Systems Analysis and the Dynamics of Epidemics

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A system is a limited section of the real world. The limits are chosen in such a way that the environment of the system does not materially influence the system. Within a system, the variables show a variety of mutual interactions. In botanical epidemiology, the main components of the system are the host crop, the parasite (sometimes also the vector), and the microclimate.

Systems analysis is a method by which complex situations can be understood and described quantitatively. A systems analysis contains some of the following elements: measurement, analysis, and simulation. The variables of the system must be measured. The quantitative relations between the variables must be analyzed. Prior to or during the measurement and analysis phases, the basic concepts evolve which are to be applied in a model that embraces all variables and their interrelationships. Simulation is the construction of the model and the study of its behavior.

The aim of systems analysis is to describe a system as a whole, the holistic approach. Advances have been made in the fields of business administration research (4), ecology (1, 16), and recently in phytopathology (15). The present contribution is mainly inspired by the author's studies on cereal rusts.

Systems analysis.—Measurement and analysis.—This paper is a theoretical study. It is not concerned with the techniques of measuring. The measurements referred to are taken from the literature. Only two

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general remarks need be made. One is that the literature teems with quantitative data that can be utilized in a holistic approach. The second is that the choice of the variables to be measured is of crucial importance; e.g., in the case of spore germination the leaf wetness period is often more relevant than the relative air humidity at standard screen height.

Analysis of measurements in phytopathology is usually of the two-factor type; one biological, one physical. For example, the duration of the latent period of stripe rust (*Puccinia striiformis*) on wheat is mainly determined by the average ambient temperature. In The Netherlands, I studied this relation under field conditions (17). The result of the analysis is tentatively laid down in the equation:

1) NLPD $= (1005 + 11.3 \times \text{TEMP})/(2.5 + 5.65 \times \text{TEMP})$ where NLPD = latent period in days, and TEMP = average daily temperature in centigrade, calculated from 24 hourly observations at standard screen height.

The limiting condition is: $4 \leqslant \text{TEMP} \leqslant 19$. In California, Tollenaar & Houston (12) studied the effect of ambient temperature on the latent period of wheat stripe rust in growth cabinets under constant conditions. The similarity between the results (Fig. 1) can hardly be due to chance. Such similarities encourage me to use the data available from the literature, at least as a first approach.

In the more complex systems of animal ecology, a multifactor analysis is more appropriate. All variables measured are subjected to partial regression analysis in order to find significant relationships between variables. These relationships are studied in more detail. Such an approach is rare in phytopathology, but may yield interesting information.

Models in epidemiology.—Stochastic models are often used in medical epidemiology where emphasis is on large numbers of small populations; e.g., families.

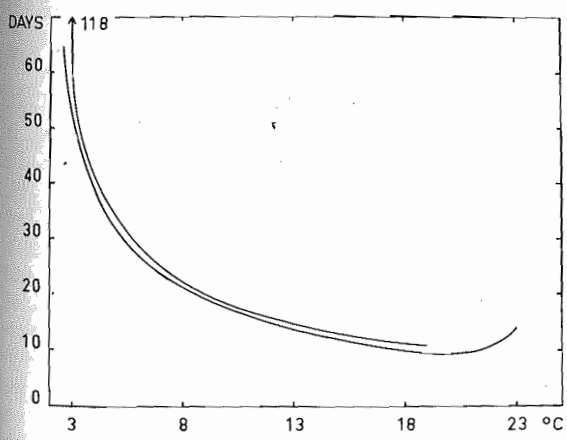

Fig. 1. Analysis of measurements: latent period in days (NLPD) versus temperature (TEMP) in *Puccinia striiformis* f. sp. *tritici*. Upper curve: The Netherlands (17), field observations, temperature is the average of hourly observations at standard screen height; see equation (1) in text. Lower curve: California (12), growth cabinet observations.

Botanical epidemiology uses deterministic models, in which each effect is unequivocally determined by known causes. The distinction between the stochastic and the deterministic approach is not always sharp. The logarithmic development in the early stage of an epidemic can be explained by deterministic, and also by stochastic, reasoning.

De Wit's (3) distinction between demonstrative and explicative models is useful. Large (7) used the cumulative normal curve as a model for the growth of a population of *Phytophthora infestans*; this model is purely demonstrative because it shows results but explains nothing. The logistic curve (equation 2) fits well with many kinds of experimental data, among others the epidemics of *Puccinia striiformis* f. sp. *tritici* (17). Nevertheless, the basic assumptions of the logistic model are rather arbitrary. Van der Plank (14) considers the good fit between observational data and model to be merely accidental. Therefore, the logistic model is also classified as demonstrative. In contrast with the foregoing the more elaborate models proposed by Van der Plank (14) (see equation 3) and Oort (9) are based on clear biological concepts: latent period, infectious period, and multiplication factor. These are explicative models. Nevertheless, even these models disregard many biological phenomena of potential importance, and their validity is restricted to some types of epidemics only.

