effects of

spatial variability in simulation models.

This paper will concentrate on the dynamic simulation of dispersal. In studies of temporal dynamics of pests and diseases insight into spatial dynamics may be crucial to explain processes on the population level and to allow the construction of realistic models (KAREIVA and ODELL, 1987). Furthermore, in a variety of systems, spatial dynamics *per se* is the topic of the study, e.g. in the case of the spread of diseases in crops.

Three examples will be given of explanatory dynamic simulation models which incorporate a detailed treatment of spatial dynamics to explain system behaviour in time and space. The first example - distribution and flight of fungal spores inside a forest - applies diffusion and flux equations without making reference to the behaviour of individual spores. In the second example - predation by carabid beetles - detailed observations on behaviour of individuals are integrated in a motivational and behavioral model as part of a population model to calculate predation and beetle population dynamics. In the last example, we describe a first attempt to model the spread of viruses in a way that accounts for the walking and feeding behaviour of the vector.

## 2. MODELLING AERIAL SPORE TRANSPORT OF SILVERLEAF FUNGUS, *Chondrostereum purpureum*

### 2.1 Introduction

'American' black cherry, *Prunus serotina*, was introduced into the Netherlands between 1920 and 1950 to improve the understory of coniferous forests on poor sandy soils. However the species became a serious competitor for native tree species and hampered forest regeneration. Cutting blackcherry trees and inoculating the stubs with silverleaf fungus, *Chondrostereum purpureum*, was considered as a possible means of biocontrol. Before this could be undertaken, the possible
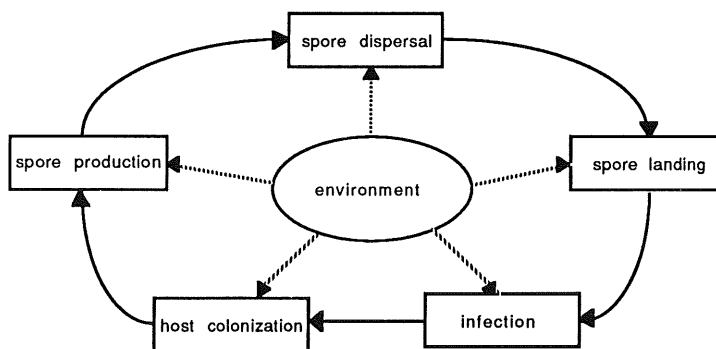


Fig. 1: Schematic presentation of life cycle of *Chondrostereum purpureum.*

escape of spores of the fungus to susceptible *Prunus* species outside forests, e.g. ornamental trees and fruit trees, had to be quantified (WAGENMAKERS, 1984; DE JONG and SCHEEPENS, 1985; DE JONG, 1988; DE JONG *et al.,*1988). To accomplish this, the life cycle of the fungus was divided into five main phases (Fig. 1). The processes in each phase were quantified. Here the approach adopted to quantify spore dispersal is outlined.

DE JONG (1988) described the dispersal of spores outside the forest with the Gaussian plume model while he calculated the place and extent of spore emission from the forest by a quantitative evaluation of the relevant spore exchange mechanisms, based on crop micrometeorological studies of GOUDRIAAN (1977, 1979). Spores can be emitted from the forest vertically by turbulent diffusion and horizontally with wind currents (mass flow) downwind of the forest and diffusion. The importance of horizontal emission decreases with forest size. Vertical and horizontal spore emission are functions of forest size and weather, notably wind speed and temperature profile. DE JONG used a multi-layer model of the forest for his calculations. Here, the essential elements of the model are illustrated by means of a one-layer model.

## 2.2 One-layer model.

As a first approximation the forest is treated as a single vegetation layer. It is assumed that the spore concentration is the same over the whole forest area. The spore concentration in the forest is the result of seven exchange processes with the environment (Fig. 2). Along the vertical axis the density of spores (spores $m^{-2}$ forest area) changes due to (1) turbulent exchange with the atmosphere, (2) sedimentation from the atmosphere, (3) sporulation by *C. purpureum* basidiocarps, (4) sedimentation on the ground and (5) deposition on branches and needles. In the horizontal plane spores are transported due to (6) air mass flow (wind) and (7) turbulent diffusion.
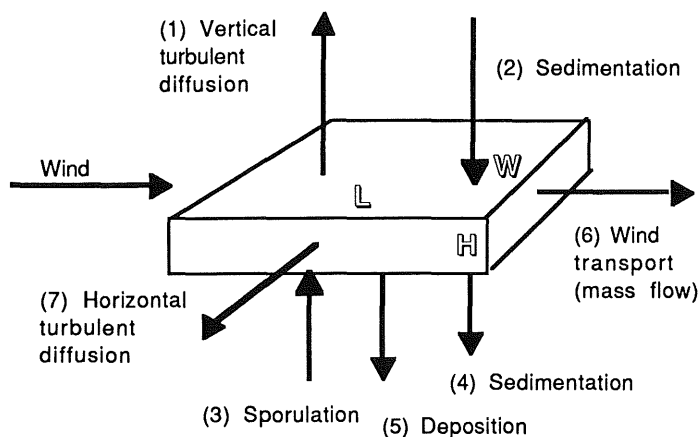


Fig. 2: Box representation of one-layer approach to modelling spore emission from a forest. H: height; L: length and W: width of forest.

Accounting for these seven processes the net rate of change of spore density (spores $m^{-2}$ $s^{-1}$) in the forest can be formulated as:

$$H * C_i' = - R_{turb} + R_{sedin} - R_{sedout} + R_{spor} - R_{dep} - R_{wind} - R_{dif} \tag{1}$$

where H is the height of the trees, $C_i$ the spore concentration in the forest and $C_i'$ the net rate of change of $C_i$.

$R_{turb}$ is the rate of vertical turbulent spore exchange between the forest and the atmosphere,

$R_{sedin}$ is the rate of spore sedimentation into the forest from the atmosphere,

$R_{sedout}$ is the rate of spore loss from the forest by sedimentation on the ground,

$R_{spor}$ is the rate of spore production by *C. purpureum* basidiocarps

$R_{dep}$ is the rate of spore loss from the forest by deposition on needles, branches and soil,

$R_{wind}$ is the rate of sidewards spore emission due to mass transport by the wind, and

$R_{dif}$ is the rate of sidewards spore emission due to turbulent diffusion.

The rate of vertical turbulent spore exchange between the forest and the atmosphere depends on the difference in spore concentration as driving force and the exchange resistance r (with dimension s $m^{-1}$):

$$R_{turb} = (C_a - C_i)/r \tag{2}$$

where $C_a$ is the spore concentration in the atmosphere.

The rate of spore sedimentation is proportional to spore concentration and the sedimentation velocity (m $s^{-1}$).

$$R_{sedin} = V_{sed} * C_a \tag{3}$$
$$R_{sedout} = V_{sed} * C_i \tag{4}$$

$R_{spor}$ is a function of temperature and basidiocarp area, derived from data of GROSCLAUDE (1969).

The rate of spore deposition in the forest depends on wind speed, u (m $s^{-1}$), the deposition efficiency of needles, branches, etc., $e_{dep}$ (-), the area of branches, needles, etc. with respect to ground area, A ($m^2$ $m^{-2}$) and the spore concentration, $C_i$:

$$R_{dep} = u * e_{dep} * A * C_i \tag{5}$$

The rate of horizontal mass transport of spores is

92

$$R_{wind} = H * C_i * u/L \tag{6}$$

in which L is the length of the forest. The rate of horizontal diffusion equals

$$R_{dif} = C_i/r_h \tag{7}$$

where $r_h$ is the resistance to horizontal diffusion, which increases linearly with the size of the forest.

Typical values for the parameters in these equation are: $R_{spor}$ = 20 spores $m^{-2} s^{-1}$, $C_a$ = 100 spores $m^{-3}$, r = 20 s $m^{-1}$, $V_{sed}$ = 1 mm $s^{-1}$, u = 2 m $s^{-1}$, $e_{dep}$ = 0.01, A = 2 $m^2$ $m^{-2}$ and $r_h$ = 1000 s $m^{-1}$.

Now equation 1 can be written explicitly

$$H * C_i' = R_{spor} + C_a/r + V_{sed} * C_a - (1/r + V_{sed} + u * e_{dep} * A + u * H/L + 1/r_h) * C_i \tag{8}$$

or, more briefly

$$H * C_i' = b - a * C_i \tag{9}$$

Here b denotes rates independent of $C_i$ while a denotes rates that vary with $C_i$. At equilibrium , when $C_i'$=0, $C_i$ equals b/a:

$$C_i = (R_{prod} + C_a/r + V_{sed} * C_a)/(1/r + V_{sed} + u * e_{dep} * L + u * H/L + 1/r_h) \tag{10}$$

$$C_i = (20 + 100/20 + .001 * 100)/(1/20 + 0.001 + 2 * 0.01 * 2 + 2 * 17/250 + 1/1000)$$

$$C_i = 25.1/0.228 = 110 \text{ spores } m^{-3}$$

For this steady state the contribution of the seven transport processes to spore emission from the forest can be calculated with Eqs. 2 - 7. Some examples of results from the one-layer model are given in Fig. 3.

This simple model gives a reasonable first impression of the importance of the different processes in spore dispersal. Apparently, under the conditions chosen, sporulation, turbulent vertical transport, horizontal mass flow and deposition are the only processes of practical importance. At very low wind speeds sedimentation may be also important.

