Development of EPIVIT, a Simulation Model for Contact- and Aphid-Transmitted Potato Viruses

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ABSTRACT

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A model (EPIVIT) was developed for the simulation of potato harvest infection (% infected tubers) with a contact- or aphid-transmitted virus. Its state variables are the efficiency of autoinfection, primary infection of plants, and tuber infection of primarily infected plants. Input variables are daily minimum and maximum temperature and data referring to aphid species presence and the fluctuation of their winged population above the respective field. The model version for contact-transmitted viruses is based on an individual plant approach, which simulates the spread of the epidemic from infectious to healthy plants. The version for aphid-transmitted viruses is based on a population approach simulating primary infection by means of the negative binomial distribution. The code of both versions includes stochastic elements. Rate parameters for the simulation of the physiological age of the crop, the susceptibility of a plant to an infection, and the state variables are temperature sensitive. Coarse and fine sensitivity analysis of the model are presented. A working version of EPIVIT was implemented for IBM PCs and compatibles. The program is menu-and-mouse or keyboard driven and displays graphical and numerical model outputs.

Additional keywords: epidemiology, model verification, pathogen × host × environment interaction, pathosystem modeling, polyethic epidemic.

The potato crop is infected by numerous viruses, which differ in characteristics such as architecture, physico-chemical properties, and mode of transmission. The understanding of virus transmission is of particular practical value, as it facilitates conceptual approaches for the management of virus diseases. Those potato viruses that are the most important in terms of global spread and yield reduction are contact- or aphid-transmitted. It is generally understood 1) that the percentage of virus-infected tubers in a seed tuber lot (virus incidence) increases if the same tuber lot serves as a source for seed tuber selection during consecutive seasons (polyethic epidemic), and 2) that this phenomenon is responsible for a progressively increasing loss of the yield potential of the seed tubers.

A predictive model for harvest infection (percentage of infected tubers in the harvest of a plot) would be beneficial for potato production in both the developed and the developing world. It would be of particular interest to seed production specialists in developed countries as a potential tool for precise determination of the haulm destruction date to avoid exposure of the crop to high aphid populations. Additionally, it would be useful for seed program managers and for seed production specialists in developing countries. In these countries, for numerous reasons, formal seed systems have not had the same impact as in developed countries. Field data on virus incidence and yield reduction caused by viruses are scarce. Many of the farmers in some of these countries live in zones where virus degeneration of seed potatoes is very low (3), degeneration being understood as the difference between the proportion of infected tubers in a seed tuber lot and its respective harvest. The above-mentioned model would allow estimation of the number of successive generations for which a high-quality seed tuber lot may be multiplied with traditional crop management practices. Zones that are suitable for the multiplication of high-quality seed tubers could be more easily demarcated.

The potato plant has received considerable attention from modelers for simulating crop growth but little for simulating virus epidemics. Several models for potato crop growth and development, with different degrees of complexity, have been published (10,21,23,28,33). Because growth is temperature-dependent (27), all calculate a temperature-related physiological age of the crop or of determined organs instead of using a calendar scale, in order to obtain a realistic scale for growth. One simulation model for potato viruses has been fully published so far (35). It is thought to improve the understanding of the complex interactions between virus, vectors, host, and environment for PVY^O in Sweden. It calculates the percentage of PVY^O-infected tubers in the harvest of a field. The state variable is the number of plants that act as a virus source in an average potato field within a region. One of the major drawbacks of the model may be that the percentage of tubers infected among those produced by primarily and secondarily infected plants is fixed at 100%. However, the forecast that this model makes was reported to be accurate enough for use by seed potato growers in Sweden (35).

Extensive quantitative epidemiological studies in contrasting agroecozones of Peru (3) point to biological variables, in addition to those considered by the above model, which are essential for understanding potato virus pathosystems in contrasting environments and are essential for their effective management. Under determined conditions, some of the daughter tubers of secondarily infected plants may be healthy. This phenomenon was attributed to a temperature response of the percentage of tuber infection of secondarily infected plants. The expression "efficiency of autoinfection" has been used for this mechanism (3). The studies mentioned above also suggest that temperature conditions affect an aphid's ability to acquire and transmit the virus. Experiments under controlled conditions (9,36,37,42) documented that many parameters of the potato virus pathosystem respond to changes of temperature.

This publication reports the development of a model that was stimulated by a potato improvement program in Peru (11). The

objective of the study was a biologically significant model that predicts degeneration represented by the change of the percentage of virus-infected tubers between planting and harvest in a determined agroecozone. The model should respond to changes in temperature conditions and plant genotype, and be suitable for use in forecasting polyethic epidemics of the most important potato viruses in the Andes. Due to its postulated flexibility in parameter fixation for the computation of the model's state variables and the response of rates to temperature, the model could be adapted to growing zones other than the Andes, and to contact- and aphid-transmitted viruses of other vegetatively propagated tuber and root crops such as cassava, sweet potato, etc. Consequently, the model was named EPIVIT (epidemics of viruses of tuber and root crops, or in Spanish, in homage to the country which gave impetus to its development: epidemía de virus de cultivos de tubérculos y raízes).

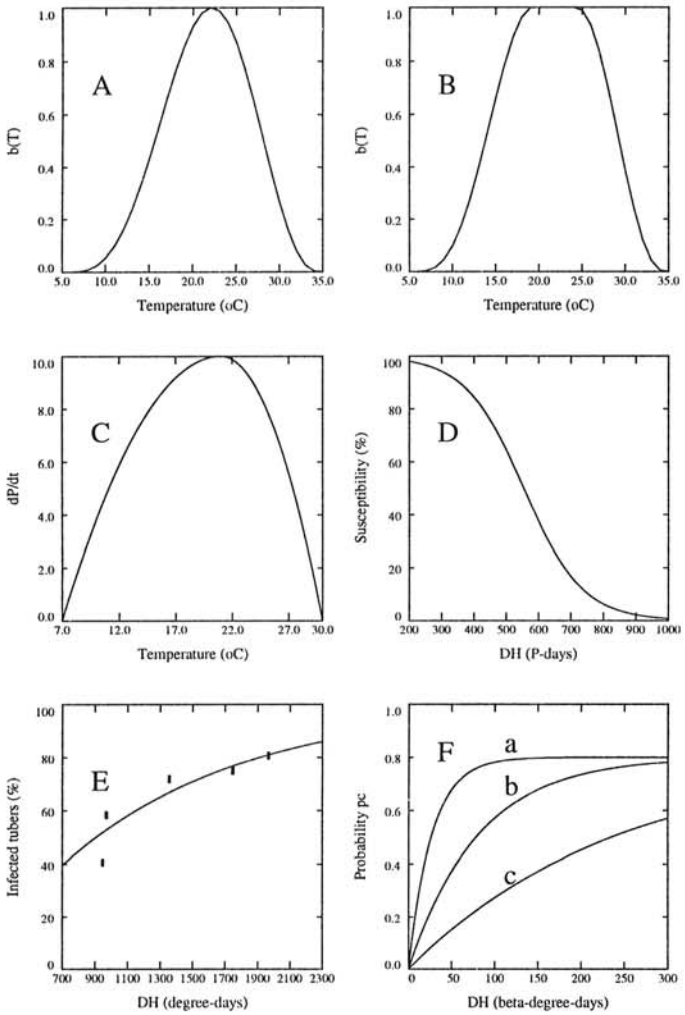

Fig. 1. Plots for the illustration of some selected models that were essential for EPIVIT's development. **A,** An example of a beta-function with temperature T as independent variable (parameters: $m = 3$, $n = 4$; cardinal temperatures of 5 and 35°C); **B,** the modified, discontinuous version of the beta-function (same parameter values and temperature range as **A,** delay range of 5°C); **C,** function for the simulation of the rate of advancement of the physiological crop age with T reported by Sands et al. (33); **D,** model for the logistic decrease of a plant's susceptibility to a virus infection with accumulated developmental heat (DH) related to physiological crop age ($DH_0 = 98$, $r = -1.0998$E-2); **E,** fit of the monomolecular function through data on the efficiency of autoinfection (tsi) with PVX plotted against DH in degree-days accumulated above 0°C in the respective agroecozones (3); model: $tsi = 1 - [(1 - 0.145) \exp(-9.13\text{E-}4 \text{ degree-days})]$; fit (with values transformed to the linear model version): $r^2 = 0.941$; **F,** selected multiple infection transformations relating DH (in beta-degree-days) to the probability of infection with a contact-transmitted virus (pc) for a healthy plant situated adjacent to a virus source plant; model: $pc = Su_c\ [1 - \exp(-r_{mi}\ DH/Su_c)]$, $Su_c = 0.8$; r_{mi} is with case a: 3.0; case b: 1.0; case c: 0.33.

Modeling terminology and symbols are used as defined elsewhere (12,30). To ensure internal consistency of the model and to guarantee the dimensionality of the model concept, dimensions and units are presented with equations and variable and parameter listings as suggested by Zadoks and Schein (43). Dimensions and units are placed in square brackets. Considered dimensions are $[Ti]$ for time, $[DT]$ for developmental time (measured in heat units such as degree-days), $[T]$ for temperature, $[N]$ for numbers, and [1] for dimensionless ratios, proportions, etc. Integer numbers are represented by symbols starting with a capital letter, whereas symbols that represent a percentage are written in lower case.

MATERIALS AND METHODS

Simulation philosophy and system boundary. The model to be developed had to be explanatory to a certain degree, mimicking biological mechanisms that are decisive for harvest infection. If this model had to explain a part of the system studied, it was also expected to have good predictive value (15) for forecasting harvest infection. The model should calculate harvest infection by assembling at the end of a season simulated output variables that were considered essential for understanding the pathosystem and for defining its state (see below).

Plot sizes are small to medium (approximately 100 m^2) in most of the target zones for which EPIVIT should work (11). Therefore, EPIVIT was intended to be used for simulations in a small to medium average potato field of a determined zone. The expression "average" refers to crop management, cultivar, vectors, climate, diseases, and pests in the simulated field that were considered representative for the respective zone. The model should address polyethic epidemics of contact- and aphid-transmitted viruses. It should be dynamic and mechanistic, including the linkage of continuous differential equations with time-discrete difference-equations.

In general, degeneration experiments may require experimental designs demanding extensive serological testing and spatial monitoring in order to yield conclusive results (3). Conventional factorial designs with repetitions cannot be realized with such complicated plot designs. Therefore, probabilistic elements that account for the fluctuation of results in a particular plot around the biological trend (29) had to be incorporated into EPIVIT.

Basic assumptions. Agroecological conditions within a field were assumed to be homogeneous. According to the model, a plant ages on a physiological, not calendar, time scale. Physiological age depends on temperature. The period from emergence to senescence was considered to be the part of a growing season that is relevant to virus spread in a field. It was assumed that degeneration does not increase greatly during tuber storage. Further assumptions were the following: 1) The growing period is initiated for computations at 50% emergence in the field and is stopped at 100% senescence (33); the crop does not suffer water stress. 2) No virus is carried into the simulated field from outside sources. 3) No differences exist among the efficiency of autoinfection of individual plants within the same field. 4) After the successful transfer of a viral particle to cells of a previously healthy plant (virus transmission), the latter becomes latently infected during a certain period of time (latent period), which implies that the virus replicates but does not yet translocate to other plant organs, that the tubers of such plants are therefore still healthy, and that the virus titer in the tissue is too low for the plant to act as a virus source plant. 5) After this latent period, the plant becomes infectious and the systemic virus movement toward the tubers begins. 6) This virus movement toward the tubers and the virus multiplication in host cells determine both the efficiency of autoinfection of secondarily infected plants and tuber infection of primarily infected plants; this mechanism is governed by the same parameters for both infection types (secondary and primary). Assumptions that are specific for the computation of a particular variable or a particular virus type are given in the respective sections below.

