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MODELS IN THE EPIDEMIOLOGY OF BEET YELLOWING VIRUSES

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Summary

Three models relating to the epidemiology of beet virus yellows are discussed and compared. The descriptive model of WATSON et al. (Ann. Appl. Biol. 81, 181-198, 1975) predicts final virus yellows incidence on the basis of weather data. HARRINGTON et al. (Ann. Appl. Biol. 114, 459-469, 1989) revised this model, incorporating biological variables in the regression equation. A second model, described by WATSON and HEALY (Ann. Appl. Biol. 40, 38-59, 1953), calculates the time-course of a virus yellows epidemic on the basis of aphid catches. This analytical model is based on a set of strongly simplifying assumptions concerning the movement of the vector and the transmission of virus. The third model simulates the development of foci of beet yellows virus on the basis of a detailed description of the behaviour of individual vectors. This model is used to study the importance of the various processes underlying virus spread.

Sommaire

Trois modèles ayant trait a l'épidemiologie des virus de la jaunisse sont discutés et comparés. Le modèle descriptif de WATSON et al. (Ann. Appl. Biol. 81, 181-198, 1975) prédit l'incidence finale des virus de la jaunisse à partir des données météorologiques. Ce modèle a été revisé par HARRINGTON et al. (Ann. Appl. Biol. 114, 459-469, 1989) qui ont introduit des variables biologiques dans l'équation de régression. Le deuxième modèle, décrit par WATSON et HEALY (Ann. Appl. Biol. 40, 38-59, 1953) calcule la dynamique temporelle d'une épidemie de jaunisse à partir des prises de pucerons. Ce modèle analytique fait des hypothèses fortement simplificatrices quant au déplacement des vecteurs et à la transmission des virus. Le troisième modèle simule le developpement des taches de jaunisse de la betterave, à partir d'une description détaillé du comportement des vecteurs individuels. Ce modèle est utilisé pour étudier l'importance relative des divers processus intervenant dans la dispersion des virus.

Zusammenfassung

Drei Modelle der Epidemiologie von Vergilbungsviren in Zuckerrüben werden beschrieben und verglichen. Das beschreibende Modell von WATSON et al. (Ann. Appl. Biol. 81, 181-198, 1975) macht eine Voraussage über die finale Vergilbungsinzidenz eines Jahres auf Grund des vorhergehenden Winterwetters. HARRINGTON et al. (Ann. Appl. Biol. 114, 459-469, 1989) passten dieses Model an und introduzierten biologische Variabelen in die Regressionsvergleichung. Das zweite Modell, beschrieben von WATSON et HEALY (Ann. Appl. Biol. 40, 38-59, 1953) berechnet den Zeitverlauf einer Vergilbungsepidemie auf Grund von Vektorfängen. Dieses analytische Modell basiert auf stark simplifizierende Annahmen über das Verhalten des Vektors und die Transmission des Virus. Das dritte Modell simuliert die Verbreitung von Vergilbungsviren auf Grund einer detaillierten Beschreibung des Verhaltens der einzelnen Vektoren. Das Modell erklärt die Entwicklung von Vergilbungsherden im Gewächs. Es wird angewand um den Belang der verschiedenen Prozesse, die der Verbreitung unterliegen, zu untersuchen.

INTRODUCTION

Virus yellows is an economically important disease of sugarbeet, *Beta vulgaris*, throughout the world, causing maximum yield losses of about 50% (DUFFUS, 1973; HEIJBROEK, 1988a,b; SMITH, 1988; VAN DER WERF, 1988). 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 together in a plant. The green peach aphid, *Myzus persicae* is the most efficient vector and the only one of practical importance (WATSON et al., 1951; BJÖRLING, 1952; HEATHCOTE, 1966). BYV is a closterovirus (BAR-JOSEPH et al., 1979) while BMYV and BWYV are both luteoviruses (DUFFUS, 1973; DUFFUS and RUSSELL, 1975). BMYV is only reported from Europe whereas BYV and BWYV occur worldwide. Strikingly, most BWYV strains from Europe do not infect sugarbeet (SMITH and HINCKES, 1985) whereas most strains from other continents do (DUFFUS, 1973). The taxonomy of luteoviruses is still problematic (ROCHOW and DUFFUS, 1981; WATERHOUSE et al., 1988).

The importance of yellowing viruses in sugarbeet varies from year to year (DUNNING, 1988; HEATHCOTE, 1988), depending on the buildup of a virus reservoir over the years and on the weather conditions in specific years. These affect the winter survival of aphids and viruses and the time of initial infections in the sugarbeet crop. Therefore, the kind of chemical control measures needed, if any, varies from year to year, and warning services have been set up to advise the growers (DEWAR, 1988). Presently, most warning services work on an empirical basis, but their advices may in the future be more and more underpinned by the results of quantitative models of the epidemiology and yield impact of the viruses.

Models are simplified representations of real systems. Distinction is made between static and dynamic models and between descriptive and explanatory models (DE WIT and RABBINGE, 1989). Dynamic models describe the way in which a system changes over time. They are generally formulated as differential or difference equations. Static models have no time-component involved. Descriptive models show the existence of relations between elements, but do not explain the relations. Explanatory models distinguish two levels of organization: the system level and the process level. The behaviour of the system is explained by quantifying the underlying processes and relations.

In this paper a review of existing models in the field of beet virus yellows epidemiology is given. First, a descriptive static model for the relation between virus yellows incidence and weather conditions, due to WATSON et al. (1975) is described. Subsequently a descriptive dynamic model for the spread of virus yellows, developed by WATSON and HEALY (1953) is discussed. Finally an explanatory dynamic model for the spread of beet yellows virus, developed by RIESEBOS (1987), is presented.