The assumption of a homogeneous spore density over the whole forest area is not justified. When air without spores is blown into the forest, $C_i$ will be very low at the upwind side while the calculated value $C_{i,eq}$ is reached at some distance in the forest. How large is this distance? This question could be answered by horizontal compartmentalization of the forest.
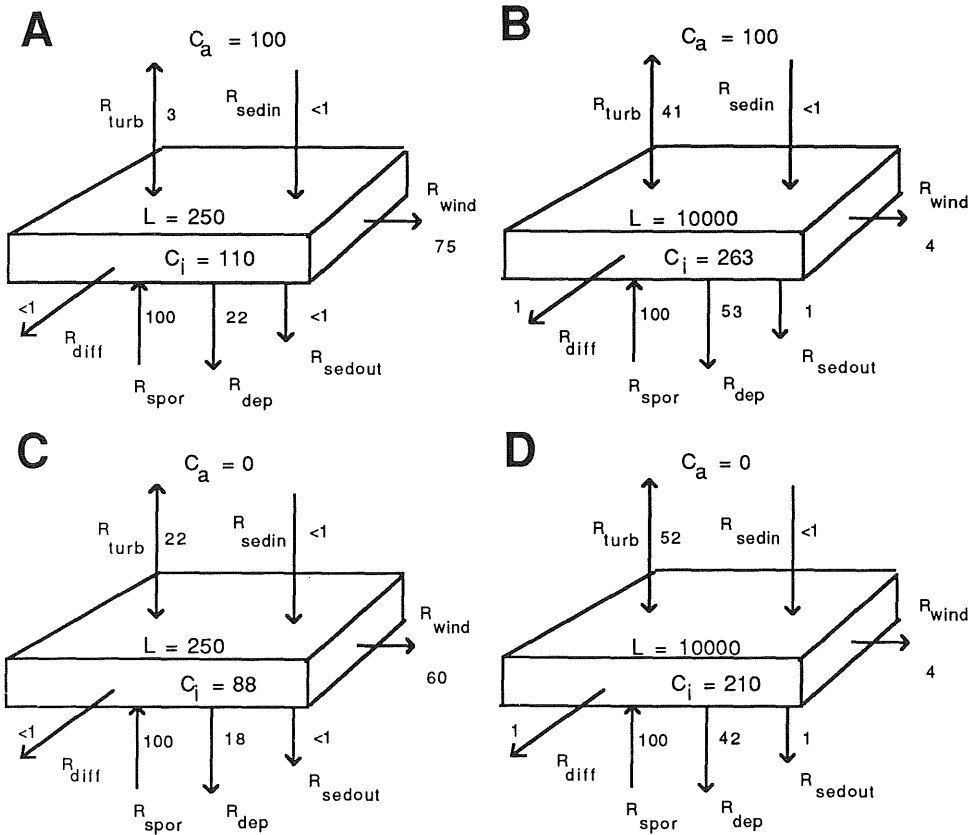
Fig. 3: Spore emission from a forest calculated with the one-layer model for two forest sizes and two atmospheric spore concentrations. A: L = 250 m and $C_a$ = 100 spores m$^{-3}$; B: L = 10000 m and $C_a$ = 100 spores m$^{-3}$; C: L = 250 m and $C_a$ = 0; D: L = 10000 m and $C_a$ = 0. In- and effluxes are expressed in %.

However, a rough estimate may be obtained in a simpler way. According to Eq. 1, the partial derivative of $C_i$ with respect to time is:

$$\delta C_i/\delta t = \{ R_{spor} + C_a/r + V_{sed} * C_a - (1/r + V_{sed} + u * e_{dep} * A + u * H/L + 1/r_h) * C_i\}/H \qquad (11)$$

The air traverses the forest at a rate dx/dt = u where x denotes distance along the wind axis with x = 0 at the upwind side of the forest. Therefore $\delta t$ equals $\delta x/u$, so that

94

$$\delta C_i/\delta x = \{ R_{spor} + C_a/r + V_{sed} * C_a - (1/r + V_{sed} + u * e_{dep} * A + u * H/L + 1/r_h) * C_i\}/(H * u) \qquad (12)$$

or shorter:

$$\delta C_i/\delta x = (b - aC_i)/(H * u) \qquad ' (13)$$

Equation 13 describes an exponential convergence of $C_i$ to $C_{i,eq}$ along the wind axis. The equilibrium concentration is closely approximated when x equals 3 times H * u/a, i.e. in the example given in Fig. 3A (L = 250 m and $C_a$ = 100 spores $m^{-3}$) at: x = 3 * 17 * 2/0.228 = 447 m

Apparently the equilibrium concentration is not reached in this small forest. The error which results from this mistake is small because $C_a$ and $C_{i,eq}$ differ little. For the large forest of Fig. 3B the assumption that air leaving the forest at the lee edge contains spores at the equilibrium concentration is fulfilled (x << L). However, in this situation the assumption implicitly made in Eq. 5 that spore emission through mass flow affects the spore concentration in the whole forest is not fulfilled. Again this problem could be solved by horizontal compartmentalization. Nevertheless, the one-layer approach outlined here allows a useful first approximation of spore emission under different circumstances. Spore concentrations and fluxes can be easily obtained with a pocket calculator. However, for a deeper insight into the spore dispersal processes in a forest a multi-layer approach is necessary.

## 2.3 Four-layer model

A more realistic representation of the system is obtained by distinguishing three vertical forest layers: (1) a spore production layer near the ground, (2) a stem layer and (3) a crown layer in which spores may be deposited on needles and branches (Fig. 4). Above the forest, distinction is made between a thick upper atmosphere layer without spores and a thinner boundary layer, just above the forest, in which air movement is slowed down by drag exerted by the canopy. The free atmosphere does not take part in the spore exchange processes and is excluded from the model equations. The resulting four-layer model is essentially the same as the one-layer model described above. The only difference is that rates are now calculated for four layers in stead of one, resulting in a matrix formulation of the problem (WAGENMAKERS, 1984; DE JONG, 1988).

With the matrix model the distribution of spores at equilibrium can be calculated while the equilibrium concentrations can be used to calculate the horizontal and vertical emission rates. However, the calculation of an equilibrium state of the system is only valid when the circumstances remain constant long enough to establish equilibrium. This will rarely be the case because wind speed and direction, the main driving forces in this system, fluctuate in time and space. Therefore, a dynamic simulation approach would be appropriate. In a dynamic simulation model horizontal dimensions could be added.

```
5
                              Free atmosphere

        ─────────────────────────────────────  25 m
4                             Boundary  layer

        ─────────────────────────────────────  17 m
3                             Crown  layer

        ─────────────────────────────────────  8.5 m
2                             Stem  layer

                   larch  trees
                                               0.25 m
1                             Spore production layer
                                               0.00 m
```
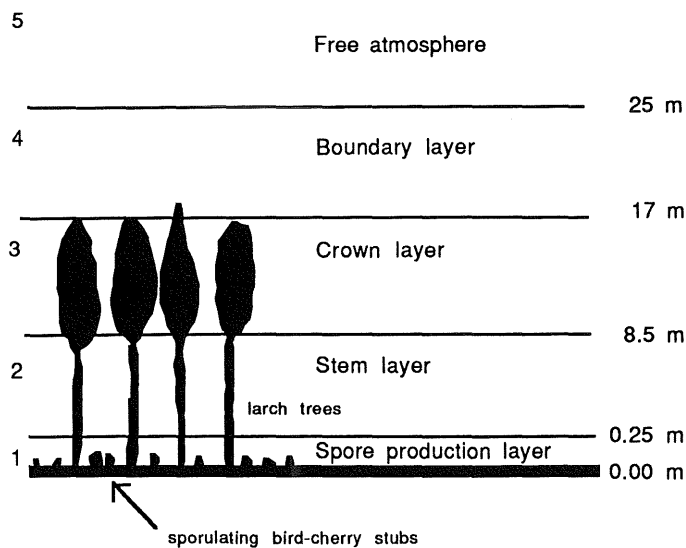
sporulating  bird-cherry  stubs

Fig. 4: Schematic representation of aerial layers in a forest in which *Prunus serotina* is controlled with *Chondrostereum purpureum*

The four-layer model, as described, enables an analysis of risks associated with biocontrol of black cherry. The transport of airborne material within forest or crop canopies plays a vital role in disease epidemiology and constitutes a factor of importance in many other fields, e.g. spraying techniques for pesticides and transport of pollen or air pollutants. Moreover, calculation of the spread in the environment of genetically engineered organisms, e.g. viruses (CORY and ENTWISTLE, 1988; HUBER, 1988), has become an important issue recently. Simulation and systems analysis may help to quantify risks and gain insight into the processes involved.

## 3. MODELLING DISPERSAL OF THE CARABID BEETLE *Pterostichus coerulescens* AND ITS CONSEQUENCES FOR PREDATION

### 3.1 Introduction

In many cases food of predators, as of other animals, is not distributed randomly throughout the habitat but shows a more or less aggregated pattern (SOUTHWOOD, 1978; UPTON and FINGLETON, 1985). As many predators actively search for food, the pattern of movement in relation to the density and distribution of the prey greatly affects the rate of feeding and as a consequence the rate of reproduction and the spatial and temporal dynamics of the population in the field.

The predation strategy of the carabid beetle *Pterostichus coerulescens* L. (=*Poecilus versicolor* Sturm) has been studied in relation to prey availability, to analyse its effect on egg production and survival of the species (MOLS, 1979, 1983, 1986, 1987 and in prep.). The beetle lives in heathland

and poor grassland where it hunts for aphids, caterpillars and maggots. The internal factors governing the beetle's behaviour are analyzed and related to components of behaviour. The results are integrated in a simulation model to predict predation, consumption and egg production under different sets of external conditions such as prey density, prey distribution and temperature.

## 3.2 Modelling motivation and predatory behaviour of *Pterostichus coerulescens*

In many species behaviour is governed by some internal 'motivational drive' which is related to gut content (HOLLING, 1966; FRANSZ, 1974; RABBINGE, 1976; SABELIS, 1981; KAREIVA and ODELL, 1987). For *P. coerulescens* the satiation level of the gut, defined as the actual gut content divided by the potential gut content or gut capacity, is a useful measure for the 'motivation' of the beetle (MOLS, 1987). The potential gut content can not exceed a physical maximum but its actual size depends on the volume of other organs and tissues. The actual gut content changes by ingestion, excretion and resorption. The rates of change are predominantly affected by ambient temperature and day length, the latter determining the pre-oviposition period. Thus the physiological drive for behaviour, i.e. the satiation level or its complement, hunger, results from various internal states which in their turn are affected by the amount of prey ingested and climatic conditions. A simulation model is developed in which the various relations are quantitatively introduced.