Temperature sensitive growth rates and developmental heat. Temperature has been proposed as the principal climatic variable for potato virus–host plant interaction (3). The response of a growth process to changes in temperature conditions can be characterized (i) by the temperature range where growth occurs, and (ii) a function determining the growth rate at any temperature within this range relative to the maximum rate. A function which is highly variable in shape and which has been used to relate temperature to the development of pathogenic fungi is the beta-function (2,5,19,20; eqs. 1 and 2). Its plot is bell-shaped with cardinal temperatures T_{min} and T_{max} (see an example in Fig. 1A). The parameters m and n are host–pathogen specific, and a is a scaling factor. The function represents physicochemical principles of the response of a growth rate to temperature.

$$b(T) = a\ T_b^n(1 - T_b)^m \quad [1]\ [-] \quad (1)$$

$$T_b = (T - T_{min})/(T_{max} - T_{min}) \quad [1]\ [-] \quad (2)$$

EPIVIT uses this function to relate rates with temperature. In a discontinuous version, $b(T)$ increases according to eq. 1, starting with $b(T) = 0$ at T_{min} and with $(T_{max} - dr)$ as cardinal temperature at the upper end of the relevant temperature range. Once the function gets to 1.0, $b(T)$ is held at this value during a given delay range of temperature degrees (dr) before it declines again, according to eq. 1, with a lower cardinal temperature of $(T_{min} + dr)$ until it falls to 0 at T_{max} (see an example in Fig. 1B).

The calculation of degree-days (or heat sums) has been used extensively for the calculation of developmental heat as a model for relating recurring phenomena of insect and plant development to environmental changes with time. Degree-days were computed by integration of the area under the sine curve through historical minima and maxima of daily temperature above a determined threshold.

EPIVIT's fundamentals. EPIVIT defines the state of the pathosystem at the end of a season with the efficiency of autoinfection (*tsi*), the percentage of primarily infected plants (*pi*), and the percentage of infected tubers of primarily infected plants (*tpi*). The model does not use a rate representing the change of harvest infection (*hi*) per season (k) but computes harvest infection at crop senescence by mechanistically computing *hi* for each season with the state variables mentioned above according to the rules derived from previous studies of the biological mechanisms which underlay the epidemic. The number of infected seed tubers in the simulated field (*Si*) is the number of seed tubers used (Ns) multiplied by harvest infection of the last season ($hi(k - 1)$; eq. 3). The number of emerged infected seed tubers (*SiE*) is the product of *Si* with the respective emergence (*esi*; eq. 4). The number of plants that do not act as a virus source until harvest (*HeE*, including latently infected plants) is the difference between the product of the number of healthy seed tubers with the respective emergence (*eh*) and primarily infected plants (*Pi*) that become infectious, i.e., a virus source plant for further spread until the end of a season (eq. 5). The efficiency of autoinfection (*tsi*) and the percentage of infected tubers of primarily infected plants (*tpi*) are averaged respectively over all secondarily and infectious primarily infected plants (%). The output variable (*hi*) is computed at crop senescence according to eq. 6, the numerator representing the simulated number of infected tubers produced in the respective field and the denominator representing the simulated total number of tubers produced. *NSi, NPi,* and *NHe* are constants for the number of tubers produced, respectively, by secondarily infected,

TABLE 1. List of EPIVIT's input, output, and auxiliary variables

Variables	Description	Dimension[a]	Units
Input variable			
T_{min}, T_{max}	Historical minimum and maximum of daily temperature	[*T*]	[°C]
Tc	Weekly aphid trap catches	[*N*]	[*aphid*]
Output variable			
hi	Percentage of infected tubers in the harvest	[1]	[%]
Auxiliary variables			
bd	Beta-degree (rate of advancement of *DH* with *t*)	[*DT*/*Ti*]	[*bdd*/*hour*][b]
DH	Developmental heat sum	[*DT*]	[*bdd*]
esi	Emergence of secondarily infected seed tubers	[1]	[%]
eh	Emergence of healthy seed tubers	[1]	[%]
Hf	No. of virus-free plants	[*N*]	[*plant*]
He	No. of noninfectious plants (includes latently infected plants)	[*N*]	[*plant*]
HeE	No. of emerged *He*	[*N*]	[*plant*]
i	Week	[*Ti*]	[*week*]
In	Simulated, performed inoculations in the field	[*N*]	[*inoc.*]
Inp	Simulated, performed inoculations per plant	[1]	[*inoc.*/*plant*]
k	Season	[*Ti*]	[*season*]
Lpw	Latent period (measured in weeks)	[*Ti*]	[*week*]
Ne	No. of planted tubers in the simulated field	[*N*]	[*tuber*]
Ns	No. of emerged tubers in the simulated field	[*N*]	[*tuber*]
pa	Probability of a landing aphid being viruliferous	[1]	[%]
pc	Probability of infection with a contact-transmitted virus	[1]	[%]
Rf	Relative efficiency factor (*T* sensitive)	[1]	[−]
Pi	No. of primarily infected, infectious plants	[*N*]	[*plant*]
pi	Percentage of primarily infected plants that are infectious	[1]	[−]
PiP	No. of newly, primarily infected plants	[*N*]	[*plant*]
r_p	Rate of advancement of physiological age with *t*	[*DT*/*Ti*]	[*P-day*/*hour*]
Si	Secondarily infected plants or seed tubers	[*N*]	[*plant*]
SiE	No. of emerged *Si*	[*N*]	[*plant*]
Sp	No. of aphid species	[*N*]	[*species*]
Vp	Simulated vector pressure	[*N*]	[*Vpu*][c]
t	Season time	[*Ti*]	[*day*]
T	Temperature	[*T*]	[°C]
Ta	Average daily temperature during aphid activity	[*T*]	[°C]
Tv_{sp}	Simulated viruliferous aphids of species *sp*	[*N*]	[*aphid*]

[a] Dimensions are: *DT*: developmental heat; *N*: numbers; *T*: temperature; *Ti*: time; 1: dimensionless (ratios, proportions, etc.).
[b] *bdd*: Beta-degree-days.
[c] *Vpu*: Vector pressure units.

primarily infected, and healthy plants.

$$Si(k) = hi(k-1)\ Ns(k) \qquad [N]\ [plants] \qquad (3)$$

$$SiE(k) = Si(k)\ esi(k) \qquad [N]\ [plants] \qquad (4)$$

$$HeE(k) = \{[100 - Si(k)]\ eh(k)\} - Pi(k) \qquad [N]\ [plants] \qquad (5)$$

$$ih(k) = [SiE(k)\ tsi(k)\ NSi + Pi(k)\ tpi(k)\ NPi]/[SiE(k)NSi + Pi(k)\ NPi + HeE(k)\ NH] \qquad [1]\ [\%] \qquad (6)$$

EPIVIT is driven by daily minimum and maximum temperatures and weekly aphid catches of an appropriate insect trap, which allow for the monitoring of the vector species present and the estimation of their respective specimen numbers for a particular week. State variables are calculated daily according to the actual set of independent variables. Primary infection of plants is computed weekly from weekly aphid input data. The remoteness of a selected production zone and/or the limited availability of trained technicians may make daily recordings of aphid data difficult in developing countries. Data collection is most probably possible every three or more days; thus, weekly input of aphid data into the model has been used.

EPIVIT's input, output, and auxiliary variables and the model's constants are listed in Tables 1 and 2. All parameters for the simulation of state variables and essential auxiliary variables and indices used are summarized in Table 3.

Physiological time. Two approaches for the simulation of the crop's physiological age were considered, which appeared to enhance the flexibility of the model and its potential to be applied to different conditions. With both, physiological age (*P-time*) until season time t is accumulated relative to real time at a rate rp, which depends on temperature T (eqs. 7 and 8).

$$r_p(t) = \frac{dP}{dt} = kp\, f[T(t)] \qquad [DT/Ti]\ [P\text{-}days/hour] \qquad (7)$$

$$P\text{-}time(t) = \int_0^t r_p\,[T(t)]\,dt \qquad [DT]\ [P\text{-}days] \qquad (8)$$

TABLE 2. List of indices and constants used by EPIVIT

Indices	Description	Constants	Description
a	Refers to *DH*[a] related to *tsi*[b]	*kp*	Scaling factor for r_p[c]
at	Refers to *DH* related to *tsi*[b]	*NHe*	No. of daughter tubers of a *He*[d]
b	Refers to *DH* related to *Pi*[e]	*NPi*	No. of daughter tubers of a *Pi*[e]
br	Between planted rows	*NSi*	No. of daughter tubers of a *Si*[f]
c	Constitutive	C_{wr}	Randomization constant or see Table 3
Lp	Relates to the latent period		
mr	Relates to mature plant resistance		
pi	Relates to primary infection		
p	Relates to physiological age		
sp	Aphid species		
wr	Within a planted row		
z	Aphid specimen or a single plant		

[a] Developmental heat.
[b] See Table 3.
[c] See Table 1.
[d] Noninfectious plant.
[e] Primarily infected, infectious plant.
[f] Secondarily infected plant.

The constant kp is a scale factor and was set to 10 (33). The first approach uses two continuous functions with adjacent upper and lower cardinal temperatures, respectively, for the nonlinear relationship between development and temperature, $f(T)$, and was taken from the literature (33). The second approach uses the beta-function as a theoretical function for $f(T)$. After preliminary comparison of the two approaches by calculating *P-time* with historical data (data not shown), the beta-function was chosen because it provided the most flexible approach for the computation of physiological time.

Host response to virus infection. EPIVIT includes elements that relate to the plant's response to the viral pathogen. This is essential for the simulation of all state variables. It is assumed that plant response is composed of a constitutive susceptibility index (Su_c) ranging from 0 to 1.0, which is provided by the genotype of the respective cultivar, and a variable susceptibility factor (Su_{mr}). The latter reflects mature plant resistance and refers to the establishment of the pathogen in the host and subsequent systemic translocation in the plant's organs towards the tubers (34). The model considers mature plant resistance to be a function of the physiological but not the calendar age of the crop. It responds therefore to changes in temperature conditions and ranges from 0 to 1.0. Su_{mr} is the difference of mature plant resistance from 1.0, and it decreases from 1.0 to 0 during plant aging. Decrease is initialized at a physiological age of *Mri* and ends at the physiological age P_{max} that a cultivar accumulates from 50% emergence until senescence. It is a logistic function, as suggested by the array of data points used by Sigvald (35) for the same purpose. The steepness of the decrease is determined by the rate parameter r_{mr} of the corresponding logistic function (see an example in Fig. 1D).

The latent period. During this period, the virus is supposed to establish and multiply in the infected and some neighboring host cells, accumulating up to a certain concentration. The plant then becomes infectious, i.e., it may act as a source for virus spread to healthy plants. Systemic spread inside the plant is assumed to start at this point. The latent period (*Lp*) is needed for the simulation of primary infection of plants. EPIVIT measures *Lp* in units of developmental heat which are computed similarly to those for the physiological age (eqs. 7 and 8). The parameters m_{Lp}, n_{Lp}, and dr_{Lp} of the respective beta-function may be equal to or different from those used for the computation of physiological age. The model multiplies the amount of developmental heat accumulated in this way by the susceptibility Su_{mr} before the product (in P_{Lp}–*days*) is assigned to *Lp*.