Cereal rust epidemics grow approximately according to the logistic model described by the equation:

2) $\dfrac{dx}{dt} = r \cdot x \cdot (1 - x)$ where x = fraction of diseased host material, the total host material being given the value 1; t = time; and r = proportionality factor, named *apparent infection rate*. Van der Plank (14), who recognized that equation 2 has heuristic value only, proposed an improved model described by the equation:

3) $\dfrac{dx_t}{dt} = R_c\,(x_{t-p} - x_{t-i-p})\,(1 - x_t)$ where x_t = fraction of diseased host material at time t $(0 \leqslant x_t \leqslant 1)$: t = time in arbitrary units; p = latent period in the same time units; i = infectious period in the same time units: and R_c = factor of proportionality, called *corrected basic infection rate*.

These models, described by differential equations, are continuous, and, because the elementary time unit is infinitely small, nondiscrete. The curve describing the epidemic as a whole is found by integration of the differential equations. Though the differential equation models have furthered clear epidemiological thinking, they have two serious limitations. One is the difficulty of integrating equations containing more than four or five variables. The other is that the equation as a description of the epidemic is relevant only when the parameters R_c, i, and p are constant throughout the epidemic period. However, epidemiological parameters are usually far from constant.

A model is the simplified representation of a system. In a simulation model, one simplification is the hypothesis that change in the condition of the system is not continuous but discrete. Changes are momentaneous

and not too frequent. In this paper, the stepwise changes are supposed to take place once a day. Another simplification is the hypothesis that changes do not depend on each other, but only on the momentaneous condition of the total system. When the calculations begin, the system is in an initial condition characterized by the numerical value of the variables involved. The change from the initial condition to the resulting condition is realized by calculating the results of a great number of equations, each relatively simple, relating the variables to each other and to external information. The resulting condition of the system serves as the initial condition for the next cycle of calculations. Simulation models as described above are continuous but discrete. They are developed especially for calculation by digital computers. Simulation models have none of the disadvantages of the differential equation models. They are open models, permitting the addition of new variables at discretion. Furthermore, the program takes care of the integration.

Terminology of simulation.—Constants, variables, and auxiliaries.—All epidemiological information is contained in real numbers and/or integers which represent constants, variables, or auxiliaries. Constants are determined before a computer run is started; for example, the daily multiplication factor (DMFR). Variables change with time, like the severity of infection (XSEV). Auxiliaries are quantities introduced to simplify the equations; they could be eliminated by substitution into the rate equations. In this paper the epidemiological constants, variables, and auxiliaries are indicated by four letter codes in which the letters are related to the verbal concept represented.

Solution interval.—In simulation models, change is not continuous but discrete. The time interval between two successive changes (integration steps) is called the "solution interval". The time interval chosen here is 1 day, because many epidemiological processes show a distinct diurnal pattern.

Levels.—The condition of the system at time I is laid down in a series of variables caled "levels". Levels are the integrals calculated during the successive changes; they result from the differences between inflow and outflow accumulated over the preceding solution intervals. Codes for levels (and auxiliaries derived from levels) begin with the letter X. Latents (XLAT) and infectants (XINF) are levels. Levels are calculated from rates and preceding levels by fixed rules, the level equations: XSEV = XINF + XCTR where the severity XSEV is the sum of the number of infectants XINF and the cumulative total of removals (XCTR).

Rates.—A rate represents the flow velocity of items from one level to the next. In each solution, interval rates are calculated from preceding levels and/or from other rates computed in the same solution interval. Codes for rates begin with the letter R. "Occupation" (ROCC), "apparition" (RAPP), and "removal" (RREM) are rates. The calculation of rates follows fixed rules, the rate equations, e.g.: ROCC = CDMU × XINF where the rate of occupation ROCC equals the corrected daily multiplication factor CDMU × the level of infectants XINF.

Delays, boxcar trains.—In epidemiology, the infections taking place in a solution interval (ROCC) will lead to the appearance of sporulating lesions (RAPP) in a solution interval one latent period (NLPD) later. There is a numerical relation (in this case equality) between ROCC at time I-NLPD and RAPP at time I, with a delay of NLPD days in the time of occurrence. What happens today is not only determined by what happened yesterday, but also by events that took place in the earlier history of the epidemic. The possibility of incorporating historical relations in the model is one of the attractions of the simulation technique. Delays are programmed by storing the information in a special structure and retrieving the information when the desired delay has passed. This structure is named "boxcar train" (10).

Runs.—A run is the full sequence of reading the input data, going through all the equations of one iteration cycle needed to calculate the auxiliaries, rates, and levels during one solution interval, repeating this a preset number of iterations NDAY, punching the output in tabular and graphical form, and finally returning to the starting point in order to be ready for the next run. A run must be initiated by feeding the computer some initial information. The essential data, like the number of iteration cycles NDAY, the latent period NLPD, the infectious period NIPD, and the daily multiplication factor DMFR are read from a master card. Before each run, all rates, levels, and boxcar train contents are equaled to zero.

Input and output.—The input of every run consists of a set of initial levels, a few epidemiological constants, and some program control constants. The output of every run consists of a table of rates and levels and a graph of epidemiological variables against time.

