A Stochastic Simulation Model of Epidemics of Arthropod-Vectored Plant Viruses

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ABSTRACT

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A generalized theoretical model was developed of the spread of a virus from a single source plant in a small field. Input parameters include probabilities of virus acquisition and inoculation, lengths of latent periods in the plant and vector, length of the vector infectious period, number of vectors per plant, and the amount of vector movement. Decisions about acquisition, inoculation, and vector movement are made on the basis of the values of pseudorandomly selected numbers. Output includes numbers of viruliferous vectors and positions of infected plants at each iteration. Simulation runs were performed for viruses with four generalized types of vector transmission: nonpersistent, semipersistent, circulative, and propagative. Model predictions were generally consistent with expected natural spread. Results of simulation runs illustrated the great effects that the amount of vector movement can have on disease dynamics and spatial distribution, particularly for diseases transmitted in a nonpersistent manner. The model will be difficult to fully validate; however, it provides a logically rigorous way of integrating knowledge about the many processes that affect virus disease epidemics and may be useful in the development of methods of analysis and in the development of less complex models.

Most plant viruses are vectored by arthropods, particularly aphids and leafhoppers. The dynamics of a particular virus disease epidemic depend on a number of factors, such as the number of vectors and their activity, sources of virus and vectors, climatic conditions, and a complex series of virus-plant-vector interactions (1,17,36,39,50,53,56). Although much is known about some of these interactions, relationships among the various factors affecting epidemics are far from clear. This is partly due to the difficulties inherent in monitoring viruses and vectors in the field and partly due to a lack of clear, testable hypotheses about the expected effects of transmission characteristics on epidemics.

Numerous techniques have been used in the management of plant virus diseases (16,23,40,52,57); however, many of them have inconsistent efficacy and/or economic justification. Much current work is being directed toward increasing the genetic resistance of plants to virus diseases. At present, however, predictions of the effects of altering cultural procedures or resistance characteristics on field epidemics are more rightly considered to be guesses than to be logical conclusions.

Methods for the interpretation of spatial data have received a great deal of attention in recent years (4,19,31,45,47,48,54). This general interest has been reflected in an interest in spatial aspects of plant diseases (3,8,10,18,19,24,25,41,55). Early analyses of spatial data from plant virus epidemics concentrated on the interpretation of disease gradients (12,13,51). More recent work has applied a variety of methods to plant virus disease data sets, including runs analysis, autocorrelation, and quadrat-based analyses (11,26–30). The unavailability of suitable data sets has been a major obstacle to progress in this area. Repeatedly assessing the infection status of all plants in even a small plot is extremely labor intensive. Furthermore, temporal variations in weather, vector populations, and plant susceptibility add to the difficulty of discerning relationships between epidemic behavior and fundamental biological processes.

Computer simulation provides a logically rigorous way to integrate knowledge about the many processes that contribute to virus disease epidemics. A number of simulation models of plant virus diseases have been developed (1,7,9,14,21,32,35,42). However, most of these models have been designed to simulate specific diseases, particularly those caused by nonpersistently transmitted viruses, which have no latent period in the vector and a short retention period. Few models of plant virus diseases have incorporated a spatial dimension. Some models of the spatial dynamics of nonviral plant diseases have been developed (20,33,34,44,46), but these do not take into account the many unique properties of virus diseases. Recently, Monestiez et al (35) reported on a model that stochastically simulates a nonpersistent virus that is being continually introduced into a field.

The generalized simulation model of the spread of plant virus diseases that is described herein was developed as an illustrative tool for a review article (2). A preliminary report has been published (6). In this paper, we describe the structure of the model and the assumptions on which it is based, variability of simulation results, limitations of the model, methods for interpretation of data obtained from the model, and some of the effects of input parameter values on simulated epidemics.

MATERIALS AND METHODS

Assumptions and parameters. The model simulates the spread of disease from a single infected plant in a field containing 425 plants (17 rows × 25 columns). The field is assumed to be isolated from other sources of virus; possible movement of the virus into or out of the field is ignored. The number of vectors is constant, and no provision is made for vector reproduction. Input parameters specify probabilities of acquisition and inoculation, durations of plant and vector latent periods, duration of the vector infectious period, the number of vectors per plant, and the amount of vector movement (Table 1). All periods begin and end in a discrete manner (e.g., a vector becomes fully able to transmit a virus once the vector latent period has passed, rather than gradually increasing in ability to transmit). Once they become infectious, plants remain so for the rest of the epidemic; there is no allowance for remission or effects of plant age. All decisions based on probabilities are made by comparison of a specified probability with

TABLE 1. Input parameters and state variables used in a stochastic simulation model of plant virus diseases

Parameter	Standard[a]	Definition
Input parameters		
Acquisition probability	0.5	Probability that a vector will acquire virus from an infectious plant that it resides on during an iteration.
Inoculation probability	0.5	Probability that an infectious vector will inoculate a noninfectious plant that it resides on during an iteration.
Plant latent period	8	Length (in iterations) of the period between inoculation of a plant and its becoming able to serve as a virus source.
Vector latent period	v	Length (in iterations) of the period between acquisition of virus by a vector and its becoming able to transmit virus.
Vector infectious period	v	Length (in iterations) of the period during which a vector is able to transmit virus.
Number of plants	425	Total number of plants in the simulated rectangular field.
Vectors per plant	1	Number of vectors per plant.
Movement probability	0.8	Probability that a vector will move from the plant it currently resides on during a movement iteration. Probability of movement to each of the four adjacent plants is (1−MoveProb)/4.
Moves per iteration	1	Number of vector movement iterations during one overall iteration. MoveProb is used to make a decision on movement for each movement iteration.
State variables		
Latent plants	...	Number of latently infected plants.
Infectious plants	...	Number of plants able to serve as a source of virus.
Infected plants	...	Total number of latently infected and infectious plants.
Latent vectors	...	Number of vectors that have acquired virus but have not yet become infectious.
Infectious vectors	...	Number of vectors able to transmit virus.
Viruliferous vectors	...	Total number of latent and infectious vectors.

[a] Standard values were used in simulation runs unless otherwise noted. v = Parameters were varied with transmission type.

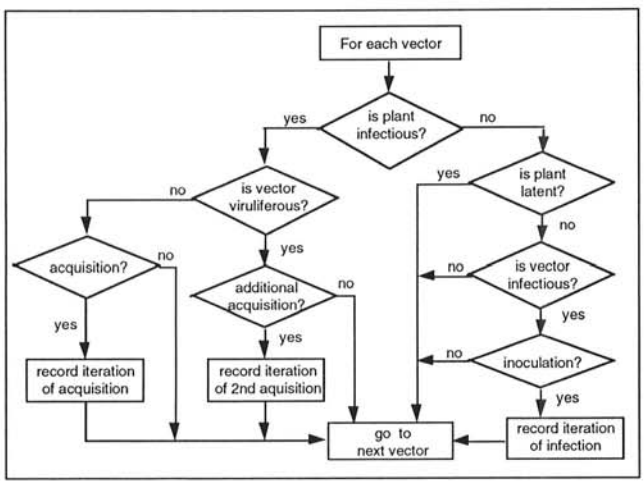

Fig. 1. Flow chart of decisions made for each vector during procedure VectorActions.