The efficiency of autoinfection. Based on epidemiological studies, a strong temperature dependence of the efficiency of autoinfection (*tsi*) was suggested (3). In a preliminary attempt to determine the nature of the relation between the efficiency of autoinfection and temperature, accumulated degree-days were calculated with temperatures >0 C for the published data set mentioned above (3). This set includes the historical weather data of the considered agroecozones and seasons, phenological data of one potato cultivar, and the measured efficiencies of autoinfection of potato X potexvirus (PVX), Andean potato mottle comovirus (APMV), potato Y potyvirus (PVY^N-strain) as co-infected with PVX, and potato leafroll luteovirus (PLRV). Total degree-days between 50% emergence and senescence were plotted and correlated against the respective reported efficiencies of autoinfection (*tsi*). The plot for PVX suggested a monomolecular relation between these variables (Fig. 1E). To improve the biological meaning of the model and the significance of correlations for all viruses studied, elements were incorporated into EPIVIT to weigh actual temperatures biologically for their significance for virus multiplication and translocation inside the plant, and for virus movement as influenced by age resistance. EPIVIT transforms temperatures with a beta-function to obtain the rate bd_a (index $_a$ relates to the efficiency of autoinfection) at which developmental heat (measured in beta-degree–day units; bdd_a) is accumulated (eq. 9). The model multiplies the rate bd_a with the age-specific susceptibility $Su_{mr}(t)$ before it integrates with time t to obtain the developmental heat DH_a (eq. 10), which serves as independent variable for the calcula-

tion of the efficiency of autoinfection (*tsi*), as explained below.

$$bd_a(t) = T(t)\ b[T(t)]_a \qquad [DT/Ti] \quad [bdd_a/\text{hour}] \qquad (9)$$

$$DH_a(t) = \int_0^t bd_a(t)\ Su_{mr}(t)\ \mathrm{d}t \qquad [DT] \quad [bdd_a] \qquad (10)$$

For obtaining significant correlations with PVY and APMV in preliminary testing of this method with the data set mentioned above, the incorporation of a further biologically meaningful parameter into the model was necessary. It was called triggering developmental heat (*TH*), measured in beta-degree-days bdd_{at}. A number of *TH* beta-degree-days are required for triggering heat accumulation for DH_a. The model uses the same parameter values for computing db_a related to DH_a and db_{at} related to *TH*. A simulation run starts with the accumulation of developmental heat (measured in beta-degree-days) with the rate bd_{at}. A particular number of *TH* beta-degree-days triggers the accumulation of developmental heat DH_a with the rate bd_a (eq. 11). If the temperature falls below or rises above developmental minimum and maximum temperatures (e.g., 0 and 40°C), the model sets the number of beta-degree-days bdd_{at} (accumulated for obtaining the *TH*) back to zero. The procedure needs to be initiated again before DH_a can be accumulated further.

$$DH_a(t) = \int_0^t bd_a(t)\ Su_{mr}(t)\ \mathrm{d}t \text{ with}$$
$$DH_{at}(t) = TH \text{ and}$$
$$T_{\min;a} <= T(t) <= T_{\max;a} \qquad [DT] \quad [bdd_a] \qquad (11)$$

$$tsi(t_{\max}) = 1 - \{(1 - tsi_0) \exp[-r_a\ DH_a(t_{\max})]\} \qquad [N^{-1}] \quad [Plant^{-1}] \qquad (12)$$

The hypothesis that underlies this model claims that the presence and production of virus-specific transport proteins in the plant cell depend on the presence of a determined quantity of products of formerly transcribed viral genes. Equation 12 presents the analytical model (monomolecular) according to which EPIVIT computes the efficiency of autoinfection at 100% senescence ($t_{\max}$) by using DH_a as the independent variable.

Primary infection of plants. The mode of transmission of a virus greatly influences the spatial pattern and sequence of spread and hence the overall dynamics of disease progress (40). Plants adjacent to infectious ones have the highest probability of being infected in the case of contact-transmitted viruses. The pattern of the spatial spread of aphid-transmitted viruses cannot be predicted easily because virus transmission depends on winged and apterous specimens of selectively transmitting aphid species which respond to climatic conditions, field management practices, and competition with other organisms of the ecosystem. Consequently, different approaches were used for developing a model for the primary spread of contact- and aphid-transmitted viruses.

Contact-transmitted viruses. For contact-transmitted viruses, EPIVIT characterizes each plant by its spatial position in the field and simulates the virus spread from infectious to adjacent healthy plants. A successful infection is the result of the interaction between plant and virus in which both respond to climatic conditions, of which temperature may be the most important variable (3). It has been documented for PLRV, although not for contact-transmitted PVX and APMV, that susceptibility of a potato plant to infection is altered by changing temperature conditions (37).

TABLE 3. The parameters of EPIVIT's state variables and of its essential auxiliary variables

Variables and parameters	Description	Dimension[a]	Unit
Efficiency of autoinfection (*tsi*) and primary tuber infection (*tpi*)		[1]	[$plant^{-1}$]
m_a, n_a, dr_a,		[1]	[−]
$T_{\min;a}$, $T_{\max;a}$	Beta function parameters	[*T*]	[°C]
TH	Trigger developmental heat	[*DT*]	[bdd_a]
r_a and	Rate parameter and y-axis intercept of the monomolecular	[DT^{-1}]	[bdd_a^{-1}]
tsi_0	function representing $tsi = f(DH)$	[1]	[%]
Primary infection of plants (*Pi*)		[*N*]	[*plant*]
General			
Lp	Latent period	[*DT*]	[P_{Lp}-*days*]
m_{Lp}, n_{Lp}, dr_{Lp}	Beta-function parameters	[1]	[−]
Contact-transmitted viruses		−	−
m_b, n_b,		[1]	[−]
$T_{\min;b}$, $T_{\max;b}$	Beta function parameters	[T]	[°C]
C_{wr}, C_{br}	Plant age at canopy closure within and between rows	[*DT*]	[*P-days*]
r_{mi}	Rate parameter of the multiple infection transformation	[*DT*]	[bdd_b^{-1}]
Aphid-transmitted viruses			
k_{pi}	Parameter of the negative binomial distribution	[1]	[−]
h_1, h_2, h_3	Average daytime when aphid activity starts, ends, and temperature reaches its maximum	[*Ti*]	[*hour*]
Rfg_{sp}	Genotypic relative efficiency factor for species *sp* ranging from 0 to 1.0 (not temperature sensitive)	[1]	[−]
m_{sp}, n_{sp},	Beta function parameters related to the temperature-	[1]	[−]
$T_{\min;sp}$, $T_{\max;sp}$	sensitive Rf_{sp} ranging from 0 to 1.0	[*T*]	[°C]
Af_{sp}	Relative attraction factor for species *sp* ranging from 0 to 1.0	[1]	[−]
q	Scaling parameter for *Rf* and *Af*	[1]	[−]
M	Number of aphid moves in a field before leaving	[*N*]	[*moves*]
Physiological time (*P-time*)		[*DT*]	[*P-days*]
m_p, n_p, dr_p,	Beta function parameters for the calculation of the rate of	[1]	[−]
$T_{\min;p}$, $T_{\max;p}$	physiological time advancement with time, $r_p(t)$	[*T*]	[°C]
Susceptibility of plants to an infection (Su_{mr})		[1]	[−]
Su_c	Constitutive susceptibility index ranging from 0 to 1.0	[1]	[−]
$P_{\max}$	Physiological age at 100% senescence of the crop	[*DT*]	[*P-days*]
Mri	Physiological age at the initialization of mature plant resistance	[*DT*]	[*P-days*]
r_{mr}	Rate parameter of the logistic function for mature plant resistance	[DT^{-1}]	[$P\text{-}days^{-1}$]
Qualitative parameter			
Spatial pattern	Seed tubers are distributed onto the (row × plants/row)-plot lattice at random, uniformly, or according to a historical field design	−	−

[a] Dimensions are: *DT*: developmental heat; *N*: number; *T*: temperature; *Ti*: time; 1: dimensionless (ratios, proportions, etc.).

Such a phenomenon may be explained in part by temperature-sensitive susceptibility such as that modeled by Su_{mr} (see above). EPIVIT relates Su_{mr} (derived from mature plant resistance) only to the biological mechanisms that operate after a successful insertion of a virus into a host cell. However, the ease with which cuticle and epidermal cells may be wounded and the conditions during the early stages of the virus insertion are likely to be important for a successful infection as well. The quantitative relationship between temperature conditions and these biological parameters of primary infection have not been elucidated so far. EPIVIT assumes that weekly degree-days provide explanatory help in this respect because they reflect the fluctuation of temperature, to which above parameters respond within a given time range in a biologically significant way.

The model relates weekly accumulated developmental heat to the probability of virus spread from an infectious to an adjacent healthy plant (eqs. 13 to 15). It multiplies the heat accumulation rate $bd_b(t)$ with Su_{mr} before integration and accumulation. Integration of bd_b starts following the time when the plant canopy closes (tc; eq. 14). The model uses C_{wr} and C_{br} for the physiological ages at which a canopy closes, respectively, within a row and between rows.

EPIVIT uses Gregory's multiple infection transformation (18) to calculate probabilities of infection. A plant z, which is healthy and adjacent to an infectious plant during week i, becomes infected with a probability of $pc_z(i)$. The computation of $pc_z(i)$ by means of the multiple-infection transformation (eq. 15) requires Su_c being the constitutive susceptibility, r_{mi} a rate parameter of the multiple-infection transformation, and $DH_b(z,i)$ the developmental heat measured in beta-degree-days accumulated until week i for the respective plant z since C_{wr} (adjacent infectious plant in the same row) and since C_{br} (adjacent infectious plant across the row). Su_c acts as asymptote of the function (for an example see Fig. 1F).

$$bd_b(t) = T(t)\ b[T(t)]_b \qquad [DT/Ti] \quad [bdd_b/hour] \qquad (13)$$

$$DH_b(t) = \int_{tc}^{t} [Su_{mr}(t)\ bd_b(t)] \qquad [DT] \quad [bdd_b] \qquad (14)$$

$$pc_z(i) = Su_c\{1 - \exp[-r_{mi}\ DH_b(z,i)/Su_c]\} \qquad [1] \quad [\%] \qquad (15)$$

$$Pi(i) = Pi(i-1) + PiP(i - Lpw) \qquad [N] \quad [Plants] \qquad (16)$$

$$PiP(i - Lpw) = \sum_{z=1}^{Hf(i-Lpw)} Hf_z(i - Lpw) \text{ if } C_z = \text{random } (C) <= C\ pc_z(i - Lpw) \qquad [N] \quad [Plants] \qquad (17)$$

Once the plants touch each other, the model simulates weekly primary infection and records the health state (healthy, latently infected, and infectious) of each spatially identified plant in the plot. The total number of primarily infected plants that are infectious (Pi) in the respective plot is calculated with a difference equation (eq. 16). The plants that became infectious during week i are added to the number Pi of the previous week. The plants $PiP(i - Lpw)$ that became newly infected one latent period (Lp) ago are the newly infectious plants, i.e., those that become infectious in week i. The letter w in Lpw indicates that Lp is transformed to week units. $PiP(i - Lpw)$ is assembled based on the probabilities pc, which refer to all those probabilities with which an individual virus-free plant (Hf_z) at the time ($i - Lpw$) may become infected. It is the total accumulated number of individual Hf_z plants of the week ($i - Lpw$) for which a random number C_z within the range 1 to C (randomization constant) is less than or equal to the product of C with the plant specific probability of infection $pc_z(i - Lpw)$ (eq. 17).