REGRESSION MODELS

WATSON et al. (1975) derived a regression equation for the prediction of virus yellows incidence on the basis of 21 years (1951-1971) of field observations:

$$y = 100 \cdot \left[\sin \left(110.5 - 77 \cdot {}^{10}\log (x_1) + x_2 \right) \right]^2 \qquad (1)$$

where

y = Virus yellows incidence (%) at the end of August,
x_1 = the number of frost-days in January, February and March, and
x_2 = the mean temperature in April.

For the years included in WATSON's analysis the formula gives accurate predictions (Fig. 1), but only one year with a high virus yellows incidence was represented in the data-set. The formula failed to predict a later outbreak in 1974 (WATSON et al., 1975). Incidences in 1975 and 1976 were also significantly underestimated (HEATHCOTE, 1986). According to WATSON et al. (1975) the failure in 1974 was due to the occurrence of unusual conditions: 'Local sources of virus will have increased during the two previous mild winters, and sugar-beet seed crops in 1974 were more heavily infected with yellows than usual.... Also, in 1974 the crop was backward and irregular due to drought ...'. These conditions had been absent in the years used for the construction of the regression equation.

HARRINGTON et al. (1989) have developed a new regression model to forecast the incidence of virus yellows in England. They used data from 1965 onwards to allow data on the time of flight of *M. persicae*, as determined by the Rothamsted Insect Survey suction traps (MACAULY et al., 1988), to be incorporated in the regression equation (2):

$$y = 100 \cdot \left[\sin \left(0.37 \cdot \arcsin \sqrt{\frac{x_1}{100}} - 25.7 \cdot x_2 - 3.125 \cdot x_3 + 0.0092 \cdot (x_3)^2 \right) \right]^2$$

where

y = predicted virus yellows incidence (%),
x_1 = virus yellows incidence in previous year (%),
x_2 = number of ground frosts in January and February, and
x_3 = day (from 1 January) when the first *M. persicae* are caught.

The revised regression model gives a better fit to the high incidences in 1974 and 1975 and it is presently used in the British warning system. Unfortunately, the data set used by HARRINGTON c.s. has the same shortcoming as that of WATSON et al., namely that few years with high incidences are represented. The new regression model can therefore, among other reasons, not be relied upon exclusively for vector control decisions. Its predictions are compared to other informations, a.o. local sources of virus and overwintering sites for aphids, to make a final integrated extension decision (HARRINGTON et al., 1989).

Attempts to establish regressions similar to those of WATSON et al. for Dutch conditions have been unsuccessful (W. HEIJBROEK, pers. comm.). Apparently, the processes underlying the epidemiology of yellowing viruses in the Netherlands are so different from those in England that they can not be described, using the same correlative approach. Within England incidences in different regions are highly correlated (WATSON et al., 1975; HARRINGTON et al., 1989).

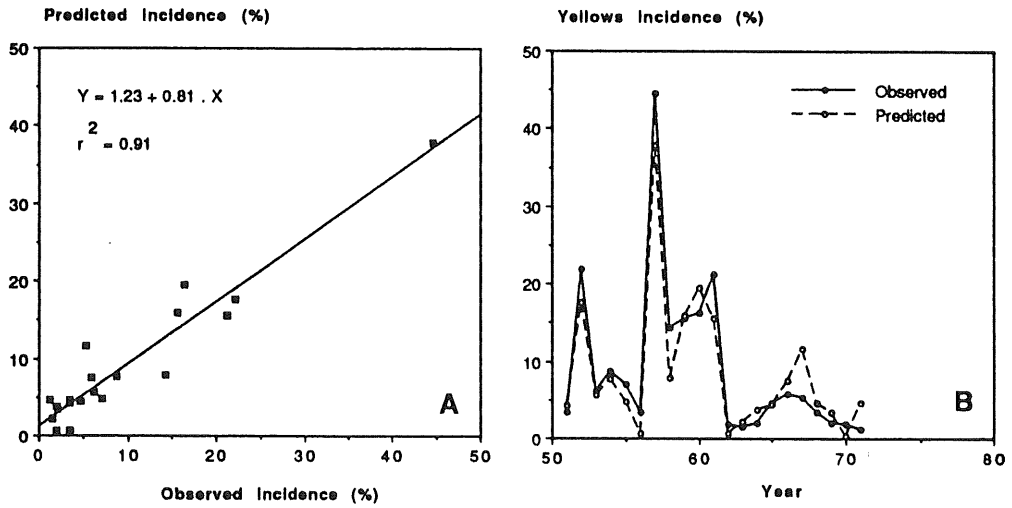

Fig. 1: Predicted versus observed virus yellows incidence in England 1951 - 1971 (A) and virus yellows incidence versus time (B) according to data (WATSON et al., 1975: Table 2) and regression model of WATSON et al. (1975).

ANALYTICAL MODELS

Analytical models characterize the structure of a system in one or a few equations. Their purpose is to study the relation between system structure (characterized by the equations) and system behaviour in a general sense. Analytical models are widely applied in theoretical ecology (e.g. MAY, 1976; EDELSTEIN-KESHET, 1988). Most analytical models are dynamic and descriptive, but as more biological realism is introduced they become gradually explanatory. A typical example of a descriptive analytical model is provided by the logistic growth rate equation in which the absolute rate of increase of a population equals:

$$\frac{dN}{dt} = \underbrace{r \cdot \left(1 - \frac{N}{K}\right)}_{\text{relative rate of increase}} \cdot N \qquad (3)$$

where
r is the relative growth rate at very low population densities,
N is the population density, and
K is the maximum population density.

The relative growth rate of the population decreases linearly with the number of organisms, N. The equation yields the symmetric S-curve of Fig. 2. Logistic equations have been used extensively in plant disease epidemiology for *descriptive* purposes (ZADOKS and SCHEIN, 1979; THRESH, 1983).