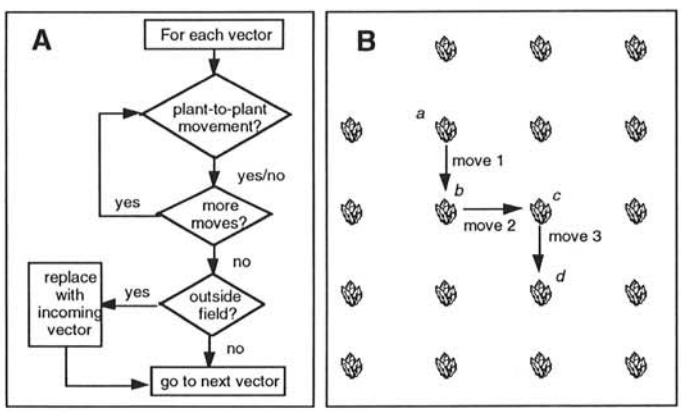

Fig. 2. Illustrations of procedure VectorMovements. **A,** Flow chart of decisions made for each vector; **B,** representative vector movements from plant a to plant d during one iteration for a model specifying three movements per iteration. In each of the three moves, the vector could have remained on the plant it was on or moved to any one of the four adjacent plants, depending on the value of a pseudorandom variable.

a pseudorandom value obtained from a mixed congruential generator. For example, if the specified acquisition probability is 0.75, then a pseudorandom value of ≤ 0.75 will result in a decision for acquisition. There are two steps in each calculation made by the generator: 1) a new seed value is calculated as (25,173 * OldSeed + 13,849) mod 65,536; and 2) the new seed is divided by 65,536 to give a value between zero and one. During a simulation run, this calculation is performed each time that a decision requires a pseudorandom value. Multiple runs with the same values of input parameters and a specified group of starting seed values are performed to obtain pseudoblocking (22).

Model structure. Epidemic behavior is simulated by two procedures: VectorActions and VectorMovements. In each iteration of a simulation run, procedure VectorActions is implemented for each vector, and then procedure VectorMovements is implemented for each vector. Decisions are made on whether each vector will acquire or inoculate virus in procedure VectorActions (Fig. 1). For each vector, a record is maintained of the position of the plant on which it currently resides, whether or not the vector is viruliferous, and the iteration numbers on which it first and last acquired virus. To simulate possible multiple acquisitions by individual vectors, the start of an infectious period (i.e., the end of a vector latent period) is determined by the iteration number of the first acquisition during the current period that a vector is viruliferous, whereas the end of an infectious period is determined by the iteration of the last acquisition. For each plant that is infected, a record is maintained of the iteration number on which it was inoculated. In procedure VectorMovements, decisions are made on the movement of each vector to other plants (Fig. 2). In the simulations discussed here, vectors move in a random walk manner. Whether a vector will move during an iteration is determined by the movement probability. If the value of a pseudorandom number is greater than the specified movement probability, then the vector does not move. If the value is less than the movement probability, then the vector moves one plant in a direction determined by the magnitude of the value, with the probabilities of movement in each of the four cardinal directions being equal. For example, if movement probability is 0.80, then values of 0.80–1.00 result in no movement, values of 0–0.20 result in movement to the left, and values of 0.20–0.40 result in movement down. For each vector during each iteration, this movement decision is made for a specified number of moves per iteration. A vector does not have any interactions with plants that it temporarily occupies during an iteration. If a vector moves to a position outside the simulated field, it is replaced by a vector

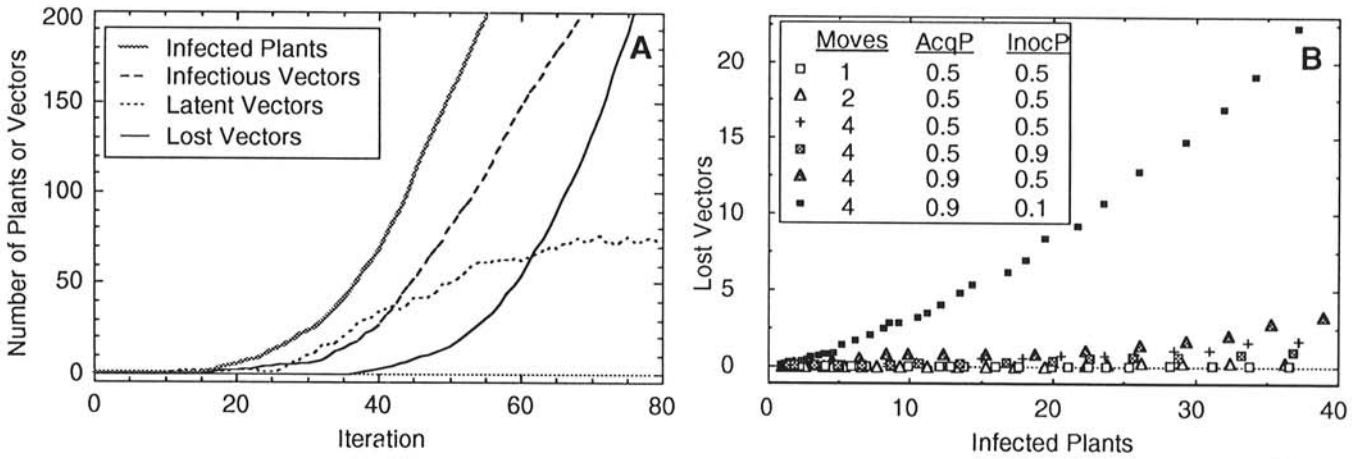

Fig. 3. A, Dynamics of infected plants and classes of vectors during simulation runs for a propagative virus with two movements per iteration, acquisition probability of 0.9, and other parameters at standard values. **B,** Relationships between number of "lost" viruliferous vectors and number of infected plants for simulation runs of a propagative virus with specified values of movements per iteration (Moves), acquisition probability (AcqP), and inoculation probability (InocP), and other parameters at standard values.

that enters from the opposite side of the field. The new vector is nonviruliferous even if the one moving outside is viruliferous. This procedure simulates a field that is part of a larger one but does not strictly simulate a real situation, since viruliferous vectors can be lost. In order to allow assessment of whether the loss of vectors is too great to produce a satisfactory simulation, a count is maintained of the number of "lost" vectors (viruliferous vectors that have moved outside the field).

The model has been implemented in Pascal on Apple Macintosh and IBM PC-compatible microcomputers.

RESULTS

Evaluation strategy. Model evaluation consisted of 1) assessment of the limits of the model and the amount of run-to-run variability, 2) evaluation of measures of model behavior, 3) assessment of the effects of input parameters, and 4) development of simplified representations of model behavior. Values were selected that approximate virus diseases characteristic for four general transmission types: nonpersistent (zero and one iterations for vector latent and infectious periods, respectively), semipersistent (zero and two), circulative (one and eight), and propagative (six and 32). Other parameters were varied one or two at a time for each of these four basic types. Standard values were used for the parameters that were not varied. Values of starting seeds for the random number generator were selected from a random number table and were used repeatedly to provide pseudoblocking. At least 10 replicate simulation runs were conducted for each combination of input parameters that was examined. The time step represented by each iteration was considered to be 1 day.

Limitations of the model. In order to determine the spatial and temporal limits of the model, simulation runs were performed with input parameter values that were expected to result in a large number of lost vectors. For most parameter combinations, lost vectors increased only after the number of infected plants had increased to more than 30 of the 425 plants in the field; however, when a propagative virus was combined with a relatively large amount of vector movement, a high acquisition probability, and a low inoculation probability, vectors were lost early in the simulation (Fig. 3). In order to avoid such invalid simulations, most analyses concentrated on phases of simulated epidemics during which there were fewer than 30 infected plants and excluded some propagative simulation runs.