Flow diagrams.—Flow diagrams illustrate the structure of the model and the interrelationships of the equations. A flow occurs into and out of a level. Rate equations, acting as valves in the flow channels, determine the rate of flow. Several types of flow systems (in a factory: materials, orders, and information) can be combined in one flow diagram, using a system of flow symbols (Fig. 2).

A simple model of an epidemic.—The model in epidemiological terms.—A simple model of an epidemic is based on the epidemiological concepts "latent period", "infectious period", and "multiplication factor" (9, 14). A spore of a pathogen arrives in a disease-free but susceptible crop. It establishes a lesion which after a latent period of NLPD days starts to sporulate and continues to sporulate during an infectious period of NIPD days. During the infectious period, the lesion constantly produces DMFR effective spores per day. DMFR is the daily multiplication factor [Van der Plank's (13) Corrected Basic Infection Rate]. Each effective spore in its turn establishes a lesion which after NLPD days will produce DMFR new effective spores per day during NIPD days, etc. This model ultimately leads to a logarithmic growth of the total number of infections. The graph representing the logarithm of the total number of lesions versus time shows

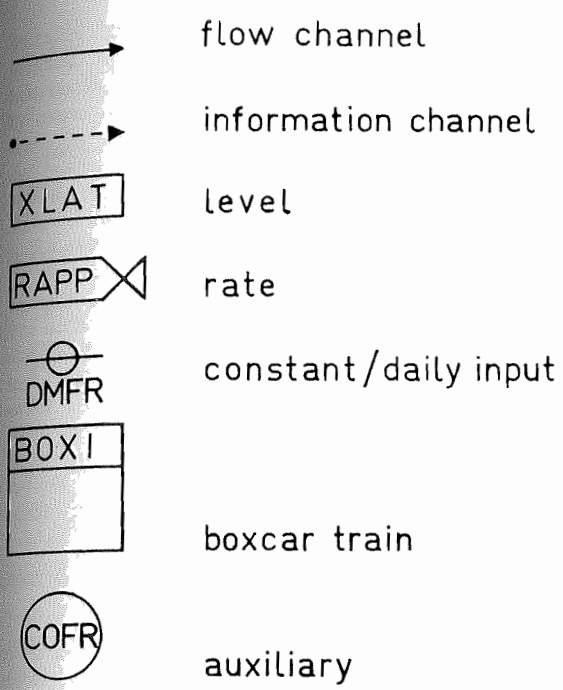

Fig. 2. Symbols in flow diagrams.

initially an undulating pattern, but gradually approaches a straight line (Fig. 8-A).

Infection sites.—The crop is thought to consist of a large but finite number of infection sites. All infection sites have equal areas and equal chances of infection. An infection site occurs under one of the following mutually exclusive conditions: vacant, latent, infectant, or removed (Table 1). The physical dimensions of an infection site roughly coincide with the reproductive unit of the parasite studied. In wheat, for example, the infection site for leaf rust (*Puccinia recondita* f. sp. *triticina*) would be ca. 4 mm^2 of leaf area.

Occupation, apparition, removal.—The biological processes of infection, opening of uredopustules, and death of pustules must be symbolized by operational concepts. Simplification is imperative. The infection process takes many hours, but is symbolized by the operational concept "occupation": an infection site becomes occupied. The process of pustule opening is operationalized by the concept "apparition". Apparition is momentaneous, and all apparitions of occupations from one age class take place at the same moment. Decline of the sporulation and death of the pustule is a gradual process. To make this biological process operational, it is symbolized by the term "removal". Removal is momentaneous and, usually, removals of occupations from one age-class take place at the same moment. Occupation, apparition, and removal are among the changes of the system, each with a rate of change (Table 1) expressed in infection sites per solution interval.

TABLE 1. Relationships between the variables of the simulation program

Conditions		Levels	Changes	Rates
Vacant		XVAC	Occupation	ROCC
Occupied	Latent	XLAT	Apparition	RAPP
	Infectant	XINF	Removal	RREM
	Removed	XCTR		

Time relations.—Change is thought of as discrete, and is calculated once a day; in a rust epidemic, infection is an essential process which usually takes place between 6 PM and 6 AM. A simplification accepted in this model is that all changes like occupation, apparition, and removal are momentaneous and occur at the same time. All changes occurring during the solution interval are projected to time 6 PM, the beginning of the solution interval, which runs from 6 PM of the preceding to 6 PM of the following day. As a consequence, a spore formed at time I will infect the host at time I + 1.

Effective spores.—Consider brown leaf rust of wheat. Most of the uredospores are wasted. Relatively few cause new infections; these are called "effective spores." The model deals with effective spores only; wasted spores are disregarded. One of the usual assumptions in epidemiological models is a constant number of effective spores produced per day per lesion, named "daily multiplication factor" (DMFR).

Latent period.—The latent period NLPD is the time interval between occupation and apparition. The latent period is measured in solution intervals (= days). The day of occupation is included in the latent period; the day of apparition is excluded from the latent period.

Infectious period.—The infectious period (NIPD) is the time interval between apparition and removal. NIPD is measured in solution intervals (= days). The day of apparition is included in the infectious period; the day of removal is excluded from the infectious period.