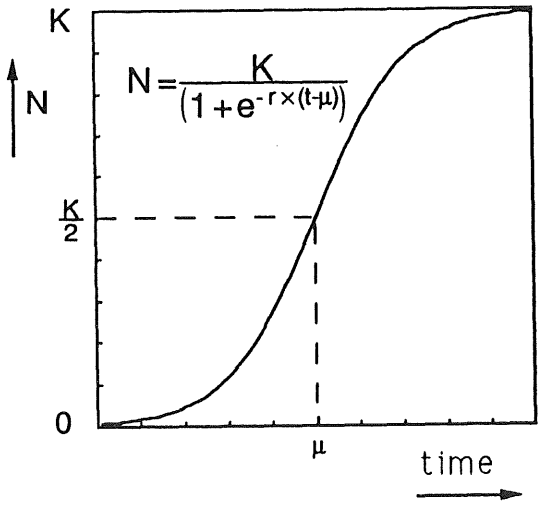

Fig. 2: Cumulative logistic growth curve. For the description of the increase in incidence of a virus disease in a crop, K equals 1 (100% infection). The 50%-point, μ, depends on the initial incidence and can be solved from $N_0 = \frac{K}{1 + e^{r \cdot \mu}}$

Only occasionally are analytical models applied for *explanation* of events in the field. MADDEN et al. (1987) give an example. There are at least two reasons for this. (1) Because of the simplifying assumptions, analytical models do not represent biological systems realistically. (2) The parameters cannot be measured at the level of the individual organisms involved. E.g. the carrying capacity, *K*, in the logistic equation depends on environmental factors and is often difficult to establish.

AN ANALYTICAL MODEL OF THE RANDOM SPREAD OF BEET YELLOWING VIRUSES

WATSON and HEALY (1953) developed their model from the following set of assumptions about the epidemiology of virus yellows in the 1940's:

1) Virus yellows is spread by winged *M. persicae* only (BYV and BMYV were not yet distinguished in 1953).
2) Infected plants are distributed at random over the field (no patches).
3) Vector aphids originate from outside the field. They have to acquire virus from an infected plant within the field before they can inoculate[1].
4) Aphids move from plant to plant at random. Every plant has the same chance to be visited, irrespective of distance.
5) The probability of acquisition of virus on an infected plant is 1. Aphids retain the virus lifelong.
6) Aphid flight and virus transmission occur in waves which are interspersed at three week intervals. At each wave, all aphids in the field make the same number of moves in the field, say *t*.
7) Plants infected in preceding waves serve as sources of virus. Thus the latency period (time between infection of a plant and possibility of acquisition of virus from infected leaves) is effectively taken to be 3 weeks.
8) Plants show symptoms at the next wave, i.e. the incubation period is effectively 3 weeks.
9) Viruliferous aphids deliver virus in a proportion, *p*, of their visits to plants. *p* is called the inoculation chance.
10) The number of immigrant aphid vectors per plant, say *N*, is estimated as 0.1 x trap-count, where trap-count is the number of winged *M. persicae* captured on sticky yellow traps exposed for 3 weeks in the field, as described by WATSON et al. (1951).

[1] Distinction is made between *inoculation* and *infection*, defining these terms as follows: *Inoculation* is delivery of virus to a plant and results in *infection* of the leaf on which the inoculation is made. *Infection* is inoculation of a healthy plant. According to these definitions *inoculation* causes *infection* of healthy plants but has no consequences for plants which have already contracted virus.

It can be shown mathematically (WATSON and HEALY, 1953) that the listed hypotheses result in a single formula relating the incidence of yellowed[2] plants after three weeks to the present incidence of yellowed plants. For simplicity, the formula is split in two parts. The first equation (Eq. 4) describes the expected number of inoculations per aphid (I), given the inoculation chance (p), the current incidence of infection (k_0) and the number of plants visited per aphid (t). The second equation (Eq. 5) describes the predicted incidence of infection *after* the infection wave (k_1), on the basis of the present incidence of disease (k_0), the average number of aphids per plant (N), and the expected number of inoculations per aphid (I), as calculated in the first equation.

$$I = \frac{p}{k_0} \cdot \left(\left(1 - k_0 \right)^t + k_0 \cdot t \quad - 1 \right) \tag{4}$$

$$k_1 = k_0 + \left(1 - k_0 \right) \cdot \left(1 - e^{-N \cdot I} \right) \tag{5}$$

where

I = expected number of inoculations per aphid,
p = probability of inoculation,
t = the number of plants visited by an aphid
k_0 = present proportion of infected plants,
k_1 = predicted future proportion of infected plants,
N = average number of immigrant winged *M. persicae* per plant,

The meaning of these formula's is most readily understood by the use of nomograms (Figs 3 and 4).

Fig. 3 shows that I, the expected number of inoculations per vector, increases curvilinearly with k_0, the incidence of infected plants. A maximum number of inoculations per vector, $p.(t-1)$ is reached when k_0 is 1. When k_0 is not very low, vectors need at an average few moves, namely $1/k_0$, to encounter an infected plant and acquire virus[3].

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[2] yellowed means showing symptoms of infection with virus yellows.

[3] The expression $1/k_0$ follows from the geometric probability distribution, which describes the number of Bernouilli experiments needed to obtain the first success. This distribution has an expectation value of $1/p$ where p is the chance on a success.

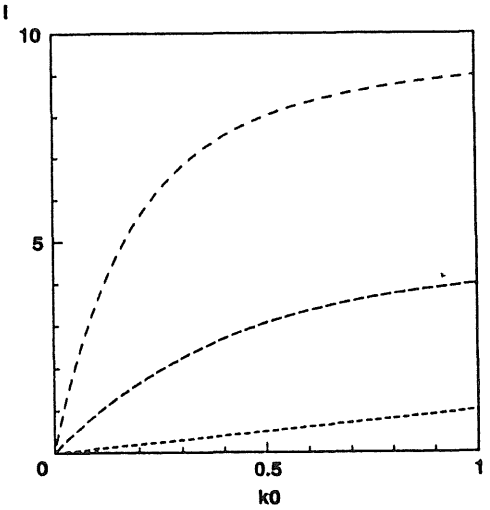

Fig. 3: Relation between I, the expected number of inoculations per vector, and k_0, the incidence of infected plants according to WATSON and HEALY's model, for 4 values of t, the number of plants visited per vector: 10 (— — —), 5 (———), 2 (----), or 1 (——— ; I = 0). The probability of inoculation, p, acts as a scaling factor along the Y-axis. For simplicity, a value of 1 is taken.