EPIVIT simulates the spread of contact-transmitted viruses only to plants directly adjacent to infectious plants within the respective row and across the row. It was assumed that reinfection of a plant has no effect on tuber infection of this same plant.

Aphid-transmitted viruses. Aphid behavior in a particular potato field (settling behavior, flight patterns, walking distances, reproduction rates, etc.) is responsible for the spread pattern of aphid-transmitted viruses, but data documenting this behavior are most often lacking for data sets available on virus incidence in the harvest of determined field plots. Consequently, it is not possible to simulate primary infection in such plots on an individual plant basis. Simulating the number, i.e., the population of plants that become primarily infected within a given time of the season appears to be more adequate.

Dispersal patterns of aphid-transmitted viruses in small plots tend to be clumped (3,39), in contrast to random or uniform (regular) patterns (6,24,25). The negative binomial distribution is commonly used by biologists as a statistical frequency distribution to represent clustered pattern (24). For EPIVIT for aphid-transmitted viruses, the model used was slightly modified from a reported model for the simulation of the impact of soybean mosaic potyvirus on yield and on the level of botanical soybean seed transmission (32). EPIVIT uses the negative binomial distribution to estimate the number of plants $PiP(i - Lpw)$ that became primarily infected during week $i - Lpw$ becoming infectious during week i. Primary infection is a function (eq. 18) of the parameter k_{pi} of the negative binomial distribution; the latent period (Lp); the trap catches (Tc) and relative efficiency factors (Rf) for virus transmission of particular aphid species; the attraction factors (Af) representing the relative attraction of an aphid species by the respective trap color; the plant's susceptibility determined by mature plant resistance (Su_{mr}); and M, which is a parameter related to aphid behavior (see below). The constitutive susceptibility, used for contact-transmitted viruses, was not incorporated into the model for aphid-transmitted viruses in the present version. The negative binomial distribution to calculate $PiP(i - Lpw)$ is represented in eq. 19. $Hf(i - Lpw)$ is again the number of virus-free plants at the end of week $i - Lpw$. It is multiplied by an expression including k_{pi} and $Inp(i - Lpw)$, the average number of inoculations per plant during week $i - Lpw$.

EPIVIT calculates $Hf(i - Lpw)$ as the difference of the number of emerged seed tubers (Ne) with the number of secondarily infected emerged seed tubers (SiE), infectious primarily infected plants [$Pi(i - Lpw)$], and latently infected plants at the end of week $i - Lpw$ (eq. 20).

$$Pi(i) = Pi(i-1) + PiP(i - Lpw) = Pi(i-1) + f(k_{pi}, Lp, Tc, Rf, Af, Su_{mr}, M) \qquad [N] \quad [plants] \qquad (18)$$

$$PiP(i - Lpw) = Hf(i - Lpw)(1 - \{k_{pi}/[k_{pi} + Inp(i - Lpw)]\}^{k}pi) \qquad [N] \quad [plants] \qquad (19)$$

$$Hf(i - Lpw) = Ne - SiE - Pi(i - Lpw) - \sum_{w=i-2\,Lpw-1}^{i-Lpw-1} PiP\,(w) \qquad [N] \quad [plants] \qquad (20)$$

$$Inp(i - Lpw) = \text{In}(i - Lpw)/Ne \qquad [1] \quad [inoc./plant] \qquad (21)$$

$$In(i - Lpw) = Su_{mr}(i - Lpw)\ Vp(i - Lpw) \qquad [N] \quad [inoc.] \qquad (22)$$

$$Vp(i - Lpw) = q \sum_{sp=1}^{Sp(i-Lpw)} \{Tv_{sp}(i - Lpw)\ Rf_{sp}(i - Lpw)/Af_{sp}\} \qquad [N] \quad [Vpu] \qquad (23)$$

$$Rf_{sp}(i - Lpw) = Rfg_{sp}\ b[Ta(i - Lpw)] \qquad [1] \quad [-] \qquad (24)$$

$$Tv_{sp}(i - Lpw) = \sum_{z=1}^{Tc_{sp}(i-Lpw)} Tc_{sp,z} \text{ if } C_z = \text{random } (C) <= C\ pa(i - Lpw) \qquad [N] \quad [aphids] \qquad (25)$$

$$pa(i - Lpw) = \{[SiE + Pi(i - Lpw)]/ Ne\}\,[M/(M+1)] \quad [1] \quad [\%] \qquad (26)$$

The model estimates $Inp(i - Lpw)$ by $In(i - Lpw)$, which is the total number of inoculations in the field during week $i - Lpw$, divided by Ne (eq. 21). $In(i - Lpw)$ is a simulated vector pressure index $Vp(i - Lpw)$ (measured in vector pressure units Vpu) multiplied by the plant's susceptibility (Su_{mr}), which is determined by mature plant resistance at the end of the respective week (eq. 22). Equation 23 explains the calculation of $Vp(i - Lpw)$ relating the simulated number of viruliferous trapped specimens $Tv_{sp}(i - Lpw)$ of Sp aphid species with the respective relative transmission efficiencies $Rf_{sp}(i - Lpw)$ and their constant factors for attraction Af_{sp} by the respective trap color. $Vp(i - Lpw)$ represents the simulated number of landing aphids which are viruliferous and accumulated during the respective week. Since both $Rf_{sp}(i - Lpw)$ and Af_{sp} are relative parameters, they need to be calibrated by q to yield an integer number of inoculations $In(i - Lpw)$ (eq. 23). An Rf_{sp} responds to changes in temperature conditions: a constant genotypic Rfg_{sp} (i.e., an Rf_{sp} that is constitutional to the respective species) is multiplied with a temperature-sensitive beta-function value $b[Ta(i - Lpw)]$ (eq. 24). $Ta(i - Lpw)$ stands for the average temperature conditions during main times of daily aphid activity in week $i - Lpw$ and is calculated as the average of daily temperature means in week $i - Lpw$ between a determined morning time (h_1) and an afternoon time (h_2), when aphid activity starts and stops respectively. The model calculates $Tv_{sp}(i - Lpw)$ stochastically. For each specimen of species sp, trapped during week $i - Lpw$, a random number C_z is selected between 1 and C (randomization constant). $Tv_{sp}(i - Lpw)$ is an accumulated number of specimens of week $i - Lpw$ if accounting among all specimens trapped ($Tc_{sp}(i - Lpw)$) only for those that are associated with a C_z that is less than or equal to C multiplied by $pa(i - Lpw)$ (eq. 25). The last variable represents the probability that a landing aphid in week i is viruliferous. For nonpersistently transmitted viruses, $pa(i - Lpw)$ reflects the direct flights of winged aphids from source plants within the field to healthy plants because specimens lose the virus particles after some probing. In the case of persistently transmitted viruses, $pa(i - Lpw)$ refers to the probability that an aphid feeds at least once on a viruliferous plant, meanwhile moving within the respective field during an average presence time that is the same for all aphids. The model estimates this probability as the proportion of source plants (emerged secondarily infected and infectious primarily infected plants) among all plants in the field, multiplied by the term $M/(M+1)$ (eq. 26). M represents the average number of moves an aphid makes within a field before leaving (32).

Primary infection of tubers. EPIVIT calculates $tpi(k)$, i.e., the percentage of tubers infected among those produced by infectious primarily infected plants during season k as an average value of the tpi of each individual primarily infected, infectious plant at 100% senescence (t_{max}) (eq. 27). Eq. 12 provides the necessary functional relationship between beta-degree-days accumulated since the week in which an individual plant became infectious and the tpi of such a plant. Parameters for computing the tpi of individual plants and the efficiency of autoinfection (tsi) are the same.

$$tpi(t_{max}) = \frac{\sum_{z=1}^{Pi(t_{max})} tpi_z\,(t_{max})}{Pi(t_{max})} \quad [N^{-1}] \quad [plant^{-1}] \qquad (27)$$

Each plant included for tpi calculation with contact-transmitted viruses is identified by its attributes related to the spatial position in the field, whereas such plants infected with aphid-transmitted viruses are indexed only with the respective week.

Computations. Temperature is the independent variable for computing beta-degrees and the rate at which physiological age increases (eqs. 7, 9, and 13). The daily temperature cycle is approximated by the sine wave through historical daily minimum and maximum temperatures. This practice (1) has gained broad acceptance for the calculation of degree-days in entomology and has also been recommended for epidemiological research with plant pathogens (13). The model computes physiological age and beta-degree-days by integration of the physiological time and beta-degrees, respectively, with time using the trapezoidal numerical integration method and a time step of 1 h.

EPIVIT for aphid-transmitted viruses needs to know the temperatures at average daily hours when aphid activity initiates and stops (h_1 and h_2; Table 3). Since the sine wave has no specific relation to a particular time of day, EPIVIT requires additionally the input of the average daily hour at which the temperature rises to its maximum (h_3) to be able to calculate the temperatures at h_1 and h_2.

The model determines, at the beginning of a season, the number of emerged healthy and secondarily infected plants. It computes the physiological age starting at 50% emergence (essential for Su_{mr}, C_{wr}, C_{br}, and Lp). Simultaneously, beta-degree-days are accumulated for the computation of the efficiency of autoinfection at harvest. Since many equations of EPIVIT contain delayed arguments, such as $PiP(i - Lpw)$ in eq. 16, the model uses the fixed boxcar train approach (16) for its calculations. The total of emerged plants is assigned to three boxcars, one of which contains the healthy plants, one the latently infected plants, and one the infectious primarily infected plants. Their contents are computed weekly. For contact-transmitted viruses, each box is divided into compartments corresponding to individual plants. For aphid-transmitted viruses, these compartments correspond to all plants falling into the respective category. Compartments for individual plants in the box for plants free of contact-transmitted viruses are filled with the accumulated amount of beta-degree-days, bdd_b, for the individual plant. The probability of infection for these plants is computed with $pc(z,i)$. For aphid-transmitted viruses, the one single compartment of this box holds the total number of virus-free plants. Each compartment of the box for plants latently infected with a contact-transmitted virus is associated with the accumulated fraction of the latent period of the respective plant. For aphid-transmitted viruses, a compartment holds the number of plants that became newly infected in a particular week, and it is associated with the respective fraction of the latent period. The box for infectious primarily infected plants is structured in the same way as the one for latent infections, but individual plants or weekly plant groups are associated with the accumulated fraction of infected tubers of the respective primarily infected plants. At the end of a season, the model relates the plant numbers in the boxes, the number of emerged secondarily infected plants, the simulated efficiency of autoinfection, primarily infected plants, and the average percentage of infected tubers of primarily infected plants according to eqs. 6, 12, 17 or 18, and 27, to calculate harvest infection hi. If desired, seed infection of the next season is computed (eq. 3).

The model code treats missing historical weather data for up to 3 days, as described elsewhere (8). Furthermore, a random-number generator was incorporated into the model (41) for the random selection of spatial positions in a field (first selection: row; second selection: plant) to assign nonemerged and, if convenient (see implementation) secondarily infected plants in the simulated plot, and for random runs according to eqs. 17 and 25.