Run variability. In order to evaluate variability among simulation runs, runs were performed for selected parameter sets with a total of 100 starting seed values selected from a random number table. In general, mean numbers of infected plants for runs with the 10 standard starting seeds were within the 95% confidence interval calculated for 100 runs, which indicated that general behavior was well-predicted by the standard runs. However, for a few parameter sets, means for 10 runs were slightly outside means for 100 runs. For most parameter sets, the mean iteration at which at least 10 plants were infected (Iter10) with 10 replicates was within four iterations of the mean for 100 replicates; the mean Iter10 for 20 replicates was always within two iterations of the mean for 100 replicates (Fig. 4). Distributions of Iter10 were asymmetrical for parameter sets with a low mean Iter10 value, and variances increased with mean Iter10 value (Fig. 5).

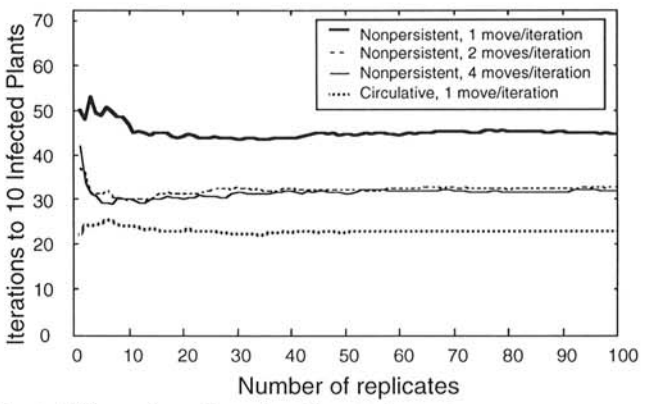

Fig. 4. Effects of number of replicated simulation runs on the mean iteration number at which at least 10 plants were infected for four sets of input parameter values. Unless noted, input parameters were at standard values.

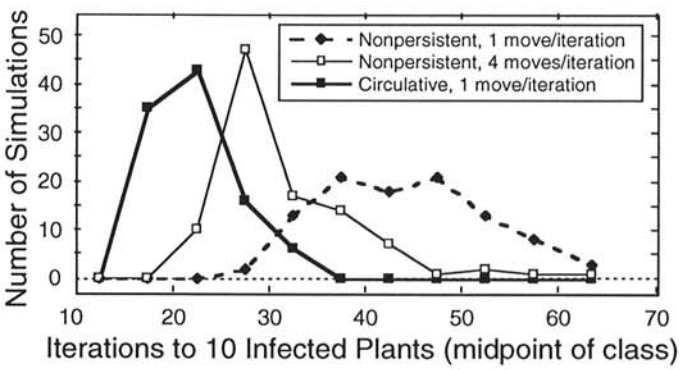

Fig. 5. Class distributions of the number of iterations to 10 infected plants for 100 simulation runs of each of three sets of input parameter values. Classes are five iterations wide. Unless noted, input parameters were at standard values.

Infection progress. In order to summarize infection dynamics, regression models of the Richards family were fitted to data for the number of infected plants. When the asymptote was fixed at 425 (the total number of plants), both the logistic and Gompertz models fitted infection progress well when equations were fitted iteratively with SAS PROC NLIN (43). When equations were fitted by linear regression on Logit- or Gompit-transformed data, the Gompertz model fitted data for most parameter sets better than did the logistic model (Fig. 6).

Effects of parameter values. The effects of varying the values of input parameters were examined for sets of runs that represented each of the four transmission types. Iter10 was significantly affected by both transmission type and variation in any of the other five input parameters (Fig. 7, Table 2). Significant interactions with transmission type occurred only for acquisition probability and inoculation probability and were due to a greater effect of varying these parameters for the nonpersistent and semipersistent transmission types. The number of infectious vectors at 10 infected plants (Vect10) was affected by acquisition probability, inoculation probability, and the number of vectors per plant. Transmission type had a marginal effect on Vect10, and plant latent period and number of vector movements per iteration had no significant effects. The number of horizontal runs of infected plants when 10 plants were infected (Run10) was affected primarily by transmission type and the number of vector movements per iteration, with marginal effects of inoculation probability and plant latent period. The effects of combinations of values of acquisition probability and inoculation probability varied greatly with transmission type (Fig. 8). For the nonpersistent and semipersistent transmission types, reduction of both acquisition probability and inoculation probability to 0.10 greatly increased Iter10; effects of this reduction were much less pronounced for the circulative and propagative transmission types. Although the effects of varying acquisition probability and inoculation probability were similar, reductions in acquisition probability generally resulted in a slightly greater increase in Iter10 than did similar reductions in inoculation probability.

Multiple regression. Multiple regression was used to develop models that predicted the behavior of simulated epidemics from values of input parameters. For each of 216 combinations of input parameters, means for 10 replicate simulation runs were calculated for Iter10, Vect10, and Run10. A "basic" model was

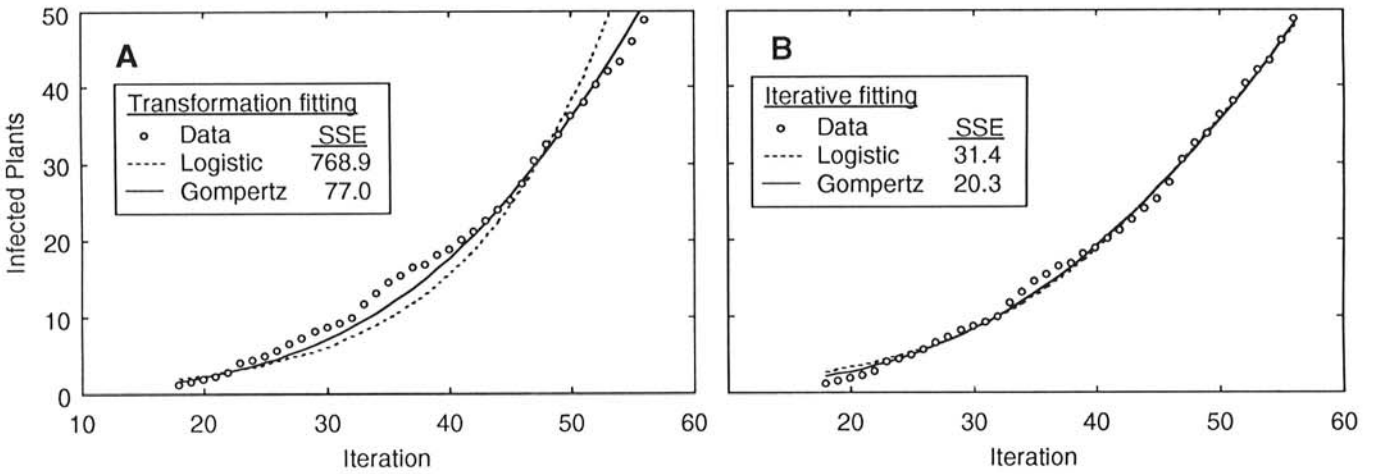

Fig. 6. Results of fitting logistic and Gompertz regression models to disease progress data by means of linear regression **A,** on transformed data or **B,** by means of a nonlinear, iterative method. Data are means from 10 replicated simulation runs for a circulative virus with standard parameter values except plant latent period = 16 iterations. SSE = the error sum of squares for comparison of nontransformed observed and predicted values.