### 3.2.1 Methods

### The motivational model

In the relational diagram of Fig. 5 the state variables describing feeding and egg production of the carabid beetle are represented. The amount of food ingested depends on the predator's hunger. After digestion in the gut, part of the food is excreted as faeces, the remainder being taken up in the haemolymph. The assimilated food is used for maintenance processes, converted into egg material in the ovaries or stored as fat. From the ovaries full-grown eggs move into the oviduct from where oviposition takes place

The five state variables defining the internal state of the predator (gut content, weight of haemolymph, ovaries, eggs in the oviduct and reserves) change with rates depending on internal and external conditions. The ovaries start to grow as soon as a critical day length of 14 h and an average daily temperature of 10 °C are exceeded. After a pre-oviposition period eggs are laid during a period of one to two months, depending on temperature. After this period the ovaries regress while most assimilated food is stored in reserves to be used during diapause. During the breeding season the ovaries monopolize the assimilated food. The concomitant increase in volume may limit full expansion of the gut, thus causing a reduction of the potential gut content during the oviposition period. Temperature is the most important external condition affecting rates of change of the internal state.
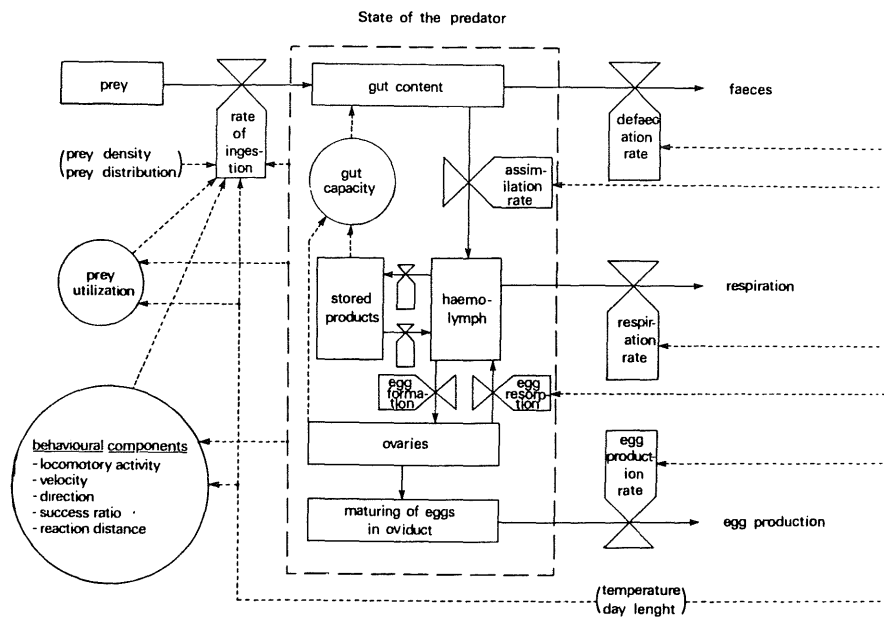
Fig. 5: Relational diagram of the motivational model of *P. coerulescens* and the behavioural components. Rectangles represent states, valves rates and circles auxiliary variables following the conventions of FORRESTER (1961). Solid lines indicate flow of matter, dotted lines flow of information.

The motivational model is verified by comparing the simulated egg production with actual data of experiments in which beetles are kept in small containers with abundant food supply to exclude aspects of searching behaviour. Model results agree with the observed egg production (not shown here; MOLS, in prep.).

## Modelling predatory behaviour of *P. coerulescens*

MOLS (1986) analyzed walking behaviour of *P. coerulescens* with video equipment. Three types of walking behaviour can be distinguished based on average linear displacement and walking velocity (Fig. 6):

a.  Straight walk. This type of walk is associated with a relatively high walking velocity (3 to 5 cm s$^{-1}$). It occurs if hunger exceeds 95%.

b.  Random walk. The walking velocity is between 2 and 3 cm s$^{-1}$. This type of walk occurs when the satiation level is below 95% and results in a winding walking path.

c.  Tortuous walk. The walking velocity is low (1 cm s$^{-1}$) and the walking path is very winding. This type of walk is found just after consumption of a prey. The duration of tortuous walk is a function of the satiation level (Fig. 7). At satiation levels of less than 10% this behaviour lasts about 11 minutes, while at satiation levels between 70 and 80% the duration is only 2 minutes.

A more detailed description of movement of the beetle, necessary for simulation of prey searching behaviour, is obtained by measuring the velocity and direction of walking during successive short periods of time, 'time steps'. The velocity of walking as a function of satiation level is shown in Fig. 8,
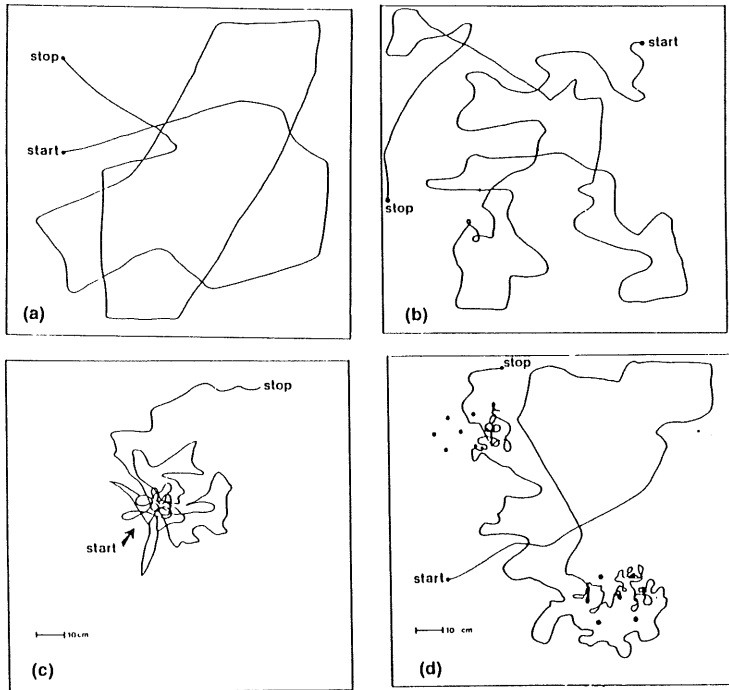
Fig. 6: Walking patterns found in *P. coerulescens:* (a) straight walk, (b) random walk, (c) tortuous walk and (d) combination of straight, random and tortuous walk in an area with prey (indicated by dots).
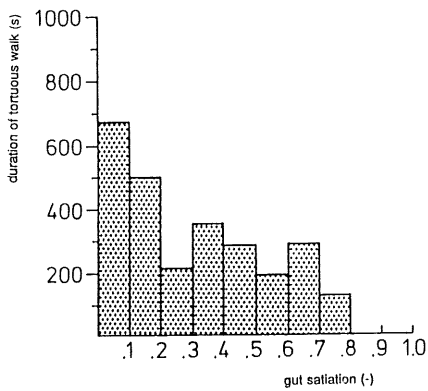


Fig. 7. Duration of tortuous walk behaviour of *P. coerulescens* following consumption of prey, as a function of the satiation level.
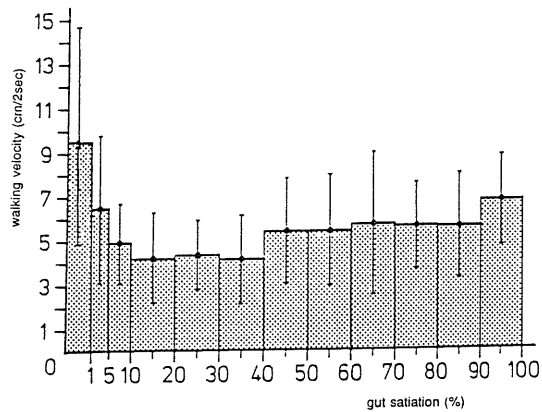
Fig. 8. Relation between the gut satiation and walking velocity of *P. coerulescens* for 'straight walk' and 'random walk'. Bars represent standard deviations.

99

not considering tortuous walk. Below a satiation level of 5%, walking velocity increases rapidly. The relative direction of walking is measured as the angular deviation from the direction in the previous time step. If a small time step is chosen, autocorrelation exists between subsequent angular deviations, which complicates the analysis unnecessarily. A large time step yields insufficient resolution. By iterative calculations it was found that a time step of 2 seconds is most suitable, yielding satisfactory walking paths without autocorrelation between successive angular deviations.

Using this time step, frequency distributions of angular deviation are constructed from the video recordings for beetles with various gut satiation levels. The frequency distributions are used to fit the parameters of the Tukey distribution, a symmetric statistical distribution characterized by three parameters (Fig. 9).
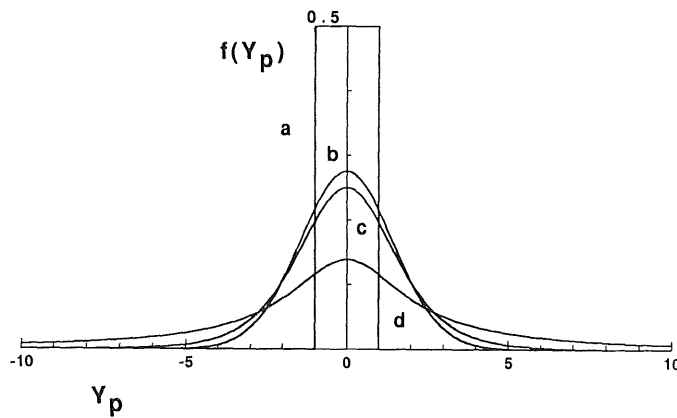


Fig. 9: Tukey probability distribution for four values of the kurtosis-parameter $\lambda$. a: $\lambda = 1$ (uniform); b: $\lambda = 0.14$ (normal); c: $\lambda = 0$ (logistic) and d: $\lambda = -0.85$ (Student's $t_1$ or Cauchy). The Tukey distribution is defined by:

$$A = \mu + \sigma.Y_p \quad \text{where}$$

$$Y_p = \begin{cases} (p^\lambda - (1-p)^\lambda)/\lambda & \text{when } \lambda \neq 0 \\ \ln(\frac{p}{1-p}) & \text{when } \lambda = 0 \end{cases}$$

A is the angular deviation (in radians), $\mu$ is the mean change in direction, $\sigma$ is a scale parameter which determines the variance of A together with $\lambda$, while the kurtosis is determined by $\lambda$. The variable p is drawn from a standard uniform distribution.

Further analysis (MOLS, 1987) shows that the parameters determining the shape of the distribution can be predicted from the walking velocity of the beetle, low velocities coinciding with a wider range of angular deviations:

100

$$\sigma = \text{EXP} ( -0.173 * V + 0.208 ) \qquad r^2=0.69$$
$$\lambda = -0.0661 * V + 0.2 \qquad r^2=0.49$$

where V is the linear displacement (cm) per time step of 2 s (see Fig. 9 for further explanation).

Thus, in the model, the motivational state of the predator determines the velocity of walking which in turn determines the concomitant probability distribution of angular deviations. By drawing from the distribution, the potential dispersal during one time step can be calculated. The actual dispersal depends on the activity of the beetle, i.e. the fraction of time the predator is walking, is negatively correlated with the satiation level.