Model evaluation. Sensitivity analysis was performed to contribute to model verification which, like model validation, is an element of model evaluation (38). Effects of systematic changes in model structure and model parameters on output variables were studied (coarse and fine sensitivity analysis; 7). With coarse sensitivity analysis, the effect of drastic changes in seed infection, temperature sensitivity of tsi and tpi, timing of aphid infestation, and spatial arrangement of infected seed tubers on harvest infection were studied. Fine sensitivity analysis was expected to help identify parameters that cause an over-proportional reaction of output variables compared to their own change, and that consequently need to be estimated precisely (7). The relative sensitivity of relevant model outputs to changes in parameter values was computed to quantify fine sensitivity. It was calculated as the

proportion between $\Delta v/v$ and $\Delta p/p$, Δv being the change in output variable v caused by a change Δp in parameter p (31).

EPIVIT had not been validated so far. Therefore, reference parameter settings were not available. It was decided to depart from a realistic initial value for each parameter and to change it within the limits of experimental precision and practical relevance. For example, the latent period was set initially to 70, which corresponds approximately to 1 wk in a site with temperature fluctuations between 13 and 23°C. This was considered a first realistic, although rough, estimate of the latent period for a modern potato cultivar in Peru (estimate based on unpublished data). Changes were then made to 80 (one additional day) and 140 (one additional week), relating to time steps of relevance for the resolution of the time scale in practical experimentation.

It was expected that the model outputs would be more sensitive to parameter changes if EPIVIT were run on plots with low seed infection. That is, since the model code is probabilistic, random discrete events may yield higher percent changes if they are calculated on low initial variable values rather than high initial variable values. Therefore, fine sensitivity analysis was applied both to plots with 2% and with approximately 20% seed infection. Other elements of model evaluation, such as parametrization (31), also called calibration or tuning (7), are presented elsewhere (4) or have yet to be performed (validation).

Plots of 300 and 216 plants were simulated for contact- and aphid-transmitted viruses, respectively (10 and six planted rows, respectively). Such plot field designs had been used for studying virus epidemics in contrasting agroecological zones in Peru (3). Adopting these designs for the sensitivity analysis was expected to provide some preliminary data that could be used later for the parametrization of the model with data from Peru. For sensitivity analyses, infected seed tubers were randomly distributed in the simulated plot, if not indicated differently.

Implementation. The source code was written in Pascal programming language, including a library of units with procedures for menu representation and graphical interfaces (P. Blaise, *unpublished*), and implemented for IBM and compatible PCs that have a math-coprocessor (Turbo-Pascal compiler, version 6.0, Borland International, 4585, Scotts Valley Drive, Scotts Valley, CA 95066). The software to be developed aimed at providing a simple and unsophisticated user interface representing a working version that allows for model parametrization and validation.

Results shown below were produced on an IBM PS/2 Model 70 386 personal computer and a compatible 486 PC. EPIVIT includes a printer interface. Graphics related to the model output were generated directly by EPIVIT, except for those related to multiple season simulations that were produced with the model outputs and a commercially available graphics package.

TABLE 4. EPIVIT's output (%) demonstration runs for contact- and aphid-transmitted viruses (means of 10 runs; random spatial distribution of infected and nonemerged seed tubers)

Virus type and variable	Mean[a]	Standard deviation[b]
Contact-transmitted (20% seed infection)[c,d]		
Efficiency of autoinfection (*tsi*)	81.6	...
Primary infection of plants (*pi*)	53.1	2.5
Tuber infection of primarily infected plants (tpi)	76.6	1.7
Harvest infection (*hi*)	57.1	2.4
Aphid-transmitted (19% seed infection)[c,e]		
Efficiency of autoinfection (*tsi*)	83.3	...
Primary infection of plants (*pi*)	32.5	7.3
Tuber infection of primarily infected plants (*tpi*)	90.4	0.7
Harvest infection (*hi*)	46.0	7.0

[a] Back-transformed means of arcsine-transformed percentages.

[b] Stochastic elements in EPIVIT's model code for calculating primary infection condition the variance of the model output.

[c] Emergence of infected and healthy seed tubers: 0.96. Temperature data of Imperial, Peru, 1988 (Fig. 2).

[d] Plot of 300 plants in 10 rows. Selected parameters for runs for contact-transmitted viruses were as follows: For *tsi*: $m_a = 2$, $n_a = 3$, $dr_a = 5$, $T_{min;a} = 5$, $T_{max;a} = 35$, $TH = 0$, $r_a = 11.17E\text{-}4$, $tsi_0 = 44.84$; for *pi*: $Lp = 70$, $m_{Lp} = 4.0$, $n_{Lp} = 5.0$, $dr_{Lp} = 3.0$, $m_b = 4.0$, $n_b = 3.0$, $T_{min;b} = 5$, $T_{max;b} = 35$, $C_{wr} = 100$, $C_{br} = 150$, $r_{mi} = 0.2$; for *P-time*: $m_p = 0.5$, $n_p = 0.5$, $dr_p = 5$, $T_{min;p} = 0$, $T_{max;p} = 35$; for susceptibility: $Su_c = 0.4$, $P_{max} = 1{,}030$, $Mri = 200$, $r_{mr} = 11.0E\text{-}3$.

[e] Plot of 216 plants in 6 rows. Hypothetical aphid data (Fig. 2). Selected parameters for runs for aphid-transmitted viruses were as follows: For *tsi*: $m_a = 1$, $n_a = 4$, $dr_a = 0$, $T_{min;a} = 5$, $T_{max;a} = 35$, $TH = 20$, $r_a = 29.88E\text{-}4$, $tsi_0 = 56.22$; for *pi*: $Lp = 70$, $m_{Lp} = 3.0$, $n_{Lp} = 4.0$, $dr_{Lp} = 3.0$, $q = 10.0$, $k_{pi} = 2.0$, $h_1 = 10$, $h_2 = 16.0$, $h_3 = 14.5$; for all aphid species: $m_{sp} = 2.0$, $n_{sp} = 4.5$, $T_{min;sp} = 5$, $T_{max;sp} = 35$, $M = 20$; for *P-time*: $m_p = 0.5$, $n_p = 0.5$, $dr_p = 5$, $T_{min;p} = 0$, $T_{max;p} = 35$; for susceptibility: $P_{max} = 1{,}030$, $Mri = 200$, $r_{mr} = 11.0E\text{-}3$. Rfg_{sp} and Af_{sp} as reported elsewhere (3).

RESULTS

Demonstration runs. The output of the implemented model is illustrated by using hypothetical parameter settings (Table 4, footnotes d and e). The generated output is artificial, i.e., does not represent any real situation and has only demonstrative purposes. Parameter values and input data sets do not relate to a particular season in a specific site but lie within roughly estimated boundaries for a temperate climate and a modern potato cultivar. The weather and aphid data used are presented in Figure 2 (aphid population 1). Aphid data are also hypothetical and represent yellow water trap catches, which may be the method most commonly used worldwide for studying aphid populations in potato fields. Parameter settings are not justified and verified in this section. The relative significance of their values compared to others may be estimated individually by considering the relative sensitivity of each parameter (see fine sensitivity analysis below).

Single season simulations. The average output of 10 runs of EPIVIT for contact-transmitted viruses is presented in Table 4 for a plot with seed tubers (20% infected) distributed at random on the (row × plants/row)-plot lattice. Means are reported, as the model output is variable due to the stochastic model code

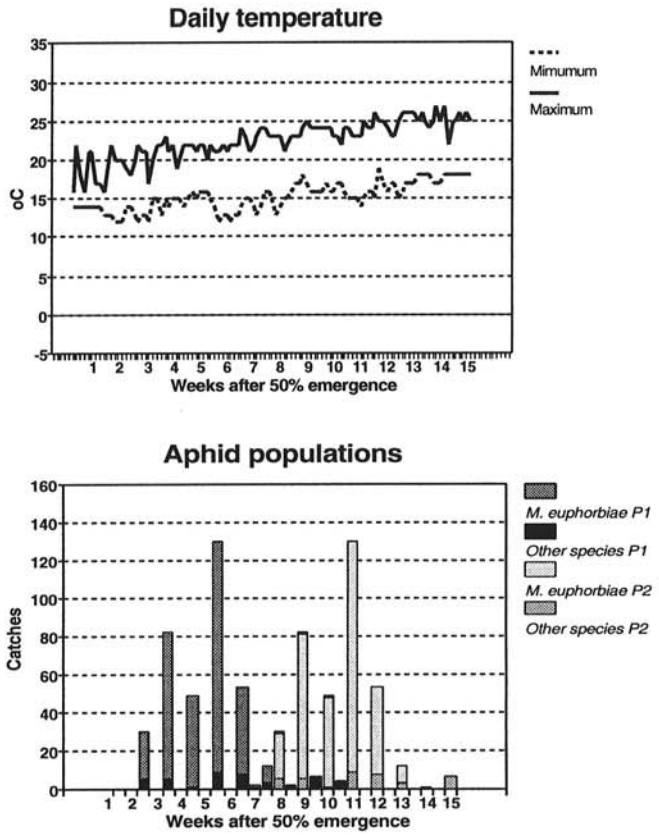

Fig. 2. Minimum and maximum daily temperature data of Imperial, Peru, 1988, and hypothetical yellow water trap catches of winged aphids (*P1* = population 1; *P2* = population 2) used for sample runs and sensitivity analysis of EPIVIT.

(eqs. 17 and 25). The increase of the percentage of infectious primarily infected plants in this plot is displayed for these 10 runs in Figure 3A.

Outputs of EPIVIT for aphid-transmitted viruses and the respective parameter values used are also shown (Fig. 3B and Table 4). Compared to the demonstration runs for contact-transmitted viruses, the high variability of primary infection of plants (standard deviation of 7.3%) is noteworthy. It conditions the variability of harvest infection (standard deviation of 7.0%). The stochastic model code (eq. 25) causes such variability of the model output. The reasons for the difference in variability of outputs between EPIVIT for contact- and aphid-transmitted viruses are discussed below.

Multiple-season simulations. The model output of five simulation runs is displayed graphically for contact- and aphid-transmitted viruses (Fig. 3C and D), assuming that seed infection in the first season is 2% and that the seed tubers for the following seven seasons are selected every season from the harvest of the simulated plot. The same parameter values were used as for single season simulations.

Coarse sensitivity analysis. Increasing seed infection from 2 to 50% greatly increases EPIVIT's output for infection by contact-transmitted viruses, i.e., primary infection of plants (*Pi,* or as percentage *pi*), tuber infection of primarily infected plants (*tpi*), and harvest infection (*hi*; Table 5). Also in reality, *tpi* responds positively to an increase of seed infection for contact- and aphid-transmitted viruses (3). Making *tsi* temperature insensitive and setting it to 100% does not affect the model's output as long as seed infection is low (2%) (Table 5). If seed infection is high (50%), however, *hi* becomes higher when using a temperature insensitive *tsi* than when using a temperature sensitive *tsi* (90.5 and 81.4%, respectively). Making *tpi* temperature insensitive and setting it to 100% shows the same overall effect as the corresponding manipulation of *tsi.*

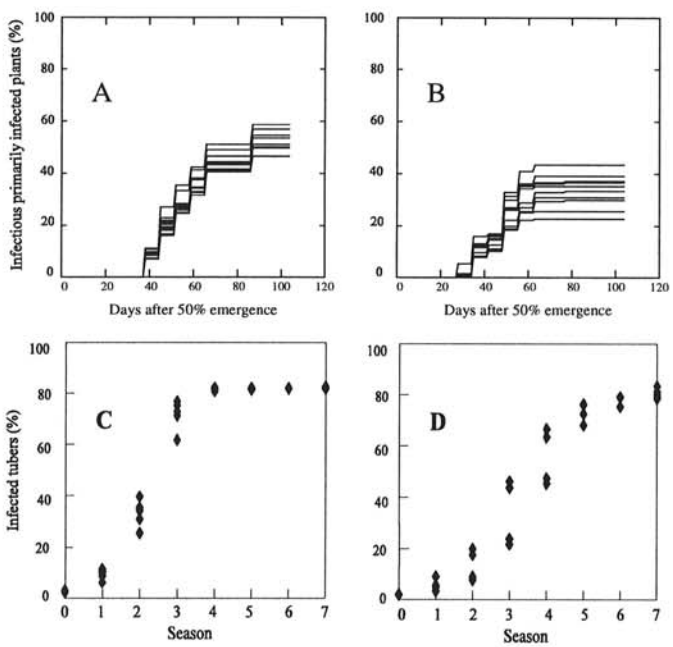

Fig. 3. Graphical output of successive demonstration runs with EPIVIT for contact- and aphid-transmitted viruses with historical weather data of Imperial, Peru, 1988, and hypothetical yellow water trap catches of winged aphids. **A,** Ten single-season simulations of the percentage of infectious primarily infected plants in a field for contact-transmitted viruses. **B,** Ten single-season simulations for aphid-transmitted viruses. **C,** Five multiple-season simulations for contact-transmitted viruses of the percentage of infected tubers in the harvest of a potato plot starting with 2% of infected seed tubers and without changing the seed in subsequent seasons. **D,** Five multiple-season simulations for aphid-transmitted viruses. Values used for the model parameters, and temperature and aphid data are presented respectively in Table 4 and Figure 2.