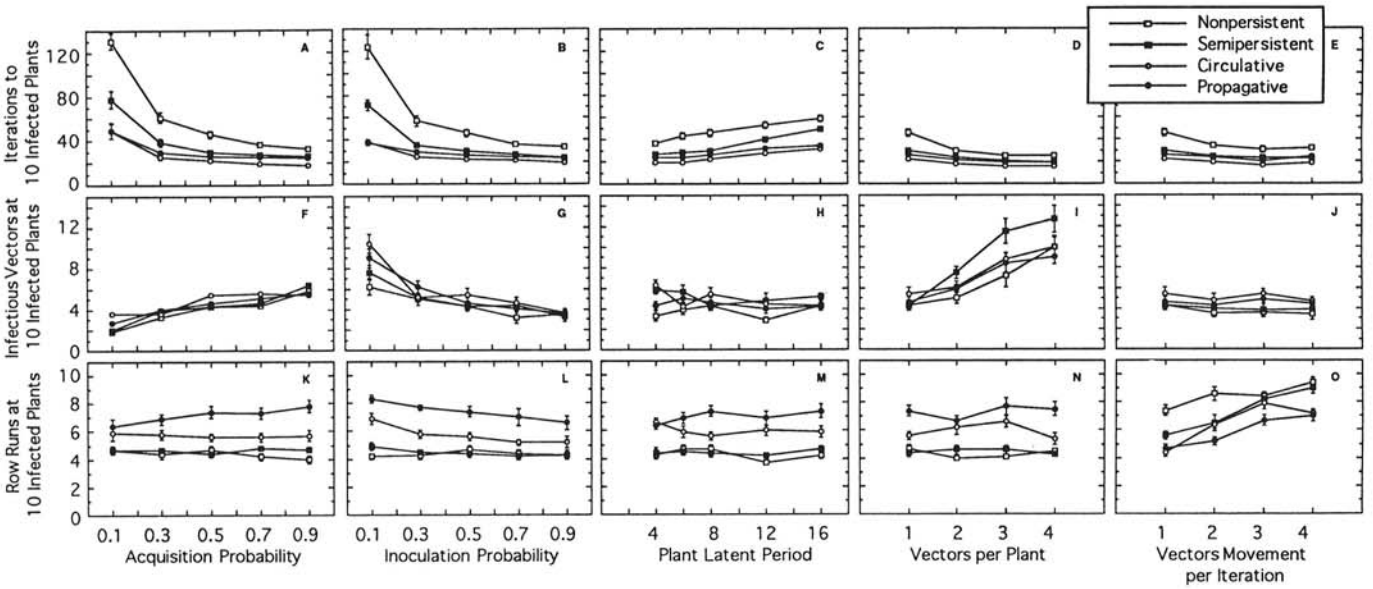

Fig. 7. A, F, and K, Effects of acquisition probability, **B, G, and L,** inoculation probability, **C, H, and M,** plant latent period, **D, I, and N,** number of vectors per plant, and **E, J, and O,** number of vector movements per iteration on the number of iterations to 10 infected plants **(A–E),** the number of infectious vectors at 10 infected plants **(F–J),** and the number of row runs of infected plants at 10 infected plants **(K–O)** in simulated virus epidemics. Bars represent standard errors for 10 replicate simulation runs. "Row runs of infected plants" is defined as the number of runs of adjacent infected plants in the simulated field when counts are made horizontally along the rows of plants.

derived that included each of the seven main input parameters or their inverses for each of these three dependent variables. Additionally, "best" models that included interaction terms were selected by use of stepwise multiple regression on the basis of coefficients of determination, R^2, and total squared error, Mallow's Cp (5). Preliminary analyses indicated that a double-log transformation of Iter10 $\{\log_{10}[\log_{10}(\text{Iter10})]\}$ and a single-log transformation of Vect10 $[\log_{10}(\text{Vect10})]$ resulted in consistently better fits. Log transformations had little effect on the fitting of models to Run10 data. For Iter10 data, all of the seven input parameters had a significant effect in the basic model (Table 3, Fig. 9). The best model contained nine independent variables that included various combinations of the seven input parameters but had only marginally better fit than did the basic model (Table 4). For Vect10 data, moves per iteration and plant latent period had no significant effect in the basic model. The best model contained seven independent variables, with vector latent period not a part of any term. For Run10 data, acquisition probability, vectors per plant, and plant latent period had no significant effect in either the basic or best models. Although it contained only four independent variables, the best model for Run10 had a slightly better fit than did the basic model.

Relationships between numbers of infectious vectors and plant infection. In order to evaluate how well increases in plant infection can be predicted from numbers of infectious vectors, the change in the number of infectious plants was calculated for the first 10 pairs of iterations past Iter10 in each of 10 replicate simulation runs. Linear correlation was used to compare this change in plant infection with the number of infectious vectors at previous iterations. In general, correlations were strongest when change in infection was compared with the number of vectors at a lag of eight iterations, equivalent to one plant latent period (Fig. 10). Correlation coefficients increased with inoculation probability. For the circulative and propagative transmission types at high inoculation probability, correlation coefficients were significant for a range of lags, probably due to the long retention periods of these types. Correlation coefficients for the nonpersistent and semipersistent transmission types were generally significant only

TABLE 2. Results of F tests from analyses of variance of the effects of parameter level and transmission type on number of iterations to 10 infected plants, and numbers of viruliferous vectors and number of runs of infected plants when 10 plants were infected[a]

Dependent variable Parameter[b]	F value for effect of parameter levels on dependent variable[c]					Transmission type[d]	Level × transmission type[e]
	Nonpersistent	Semipersistent	Circulative	Propagative	Overall		
Number of iterations to 10 infected plants							
Acquisition probability	60.97***[f]	44.75***	41.66***	18.53***	158.52***	144.17***	2.48**
Inoculation probability	96.32***	79.15***	37.18***	16.67***	207.55***	241.24***	7.25***
Plant latent period	10.25***	25.28***	21.98***	11.54***	65.42***	158.39***	1.31 ns
Vectors per plant	38.36***	21.27***	22.48***	12.84***	90.82***	129.58***	1.37 ns
Movements per iteration	10.69***	5.98**	7.71***	4.23**	25.58***	94.16***	1.01 ns
Number of infectious vectors at 10 infected plants							
Acquisition probability	10.58***	12.12***	2.69*	4.83**	26.67***	3.88*	1.18 ns
Inoculation probability	7.23***	6.89***	8.80***	8.61***	29.16***	4.69**	0.79 ns
Plant latent period	2.25 ns	1.6 ns	2.14 ns	0.58 ns	2.19 ns	8.35***	1.46 ns
Vectors per plant	12.74***	19.42***	8.89***	9.42***	47.36***	5.42**	1.00 ns
Movements per iteration	0.77 ns	0.77 ns	0.34 ns	0.14 ns	1.00 ns	5.36**	0.34 ns
Number of runs of infected plants at 10 infected plants							
Acquisition probability	1.59 ns	0.41 ns	0.06 ns	1.45 ns	0.04 ns	60.51***	1.16 ns
Inoculation probability	0.63 ns	1.21 ns	3.54**	2.42 ns	4.69**	81.73***	1.03 ns
Plant latent period	2.56*	0.64 ns	0.89 ns	0.74 ns	1.28 ns	65.78***	1.19 ns
Vectors per plant	1.74 ns	0.29 ns	2.06 ns	0.73 ns	0.58 ns	66.70***	1.41 ns
Movements per iteration	12.13***	22.24***	14.49***	2.70*	42.60***	31.70***	2.99**