Therefore, when the number of moves, t, is large, the maximum number of inoculations per vector is already approached for small values of k_0. As a consequence, for large t, small incidences of infection can increase quickly, provided that enough vectors are present.

The model proposed by WATSON and HEALY predicts sharp increases in the incidence of infection when the expected number of viruliferous vector moves (= inoculations) per plant, $N.I$, is greater than 1. This is illustrated in Fig. 4. Because the disease is assumed to be spread at random over the field, and not patch-wise, multiple infections do not severely limit the effectiveness of the individual inoculations.

Predicted epidemics are given in Fig 5. This figure shows the calculated course of virus yellows epidemics according to WATSON and HEALY's model for an initial proportion of infection of 0.003 (i.e. ± 200 plants/ha) and 3 aphid densities, differing by a factor 3. The initial infections are made on 20 May (day 140), 10 June (day 161) and 1 July (day 182). The aphid population is assumed to start on 20 May, grow exponentially to a peak density on 22 July, subsequently collapse and reach a smaller autumn peak on 23 September (day 266). This time-course of aphid numbers is consistent with observations by WATSON et al. (1951) in the years 1943 to 1948. Figure 5 shows that the mechanisms of spread postulated by WATSON and HEALY result in explosive epidemics. A selection of field data for the 1940's was adequately fitted with WATSON and HEALY's model, taking a value of 5 for t (WATSON and HEALY, 1953). Further comparisons of the model with field data have not been made. Therefore, it is not clear whether the strongly simplified representation of the system in WATSON and HEALY's model results in inaccurate predictions.

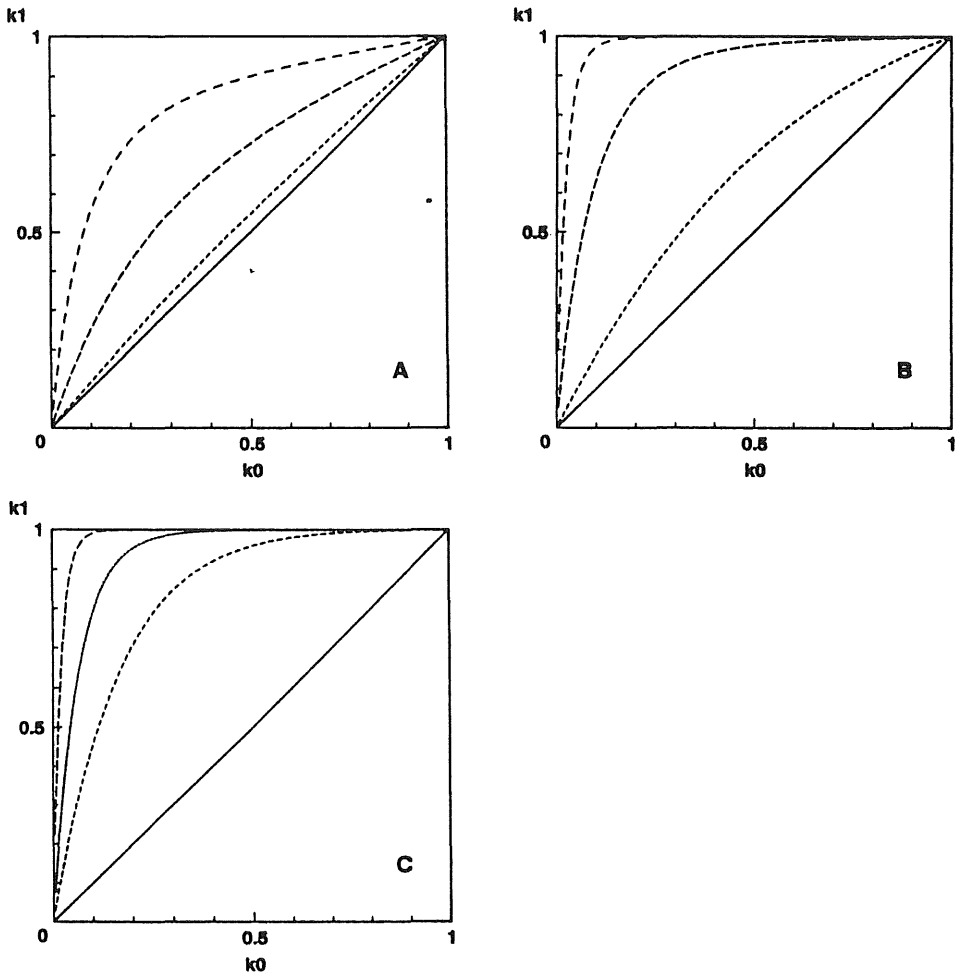

Fig. 4: Relation between predicted proportion of yellowed plants, k_1, and the present incidence, k_0, according to WATSON and HEALY's model, for 5 values of t, the number of plants visited per vector: 10 (— — —), 5 (———), 3 (———), 2 (----), or 1 (——— ; $k_1 = k_0$). The number of immigrant aphids per plant, N, is 0.2 in Fig. 3A, 1.0 in Fig. 3B and 5.0 in Fig. 3C, respectively. The inoculation chance, p, is 1.