The tendency of the response of EPIVIT's output for aphid-transmitted viruses to seed infection increasing from 2 to 19% is similar to that for contact-transmitted viruses (Table 5). A later aphid immigration, simulated by replacing aphid population one with population two (Fig. 2) reduces *pi, tpi,* and *hi* (Table 5).

Model outputs (harvest infections) using a temperature-sensitive and a constant efficiency of autoinfection (*tsi* = 100%) were also studied over time. The model was set to the mode for multiple season simulations, assuming that the same tuber lot serves as a source for seed selection during consecutive seasons. One of the cases presented in Figure 4 corresponds to a high seed infection of 98% (case A) in the first season and another to a seed infection of 2% (case B). In the first case, harvest infection does not increase further. Because of *tsi,* it decreases to the level case B reaches after four seasons (82%). Without temperature-sensitive *tsi,* however (i.e., *tsi* = 100%), harvest infection increases to 100% at the end of the fifth season.

EPIVIT's output of runs on plots with low (2%) and moderate (approximately 20%) seed infection with a random spatial distribution of secondarily infected seed tubers was compared with the outputs obtained with a uniform spatial distribution of infected seed tubers. Twenty successive runs were executed. No significant difference was determined between means of arcsine-transformed percentages obtained for the state variables and output variables with random and uniform distribution of infected seed tubers. Overall, EPIVIT's response to these large changes in the model structure and input variables is biologically meaningful.

Fine sensitivity analysis. *Contact-transmitted viruses.* It was generally observed that the efficiency of autoinfection (*tsi*) and tuber infection of primarily infected plants (*tpi*) respond with a low percent change compared with the change in parameter values (Table 6). Tuber infection values on which the percentages of change were calculated were always higher than 70% for both variables with the utilized parameter values of m_a, n_a, dr_a, $T_{min;a}$, and $T_{max;a}$ (data not presented). High relative sensitivities in such a situation would be obtained only by a large change of v as a result of the parameter value change. Such large changes were generally not observed with the changes in parameter values

TABLE 5. The reaction of EPIVIT's output to large changes in the model structure or of input variable values (means of 10 runs; random spatial distribution of infected and nonemerged seed tubers)

Virus type and manipulated variables	Seed infection (%)	Variables[a] *tsi* (%)	*pi* (%)	*tpi* (%)	*hi* (%)
Contact-transmitted[b,c]					
Reference runs[d]	2	81.6[e]	12.3	73.6	10.7
Increased seed infection[d]	50	81.6	*49.1*	*82.8*	*81.4*
Efficiency of autoinf. (*tsi*) of 100%	2	*100.0*	13.1	73.9	11.7
	50	*100.0*	*48.4*	*83.0*	*90.5*
Tuber infection of *Pi* (*tpi*) of 100%	2	81.6	11.4	*100.0*	13.0
	50	81.6	*49.0*	*100.0*	*89.8*
Aphid-transmitted[b,d,f]					
Reference runs; aphid population 1	2	81.6[d]	5.1	89.9	3.6
Increased seed inf.; aphid population 1	19	81.6	*32.8*	*90.4*	*45.9*
Aphid population 2	19	81.6	*3.8*	*81.2*	*19.1*

[a] Numbers are back-transformed means of arcsine-transformed percentages. Numbers in italics are significantly different from the reference runs mean according to the LSD test ($P < 0.05$).

[b] Emergence of infected and healthy seed tubers: 0.98. Exemplary temperature data from Imperial, Peru, 1988 (Fig. 2).

[c] Plot of 300 plants in 10 rows. The model parameters were set to the values presented in Table 4, except $m_b = 4.5$, $n_b = 2.0$.

[d] Temperature-sensitive efficiency of autoinfection (*tsi*).

[e] The efficiency of autoinfection (*tsi*) does not respond to changes of the manipulated variables neither biologically nor according to the model code.

[f] Plot of 216 plants in six rows. Hypothetical aphid data (Fig. 2). The model parameters were set to the values indicated in Table 4. Rfg_{sp} and Af_{sp} as reported elsewhere (3).

applied. It was concluded that the accuracy of estimation of the respective parameters is sufficient for these state variables if parameter values lie within the range of changes used in this sensitivity analysis.

Pi showed a high relative sensitivity to changes of some specific parameters. With 2% seed infection, moderate changes of the variables *Mri*, C_{wr} (parallel with C_{br}), $T_{max;b}$, n_b, Lp, and dr_{Lp} yielded a considerable percent change of *Pi* (Table 6). If relative sensitivity of the model output for harvest infection (*hi*) was high, it could be explained by the high sensitivity of *Pi*. The relative sensitivity for *hi*, however, was always lower than that for *Pi*. The relation of the percent change of the parameters with the percent change in the respective output variable was observed to be nonlinear (e.g., an increase of C_{wr} of 10 and 50% yielded a relative sensitivity of −2.41 and −0.78, respectively).

The tendency of the model response to changes in parameter values is similar for model runs on a plot with 2 and 20% seed infection (same sign of the respective relative sensitivities with absolute values $\geq |0.3|$). In all cases, the relative sensitivity was lower in the plot with 20% seed infection. The signs of the relative sensitivities are biologically meaningful. Only in a few cases, in the plot with 2% seed infection, was the sign of the relative sensitivity not as expected biologically (e.g., relative sensitivity of +0.77 for a decrease of C_{wr} by 10%, which means less primary infection despite slightly earlier canopy closure).

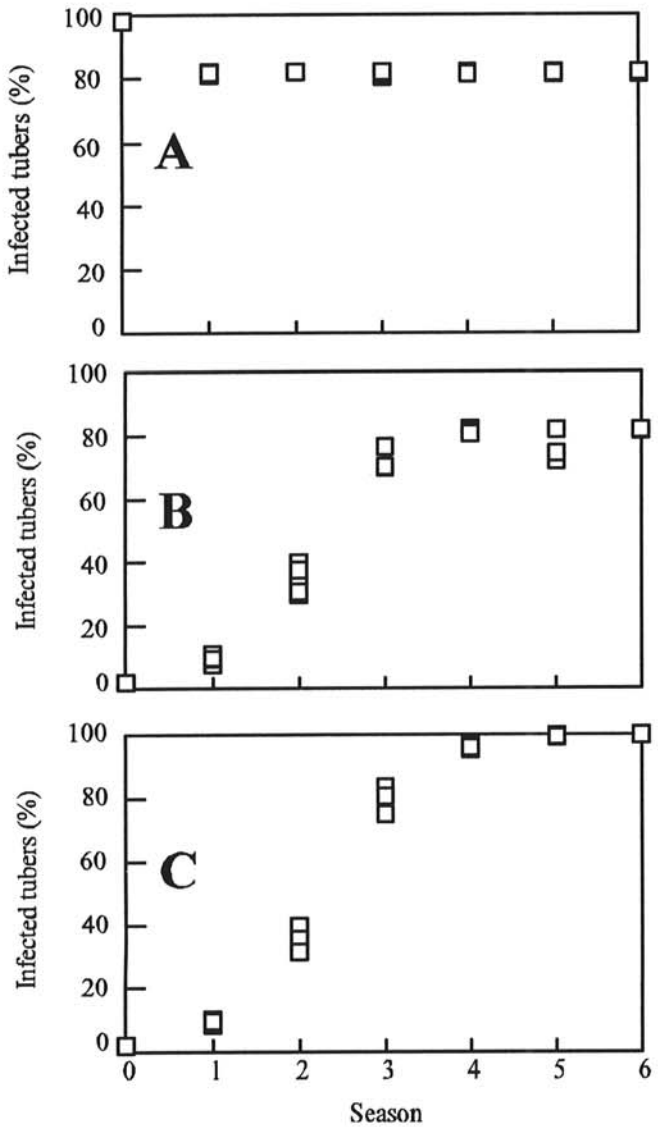

Fig. 4. Scatter plots simulating harvest infections (five infected tubers) produced with five successive demonstration runs of multiple-season simulations for six consecutive seasons. **A,** Seed infection of 98%, temperature-sensitive efficiency of autoinfection. **B,** Seed infection of 2%, temperature-sensitive efficiency of autoinfection; **C,** Seed infection of 2%, efficiency of autoinfection fixed at 100%. The values of the model's parameters used are listed in Table 4; temperature data are presented in Figure 2.

Aphid-transmitted viruses. EPIVIT for aphid-transmitted viruses reacts similarly to parameter changes as the version for contact-transmitted viruses. General observations coincide, such as those related to the relative sensitivity of *tsi* and *tpi*, and the difference in response of the model to plots with low and high seed infection (Table 7). In contrast to the version for contact-transmitted viruses, however, there are more parameters with a high relative sensitivity, which is best noted by comparing the relative sensitivities of both versions run on the plot with approximately 20% seed infection (Tables 6 and 7). Most of these parameters correspond to aphid behavior and characteristics related to their attraction by a trap color and the aphid's capacity for virus transmission (Table 7): the model output is sensitive to changes of the temperature range within which the relative efficiency is modeled (T_{max};sp), but also to changes of Af_{sp}, h_1 (together with h_2 and h_3), n_{sp}, and m_{sp}. Other parameters with a considerable relative sensitivity are the calibration parameter q, or are related to the host plant (*Mri*). The model's reaction to these parameters is over-proportional (relative sensitivity $> |1.0|$) even if the model is run on a plot with 19% seed infection. The model's sensitivity to changes in q is linear: its relative sensitivity is approximately the same regardless of the amount of change in parameter value. Such information is useful for the later calibration of the model to real data because it facilitates estimation of the precision and range of parameter values to be tested.

Implementation. A simulation run with EPIVIT for contact-transmitted viruses for a season with 104 days, from 50% emergence to senescence, requires 24 s on the 386 PC used, and 4 s on the 486 PC (both with mathematical coprocessors). EPIVIT for aphid-transmitted viruses uses less computer time (17 s with the 386 PC) because no spatial simulation is performed. The program is menu-driven via keyboard or mouse. A manual for this working version of EPIVIT that explains the menu options (many of them are self-explanatory) is presently not available. Parameters and input data can be changed interactively. Secondarily infected seed tubers may be distributed according to a historic field design, at random, or uniformly (24,25) onto the (row × plants/row)-lattice pattern of a field. Nonemerging seed tubers are randomly distributed onto all available positions.