[a] Each line represents results of an analysis of variance of data from 10 replicate simulation runs for each level-transmission type combination. Data for iterations to 10 infected plants were subjected to a double log $\{\log_{10}[\log_{10}(x)]\}$ transformation to reduce heterogeneity of variance.
[b] Levels of the parmeters were 0.1, 0.3, 0.5, 0.7, and 0.9 for inoculation probability and acquisition probability; 4, 6, 8, 12, and 16 iterations for plant latent period; 1, 2, 3, and 4 vectors per plant; and 1, 2, 3, and 4 movements per iteration.
[c] The effects of the levels of the parameter for each of the four transmission types and the effect of levels of the parameter over all four transmission types.
[d] The overall effect of transmission type.
[e] The effect of the interaction between transmission type and parameter level.
[f] ns = Not significant at $P > 0.05$; *, **, and *** = significant at $0.05 \geq P > 0.01$, $0.01 \geq P > 0.0001$, and $P \leq 0.0001$, respectively.

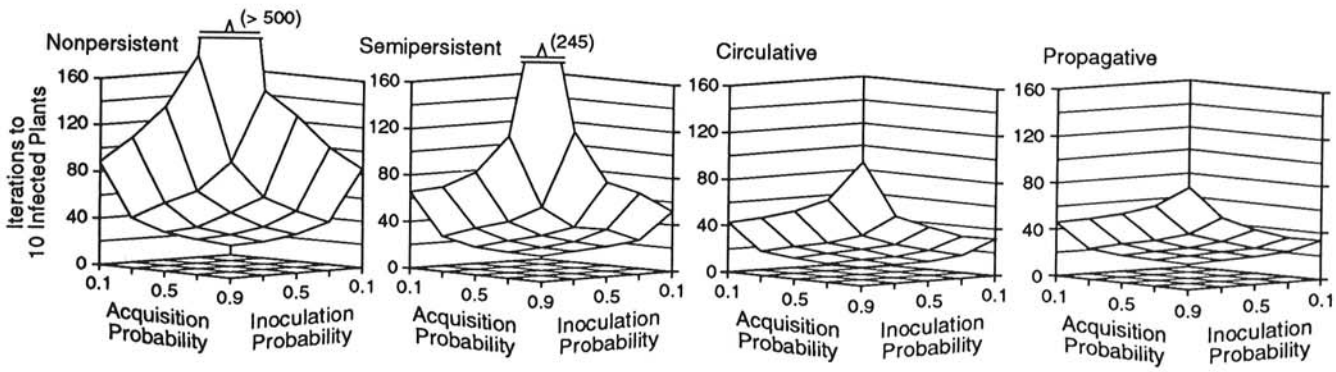

Fig. 8. Effects of combinations of values of acquisition probability and inoculation probability on the number of iterations to 10 infected plants. All other input parameters were at standard values.

for an 8-day lag.

Infection gradients. With most parameter sets, infected plants were clustered around the initially infected plant. Gradients were steepest with the nonpersistent and semipersistent transmission types and most shallow with the propagative transmission type (Fig. 11). Increasing acquisition probability steepened gradients, and increasing the number of vector movements per iteration made gradients more shallow (Fig. 12).

Runs of infected plants. Numbers of runs of adjacent plants were similar when the counts were made horizontally (along the rows of the simulated field) or vertically (down columns). The number of runs of infected plants was well below the level that would be predicted for a random distribution for the standard parameter sets of all four transmission types (Fig. 13). For similar numbers of infected plants, the number of runs was consistently greatest for the propagative transmission type and least for the nonpersistent and semipersistent transmission types (Fig. 14). Increasing acquisition probability resulted in small decreases in numbers of runs for both the nonpersistent and propagative transmission types (Fig. 15A and B). Increasing the number of vector movements per iteration had a much greater effect; if two or more movements were specified for the propagative transmission type, the number of runs approached that expected with a random distribution (Fig. 15C and D).

Time to infection. Plotting the average number of iterations until infection against distance from the initially infected plant gave near-linear relationships for standard parameter sets of all four transmission types (Fig. 16). Increasing acquisition probability tended to decrease the intercept of these straight lines for both the nonpersistent and propagative transmission types and decreased slopes for the nonpersistent transmission type (Fig. 17A and C). Increasing the number of vector movements per iteration had more of an effect on slope than on intercept; slopes approached zero for the propagative transmission type with three or four movements per iteration (Fig. 17B and D).

TABLE 3. "Basic" multiple regression models using parameter levels to predict number of iterations to 10 infected plants, and numbers of viruliferous vectors and runs of infected plants when 10 plants were infected[a]

Dependent variable Parameter	Coefficient ± SE	t for H_0: coefficient = 0[b]
Log_{10}[(Log_{10}(number of iterations to 10 infected plants)][c]		
Intercept	−0.0935 ± 0.0128	−7.31***
1/Inoculation probability	0.0105 ± 0.0005	20.28***
1/Acquisition probability	0.0126 ± 0.0004	28.25***
1/Vectors per plant	0.0718 ± 0.0090	7.94***
1/Moves per iteration	0.0479 ± 0.0044	10.80***
Plant latent period	0.0055 ± 0.0010	5.74***
Vector latent period	0.0043 ± 0.0007	6.15***
1/Vector infectious period	0.0964 ± 0.0046	20.99***
Log_{10}(number of infectious vectors at 10 infected plants)[d]		
Intercept	1.1709 ± 0.0536	21.83***
1/Inoculation probability	0.0322 ± 0.0022	14.48***
1/Acquisition probability	−0.0393 ± 0.0019	−20.61***
1/Vectors per plant	−0.4125 ± 0.0378	−10.90***
1/Moves per iteration	0.0319 ± 0.0186	1.71 ns
Plant latent period	−0.0040 ± 0.0041	−0.98 ns
Vector latent period	−0.0085 ± 0.0030	−2.86 ns
1/Vector infectious period	−0.1681 ± 0.0193	−8.69***
Number of runs of infected plants at 10 infected plants[e]		
Intercept	9.1704 ± 0.3922	23.38***
1/Inoculation probability	0.1013 ± 0.0163	6.23***
1/Acquisition probability	−0.0179 ± 0.0139	−1.28***
1/Vectors per plant	−0.3129 ± 0.2766	−1.13***
1/Moves per iteration	−3.5894 ± 0.1360	−26.40***
Plant latent period	0.0058 ± 0.0296	0.20 ns
Vector latent period	0.2239 ± 0.0216	10.34***
1/Vector infectious period	−1.3364 ± 0.1415	−9.45***

[a] Data were means from 216 simulated epidemics representing all transmission types. Parameters included in the models were selected from among simple parameters and their inverses. For iterations to 10 infected plants and number of infectious vectors, R^{*2} is the proportion of nontransformed variance explained by the model (model sum of squares divided by total sum of squares).

[b] ns = Not significant at $P > 0.05$; *, **, and *** = significant at $0.05 \geq P > 0.01$, $0.01 \geq P > 0.0001$, and $P \leq 0.0001$, respectively.

[c] $R^2 = 0.9050$; $R^{*2} = 0.8155$.

[d] $R^2 = 0.8189$; $R^{*2} = 0.8220$.

[e] $R^2 = 0.8690$.