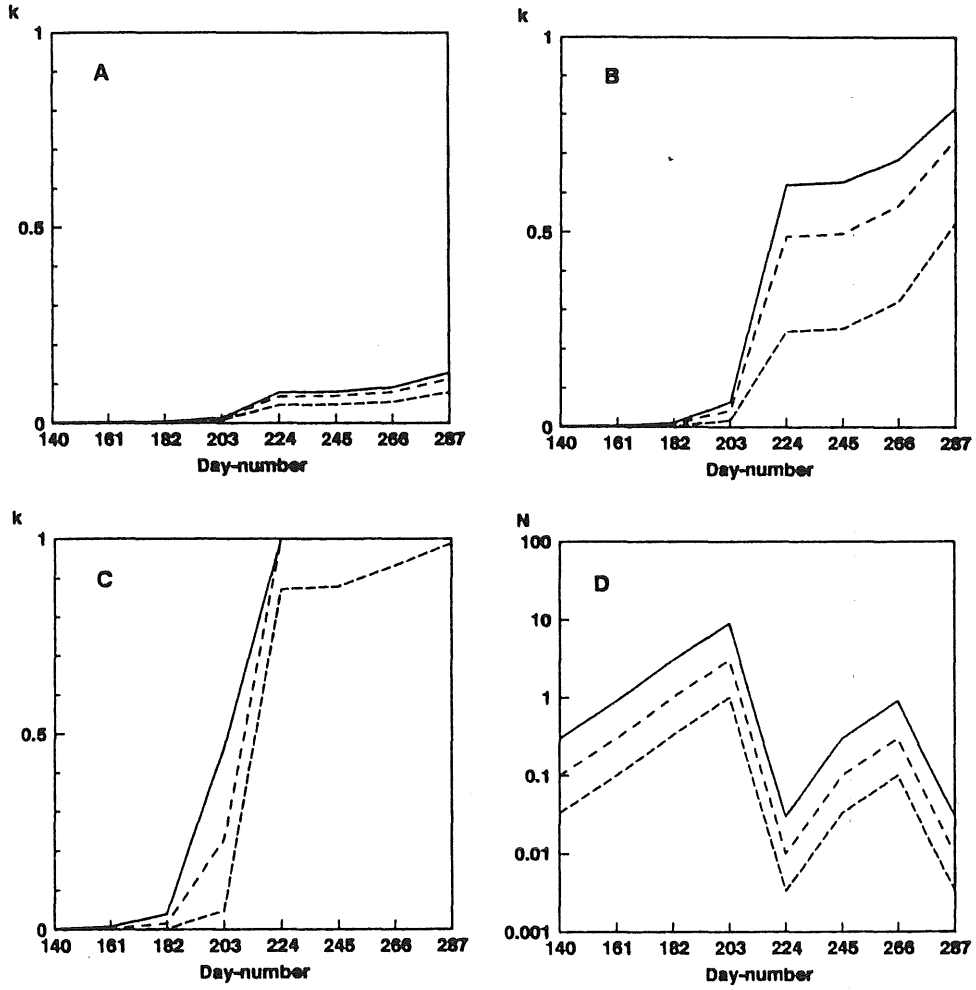

Fig. 5: Epidemics of virus yellows predicted by the model of WATSON and HEALY (1953) for an initial incidence of infection of 0.003 (i.e. ± 200 plants/ha) and maximum aphid densities of (A) 1, (B) 3 and (C) 9 *Myzus persicae* per plant. The postulated seasonal courses of aphid density for the 3 peak densities 9 (———), 3 (— — —) and 1 (————) are shown in Fig. 4D. In Figs A, B and C, the initial infections are made on day 140 (20 May; ———), 161 (10 June; — — —) or 182 (1 July; ————). The inoculation chance, *p*, is 1.

A SIMULATION MODEL OF THE DEVELOPMENT OF PATCHES OF BEET YELLOWS VIRUS

A Poisson distribution of infected plants and aphids over the field, as assumed by WATSON and HEALY (1953), has only in few cases been observed in the field. Virus yellows occurs more often in patches varying in size from 1 to 30 m diameter, depending on the date of primary infection (VAN DER WERF, 1988). The different patterns of spread, *patchwise* and *scattered,* may be related to distinct phases in beet virus yellows epidemics (WATSON et al., 1951; VAN DER WERF, 1988):

(1) *Introduction phase*. In May or early June, vectors and viruses enter the crop and start to multiply.
(2) *Establishment phase*. After adjacent plants have made leaf contact (early or mid-June) dispersal of wandering aphids is facilitated, resulting in incipient patchwise within-field dispersal of vectors and virus.
(3) *Dissemination phase*. In the course of July, the vector population reaches a maximum after which the numbers decrease and the individuals disperse due to a combination of factors: decreasing nutritional quality of the ageing plants, increased proportion of winged offspring, and increased mortality and disturbance due to the aggregation and increase in numbers of predators, pathogens and parasites. The relatively short-range dispersal by wingless aphids probably gives rise to patches whereas the long(er)-range dispersal by alatae results in the scattered spreading pattern.

As WATSON and HEALY's model is valid only in the rare situation of random spread, a model must be developed that takes account of the patchy nature of the dissemination of yellowing viruses under most circumstances. In the following, an explanatory dynamic simulation model for the development of beet yellows virus patches in sugarbeet is presented. The model gives a much more detailed representation of the system than WATSON and HEALY were able to do in 1953. Aim of the model is to increase insight in the development of patches by comparing simulation results (which reflect our conception of the system) and experimental data and by performing a sensitivity and uncertainty analysis of the model. Full details of the model are given by RIESEBOS (1987).

Structure of the model

The simulation model describes the events occurring in a 12 x 12 m^2 sugarbeet plot with 7.8 plants m^{-2} and a time-varying number of aphids. The model is object-oriented. Two types of objects are distinguished: plants and aphids. Individual plants are defined by 7 attributes:
1) infection class (% of leaf area infected),
2) date of infection,
3) number of aphids on the plant,
4) number of healthy leaves,

5) presence or absence of symptoms,
6) x-coordinate (row-number),
7) y-coordinate (position in the row).
Some of these attributes are interdependent. The attributes 1, 4 and 5 can be calculated from the date of infection, attribute 2. Attribute 3, the number of aphids on a plant, follows from the position of the individual aphids.