A simulation run usually starts with the reading of the spatial position of secondarily infected and nonemerged seed tubers in a selected historic field. Weather and aphid data are then looked up. Two basically different options may be chosen in reference to the increase of infection with time: if the simulation for a single season is preferred, the increase of the number of infectious primarily infected plants during the season is displayed graphically. Multiple season simulations may be chosen, however, assuming that the harvest of one season serves as seed source for the next season. In this case, the increase of harvest infection during successive growing seasons is displayed. Results are also displayed numerically. A representation of the spatial pattern of nonemerged, healthy, latently infected, infectious primarily infected, nonemerged secondarily infected and harvested secondarily infected plants can be produced for contact-transmitted viruses at the end of a simulation run (Fig. 5).

The number of tubers produced by plants of different health states *NSi*, *NPi*, and *NHe* is not treated as a variable in the present version but as a constant set to 20, since no significant effect of the plant's health state on the tuber number has been observed in the Andes (L. Bertschinger, *personal observation*). The rate parameter of the logistic function for mature plant resistance, r_{mr}, is calculated by EPIVIT's actual implementation version to produce a symmetrical sigmoid decrease of Su_{mr} between initialization of mature plant resistance at *Mri* and maximal physiological age P_{max}.

DISCUSSION

Model structure. EPIVIT's structure is based on general knowledge of the mechanisms involved in potato virus epidemiology and the findings of studies of potato virus epidemics in different agroecozones of Peru (3). The coarse sensitivity analysis demonstrated a reaction of the model to changes in the model's structure which is compatible with this knowledge. Stochastic elements of the model determine the variability of some of its outputs. Other components, however, may also have probabilistic distributions (such as the settling behavior of aphids, for example), which may cause significant fluctuations in the outputs of the real system. EPIVIT's validation with experimental data must prove whether the variability produced by the model meets that of the real system or if other probabilistic elements need to be incorporated into the model code.

Input variables. Temperature and aphid population are the only input variables of the model, i.e., the degrees of freedom of the model are low. Other variables, such as humidity and precipitation, may have theoretical importance for the output of the state variables, but only in an indirect way through aphid population and plant growth on which virus transmission, multiplication, and translocation depend. EPIVIT uses trap catches as a parameter variable for the aphid population present above a field. It does not in its current version simulate either the population itself or plant growth and development in detail, which would require additional input variables such as precipitation and humidity. In view of the indirect involvement of additional input variables in the modeled pathosystem, it appears appropriate in a first step to consider only temperature as the principal climatic variable.

Fine sensitivity analysis demonstrated that with EPIVIT for aphid-transmitted viruses, a precise estimation of variables relating to aphid behavior (h_1 and h_2) and virus transmission efficiency of different species is essential. Experimental data need to confirm whether the concept of relating the transmission efficiency with the weekly average of daily means between temperatures at h_1 and h_2 is correct. Aphid activity, settling behavior, and behavior on the crop (alatae and apterous) in relation to virus spread is related to other variables such as wind (22), in certain cases precipitation (14), humidity, and others. A conflict between precision and simplicity of the model arises. EPIVIT's validation may indicate whether the model suffices for the demands of the problems for which it was developed.

It should be emphasized that the model can be applied easily to aphid catch data other than those from yellow water traps. By setting the attraction factors Af_{sp} to 1.0, EPIVIT may handle data from traps and methods that are unbiased, such as from suction traps, nets, or leaf counts.

The simulation of state variables and essential auxiliary variables. According to the sensitivity analysis of the model, hypotheses made for formulation of the simulation code for state variables appear to be reasonable. Validation of EPIVIT will provide further information on their correctness. Proof of their suitability, however, can be obtained only by analytical experimentation of the real biological system.

Two hypotheses in particular must be verified in this respect: first, the multiple-infection transformation for the description of

TABLE 6. Relative sensitivity of EPIVIT's state and output variables to changes in parameter values for contact-transmitted viruses (calculated on means of 10 runs per parameter combination[a]; infected seed tubers with random spatial distribution[b])

Parameter[c]	Unit	Parameter: Value Initial	Parameter: Value New	Parameter: Change (%)	Relative sensitivity[d]: Variables (seed infection 2%) *Pi*	*tpi*	*tsi*	*hi*	Variables (seed infection 20%) *Pi*	*tpi*	*tsi*	*hi*
Lp	P_{Lp}-days	70	80	+14	−1.16	+0.55	–[e]	−1.03	−0.21	+0.05	–	−0.09
			140	+100	−0.58	+0.08	–	−0.48	−0.13	−0.01	–	−0.07
m_{Lp}	–	4	3	−25	+0.29	−0.14	–	+0.15	+0.06	−0.01	–	+0.02
n_{Lp}	–	5	6	+20	−0.43	−0.02	–	−0.39	−0.01	−0.01	–	0.00[f]
$drLp$	°C	3	0	−100	+1.00	+1.00	–	+0.85	+1.00	+1.00	–	+0.50
		3	6	+100	−0.26	+0.06	–	−0.11	0.00	+0.01	–	0.00
m_b	–	4.5	3.5	−22	+0.81	+0.01	–	+0.78	−0.01	0.00	–	−0.01
n_b	–	2.0	3.0	+50	−1.19	−0.01	–	−0.18	0.00	0.00	–	−0.01
$T_{min;b}$	°C	5	3	−40	+0.19	−0.48	–	+0.08	0.00	−0.01	–	−0.01
$T_{max;b}$	°C	35	30	−14	+1.19	+0.05	–	+1.06	+0.02	+0.08	–	+0.07
C_{wr}[g]	P-days	100	110	+10	−2.41	+0.72	–	−1.62	−0.33	+0.05	–	−0.15
			150	+50	−0.78	+0.06	–	−0.65	−0.09	−0.04	–	−0.06
			90	−10	+0.77	−0.21	–	+0.52	−0.13	−0.07	–	−0.07
			50	−50	+0.04	−0.17	–	−0.11	+0.01	−0.05	–	−0.01
Su_c	–	0.4	0.8	+100	+0.17	+0.01	–	+0.15	+0.03	+0.01	–	+0.02
			0.2	−50	+0.79	0.00	–	+0.67	+0.09	+0.04	–	+0.12
Mri	P-days	200	190	−5	+6.60	−1.60	+0.04	+4.81	+0.48	−0.18	+0.04	+0.22
			130	−35	+0.66	−0.22	+0.04	+0.41	+0.05	−0.02	+0.04	+0.05
r_{mi}	bdd_b^{-1}	0.5	1.0	+100	+0.09	−0.01	–	+0.07	+0.02	0.00	–	+0.01
			0.1	−80	+0.65	0.00	–	+0.55	+0.14	+0.06	–	+0.10
m_a	–	2.0	1.0	−50	–	+0.08	+0.14	+0.20	–	+0.08	+0.14	+0.11
n_a	–	3.0	2.0	−33	–	+0.01	−0.08	+0.11	–	−0.01	−0.08	−0.05
dr_a	°C	5.0	3.0	−40	–	+0.03	+0.03	+0.18	–	+0.02	+0.03	+0.02
			7.0	+40	–	+0.02	+0.03	−0.21	–	+0.01	+0.03	+0.02
$T_{min;a}$	°C	5	3	−40	–	−0.04	−0.03	+0.13	–	−0.01	−0.03	−0.03
$T_{max;a}$	°C	35	30	−14	–	+0.04	−0.16	+0.26	–	+0.02	−0.16	−0.16
TH	bdd_a	3[h]	5	+67	–	−0.01	0.00	+0.01	–	0.00	0.00	+0.01
			10	+233	–	+0.01	+0.01	−0.02	–	0.00	+0.01	0.00

[a] Back-transformed means of arcsine-transformed relative sensitivities.
[b] Plot of 300 plants in 10 rows of 10 m. Emergence of infected and healthy seed tubers: 0.96. Exemplary temperature data of Imperial, Peru, 1988 (Fig. 2).
[c] The model parameters were set to the values presented in Table 4, except $m_b = 4.5$, $n_b = 2.0$, and $r_{mi} = 0.5$.
[d] Relative sensitivity: $(\Delta v/v)/(\Delta p/p)$, with Δv for the change in output variable v caused by a change of Δp in parameter p.
[e] Dashes indicate that the manipulated parameter has no relation to the respective variable according to EPIVIT's code.
[f] A value of 0.00 means that the relative sensitivity is <0.005 and >-0.005.
[g] C_{br} was changed proportionally to the changes of C_{wr}, from 150 to 165, 225, 135, and 75, respectively, in the order indicated from top to bottom.
[h] To allow the parameter change to be expressed as a percentage, TH was set to an initial value of 3 instead of 0 as for the other model runs (footnote c).

the relation of the probability of a plant becoming infected by a contact-transmitted virus using beta-beta-degrees as the independent variable; and second, the application of the monomolecular model to the relation of the efficiency of autoinfection to accumulated beta-degree-days.

The use of the negative binomial distribution for the simulation of primary infection by aphid-transmitted viruses may be controversial. First, the system for which EPIVIT was developed was limited to cases without virus input from outside a field for which the negative binomial distribution is the distribution of choice. Second, the negative binomial distribution provides more flexibility than the Poisson distribution commonly used for modeling purposes under the assumption that within the region of interest, events (e.g., aphid settling from outside a field) are randomly distributed in space. The negative binomial distribution approaches the Poisson distribution if the parameter k_{pi} is increased. An erroneous application of the negative binomial distribution instead of the Poisson distribution yields large differences between the simulated outputs of these distributions only if $k_{pi} < 2$ and if the simulated respective proportion (in EPIVIT's case *Pi*) is greater than 0.75 (26).

The model does not differentiate between persistently and nonpersistently transmitted viruses. No reference has been found that compares the modeling of two such pathogens with practical and quantitative model experimentation. Some of EPIVIT's parameter variables may have distinct meaning for both cases, such as the probability *pa(i)* as explained above. The assumptions made in relation to the use of the negative binomial distribution (which is the central concept of the model) are not incompatible ex ante with the two virus types. Again, future validation will elucidate whether the model concept needs to be more specific to each of the two virus types.