DISCUSSION

In general, the relative effects of different parameter values in simulation runs were consistent with what would be expected from an informal consideration of the system. Epidemics progressed more rapidly when there were more vectors, when there was a greater amount of vector activity, when the plant latent period was shorter, or when probabilities of inoculation or acquisition were higher. Plant latent period and vector activity had little or no effect on the number of infectious vectors per infected plant. The amount of spatial aggregation, as measured by runs of infected plants, was strongly affected only by transmission type and the amount of vector activity. The similarity of these results with common sense evaluations indicates that the simulation model is a basically valid representation of actual epidemics. However, the more complex predictions of the model, such as those involving time to infection and specific combinations of input parameter values, are not intuitively obvious.

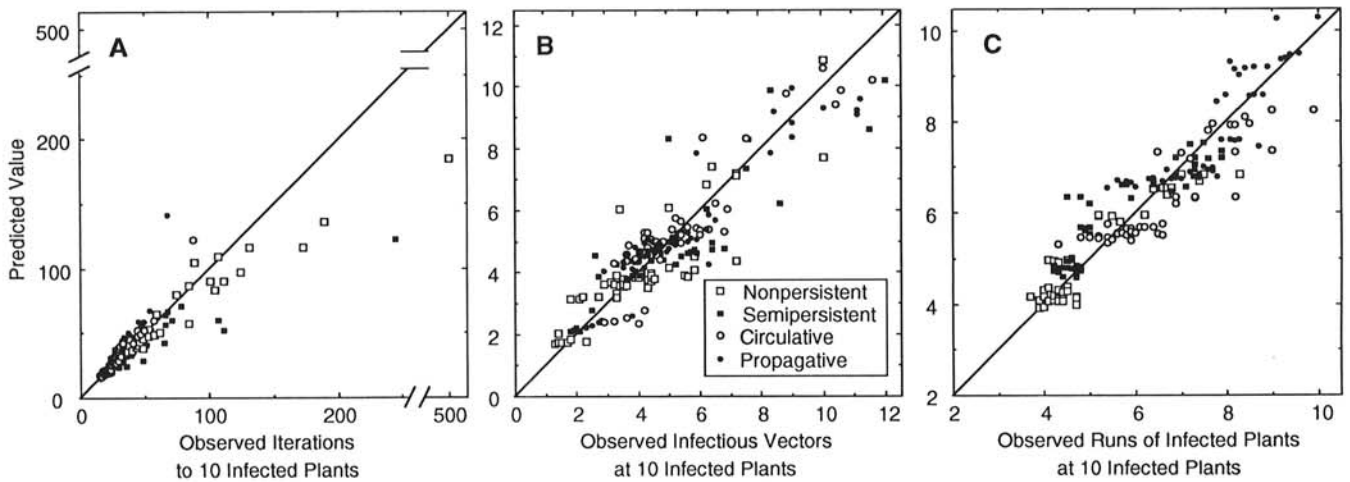

Fig. 9. Relationships between observed values of variables and values predicted by the "basic" multiple regression models for 216 simulated epidemics. **A,** Iterations to 10 infected plants; **B,** number of infectious vectors at 10 infected plants; **C,** number of horizontal runs of infected plants at 10 infected plants. Diagonal lines represent a 1:1 correspondence. Fitted models are presented in Table 3.

A simulation model can be considered to represent a body of scientific theory (37). The formalization of knowledge in simulation models may be necessary for significant progress to be made in our understanding of complex systems such as plant virus epidemics. However, as the amount of detail in a model increases, so does the difficulty of validation (15,49). For the model that we developed, it may not be possible to accurately estimate the values of the input parameters that are needed to perform runs simulating a particular field situation. Thus, it may not be possible to fully test the validity of the model by performing simulation runs with values of input parameters that have been measured in a field situation and then comparing field results with results of the simulation. It is difficult to measure acquisition and inoculation probabilities under controlled conditions; producing accurate estimates of their values in actual field situations may be impossible with current techniques. Similarly, it is very difficult to keep track of individual vectors in controlled-release field dispersal experiments; documenting the movements of all individuals within a naturally occurring vector population may be impossible. These problems bring into question the value of the model, since it cannot be validated by comparison with observations of actual epidemics. However, it may be possible to validate the model in a general manner, concentrating on the comparison of generalized model predictions with the behavior of epidemics among which only a few input parameter values are known to vary.

The slow progress of many simulated epidemics of nonpersistent viruses is an apparently counterintuitive result of the simulation runs, since many nonpersistent viruses can cause devastating epidemics (38,40). However, a closer examination of the results

TABLE 4. "Best" multiple regression models using parameter levels to predict number of iterations to 10 infected plants, and numbers of viruliferous vectors and runs of infected plants when 10 plants were infected[a]

Dependent variable parameter	Coefficient ± SE	t for H_0: coefficient = 0[b]
Log_{10}[(Log_{10}(number of iterations to 10 infected plants)][c]		
Intercept	0.1197 ± 0.0183	6.54***
1/(Vectors per plant × moves per iteration)	0.0511 ± 0.0041	12.55***
Plant latent period/acquisition probability	0.0011 ± 0.0001	11.03***
Plant latent period/inoculation probability	0.0009 ± 0.0001	8.12***
Inoculation probability × acquisition probability	−0.0710 ± 0.0119	−5.96***
Vector latent period	0.1087 ± 0.0278	3.91***
Vector infectious period	−0.0218 ± 0.0058	−3.77**
1/(Vectors per plant × vector infectious period)	0.0432 ± 0.0146	2.95**
1/(Inoculation probability × acquisition probability)	0.0004 ± 0.0002	2.73**
Plant latent period/vectors per plant	0.0013 ± 0.0008	1.65 ns
Log_{10}(number of infectious vectors at 10 infected plants)[d]		
Intercept	0.6410 ± 0.0353	18.16***
Vectors per plant × acquisition probability	0.2535 ± 0.0183	13.83***
Plant latent period/acquisition probability	−0.0027 ± 0.0003	−10.14***
1/(Vectors per plant × vector infectious period)	−0.1741 ± 0.0173	−10.07***
1/(Inoculation probability)	0.0199 ± 0.0033	6.07***
Inoculation probability	−0.1586 ± 0.0407	−3.90***
1/(Vectors per plant × moves per iteration)	0.0585 ± 0.0161	3.62**
Vector infectious period	−0.0017 ± 0.0005	−3.26**
Number of runs of infected plants at 10 infected plants[e]		
Intercept	7.8601 ± 0.1811	43.41***
1/Moves per iteration	−3.4273 ± 0.1270	−27.00***
Vector infectious period	0.5358 ± 0.0537	9.98***
Vector latent period	−2.3313 ± 0.2708	−8.61***
Inoculation probability	−1.3870 ± 0.1935	−7.17***

[a] Data were means from 216 simulated epidemics representing all transmission types. Parameters included in the models were selected from among simple parameters, their inverses, and combinations of parameters. "Best" models were selected on the bases of coefficient of determination (R^2) and total squared error (Cp). For iterations to 10 infected plants and number of infectious vectors, R^{*2} is the proportion of nontransformed variance explained by the model (model sum of squares divided by total sum of squares).

[b] ns = Not significant at $P > 0.05$; *, **, and *** = significant at $0.05 \geq P > 0.01$, $0.01 \geq P > 0.0001$, and $P \leq 0.0001$, respectively.

[c] $R^2 = 0.9202$; $R^{*2} = 0.8382$; Cp = 8.4.