Flow diagrams.—The model symbolizes the epidemic process as a flow of items through successive levels. The items are the infection sites; the levels store the numbers of infection sites in the various epidemiological conditions: XVAC, XLAT, XINF, and XCTR. The flow of items can be visualized in a flow diagram (Fig. 3-5). At the beginning of the epidemic, the crop is healthy, all infection sites being vacant; they are stored in the level XVAC. At time I, some infection sites are still vacant, others are latent, some previously latent have become infectant, and finally a number of infectant sites are removed. Infection sites flow from the level XVAC through XLAT and XINF to level XCTR. The epidemic stops when all infection sites have flowed from XVAC into XCTR. XVAC is the number of vacants at time I; XLAT is the number of latents at time I; XINF is the number of infectants at time I; and XCTR is the cumulated total of removals at time I.

In every solution interval, items (infection sites) flow from one level to the next. The number of items per solution interval flowing from one level to the next is called the rate of flow (Table 1). Rate equations

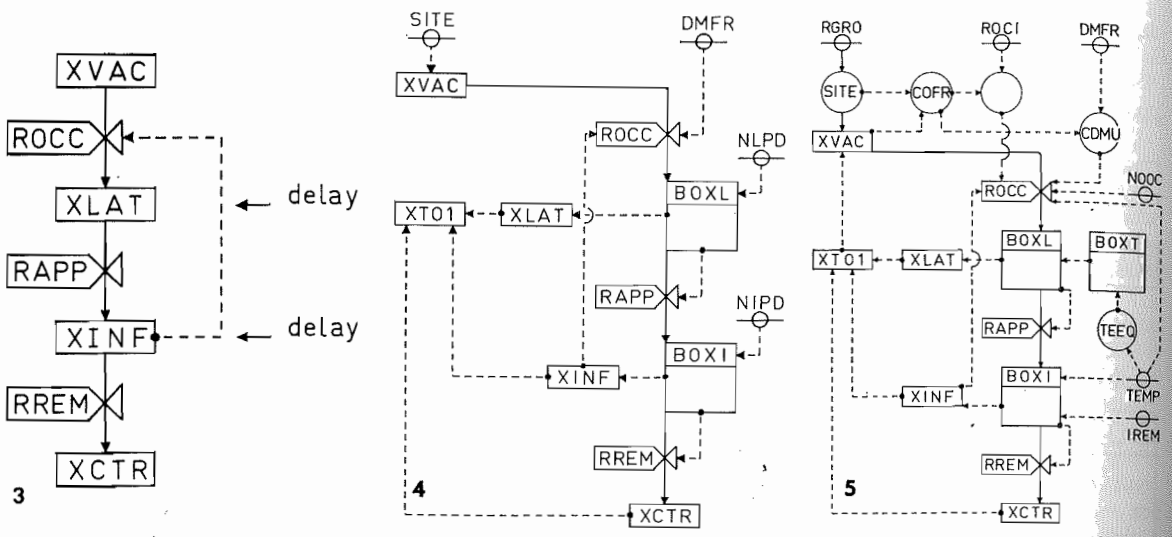

Fig. 3-5. Models of epidemics: **3)** a simple model, elementary flow diagram; **4)** a simple model, detailed flow diagram; and **5)** advanced model, detailed flow diagram.

can be visualized as valves regulating the flow through a pipeline system. ROCC is the rate of occupation; this is the number of infection sites changing from vacant into latent during the solution interval. RAPP is the rate of apparition; this is the number of infection sites changing from latent into infectant during the solution interval. RREM is the rate of removal; this is the number of infection sites changing from infectant into removed during the solution interval.

Levels and rates are interconnected by a network of information channels. The vital information channel in the epidemiological model is the feedback from the level of infectants XINF to the rate of occupation ROCC. In epidemiological terms, the amount of infection is a function of the number of lesions producing inoculum. Delays must be located in the flow diagram (Fig. 3) which gives the elementary flow diagram of our simple model.

The detailed flow diagram of the simple model (Fig. 4) shows the complete picture of the flow channel, the information network, and constants. The flow channel now contains two delays effectuated by means of the boxcar trains BOXL and BOXI for latents and infectants, respectively. Their summarized contents give the information for XLAT and XINF. The information channels needed to calculate the rates are indicated. Usually, only the cumulative total of infections (XTO1) is calculated (9, 14). This is the total number of occupations, the sum of latents, infectants, and cumulative total of removals: XTO1 = XLAT + XINF + XCTR.

Dynamic structure of an epidemic.—The simple model of Fig. 4 does not contain more than the dynamic structure of an epidemic in its most elementary form. This structure was accepted as a working hypothesis without questioning its truth. The dynamic structure discussed so far approximates to reality in the case of air-borne plant pathogens of the local lesion type, like *Puccinia recondita* and *P. graminis;* however, the structure is too rigid. Before each run, the variables NLPD, NIPD, and DMFR are set at fixed values which are, during the run, unchangeable. In nature, epidemics do not develop according to such a rigid pattern. Variations occur in response to changes in the biotic or abiotic environment. These variations can be considered to be superimposed on the existing dynamic structure which continues to operate. Examples will be given later in an advanced model of an epidemic.