The simulation of the potato plant's physiological age may need improvement in the future. It has been argued (10) that an approach such as the one used could produce lower aging rates at high temperature compared to rates at low temperature (e.g., 36°C and 11°C). A linear relation to a rate increase to lethal temperatures has been proposed (10). Even if this seems

TABLE 7. Relative sensitivity of EPIVIT's state and output variables to changes in parameter values for aphid-transmitted viruses (calculated on means of 20 runs per parameter combination[a]; infected seed tubers with random spatial distribution[b])

		Parameter			Relative sensitivity[d]							
		Value		Change	Variables (seed infection 2%)				Variables (seed infection 19%)			
Parameter[c]	Unit	Initial	New	(%)	*Pi*	*tpi*	*tsi*	*hi*	*Pi*	*tpi*	*tsi*	*hi*
Lp	P_{Lp}-*Days*	70	80	+14	+1.89	+0.01	–[e]	+1.33	+0.16	0.00	–	+0.13
			140	+100	+0.11	−0.02	–	+0.05	+0.13	+0.10	–	−0.09
m_{Lp}	–	3	2	−33	+0.37	−0.01	–	+0.24	+0.10	0.00[f]	–	+0.06
n_{Lp}	–	4	5	+25	+0.07	−0.02	–	−0.01	−0.07	0.00	–	−0.05
dr_{Lp}	°C	3	0	−100	+1.00	+1.00	–	+0.72	+1.00	+1.00	–	+0.61
		3	6	+100	+0.51	0.00	–	+0.02	−0.03	0.00	–	−0.01
q	–	10	5	−50	+1.30	−0.01	–	+0.95	+0.95	0.00	–	+0.59
			1	−90	+1.10	+1.00	–	+0.79	+1.01	0.00	–	+0.63
			20	+100	+1.04	0.00	–	+0.74	+0.65	0.00	–	−0.80
kpi	$plant^{-1}$	2.0	0.2	−90	+0.09	+0.05	–	+0.07	−0.07	−0.03	–	+0.07
			0.02	−99	+0.38	0.00	–	+0.27	+0.62	0.00	–	+0.38
			20.0	+900	−0.03	−0.02	–	−0.03	0.00	0.00	–	−0.01
h_1[g]	–	10	9	−10	−1.07	−0.02	–	−0.68	−0.76	0.00	–	−0.52
			11	+10	+1.46	0.00	–	+1.01	−1.52	0.00	–	−0.91
m_{sp}[h]	–	2.0	3.0	+50	+1.55	−0.22	–	+1.09	−1.13	0.00	–	+0.71
n_{sp}[h]	–	4.5	5.5	+22	−2.55	0.00	–	−1.81	−1.37	0.00	–	−0.84
$T_{min;sp}$[h]	°C	5	0	−100	−1.19	+0.03	–	−0.79	−0.45	0.00	–	−0.27
$T_{max;sp}$[h]	°C	35	30	−14	−5.62	+0.10	–	−4.01	−3.88	0.00	–	−2.41
M	–	20	10	−50	+0.81	+0.01	–	+0.61	+0.74	0.00	–	+0.45
			2	−90	−0.08	0.00	–	−0.05	−0.01	0.00	–	0.00
Rf_{sp}[i]	–	0.1	0.2	+100	+0.09	0.00	–	+0.05	+0.68	0.00	–	+0.41
			0.5	+400	+1.20	0.00	–	+0.85	+0.32	0.00	–	+0.20
Af_{sp}[i]	–	0.588	0.688	+17	−2.65	−0.03	–	−1.70	−1.83	+0.01	–	−1.11
			0.988	+68	−1.18	−0.29	–	−0.81	−0.75	0.00	–	−0.47
Mri	*P-days*	200	190	−5	+6.18	+0.08	+0.04	+4.60	+1.09	0.00	+0.04	+0.64
			130	−35	+1.24	0.00	+0.04	+0.89	+0.10	0.00	+0.04	+0.10
m_a	–	1.0	2.0	+100	–	+0.06	+0.11	+0.29	–	+0.06	+0.11	+0.02
n_a	–	4.0	5.0	+25	–	−0.25	−0.37	−0.33	–	−0.25	−0.37	−0.29
dr_a	°C	3.0[j]	5.0	+67	–	−0.06	+0.05	+0.20	–	+0.03	+0.05	+0.02
$T_{min;a}$	°C	5	3	−40	–	−0.04	−0.10	−0.15	–	−0.04	−0.10	−0.29
$T_{max;a}$	°C	35	30	−14	–	−0.37	−0.65	−2.14	–	−0.36	−0.65	−0.35
TH	bdd_a	20	10	−50	–	+0.01	0.00	−0.20	–	0.00	0.00	+0.04
			0	−100	–	−0.01	−0.01	+0.05	–	−0.01	−0.01	−0.03
		20[k]	10	−50	–	−0.01	−0.01	+0.38	–	0.00	−0.01	−0.13

[a] Back-transformed means of arcsine-transformed relative sensitivities.
[b] Plot of 216 plants in six rows. Emergence of infected and healthy seed tubers: 0.96. Temperature data of Imperial, Peru, 1988 (Fig. 2). Hypothetical aphid data (Fig. 2).
[c] The model parameters were set to the values which are presented in Table 4.
[d] Relative sensitivity: $(\Delta v/v)/(\Delta p/p)$, with Δv for the change in output variable v caused by a change of Δp in parameter p.
[e] Dashes indicate that the manipulated parameter has no relation to the respective variable according to EPIVIT's code.
[f] A value of 0.00 indicates that the relative sensitivity is <0.005 and >-0.005.
[g] h_2 And h_3 were simultaneously changed from 16 and 14.5 to 15 and 13.5, and 17 and 15.5, respectively.
[h] These parameters were attributed to all species of the aphid population.
[i] The values correspond to the most important species of the applied population, *Macrosiphum euphorbiae*. Rfg_{sp} and Af_{sp} of a species were not allowed to become higher than 1.0 even after addition of the indicated difference.
[j] To allow the parameter change to be expressed as a percentage, dr_a was set to an initial value of 3 instead of 0 as for the other model runs (footnote [c]).
[k] Results of further runs with $m_a = 2.0$ and $n_a = 2.0$ (instead of 0.1 and 4.0, respectively) to obtain more information on the relative sensitivity to changes of *TH*.

biologically unlikely in the case of Andean cultivars, the above concern needs to be further evaluated. However, EPIVIT is not a plant growth model (even if it may be linked to one in the future). It attempts to incorporate a robust and flexible model for the advancement of physiological time for which the beta-function actually appears to be most suitable.

The relative sensitivities and the variability of EPIVIT's outputs. The obtained higher relative sensitivities in plots with low seed infection compared to plots with high seed infection match the theoretical reflections made during conceptualizing sensitivity analysis of EPIVIT, as mentioned above. Expectations meet model behavior, which verifies the implemented concept. Because of the lower relative sensitivities at higher seed infection levels, data from plots with moderate to high seed infection should be used for future, more accurate model calibrations.

The greater variability of outputs of the version for aphid-transmitted viruses and the larger number of parameters with a high relative sensitivity are conditioned by the nature of the stochastic elements of EPIVIT for contact- and aphid-transmitted viruses; the probabilistic selection of aphids belonging to different species with different relative efficiencies for virus transmission is opposed to the probabilistic selection of individual plants that are assumed to be uniform, i.e., not variable in their reaction to contact-transmitted viruses.

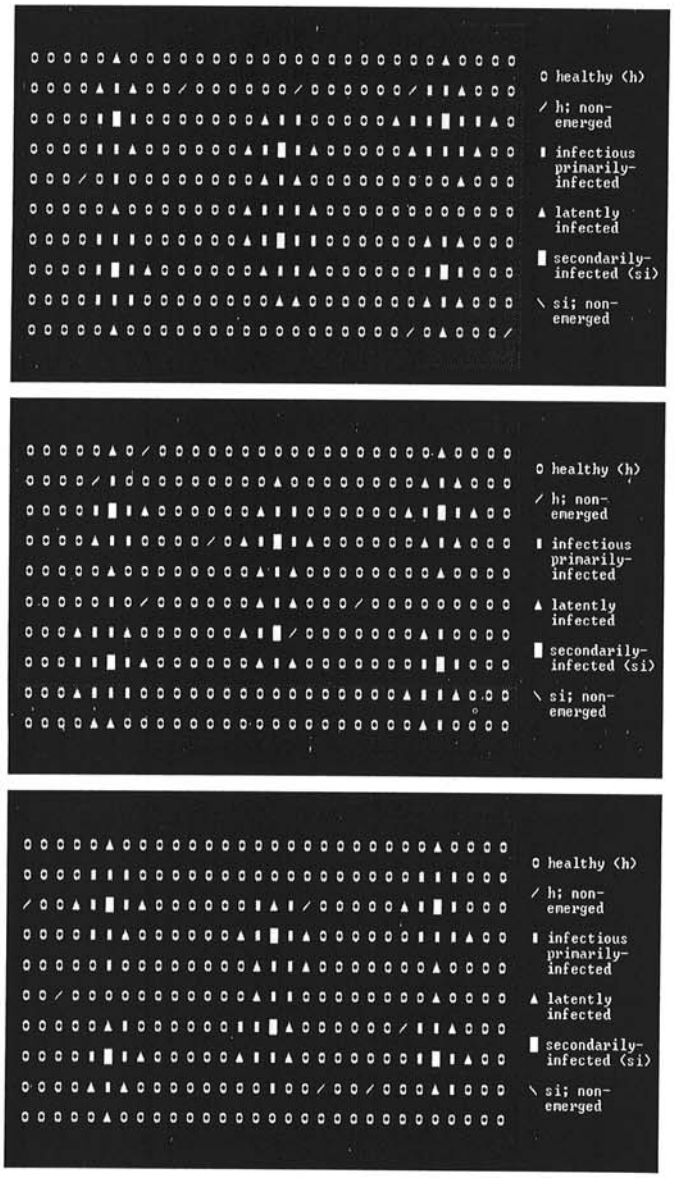

Fig. 5. Black-and-white screen captures of the spatial pattern in a simulated potato plot with 300 plants and 2% of secondarily infected seed tubers, generated by EPIVIT for contact-transmitted viruses. Healthy, non-emerged, infectious primarily infected, latently infected, emerged secondarily infected, and nonemerged secondarily infected plants are represented.

Prospects for model application. EPIVIT's development was aimed at an explanatory, biologically significant simulation model that responds to changes in temperature conditions and plant genotype, and that is adaptable to changing agroecological conditions. EPIVIT satisfies these conditions. Genotype-sensitive parameters are those for the simulation of the physiological time, susceptibility, and state variables. The model code allows for high model flexibility and adaptability by changing parameter values. The attempt was made to overcome, as far as possible, a conflict inherent to some extent in the objectives of EPIVIT's development: the conflict of simplicity with flexibility and explanatory character of the model. The latter leads in the case of potato viruses to a considerable resolution of the pathosystem, whereas simplicity would call for a low intricacy.

The purpose of the model construction was to obtain a tool for forecasting epidemics of the most important viruses of the potato crop in the Andes. How could EPIVIT simulate long-term (i.e., polyethic) epidemics in the Andes? If future validation demonstrates that the stochastic elements of the model are appropriate, predictions may be made by applying EPIVIT that are more realistic than those obtained from field experiments. Those are always subject to probabilistic events that might not be controlled by experimental design, and they call for many replicates until an acceptable mean result may be obtained. Repetitive model runs, however, with data sets of several seasons, may result in a forecast including a range of possible outputs distributed around the trend one is interested in. Forecasts may also be made for single seasons by repetitive runs with data of a particular season. This may be useful for seed production specialists in developed countries. The high variability and relative sensitivity of the model at low levels of seed infection may be a problem because of the need in these countries for precise prediction at low levels of seed infection. However, because of its stochastic elements, EPIVIT may possibly predict the range within which an output of the real system may lie. This range is relevant for seed production specialists wishing to determine haulm destruction dates and to demarcate zones of low risk of virus infection.

The incorporation of spatial components into EPIVIT's model code for contact-transmitted viruses permits model verification and validation that is innovative for quantitative plant disease epidemiology. If the spatial elements of the model are confirmed by validation, spatial pattern of disease spread may be modeled and better understood. For example, it can be tested whether harvest infection is not affected by changing the spatial distribution of infected seed tubers from uniform to random in a large plot, as is observed in small plots (300 plants). Furthermore, the individual plant approach of EPIVIT for contact-transmitted viruses will allow for using known spatial techniques, such as the calculation and comparison of gradients and the analysis of distance class frequencies (17), as innovative validation tools.

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