[d] $R^2 = 0.8712$; $R^{*2} = 0.8831$; Cp = 3.7.

[e] $R^2 = 0.8776$; Cp = 4.5.

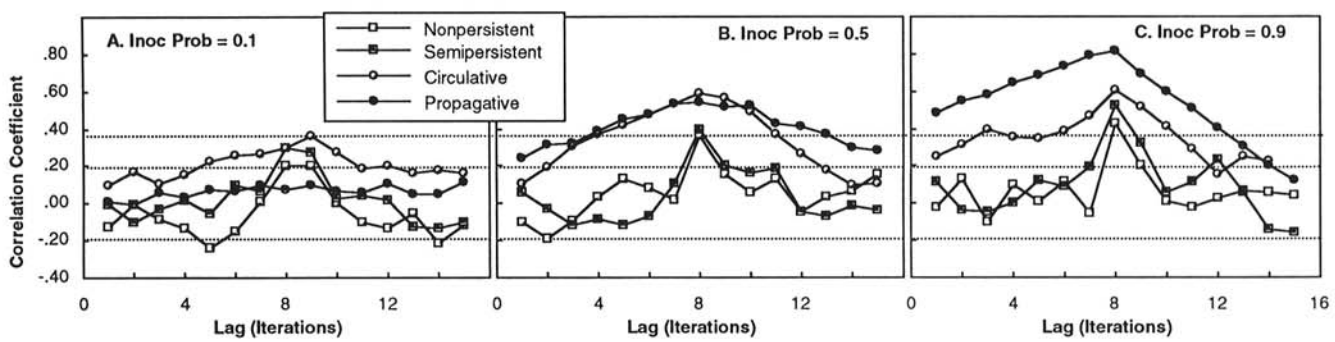

Fig. 10. Effects of lag on correlations between the change in the number of infectious plants and the number of infectious vectors at earlier iterations. Each data point represents the correlation coefficient from 10 iterations in each of 10 replicate simulation runs (n = 100) with a plant latent period of eight iterations, other standard parameter values, and inoculation probabilities (Inoc Prob) of **A,** 0.10, **B,** 0.50, and **C,** 0.90. Dashed lines represent standard limits for significance at $P = 0.05$, 0.01, and 0.0001.

indicates that simulated epidemics of nonpersistent viruses may simply have been more strongly affected by values of input variables. Compared with the other transmission types, simulated nonpersistent epidemics were more affected by variations in vector numbers and activity and probabilities of acquisition and inoculation (Figs. 7 and 8). If high values were specified for all of these input parameters, then simulated nonpersistent epidemics progressed about as quickly as those of the other types. In actual field situations, devastating epidemics of nonpersistent viruses may be due to influxes of large populations of infectious vectors and/or to very frequent vector-movements situations that are not directly addressed by the current model.

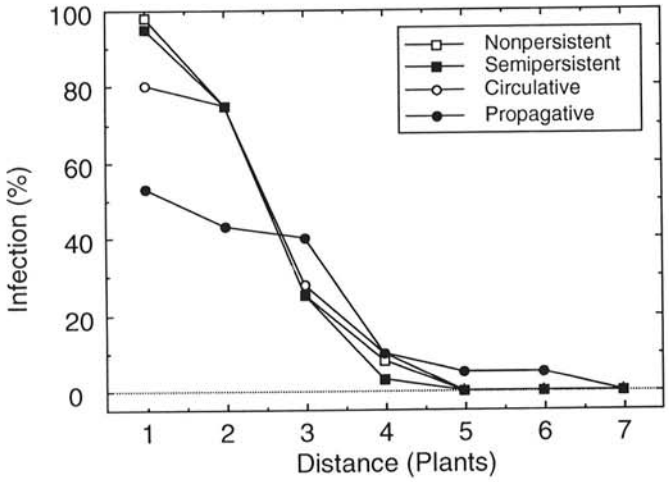

Fig. 11. Relationships between percentage of infection and distance from the initially infected plant for four virus transmission types using standard input parameter values. Percentages of infection are averages of the four cardinal directions for 10 replicate simulation runs at the first iteration on which at least 20 plants were infected.

The model described here has a number of inherent limitations, including small field size, a limited possible number of vectors, and a static vector population. Although additional simulation runs could be conducted with the current model and further analyses of existent data sets could be undertaken, further development of the model should probably be done with a new implementation. If the source code were rewritten, comparisons of results of the old and new models could be used to detect errors, new features could be incorporated, and the code could be made to be more easily modified. Improvements might include additional parameters, variable probabilities of acquisition and inoculation, larger field size, more realistic simulation of vector populations (including reproduction, development, death, and immigration), the ability to conduct large batches of simulation runs, and improved recording of output data. If the code for the simulation were separated from the code for the user interface, models could be produced that are easily transported between computer hardware platforms. Since we are not able to continue work on the model, we hope that others will continue its development.

Models such as the one we developed appear to be poorly suited to producing accurate predictions about field epidemics; however, we believe that they are a necessary intellectual exercise, and they may be a useful tool in the development of simplified models that can be applied to the field. Unless we consider all aspects of a disease system at one time, we cannot hope to understand the complete meaning of any particular piece of information. Even though models such as the one we developed cannot be validated in the usual manner, they provide a logically rigorous way of integrating knowledge about the many processes that affect virus disease epidemics.

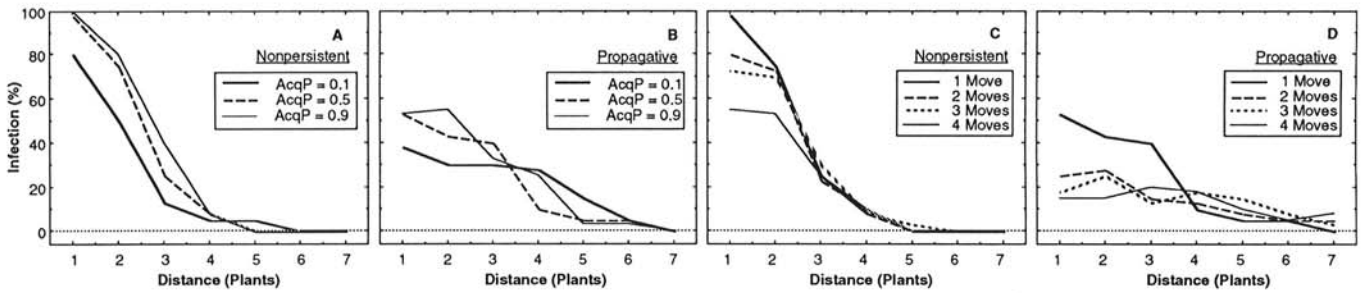

Fig. 12. Effects of variation in acquisition probability (AcqP) and number of vector movements per iteration (Moves) on relationships between percentage of infection and distance from the initially infected plant for **A and C,** nonpersistent and **B and D,** propagative transmission types. Percentages of infection are averages of the four cardinal directions for 10 replicate simulation runs at the first iteration on which at least 20 plants were infected.