Given the distribution of prey, predation occurs if the distance between the predator and the nearest prey is less than the reaction distance, the largest distance at which a prey is recognized. Experiments by MOLS (in prep.) show that the percentage beetles reacting to prey decreases with increasing distance to the prey and that the reaction is hardly affected by the level of gut satiation.

Not all encounters with prey result in prey consumption. The success ratio, defined as the proportion of encounters resulting in prey consumption, depends on the satiation level of the predator because a hungry predator is more eager to attack a prey and will continue the attack longer.
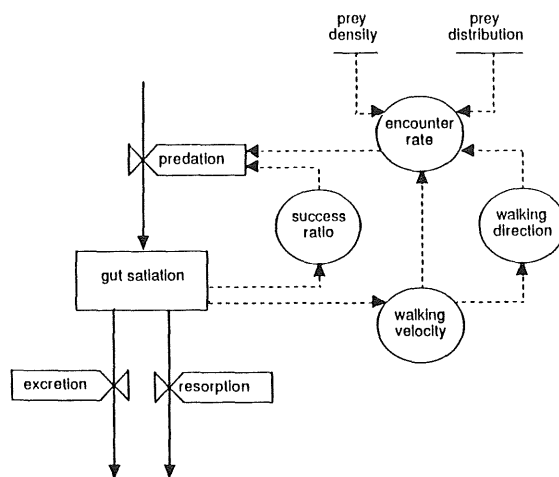


Fig. 10: Diagram illustrating relations between the motivational model (represented by the state variable gut satiation) and the behavioural components. For explanation see text.

These components of behaviour are integrated in a computer model which is linked to the model describing the motivation of the predator (Fig. 10). Prey is offered in various distributions. For P.

*coerulescens* the most important prey items are small caterpillars, aphids and maggots (HENGEVELD, 1980), which exhibit a very low walking velocity compared to the beetle's. Therefore dispersal of the prey can be neglected in the model.

The simulation model describing the internal state of the carabid and its interaction with predatory behaviour is run for aggregated and random prey distribution. Aggregated prey occur in circular clusters, each cluster containing 20 prey of 2 mg each. The distribution of prey within the clusters is random and the clusters are distributed at random over a large area. Prey density in the clusters is not affected by possible cluster overlap. Temperature is 20 °C. Overall prey densities and cluster diameters are varied to study the effect on the beetle's egg production during a season of 51 days. Each run is repeated at least ten times with different 'seeds' for the random number generator in the behavioural model. The average model outputs represent estimates of population averages because interaction between beetles is absent due to their low density.

## 3.2.2 Results

Model results (Fig. 11) show that the effect of prey density on average egg production is represented by saturation type curves which resemble the Holling type 2 functional response. Aggregated prey distributions result in higher average predation and egg production than random distributions. The greatest egg production is obtained with a cluster diameter of 40 cm. The representation of these results in Fig. 12 shows that cluster diameters from 40 to 160 cm result in similar egg productions. Cluster diameters below 40 cm result in a lower average egg production due to a low rate of prey encounter. At cluster diameters above 160 cm, egg production is low as the dispersion pattern of prey approaches the random distribution. Tortuous walk after finding a prey, which leads to more frequent visitation of previously searched area, constitutes a disadvantage to the beetle when prey is distributed at random.
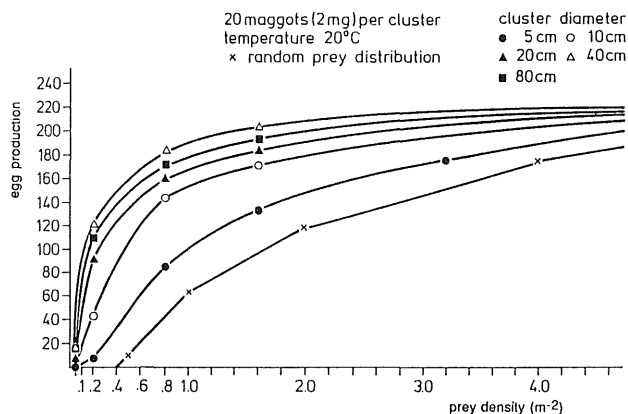


Fig. 11. Average simulated egg production of *P. coerulescens* during a 51 day period as a function of prey density for random and aggregated prey distribution. Temperature is 20 °C. Curves are eye-fitted to the simulated results; see also Fig. 12.
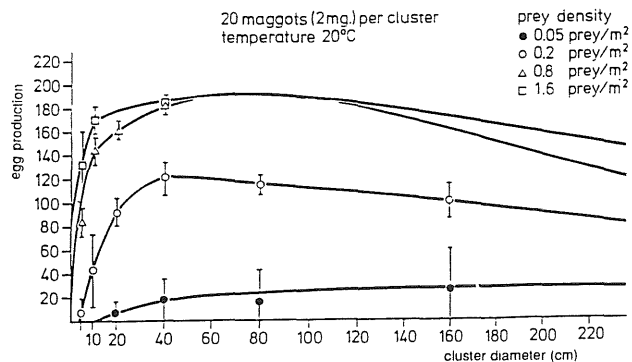
102

Fig. 12. Average simulated egg production of *P. coerulescens* during a 51 day period as a function of cluster diameter at various overall prey densities. Each cluster contains 20 prey of 2 mg each. Temperature is 20 °C. Each value represents the average of 10 runs with the stochastic model of walking behaviour. Bars represent standard deviations. Curves are eye-fitted to the simulated results Data pertain to the same simulation results as used in Fig. 11.

## 3.3 Simplification of stochastic motivation and behavioural model by means of compound simulation

The models described above can be used to analyze the effect of *P. coerulescens* on the dynamics of a pest population. Due to the stochastic nature of the model for walking behaviour and the small time-step (2 s.) this requires many simulation runs and a large amount of computer time. To avoid this, average values for predation by individual beetles, calculated with the stochastic model, could be used as input for the predator-pest model. Such an approach assumes that the model outcome resulting from average input values is identical to the average outcome with variable inputs. This is generally only true when the variation in input parameters is small or when the model is linear in its variables. In this case, however, there is a large variation in behaviour between individual beetles, especially at low prey densities (Fig. 12) while several relations (a.o. the functional response) in the model are non-linear. Thus averaging before simulation is impossible. This problem is addressed by compound simulation (FRANSZ, 1974; RABBINGE et al., 1989)

### 3.3.1 Methods

The population of beetles is divided into three classes with walking velocity as classifying criterion. Within a class the beetles are assumed to behave identically. To account for the aggregated distribution of prey, each class is subdivided into two, corresponding to beetles inside and outside a prey cluster. Thus individual beetles belong to one of six classes (Fig. 13).
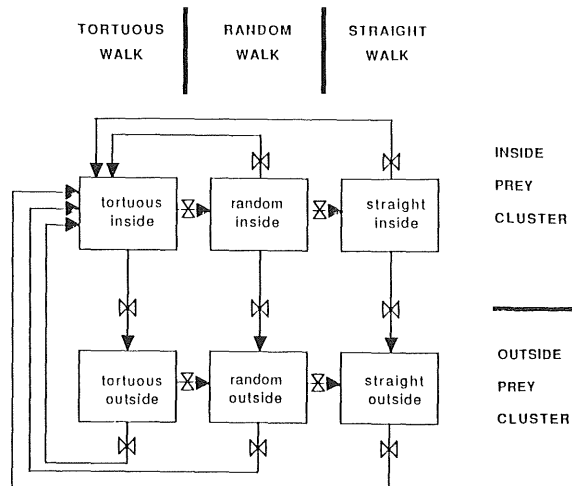
Fig. 13. Schematic representation of behavioural classes of the predator distinguished in the compound simulation model.

The complex stochastic model describing the walking behaviour of individual beetles is replaced by relations, calculated from a large number of runs with this model, which describe residence time within a prey cluster as a function of beetle walking velocity and cluster diameter (Table 1). The rate of encounters with prey clusters and prey within a cluster is approximated with analytical equations for the rate of predation on sessile prey, developed by SKELLAM (1958) and SABELIS (1981).

Table 1: Average number of steps before leaving a cluster, as a function of cluster radius and predator walking velocity, computed with the stochastic model for walking behaviour of *Pterostichus coerulescens*.

| cluster radius (m) | Predator velocity (cm s$^{-1}$) | | | | | |
|---|---|---|---|---|---|---|
| | 0.25 | 0.50 | 1 | 2 | 4 | 8 |
| 0.1 | 245 | 59 | 16 | 5 | 2 | 1 |
| 0.2 | 919 | 209 | 53 | 12 | 4 | 2 |
| 0.4 | 3335 | 886 | 189 | 40 | 9 | 3 |
| 0.8 | | 3176 | 718 | 141 | 26 | 7 |
| 1.6 | | | 2635 | 524 | 88 | 19 |
| 3.2 | | | | 2021 | 307 | 52 |
| 6.4 | | | | | 1144 | 163 |

## 3.3.2 Results

To check the validity of the simplifications, the compound simulation model is run at prey densities varying between one and 100 individuals per $m^2$. Prey, weighing 0.7 to 2 mg, is distributed at Each time step, the simulation model is run for each class of beetles separately and beetle numbers in each class are adjusted according to the outcome. Values for output variables of interest are obtained by averaging the outputs of the classes, weighed by the respective number of beetles. In this way the stochastic model of the individuals' walking behaviour is replaced by a deterministic model in which the motivational state of all beetles in a class changes by the same average rate. random. Temperature is 20 $^{\circ}$C yielding an egg production period of 51 days. Competition between carabids is assumed absent. Egg production per carabid is found to be in close agreement with the results of the stochastic model for predation.