Van der Plank (14) showed that every epidemic has an inherent max speed. The dynamic structure will, in the following, represent the epidemic at max speed or, in other words, an epidemic under opt environmental conditions. Any deviation from the opt environment tends only to slow down the speed of the epidemic.

An advanced model of an epidemic.—Variation of the dynamic structure.—An advanced model is proposed to overcome the limitation of the simple model caused by the rigidity of its dynamic structure. The simulation technique ideally meets this requirement because a program can be written to make logical decisions based on internal or external information.

An epidemic stops when all available host material is diseased. Usually, the epidemic has already slowed long before all host material becomes diseased. This fact is recognized by the term $(1 - x)$ in equations 2 and 3. The term $(1 - x)$ is called the "correction factor". It indicates that the speed of the epidemic is proportional to the healthy fraction of the host material, the total amount of host being given the value 1.

External information is needed to modify the dynamic structure of the epidemic. The latent period NLPD depends on temperature. The infectious period NIPD may be shortened or interrupted by very low temperatures. The daily multiplication factor is influenced by the amount of dew or by fungicides. For every effect, a short program can be written and woven through the main program. Such short programs will

be called "features". As the shortest time unit of the program, the solution interval, is 1 day, the external information must be presented per day and digested by means of "daily input" features. External information may contain data on the host, the parasite, the environment, etc.

The correction factor.—Many infections may have occurred at the place of a lesion, but usually the lesion is the result of one effective spore only. Because we are interested in effective spores, the infection site coincides with a lesion in the local lesion diseases. For each host-pathogen combination, a standard lesion size can be roughly estimated. Uredopustules of brown leaf rust on wheat (*Puccinia recondita* f. sp. *triticina*) cover about 4 mm^2 of leaf area. Lesions of yellow stripe rust on wheat (*P. striiformis* f. sp. *tritici*) are quite variable in size because of their semisystemic growth, but as a first approximation, 10 mm^2 is adequate. Black stem rust on wheat (*P. graminis* f. sp. *tritici*) has an approx lesion size of 10 mm^2: ca. 1,000 uredopustules/stem (6).

The total amount of available host material is symbolized as the total number of available infection sites (SITE). At the beginning of each run, SITE is read from the master card to serve as internal information during the run. SITE depends on size of the infection site (standard lesion size), the number of infection sites per plant, the number of plants per unit area, and the total crop area under consideration. Admittedly, each of these items is variable, but estimates can be made for specific cases. The total crop area may be the area of an experimental plot (e.g., 20 m^2), a field (e.g., 2.5 hectare), or the wheat area of a country (e.g., 120,000 hectare of wheat in The Netherlands). For wheat under Dutch conditions, reasonable estimates are: 320 stems/m^2 and a leaf area index (LAI) = 4 at the peak of the vegetation period. These assumptions lead to the following estimated infection sites per hectare: *P. graminis,* 3.2×10^9; *P. recondita,* 1.0×10^{10}; and *P. striiformis,* 4.0×10^9. In the following, only fields measuring one hectare with SITE = 5×10^9 will be considered.

The correction factor COFR is constructed by a feed-back from the total number of occupations XTO1 to the number of vacants XVAC:

$$XVAC = SITE - XTO1$$

$$COFR = XVAC/SITE = (SITE - XTO1)/SITE = 1 - (XTO1/SITE) = 1 - x$$

The derivation shows that COFR is identical to the correction factor of the logistic equation. The information feedback needed to construct COFR is shown in Fig. 5.

The corrected rate of occupation.—The correction factor COFR and the daily multiplication factor DMFR are combined into an auxiliary variable, the corrected daily multiplication factor CDMU. CDMU is multiplied by the number of infectants XINF to obtain the corrected rate of occupation ROCC (Fig. 5); CDMU = COFR × DMFR; ROCC = CDMU × XINF.

Daily input features.—A distinctive character of the simulation model is the possibility of introducing a daily input feature. The quantitative effect of the environmental variations must be expressed in numerical changes of one or more of the variables of the dynamic structure. The time dimension of all sudden or gradual variations must be expressed in solution intervals. In every solution interval, the program goes through a DO-loop which permits a card with external information (daily input) to be read.

There are four types of daily input features differentiated according to their way of handling the information. The additive features simply add or subtract quantities to or from levels. The on-off features determine whether the program follows the main or an alternative pathway. In a tabulation feature, the information refers to a position in a table, presented as initial information at the start of the run. The equation feature uses the external information to calculate a variable by means of a mathematical equation.

"No-occupation" feature (NOOC).—In many fungal diseases, infection depends on the presence of free water on the leaf surface, preferably in the form of dew. The working hypothesis, "if dew, then maximum infection; if no dew, then no infection" leads to an on-off feature. The external information NOOC = 1 means that no occupation will take place (ROCC = 0) no matter what value of ROCC has been calculated by the dynamic structure. NOOC = 0 means that the normal procedure is to be followed. Internal information can also govern the no-occupation feature. Temperature data as external information could lead to nonoccupation below a preset min or above a given max temperature. The preset temperature limits serve as internal information. When temperatures are so low that infectants are removed (sporulating lesions being winter-killed), the temperature is also too low for infection: NOOC is switched to 1.