Individual aphids are defined by:
1) age (h),
2) time elapsed since latest virus acquisition (h),
3) time since latest walk,
4) x-coordinate,
5) y-coordinate,
6) morph; wingless or winged.

All plants have the same number of leaves. The area covered by a plant is represented by a rectangle of 50 x 25.6 cm^2, being the row- and plant distance, respectively, for a plant density of 7.8 plants m^{-2}. Aphids within the rectangle are 'on' the plant.

The model is formulated such that its predictions can be compared to results from a field experiment in which the effects of sowing date and number of introduced *M. persicae* on the spread of BYV were studied (VAN DER WERF, 1988). In this experiment, three plants in 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).

Hourly temperature is interpolated along a sine through measured daily minimum and maximum temperatures. Plant leaf number, a measure of physiological age, is a two-phase linear function of accumulated temperature (MILFORD et al., 1985a,b; VAN DER WERF, 1988).

BYV is a semi-persistently transmitted virus. In accordance with this, aphids in the model lose the ability to transmit virus at moulting. An aphid moults every 36 hours. Essentially following SYLVESTER (1961), the retention period (RP) is taken as 12 h after virus acquisition. Based on data of SYLVESTER (1956a,b) and of 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 walking) on a given plant (rectangle) equals or exceeds the TFP or AFP without interruption, virus acquisition occurs when a non-viruliferous aphid feeds on an infectious leaf or transmission occurs when an infectious aphid feeds 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) were simulated (see sensitivity analysis).

Following infection, plants become infectious after a latency period (LP) which increases linearly with time, from 4 days in the seedling phase to 12 days in old plants having ca. 40 leaves (VAN DER WERF, 1988). Counting from the date of 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 et al., 1989a). 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[4] infected plant or on an infectious plant from leaves that appeared before the infection date (VAN DER WERF et al., 1989b; ROSEBOOM and PETERS, 1984).

Aphid population dynamics is not simulated because insufficient data are available to do this with sufficient precision, due to the difficulty of quantifying predation and immigration. If a population dynamics module were included in the model, it would be impossible to determine whether wrong model predictions of virus spread resulted from errors in the virus dissemination part of the model or from incorrectly simulated population dynamics of the vector. Therefore, the model mimics the population development observed in the field experiment. Aphid numbers in the model are adjusted to field-counts when the difference exceeds 10%. Newly added aphids are 0 h old and non-viruliferous, mimicking births. They are 'borne' on plants which are already infested by aphids, proportional to the number of aphids on the plant. Predated 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, their number being proportional to the number of aphids). Winged aphids leave the field. No immigration occurs.

The form of the aphid age distribution was taken as a criterion to determine the rate of predation in the model. With high predation rates older life stages are less represented than at low rates of predation. A predation rate of 1% per hour gave the best agreement between the simulated and observed age distribution.

The dispersal and feeding behaviour of *M. persicae* plays a central role in the model. Aphid dispersal activity is expressed in 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 in accordance with the almost complete absence of dispersal in young crops, observed in the field (VAN DER WERF, 1988). This is probably due to the high nutritional quality of young plants as well as to lack of leaf contact between them, which hampers aphid movements between plants. When the number of leaves is greater than 12, leaves start to touch and *P* is calculated as:

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[4]The phytopathological definition of latent is used: infected but not yet infectious (ZADOKS and SCHEIN, 1979).

$$P = 0.01 \cdot \left(65 - \frac{N}{2.5}\right) \qquad (6)$$

where N is the number of leaves on the plant. P is a decreasing function of N to account for aphids spending more time to walking on different leaves belonging to the same plant when the plants have more leaves. This causes the residence time on older plants to be greater than on young plants with more than 12 leaves.

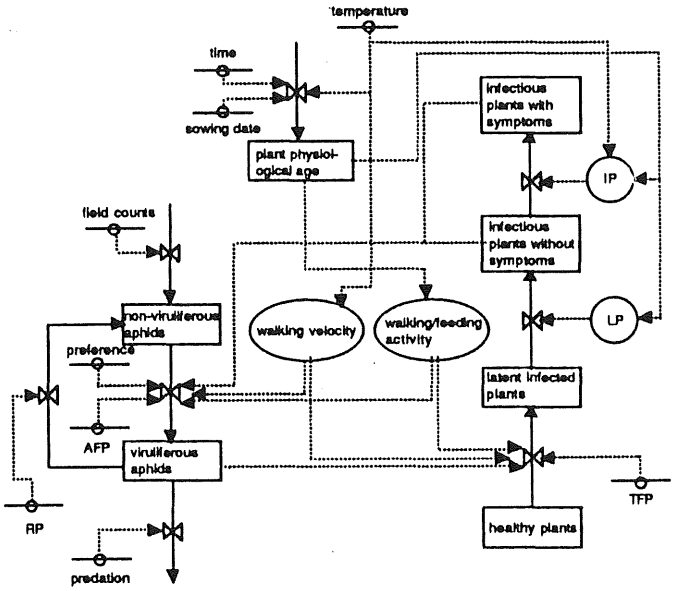


Fig. 6: Relational diagram of model of virus spread by *Myzus persicae* in sugarbeet, drawn according to conventions of FORRESTER (FORRESTER, 1961; DE WIT and GOUDRIAAN, 1978). Boxes (▭) represent states, solid arrows (⟶) represent fluxes between states, valves (⋈) in the solid arrows denote rates while hatched arrows (----▶) pointing to valves denote control of rates by state or other variables. Circles or ovals denote auxiliary variables. External variables, not influenced by the model, are denoted by —o— .