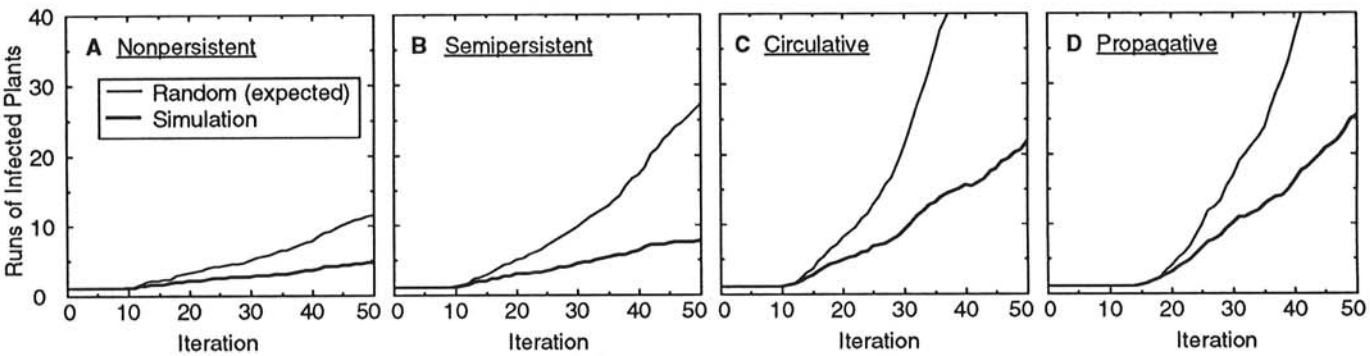

Fig. 13. Relationship between number of horizontal runs of infected plants and iteration for four transmission types using standard parameter values and for theoretical random distributions of the same numbers of infected plants. **A,** Nonpersistent; **B,** semipersistent; **C,** circulative; and **D,** propagative. Data are means of 10 replicate simulation runs.

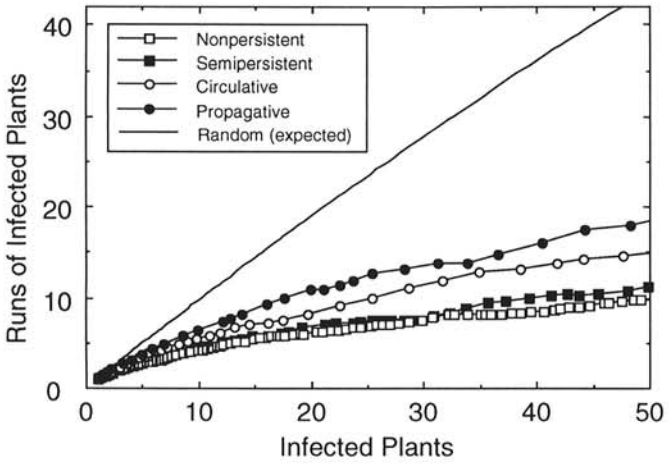

Fig. 14. Relationships between horizontal runs of infected plants and number of infected plants for four transmission types using standard parameter values and for a theoretical random distribution of infected plants.

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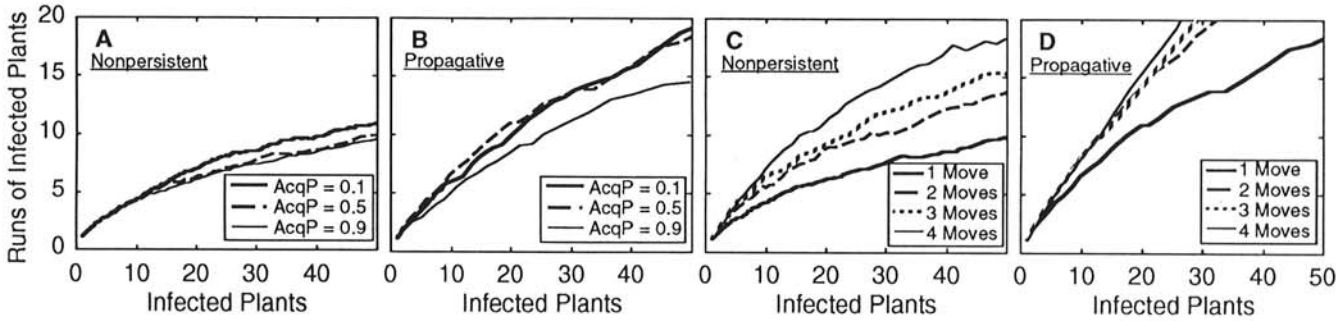

Fig. 15. Effects of variation in acquisition probability (AcqP) and number of vector movements per iteration (Moves) on relationships between number of horizontal runs of infected plants and number of infected plants for **A and C,** nonpersistent and **B and D,** propagative transmission types.

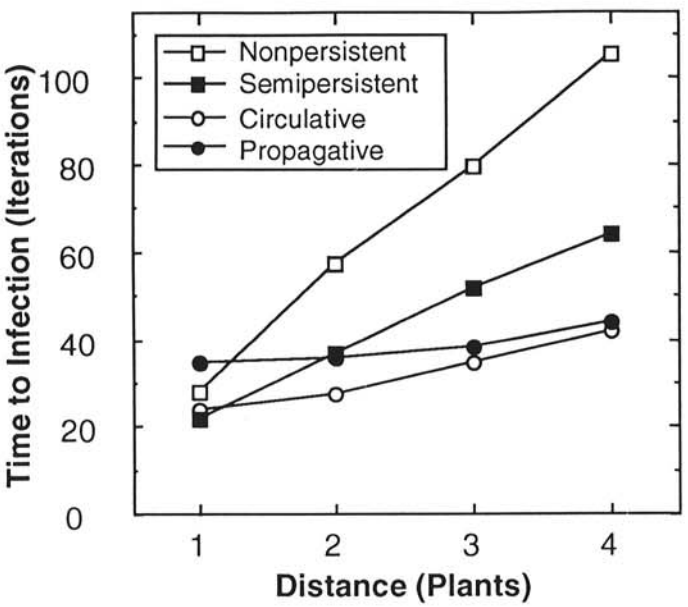

Fig. 16. Relationships between time to infection and distance from the initially infected plant for four virus transmission types. Values are averages of the four cardinal directions for 10 replicate simulation runs.

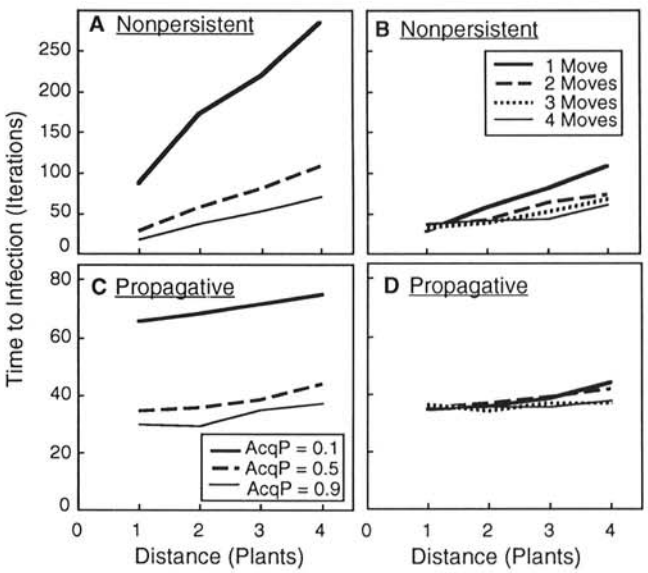

Fig. 17. A and C, Effects of variation in acquisition probability (AcqP) and **B and D,** number of vector movements per iteration (Moves) on relationships between average time to infection and distance from the initially infected plant for **A and B,** nonpersistent and **C and D,** propagative transmission types. Values are averages of the four cardinal directions for 10 replicate simulation runs.

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