In many cases, environment leads to a rate of occupation ROCC which is neither zero nor max, but intermediate. When a simple mathematical relation between the external factor and ROCC cannot be found, the tabulation feature may help. For example, a protective fungicide reduces spore germination considerably during a couple of days, then gradually degrades. The percentage reduction of spore germination found in experiments during a number of days after treatment could be stored in a table. The tabulated information is retrieved by entering the corresponding code number in the daily input cards on the days following fungicidal treatment. The NOOC column of the daily input card with its 10 positions can be used in a tabulation feature governing NOOC.

"Infectants removed" feature (IREM).—IREM is again an on-off feature. The external information must be provided by the user. If IREM = 0, the program follows its normal course. When IREM = 1, all infectants will be removed. Removal of infectants by means of external information is not an elegant method. The feature can also be governed by internal information; e.g., a preset temperature limit switching IREM from 0 to 1. In *P. striiformis* of wheat, two temperature limits may occur. Below −5 C, sporulating lesions are winter-killed; over 25 C, some cultivars show a heat-

induced resistance with sharply reduced sporulation (17).

"Additional occupations" feature (ROCI).—Assume that a cereal rust epidemic is developing from local endemic inoculum according to the known dynamic structure. Suppose that fresh inoculum is blown in by the wind from a far source. This inoculum from outside consists of a certain number of effective spores per solution interval, ROCI, which must be taken into consideration by means of an additional occupations feature. This is an additive feature consisting of a quantified signal ROCI, the rate of additional occupation at the day under consideration, all infection sites being vacant. The latter condition usually is not satisfied; therefore the correction factor COFR is applied. The corrected rate of additional occupations is added to the rate of occupations ROCC of the dynamic structure. The resulting sum is then used as an input to the dynamic structure; ROCI = COFR × ROCI; ROCC = ROCC + ROCI. There is a timing problem. The spore invasion leading to ROCI may have taken place during the preceding day(s), but should be booked in the solution interval (running from 6 PM to 6 PM) in which the occupation takes place.

"Growth" feature (RGRO).—The growth feature is a quantified signal RGRO, indicating the number of new and vacant infection sites created during the day under consideration; SITE = SITE + RGRO.

"Variable temperature" feature (TEEQ, TEMP).—Suppose the latent period at a constant temperature of 10 C is 20 days; 1 day at 10 C then represents 1/20 of a latent period. This inverse of the latent period is called the "temperature equivalent" (TEEQ). The unit of time is the solution interval (day). When temperature varies during the latent period, every day produces its own temperature equivalent. After occupation, the cumulative sum of the temperature equivalents is determined daily. When the sum is one or

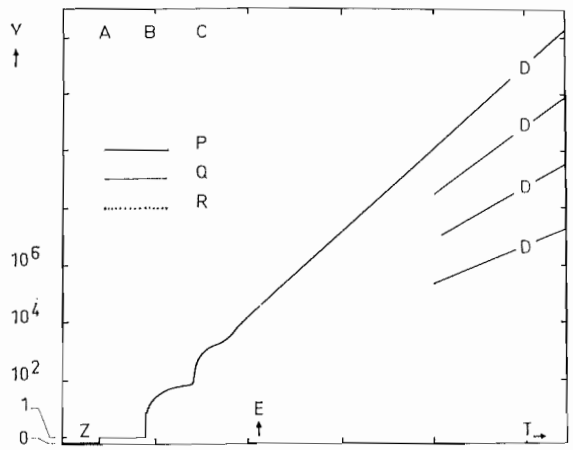

Fig. 6. Symbols used in the graphs of the simulated epidemics, Fig. 7 to 15: (A) latent period NLPD; (B) infectious period NIPD; (C) daily multiplication factor DMFR; —, one of the previous variables is specified in D; (D) specification of the variables when depicted for more than one numerical value; (E) duration of an interruption period; (P) severity curve XSEV; (Q) curve for the cumulative total of removals XCTR; (R) infectants curve XINF; (T) abscissa, time in 10-day units; (Y) ordinate, number of infection sites occupied on a logarithmic scale (zero line at the bottom of the graph, units represent 100-fold increase with base line for the number); (Z) all simulated epidemics start with the influx of one effective spore into a disease-free crop; ↑ = first day of an interruption period; ↓ = day of influx of inoculum.