The linear displacement per hour, D, is a parabolic optimum function of aphid age and temperature:

$$D = D_{max} \cdot \left(1 - \left(\frac{A - 25}{25}\right)^2\right) \cdot \left(1 - \frac{9}{4} \cdot \left(\frac{T - 30}{30}\right)^2\right) \qquad (7)$$

where A is the age of the aphid in days ($0 < A < 50$) and T the temperature in ^{o}C ($10 < T < 50$). The value of D_{max}, 125 cm was estimated from 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 relational diagram of Fig. 6. Runs with the model were made with two purposes: (1) to compare simulation results with field data and thus study the validity of the described conception of the system, and (2) to determine which parameters have the largest effect on model behaviour and need to be determined accurately in (new) experimental studies to obtain accurate model predictions.

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

Comparison of model results and field data

Simulation results and field data are compared in Figs. 7A and B, respectively, which show the time-course of the number of yellowed plants per plot for each of the six treatments. Fair overall agreement exists, 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 our conception of the system, as laid down in the present model.

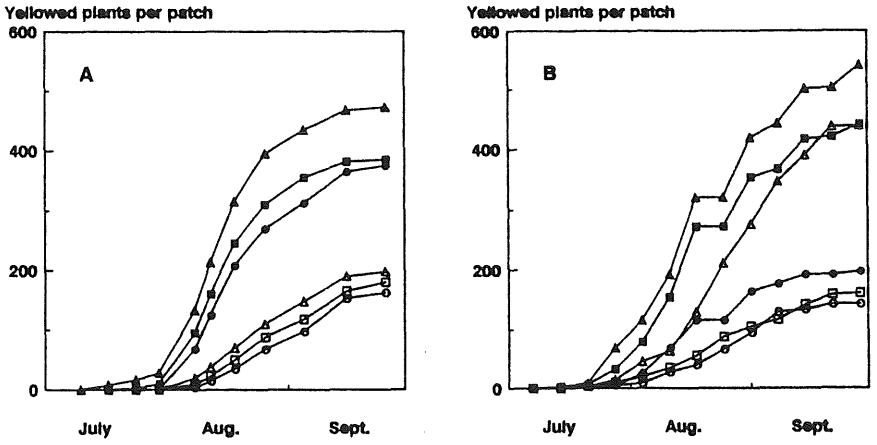


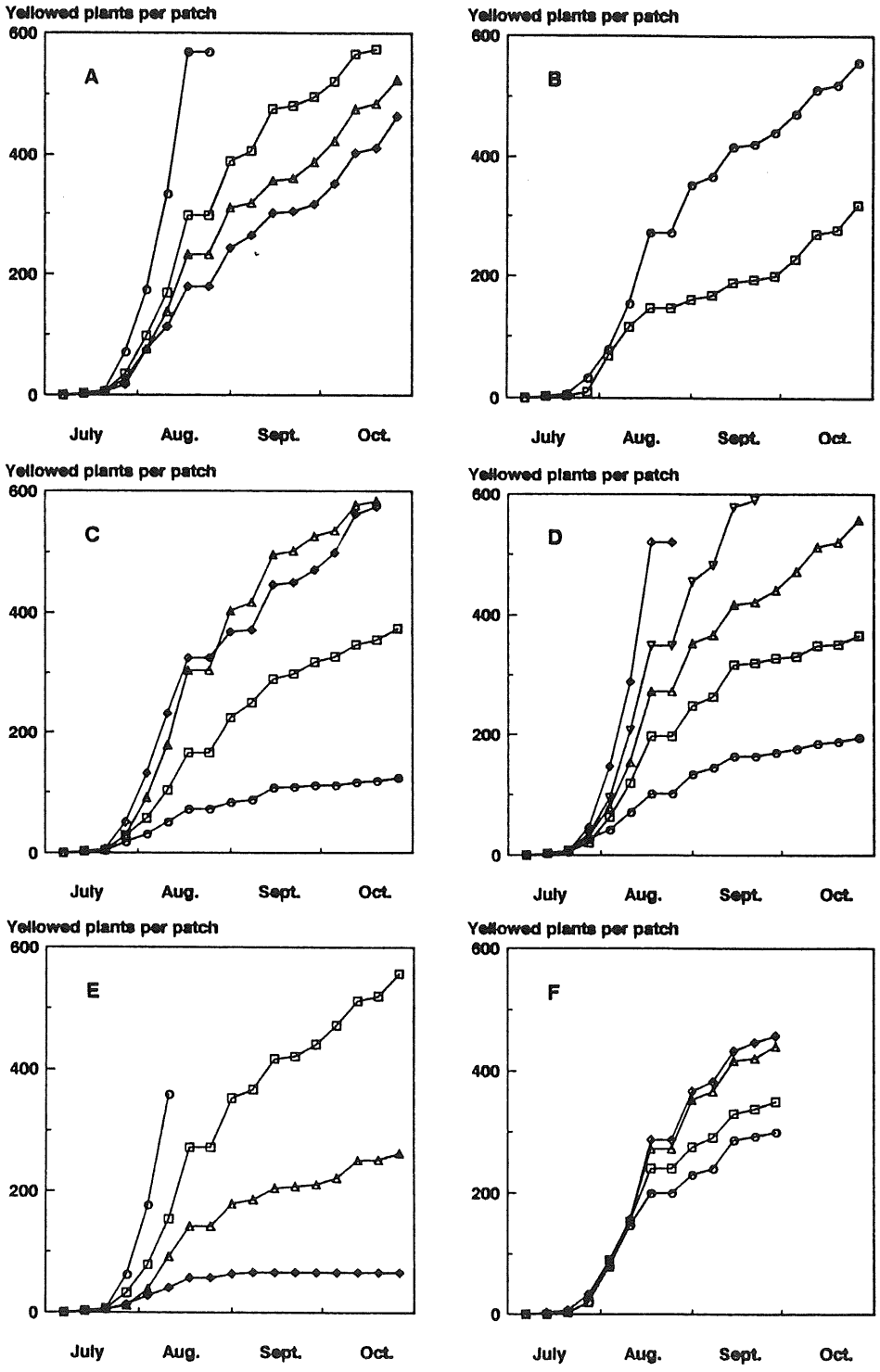
Fig. 7: Comparison of observed (A) and simulated (B) increase of number of BYV-infected plants with symptoms, in relation to crop sowing date (open symbols: 18 April; solid symbols: 20 May) and numbers of *Myzus persicae* introduced on 25 June: 2 (○, ●), 9 (□, ■) or 65 (△, ▲). Experimental data from VAN DER WERF (1988).