Next, the effect of dividing the beetle population into 6 classes by two criteria is evaluated for an overall prey density of 5 $m^{-2}$, each prey weighing 2 mg. Once per day, the initial prey density is restored. In Fig. 14 the fraction of total simulation time spent to 'random walk' or 'tortuous walk' is shown. 'Straight walk' is not represented in the figure as this type of behaviour does not occur under the simulation conditions. Although tortuous walk occurs after each prey consumption, it is maintained during 11 minutes at most. Overall, tortuous walk is found during less than 5% of the time and mainly within prey clusters. The beetles predominantly exhibit random walk. As expected, the time spent in clusters increases with cluster size and density.
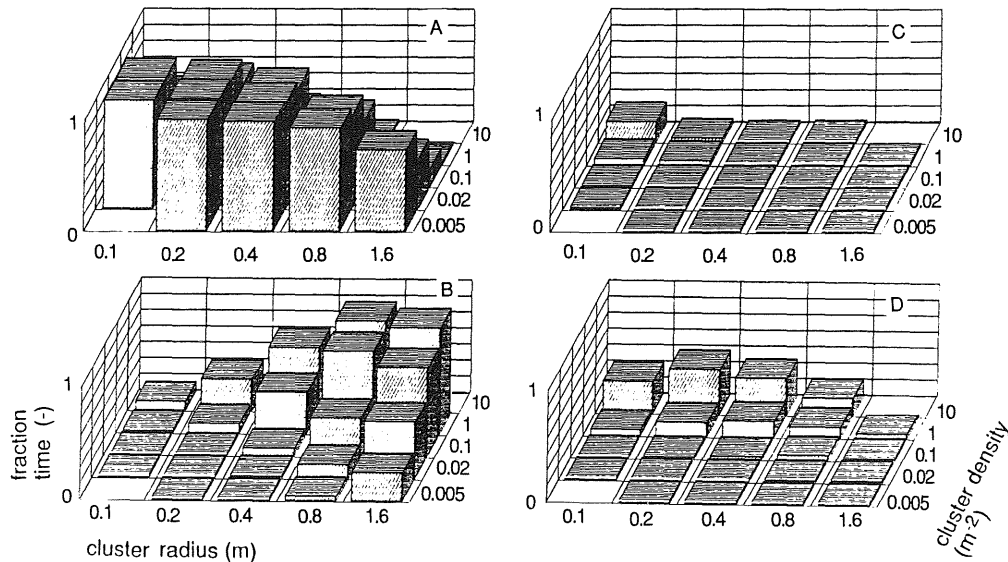


Fig. 14: Fraction of time an individual carabid spends in each of the classes of the compound simulation model as a function of cluster radius (m) and cluster density ($m^{-2}$) at an initial prey density of 5 $m^{-2}$, each prey weighing 2 mg. The initial prey density is restored at the onset of each simulated day. The graphs shown pertain to (A) random walk outside a cluster; (B) random walk inside a cluster; (C) tortuous walk outside a cluster and (D) tortuous walk inside a cluster. As 'straight walk' behaviour never occurred, the respective graphs are omitted. Not all combinations of the independent variables are represented.

Preliminary runs show that at low prey densities most time is spent to straight walk behaviour as gut satiation is low. The results suggest that, when prey density is constant, the behavioural classes can be removed from the model as beetles predominantly exhibit one type of behaviour. Then, only the location of the predator determines predation, consumption and egg production.

BAARS (1979) followed radioactively labelled *P.coerulescens* in heathland and found alternatingly straight and tortuous walk. From the insight in the dispersal of the beetle gained by the approach presented here, this can be explained by the strongly aggregated distribution of prey in such habitats. Experiments of the type BAARS (1979) carried out are extremely time consuming and do not yield information on the causes of the phenomena observed. Detailed experiments in combination with systems analysis and simulation leads to an understanding of the underlying processes and conclusions with greater general applicability.

# 4. MODELLING DISPERSAL OF APTEROUS GREEN PEACH APHID, *Myzus persicae*, AND VIRUS SPREAD IN SUGARBEET

## 4.1 Introduction

Virus yellows is an economically important disease of sugarbeet, *Beta vulgaris*, throughout the world, causing yield losses up to 60% (DUFFUS, 1973). The disease can be caused by three different viruses: beet yellows virus (BYV), beet mild yellowing virus (BMYV) and beet western yellows virus (BWYV). These viruses can occur singly or in mixed infections. BYV is a closterovirus (BAR-JOSEPH *et al.,*1979) while BMYV and BWYV are luteoviruses (DUFFUS, 1973; DUFFUS & RUSSELL, 1975). The only important vector of these viruses is the green peach aphid, *Myzus persicae*.

The disease typically occurs in patches which vary in size from 1 to 30 m diameter, depending on the date of infection (VAN DER WERF, 1988). Each patch is presumably initiated by one infectious aphid which infects one or few beet plants and starts a vector colony in the early stages of crop growth, during May or June. Subsequently, a reservoir of infectious plants is formed around the primarily-infected plant(s) while the aphid population builds up. Aphid population growth is promoted by the improved nutritional quality of virus-infected plants. Massive aphid dispersal and virus spread occurs after mid-June when adjacent plants have made leaf-contact. The aphid populations collapse in July due to predation, diseases, parasitism and decreasing suitability of the beet plant, resulting in an arrest of virus spread.

Spread of viruses by vectors constitutes a complicated three-species interaction between a plant-host, a virus-pathogen and an insect-vector. Often natural enemies are also involved in this interaction as they can reduce the number of vectors and alter their behaviour (ROITBERG *et al.,* 1979). Because of this complexity, analysis of processes in the field is difficult and observations can often be explained in more than one way. Modelling the dispersal and feeding behaviour of *M. persicae* in relation to the development of plant infectivity, provides a means of studying the importance of the various processes which are involved in virus spread. Such an approach

complements field studies by integrating results of experiments on the process-level. The latter experiments concern e.g. walking and feeding behaviour of the vector, virus transmission characteristics, latency period and incubation period.

Here a brief account is given of such a simulation study of virus spread. A more detailed description of the model and a program listing is given by RIESEBOS (1988).

## 4.2 Structure of the model

The model describes a 12 x 12 $m^2$ area with 1152 sugarbeet plants (7.8 plants $m^{-2}$) and a time-varying number of aphids. All plants have the same number of leaves and are equidistantly spaced. The status of each plant and each aphid is characterized by a memory structure encompassing a small number of variables. For a plant, these variables describe (1) the infection date, (2) the number of healthy leaves (i.e. those emerged before infection), (3) the proportion of leaves infected, (4) the presence of symptoms and (5) the number of aphids on the plant. Variables describing an aphid are (1) the time of birth, (2) the time of virus acquisition, (3) the time of latest displacement, (4) the position in the field, and (5) morph, apterous (wingless) or alate (winged).

Model results are compared to data from an experiment in which the effect of sowing date and number of introduced *M. persicae* on spread of BYV was studied (VAN DER WERF, 1988). In this experiment three plants located near the centre of each plot were infected with BYV on 23 June. On these plants 2, 9 or 65 *M. persicae* were released on 25 June. The plants had been sown on either 18 April (regular) or 20 May (late). Each of the six treatments was replicated in four plots.

Momentaneous temperatures are calculated by fitting sinusoids between measured minimum and maximum temperatures (ANONYMUS, 1986). Plant leaf number, which provides an indication of physiological age, is a function of accumulated temperature (MILFORD *et al.*, 1985a,b; VAN DER WERF, 1988).

BYV is a semi-persistent virus. In accordance with this, aphids in the model loose the ability to transmit virus at moulting. An aphid moults every 36 hours. Simplifying from SYLVESTER (1961), the retention period (RP) is taken as exactly 12 h. following acquisition. Based on data of SYLVESTER (1956a,b) and HEATHCOTE and COCKBAIN (1964), the acquisition feeding period (AFP), i.e. the feeding time needed for an aphid to acquire virus from an infected plant, is set at 2 h. while the transmission feeding period (TFP) is estimated as 1 h. Whenever the feeding time (which in the model equals the time not spent to walking) on a leaf equals or exceeds the TFP or AFP, virus acquisition or transmission occurs, provided that a non-viruliferous aphid feeds on an infectious leaf or an infectious aphid on a healthy plant. *M. persicae* shows preference for young beet leaves (JEPSON, 1983). Various degrees of aphid preference for heart leaves (those smaller than 10% of their final area) can be simulated (see below).

Following infection, plants become infectious after a latency period (LP). The LP increases linearly with time, from 4 days in the seedling phase to 12 days in old plants (VAN DER WERF, 1988). Counting from infection, symptoms become apparent after the incubation period (IP), which is

influenced by plant physiological age and temperature. The IP increases from *ca.* 3 weeks in June to 2 months in August (VAN DER WERF, 1988). Aphids can acquire virus from the infectious leaves on infected plants, irrespective of the presence of symptoms. No virus can be acquired from a healthy or a latent infected plant or from those leaves on an infectious plant that appeared before infection.

Aphid population dynamics is not simulated in this model because insufficient data are available to do this with sufficient precision, due to the difficulty of quantifying predation.and immigration. Therefore, if a population dynamics module were included it would be difficult to determine whether possibly aberrant model outcomes with respect to virus spread resulted from errors in the virus dissemination part of the model or from incorrectly simulated population dynamics of the vector. To avoid these problems, the model mimics the population development observed in the field. Every simulation-day, simulated aphid numbers are compared to field-counts made in the centre of the plot or interpolations from these numbers. When the difference exceeds 10%, new aphids are added or existing ones removed to compensate the difference. Newly added aphids are 0 h. old and non-viruliferous, mimicking births. They are borne on plants which are already infested by aphids, in proportion to the number of aphids already present on the plant. Aphids are removed at random, assuming that predators do not discriminate between aphid instars and cause a fixed relative mortality rate, irrespective of aphid density. (This means that predators aggregate in areas with high aphid density, approximately in proportion to the number of aphids present). Winged aphids leave the field. No immigration occurs.

Predation by ladybirds reduces the number of viruliferous aphids in the field. It is difficult, however, to estimate the impact of predators on the basis of predator density (FRAZER, 1988). Therefore, the predation rate in the model, 1% predation per hour, was determined in another way, by iteratively maximizing the agreement between the simulated and the observed age distribution of the aphid population, throughout the season. At lower relative predation rates than .01 h.$^{-1}$ the proportion of older nymphal stages was overestimated while at higher relative predation rates the proportion of older stages was too low.