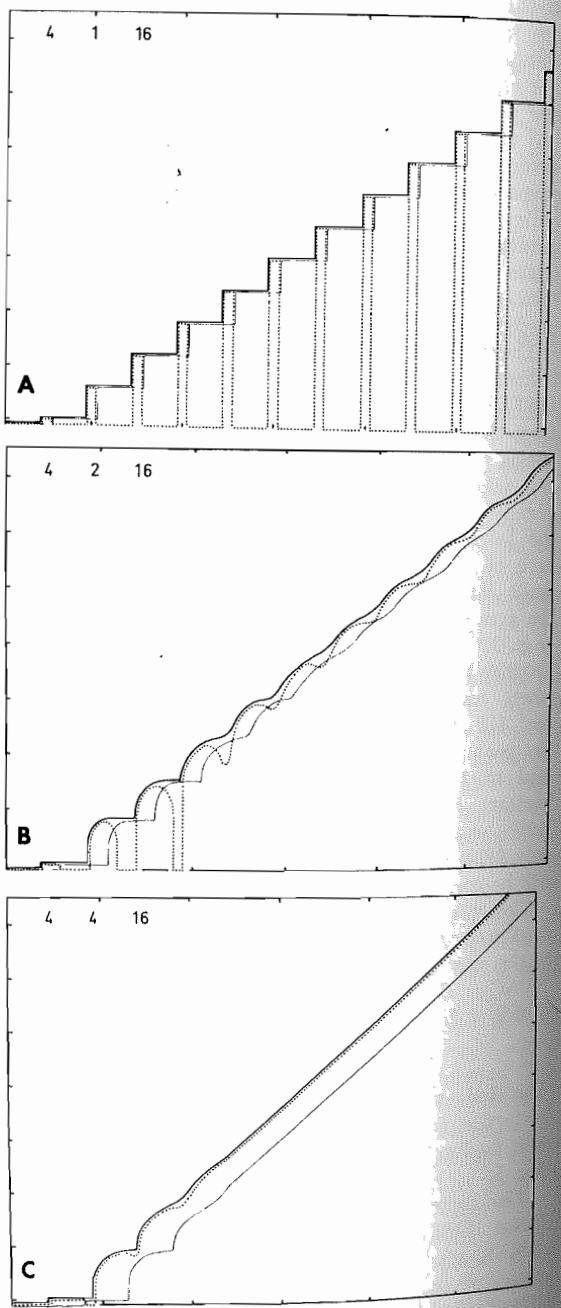

Fig. 7. Simulated epidemics. **A)** Effect of an infectious period of 1 day, staircaselike severity curve and interrupted infectants curve; **B)** 2 days, undulating severity curve, three interruptions in infectants curve; and **C)** 4 days, smooth curves.

more, the latent period comes to an end, and latents become infectants. The hypothesis of the additivity of temperature equivalents is not more than a useful approximation. It has shown its value in the analysis of yellow rust epidemics (latent period) and in the forecasting of insect flights (diapause period).

A special delay in the form of a boxcar train (BOXT) is used to store information on temperature equivalents; BOXT is parallel to BOXL. When a constant latent period is needed, part of the variable temperature feature is bypassed. TEEQ can be punched on the daily input cards. Alternatively, it can be calculated from the original temperature data, punched on the cards, by means of equation (1) and the equation: TEEQ = 1/NLPD.

RESULTS.—*The dynamic structure.*—Results obtainable with the advanced model are demonstrated by means of imaginary epidemics and the use of fictitious parameters. The parameters chosen approximate the range found in potato late blight (*Phytophthora infestans*) and the wheat rusts (*Puccinia graminis, P. recondita,* and *P. striiformis*). The results are given as graphs, redrawn from the computer-made originals, with short comments. Figure 6 shows the symbols used in the graphs.

The dynamic structure of the model is illustrated in Fig. 7-A. A latent period NLPD = 4 alternates with an infectious period NIPD = 1. The resulting severity XSEV (= XINF + XCTR) follows a typical staircase pattern on the logarithmic scale, each step representing a multiplication by DMFR = 16. NIPD being 1, an infectious lesion is removed after 1 day. The change does not affect the disease severity XSEV because this variable includes both living and dead lesions. The curve for the cumulative total of removals XCTR follows the severity line with a delay of 1 day (NIPD = 1).

In Fig. 7-B, NIPD = 2. The staircase pattern now becomes an undulating curve which gradually flattens out. The change is due to the overlap of infectious periods, leading to only three periods with XINF = 0. When NIPD = 4 (Fig. 7-C), the severity line becomes a straight line after only three or four recognizable infection waves. This is in complete accordance with theory (9, 14), and with experimental data obtained by Zadoks (17, Fig. 34.2) with *P. striiformis* on wheat.

A change of the latent period NLPD (Fig. 8-A) mainly leads to a change in the speed of the epidemic (= average slope of the severity curve). Multiplication of NLPD with a factor q leads to the exponentiation