Sensitivity analysis

Sensitivity analysis provides a means to determine the influence of model structural components and parameter estimates on the results. Here a sensitivity analysis for six parameters is presented (Fig. 8A-F). The analysis has been made for late-sown sugarbeet infested with 9 *M. persicae*. For the LP reasonably precise values were available (VAN DER WERF, 1988). Thus, Fig. 8A illustrates the large impact of the LP on the rate of spread, which confirms results obtained with simpler epidemiological models (ZADOKS and SCHEIN, 1979).

For the other five parameters, only rough estimates could be derived from the literature. Therefore, the Figures 8B-F give an indication of the uncertainty in predicted epidemics resulting from the imprecision of the parameter estimates in the model. For instance, Fig. 8B shows that virus acquisition and transmission times have a considerable effect on spread. While several authors have 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. The topic warrants further study. 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. 8C,D). Predation appears to be a particularly important factor in the model (Fig. 8E), indicating that it deserves more attention in experimental research. Predation not only influences aphid numbers, but it also affects vector longevity and the proportion of aphids that live long enough to transmit virus successfully. The effect of leaf age preference is relatively small (Fig. 8F).

Fig. 8 (next page): Sensitivity analysis of simulation model of patchwise spread of beet yellows virus by *Myzus persicae* in sugarbeet; (A) latency period: 0 (O), 4 (□), 8 (△) or 12 days (◇); (B) virus transmission characteristics: AFP = 2 h and TFP = 1 h (O) or AFP = 6 h and TFP = 4 h (□); (C) proportion of aphids walking per hour (p in Eq. 6): 20 (O), 40 (□), 60 (△) or 80 (◇); (D) walking distance per hour (D_{max} in Eq. 7): 75 (O), 100 (□), 125 (△), 150 (▽) or 175 cm (◇); (E) predation mortality: 0.005 (O), 0.01 (□), 0.02 (△), or 0.04 h^{-1} (◇); and (F) leaf age preference as indicated by the factor f in the formula $p = \min (1, f \cdot Y)$, where p is the proportion of aphids feeding on young leaves and Y is the proportion of young leaf area on the plant. (O) f = 1, (□) f = 2, (△) f = 5, and (◇) p = 1.


Yellowed plants per patch
600
400
200
0
A
July
Aug.
Sept.
Oct.
Yellowed plants per patch
600
400
200
0
B
July
Aug.
Sept.
Oct.
Yellowed plants per patch
600
400
200
0
C
July
Aug.
Sept.
Oct.
Yellowed plants per patch
600
400
200
0
D
July
Aug.
Sept.
Oct.
Yellowed plants per patch
600
400
200
0
E
July
Aug.
Sept.
Oct.
Yellowed plants per patch
600
400
200
0
F
July
Aug.
Sept.
Oct.

CONCLUDING REMARKS

The three models discussed in this paper have different structure, different goals and different merits. The regression models of WATSON et al. (1975) and HARRINGTON et al. (1989) are purely descriptive, relating final virus incidence to weather conditions. Their predictions are very useful in the practice of warning systems. The generality is limited, however. WATSON et al.'s model was only valid for British conditions, and even there it failed in some years. The quality of a regression model depends i.a. on the selection of the right independent variables. This selection can be made by trial and error, optimizing the fraction of variance 'explained' by the model (WATSON et al., 1975; HARRINGTON et al., 1989). Explanatory models in conjunction with experiments on process and system level can help to gain insight in the system such that key factors determining system behaviour can be identified. These key factors may then be used in a descriptive model.

WATSON and HEALY developed their analytical model for random spread to obtain support for their hypothesis that virus yellows was mostly spread by flying vectors. Model results do not give firm support to this idea for 3 reasons: (1) the model is too simplistic as it does not account for patchwise spread, (2) insufficient comparisons between model calculations and field observations are given, and (3) similar results could have been obtained by substituting wingless aphid counts in the equations, as numbers of winged and wingless aphids were closely correlated. Nevertheless, the model gives an interesting concise representation of a virus-vector-host plant relationship that may be useful in other systems, e.g. non-persistently transmitted viruses, where the assumption of randomness seems more reasonable.

The patch model of RIESEBOS (1987) is a very useful instrument in studies of virus epidemiology. It is more comprehensive in its description of the system than any other published model of virus spread (MCLEAN et al., 1986). Despite this, some elements in the model are crude simplifications of reality, e.g. virus acquisition and transmission, and the walking and feeding behaviour of the vector. Furthermore the model should be compared with other field data. More important than the shortcomings of the model and its validation are the shortcomings in our knowledge of the system. This is demonstrated by the sensitivity analysis. Variation of the parameters related to walking tendency, walking distance and predation rate within the biologically plausible range results in a vast variation in predicted system dynamics. This variation pinpoints our ignorance of these processes. Quantitative knowledge of these processes is needed to understand virus yellows epidemiology better, such that warning thresholds and spraying advice can be improved.

Optimization and management models (RABBINGE and ROSSING, 1987; ROSSING, 1989; DRENTH et al., 1989) have not been discussed because they have not been used yet for evaluating and improving decision support systems for virus yellows control. When more knowledge of virus yellows epidemiology

becomes available, application of these methods is worthwhile.

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