The dispersal and feeding behaviour of *Myzus persicae* plays a central role in the model. Aphid dispersal activity is expressed by the variable P, the proportion of aphids walking within an hour. When the number of leaves is smaller than 12, P has a small value, 0.025. This gives expression to the almost complete absence of dispersal in young crops, observed in the field (VAN DER WERF, 1988), which is probably due to the high nutritional quality of young plants as well as to lack of leaf contact between them. When the number of leaves is greater than 12, P is calculated as

$$P = .01 \times (65 - \frac{N}{2.5}) \tag{14}$$

where N is the number of leaves on the plant. As 2 to 3 leaves emerge each week, aphid activity decreases slowly during the season. Equation 14 is based on calculations of the proportions of newly-emerged, expanding and fullgrown leaves on the beet plant throughout the season (MILFORD *et al.*, 1985b) and estimates of the dispersiveness or aphids on these three types of
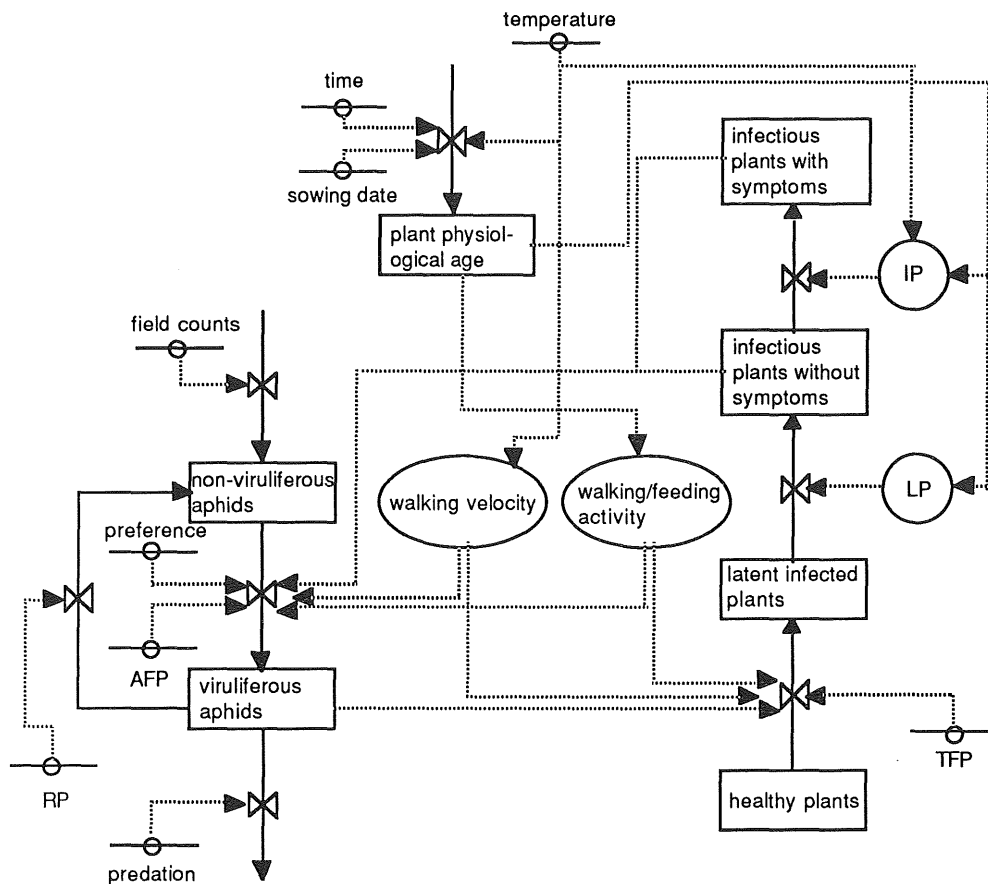
108

Fig. 15: Relation diagram of model of virus spread by Myzus persicae in sugarbeet, drawn according to conventions of FORRESTER (FORRESTER, 1961; DE WIT and GOUDRIAAN, 1978). Boxes ( ☐ ) represent states, solid arrows ( ——▶ ) represent fluxes between states, valves ( ⋈ ) in the solid arrows denote rates while hatched arrows ( ······▶ ) pointing to valves denote control of rates by state variables. Circles or ovals denote auxiliary variables. External variables, not influenced by the model, are denoted by ——○—— .

leaves (JEPSON, 1983), assuming a homogeneous distribution of aphids over the plant (RIESEBOS, 1988).

The distance covered each hour (D; linear displacement), is an optimum type function of aphid age, adult aphids walking more rapidly than young or aged ones, and temperature:

$$D = D_{max} \times \left(1 - \left(\frac{A - 25}{25}\right)^2\right) \times \left(1 - \frac{9}{4} \times \left(\frac{T - 30}{30}\right)^2\right) \tag{15}$$

where A is denotes the age of the aphid in days and T the temperature in °C. The value of $D_{max}$ is 125 cm, in accordance with data of FERRAR (1968). The walking direction is drawn from a uniform distribution over $(-\pi, \pi)$

The main components and relations of the model are summarized in the relation diagram of Fig. 15. Runs with the model were made with two purposes: (1) to compare simulation results with field data and thus test our conception of the system, and (2) to determine which parameters have the largest effect on model behaviour and should therefore be studied in more detail experimentally.

The model is written in the programming language C (KERNIGHAN & RITCHIE, 1978). The time step is one hour.

## 4.3 Results

Simulation results and field data are compared in Figs. 16A and B which show the time-course of the number of yellowed plants per plot for each of the six treatments. Fair overall agreement exists between the two figures. Agreement is very good in 4 treatments but the model underestimates virus spread resulting from release of two *M. persicae* in late-sown sugarbeet while it overestimates the spread resulting from release of 65 *M. persicae* in early-sown sugarbeet. This suggests that parameters related to plant age are more important in reality than they are in the model in its present form.

The overall similarity of simulated and observed spread is a promising result as model structure and parameter estimates on the process level are in agreement with the literature. Nevertheless, further model validation is needed to obtain a better insight in possible shortcomings of the model.
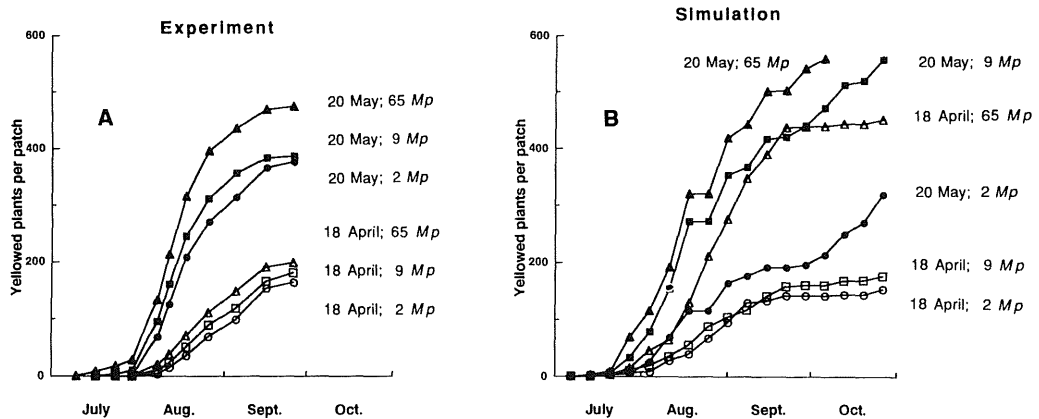


Fig. 16: Comparison of observed (A) and simulated (B) increase of number of BYV-infected plants with symptoms, in relation to crop sowing date and numbers of *Myzus persicae* introduced on 25 June
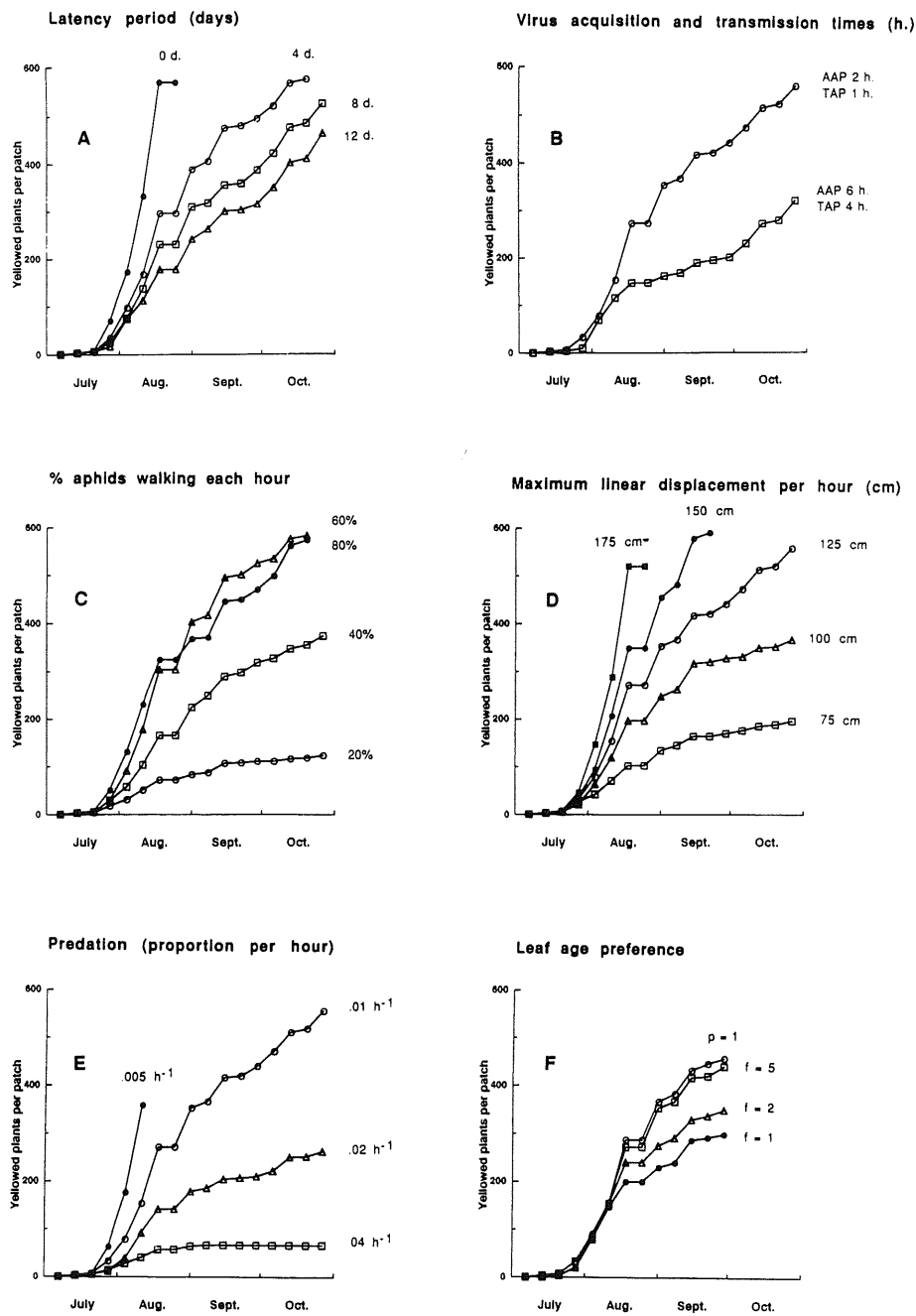
Fig. 17: Sensitivity analysis of model of virus spread by *Myzus persicae* in sugarbeet. (A) latency period, (B) virus transmission characteristics, (C) walking activity, (D) walking distance per hour, (E) relative predation mortality and (F) leaf age preference as indicated by the factor $f_{pref}$ in the formula $P = f_{pref} \times Y$, where p is the proportion of aphids feeding on young leaves and Y is the proportion of young leaf area on the plant.

Sensitivity analysis provides a means to determine the influence of model structural components and parameter estimates on the results. Here a 'fine' sensitivity analysis (RABBINGE *et al.,* 1989) for six parameters is presented (Fig. 17A-F). The analysis applies to sugarbeet sown on 20 May and infested with 9 *M. persicae.* For the LP reasonably precise values were available so that Fig.17A illustrates its theoretical importance. The LP has a large impact in our model which confirms results obtained with more basic epidemiological models (ZADOKS and SCHEIN, 1979). For the other five parameters, only rough estimates could be derived from the literature. The figures 17B-F give therefore an indication of the uncertainty in predicted epidemics resulting from the imprecision of the parameter estimates in the model. For instance, fig. 17B shows that virus acquisition and transmission times have a considerable effect on spread. While several authors determined feeding times needed for the acquisition and transmission of beet viruses under greenhouse conditions with several species of test plants, possible influences of sugarbeet host plant quality (age) in the field have been neglected. Therefore, those studies may have limited relevance for the prediction of processes occurring in the field. Aphid behaviour, here expressed by two parameters, activity and distance covered, has been very little studied, resulting in a considerable uncertainty about model predictions (Fig. 17C,D). Predation appears to be a particularly important factor in the model (Fig. 17E), indicating that it deserves more attention in experimental research, the more so because in the model predators influence only the proportion of aphids which are viruliferous and not the total number. Thus, in reality, the impact of natural enemies on virus spread may be even greater than indicated by the simulation results. The effect of leaf age preference is relatively small (Fig. 17F).

Figs. 18 and 19 show the spatial distribution of the aphids and the virus-infected plants at two-week intervals in sugarbeet sown on 18 April and 20 May, respectively. Aphids become more numerous and infect a greater number of plants in the late-sown crop. These plots clearly show the temporal separation of the presence of the aphids and the resulting increase in the number of plants with symptoms. Virus spread occurs when the aphids are numerous and disperse over the plot, predominantly between mid-June and mid-July in this case. Symptoms only become obvious at the end of this period. Spraying against aphids at this point is useless as further spread will be negligible, while plants infected at this advanced development stage suffer little damage.The appearance of a 'secondary' focus in top right-hand corner of Fig. 18 is worth noting. In field studies this would generally be regarded as the result of virus spread by alatae, 'hopping' in the field. This simulation shows that such a secondary patch may be also initiated by apterous aphids.

Figs. 18 and 19 (next pages): Simulated spatial distribution of aphids and virus-infected plants at two-week intervals in sugarbeet sown on 18 April or 20 May. In the model the plots are inoculated with BYV on 23 June and infested with 9 *Myzus persicae* 2 days later. Dates: (a) 23 June; (b) 7 July; (c) 21 July; (d) 4 August; (e) 18 August; (f) 1 September; (g) 15 September; (h) 29 September; (i) 13 October and (j) 27 October. The upper plot of each date denotes the spatial arrangement of BYV-infected plants in a 12 x 12 $m^2$ field plot. Numbers indicate the percentage of infected leaf area on plants without symptoms: (1) 0-10%, (2) 10-20%, etc. Plants with symptoms are indicated by shaded blocks, the four degrees of shading indicating percentages infected leaf area of ( ∷ ) 0-30%, ( ▦ ) 30-60%, ( ▦ ) 60-90% and ( ▉ ) 90-100%. The lower plot denotes the number of aphids per plant. Numbers of aphids per plant exceeding 9 are indicated by a shaded area ( ∷ ).
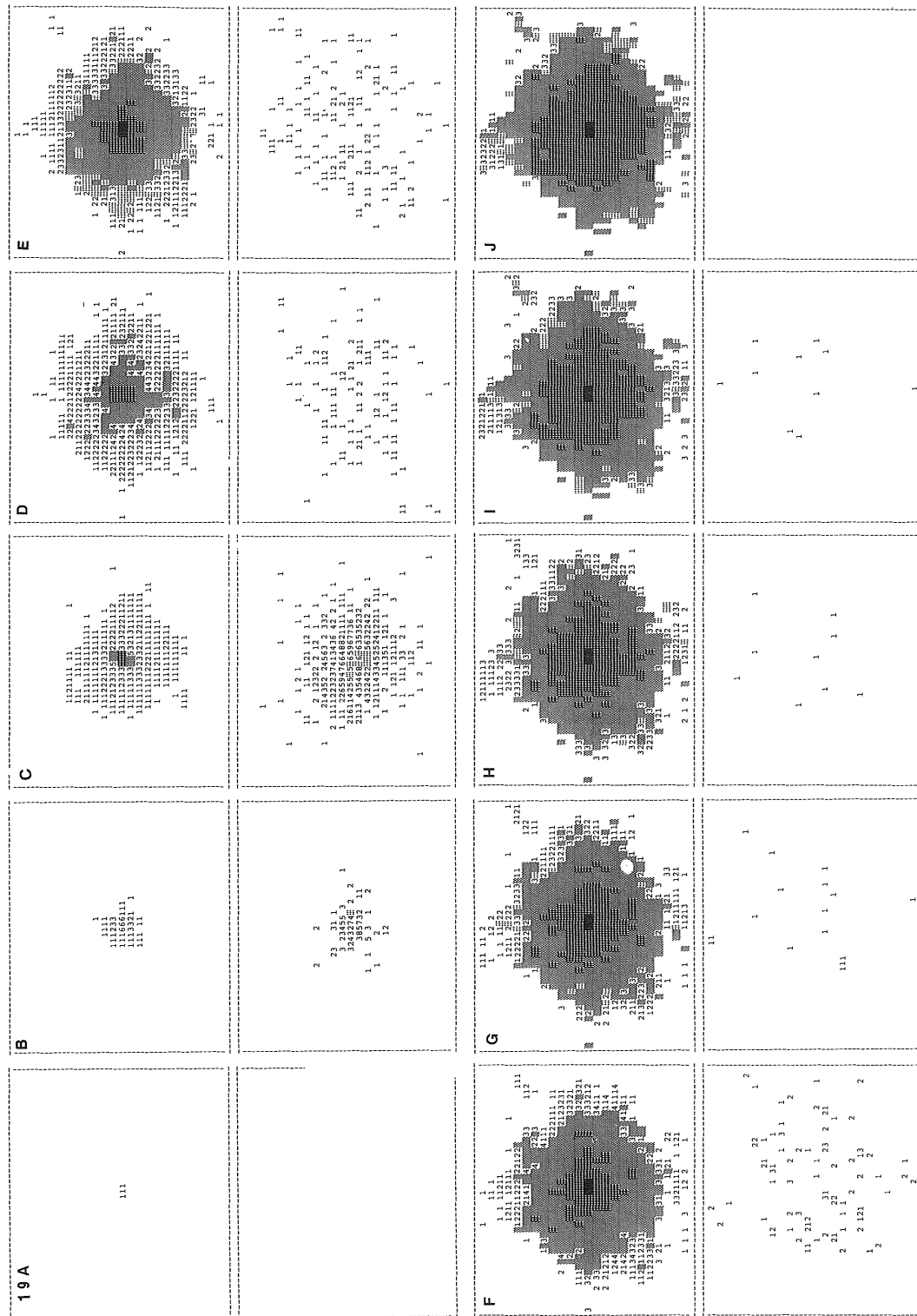
# 5. CONCLUDING REMARKS

The three spatial models of population dynamics, described in his paper, simulate the processes underlying system behaviour at different integration levels. The first model - spore dispersal in a forest - describes the system with diffusion and flux equations which apply to spore 'masses', while neglecting the behaviour of individual spores. In the second example - predation by carabids - a detailed analysis of the individuals' motivation and resulting predatory behaviour is made. Subsequently, general rules derived from this analysis are used in a compound simulation population model which distinguishes six classes of beetles but no individuals. In the third example - spread of beet viruses - the behaviour of the individual virus vectors is simulated. Computation time increases with the amount of detail included, from example 1 to 3.

The level of complexity that must be simulated in a given case, depends upon the aim of the study and on the available experimental data and computing resources. In the risk analysis pertaining to biocontrol with silverleaf fungus, simulation of individual spore trajectories would have been cumbersome while enormous computing times would have been needed to account for the large number of individual spores involved. The simpler approach adopted by DE JONG (1988) suffices to obtain the desired estimates of spore escape. In the case of the carabids, detailed information on the beetle's motivation and predatory behaviour was obtained in studies aiming at a better understanding of the survival strategy of the species in a temporally and spatially variable habitat. To evaluate the potential for natural aphid control by populations of a closely related *Pterostichus* species, living in arable lands and exhibiting similar behaviour as the one studied in heathlands, the complicated models of the motivational and behavioural processes had to be discarded because of their short time step (2 s.). The most important trait, the beetle's residence time in a prey colony, is captured in a two way table with the beetle's walking velocity and the prey colony's diameter as entries. This enables compound simulation, distinguishing only six classes of beetles. Finally, in the beet virus model, the state and behaviour of the individual vectors is accounted for. This is necessary because in this case every single vector has a potentially large impact, each infective puncture leading to the infection of a whole plant. The relatively large time step in this model (1 h.), which is made possible by the large size of the objects the vector is encountering, beet plants, allows a detailed representation of the system.

Simulation models offer the opportunity to integrate large amounts of information, gathered on the process level, in a simulation model with behaviour comparable to the real world. In this way the correctness of the conception of the system can be tested. It is often found that the behaviour of systems which are commonly regarded as 'well understood' cannot be easily predicted. Thus, gaps in knowledge are located. If, on the other hand, model predictions are satisfactory, the model can be simplified to a tool for decision making. Another merit of the modelling approach is that imaginary experiments can be performed. Such imaginary experiments can be used for orienting experimental research (beet virus model) or risk analysis (silverleaf fungus model). Experiments with the carabid model suggest that *Pterostichus* species have potential to suppress aphid outbreaks in arable crops.

Models including spatial dynamics and variability have been developed more recently than 'homogeneous' models, due, in part, to the large amounts of computing time needed. If a homogeneous model for the development of a disease is converted into a 'heterogeneous' model, accounting for 1000 interacting patches in a field, the number of state variables and rate equations in the model increases also a 1000-fold. An even greater number than that is probably needed in heterogeneous models to account for migration. As computers have become faster (some researchers even use 'supercomputers'; ONSTAD, 1988) the possibility for simulating pests and diseases in a spatially realistic way becomes much greater. This results in the ability to account for density-dependent processes in a more realistic way such that better population models may result.

## ACKNOWLEDGEMENTS

# REFERENCES

ANONYMOUS, 1986. Maandoverzichten van het weer, meteostation Swifterbant, issued by the Department of Physics and Meteorology, Agricultural University, Wageningen, the Netherlands.

BAARS, M.A., 1979. Patterns of movement of radio-active carabid beetles. Oecologia 44, 125-140.

BAR-JOSEPH, M., S.M. GARNSEY AND D. GONSALVES, 1979. The closteroviruses: a distinct group of elongated plant viruses. Adv. Virus Res. 25, 93-168.

CORY, J.S. and P.F. ENTWISTLE, 1988. Ecological considerations and research ramifications of releasing genetically manipulated viruses. Proc. 18th int. Congr. Entomol., 252.

DAAMEN, R.A., 1986a. Measures of disease intensity in powdery mildew (*Erysiphe graminis*) of winter wheat. 1. Errors in estimating pustule number. Neth. J. PL. Path. 92, 197-206.

DAAMEN, R.A., 1986b. Measures of disease intensity in powdery mildew (*Erysiphe graminis*) of winter wheat. 2. Relationships and errors of estimation of pustule number, incidence and severity. Neth. J. PL. Path. 92, 207-222.

DE JONG, M.D., 1988. Risk to fruit trees and native trees due to control of black cherry (*Prunus serotina*) by silverleaf fungus (*Chondrostereum purpureum*). PhD. thesis, Agricultural Univ. Wageningen, the Netherlands, 138 pp. (in Dutch; summary and description of computer programs in English).

DE JONG, M.D. and P.C. SCHEEPENS, 1985. Analyse van risico bij bestrijding van Amerikaanse vogelkers (*Prunus serotina*) met (*Chondrostereum purpureum* voor planten waartegen de bestrijding niet is gericht. Internal Rep. nr. 58 of Centre for Agrobiological Res., Wageningen, the Netherlands, 35 pp.

DE JONG, M.D., P.C. SCHEEPENS and J.C. ZADOKS, 1988. Risk analysis for biological control. A Dutch case study in biocontrol of *Prunus serotina* by means of the fungus *Chondrostereum purpureum*. Submitted.

DUFFUS, J.E., 1973. The yellowing virus diseases of sugar beet. Adv. Virus Res. 18, 347-386.

DUFFUS, J.E. and G.E. RUSSELL, 1975. Serological relationship between beet western yellows and beet mild yellowing viruses. Phytopath. 65, 811-815.

FERRAR, P., 1968. Interplant movements of apterous aphids, with special reference to *M. persicae* (Sulz.) (Hom. Aphididae). Bull. entomol. Res. 58, 653-660.

FORRESTER, J.W., 1961. Industrial dynamics. MIT-press, Boston.

FRANSZ, H.G., 1974. The functional response to prey density in an acarine system. Simulation Monographs, PUDOC, Wageningen, 149 pp.

FRAZER, B.D., 1988. Coccinellidae. In: A.K. MINKS and P. HARREWIJN (Eds.): Aphids, their biology, natural enemies and control. Elsevier, Amsterdam, p. 231-247.

GOUDRIAAN, J. , 1977. Crop micrometeorology: a simulation study. Pudoc, Wageningen, the Netherlands, 249 pp.

GOUDRIAAN, J., 1979. MICROWEATHER simulation model, applied to a forest. In: S. HALLDIN (Ed.): Comparison of forest water and energy exchange models. Int. Soc. Ecol. Modelling (ISEM), Copenhagen, 47-57.

GROSCLAUDE, C., 1969. Le plomb des arbres fruitiers. VII. Observations sur les carpophores et les spores du *Stereum purpureum*. Ann. Phytopath. 1, 75-85.

HEATHCOTE, G.D.and A.J.COCKBAIN, 1964. Transmission of beet yellows virus by alate and apterous aphids. Ann. appl. Biol. 53, 259-266.

HENGEVELD, R., 1980. Poliphagy, oligophagy and food specialisation in ground beetles (Coleoptera, Carabidae). Neth. J. Zool. 30, 564-584.

HOLLING, C.S., 1966. The functional response of invertebrate predators to prey density. Mem. entomol. Soc. Canada 48, 1-86.

HUBER, J., 1388. Assessing the risks of releasing genetically modified insect pathogens. Proc. 18th int. Congr. Entomol., 250.

JEPSON, P.C., 1983. A controlled environment study of the effect of leaf physiological age on the movements of apterous *Myzus persicae* on sugar-beet plants. Ann. appl. Biol. 103, 173-183.

KAREIVA, P. and G. ODELL, 1987. Swarms of predators exhibit 'preytaxis' if individual predators use area-restricted search. Amer. Nat. 130, 233-270.

KERNIGHAN, B.W. and D.M. RITCHIE, 1978. The C programming language. Englewood Cliffs: Prentice-Hall, 228 pp.

MILFORD, G.F.J., T.O.POCOCK and J. RILEY, 1985a. An analysis of leaf growth in sugarbeet. II. Leaf appearance in field crops. Ann. appl. Biol. 106, 173-158.

MILFORD, G.F.J., T.O.POCOCK, J. RILEY and A.B. MESSEM, 1985b. An analysis of leaf growth in sugarbeet. III. Leaf expansion in field crops. Ann. appl. Biol. 106, 187-203.

MOLS, P.J.M., 1979. Motivation and walking behaviour of the carabid beetle *Pterostichus coerulescens* L. at different densities and distributions of the prey. Miscell. Papers Agric. Univ. Wageningen 18, 185-198.

MOLS, P.J.M., 1983. Simulation of the motivation and egg production of the carabid beetle *Pterostichus coerulescens* L. Rep. 4th Symp. Carabids 1981, 35-43.

MOLS, P.J.M., 1986. Predation in the carabid beetle *Pterostichus coerulescens* L. In: P.J.DEN BOER, L. GRUM & J. ZYSZKO (Eds.): Feeding behaviour and accessibility of food for carabid beetles. Rep. 5th ~Meeting Europ. Carabidologists. Warsaw Agric. Univ.-SGGW-AR, 49-58.

MOLS, P.J.M., 1987. Hunger in relation to searching behaviour, predation and egg production of the carabid beetle *Pterostichus coerulescens* L.: Results of simulation. Acta Phytopathologica et Entomologica Hungarica 22, 187-296.

ONSTAD, D.W., 1988. Simulation model of the population dynamics of *Ostrinia nubialis* (Lepidoptera: Pyralidae) in maize. Environm. Entomol. In press.

RABBINGE, R., 1976. Biological control of the fruit tree-spider mite. PUDOC, Wageningen, 228 pp.

RABBINGE, R., S.A. WARD & H.H. VAN LAAR (Eds.), 1989. Simulation and systems management in crop protection. PUDOC, Wageningen. (in press).

RIESEBOS, W., 1988. Een model voor de verspreiding van beet yellows virus door *Myzus persicae*. M.Sc. thesis, Department of Theoretical Production Ecology, Agricultural University, Wageningen, the Netherlands, 140 pp. + appendices (in Dutch).

ROITBERG, B.D., J.H. MYERS and B.D. FRAZER, 1979. The influence of predators on the movements of apterous pea aphids between plants. J. Anim. Ecol. 48, 111-122.

SABELIS, M.W., 1981. Biological control of two-spotted spider mites using phytoseiid predators. Part 1. Agricultural Research Report, 910, PUDOC, Wageningen, 242 pp.

SKELLAM, J.G., 1958. The mathematical foundations underlying line transects in animal ecology. Biometrics 14, 385-400.

SOUTHWOOD, T.R.E., 1978. Ecological methods. 2nd ed., Chapman and Hall Publ., London, 524 pp.

SYLVESTER, E.S., 1956a. Beet yellows virus transmission by the green peach aphid. J. econ. Entomol. 49, 789-800.

SYLVESTER, E.S., 1956b. Beet mosaic and beet yellows virus transmission by the green peach aphid. J. Amer. Soc. Sugar Beet Technol. 9, 58-61.

SYLVESTER, E.S., 1961. Re-evaluation of retention of beet yellows virus by green peach aphids under fasting and feeding conditions. Virology 14, 467-479.

UPTON, G.J.G. and B. FINGLETON, 1985. Spatial data analysis by example. Wiley, New York, 410 pp.

VAN DER WERF, 1988. Yellowing viruses in sugarbeet; epidemiology and damage. Ph. D. thesis Agric. Univ. Wageningen, the Netherlands, 152 pp.

WAGENMAKERS, P.S., 1984. Simulatie van de sporeverspreiding van *Chondrostereum purpureum* in een bos. M.Sc. Thesis, Agicultural University, Department of Theoretical Production Ecology, Wageningen, the Netherlands. 58 pp.

WARD, S.A., R. RABBINGE and W.P. MANTEL, 1985a. The use of incidence counts for estimation of aphid populations. 1. Minimum sample size for required accuracy. Neth. J. Pl. Path. 91, 93-99.

WARD, S.A., R. RABBINGE and W.P. MANTEL, 1985b. The use of incidence counts for estimation of aphid populations. 2. Confidence intervals from fixed sample sizes. Neth. J. Pl. Path. 91, 100-104.

WARD, S.A., K.D. SUNDERLAND, R.J. CHAMBERS AND A.F.G. DIXON, 1986. The use of incidence counts for estimation of aphid populations. 3. Population development and the incidence-density relation. Neth. J. Pl. Path. 92, 175-183.

ZADOKS, J.C. and R.D. SCHEIN, 1979. Epidemiology and plant disease management. Oxford Univ. Pr., New York, 427 pp.