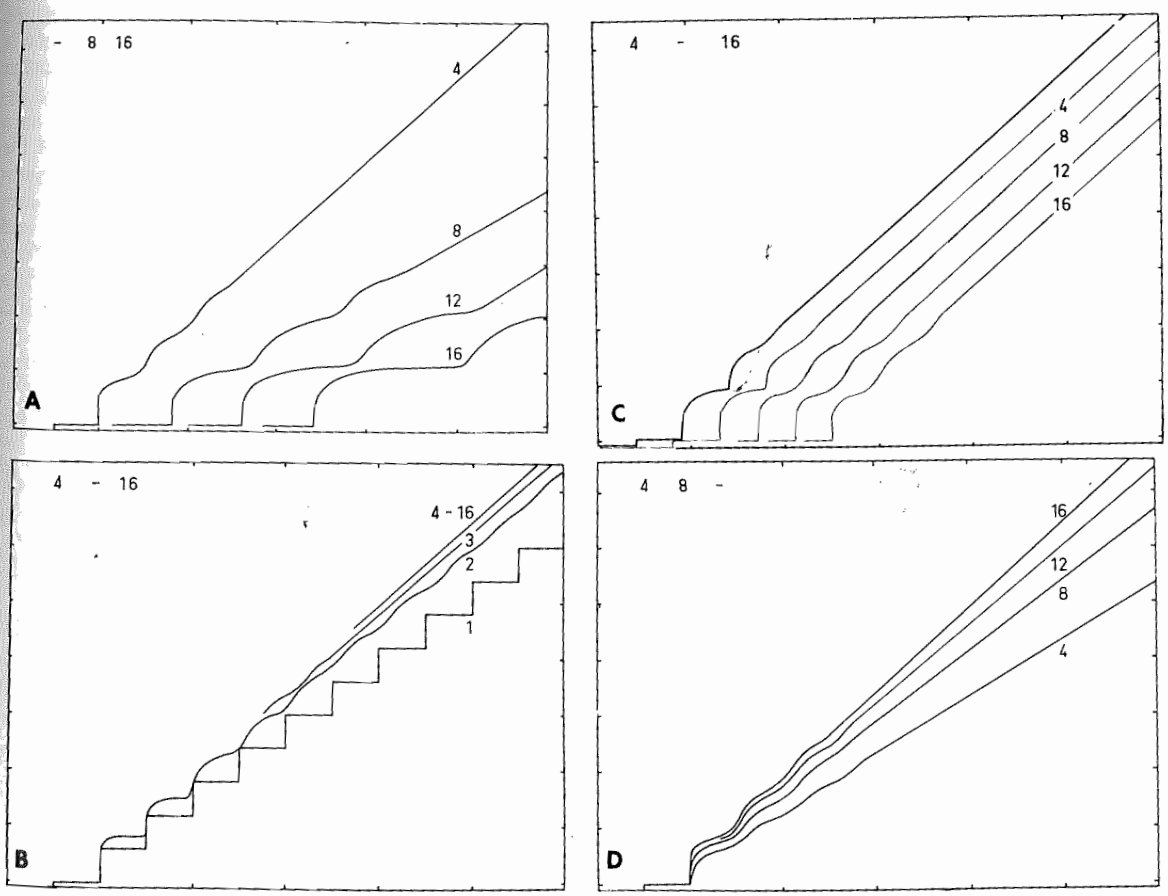

Fig. 8. Simulated epidemics. **A)** Effect of various latent periods on the severity curve; **B)** effect of various infectious periods on the severity curve and **C)** on the curve for the cumulative total of removals; **D)** effect of various daily multiplication factors on the severity curve.

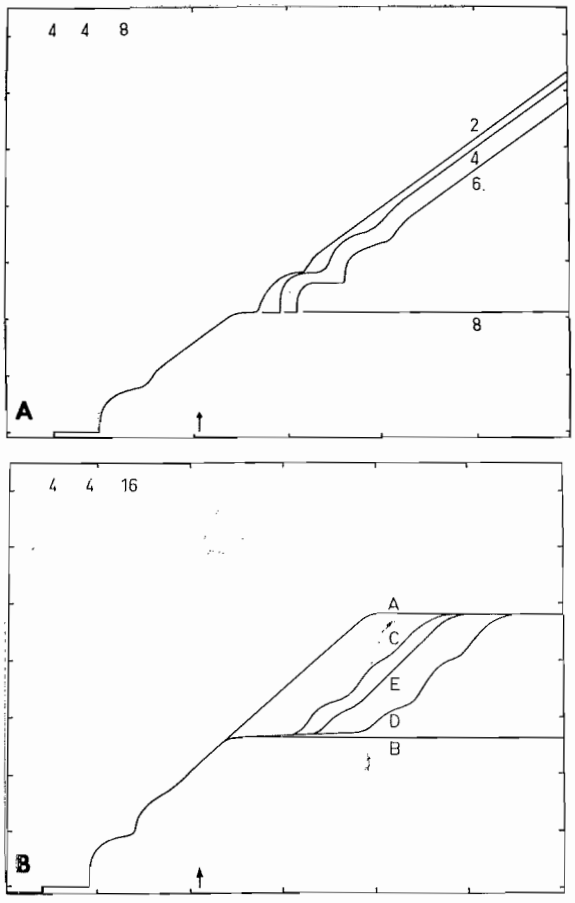

Fig. 9. Simulated epidemics. **A)** Effect of the inhibition of infection (NOOC = 1) on the severity curve during interruption periods of 2 to 8 days; **B)** effect of reduction of the severity curve; e.g., by a protective fungicide, applied at day 21. SITE = 5×10^9. Line A, no reduction; Line B, complete inhibition of infection during 8 days; Line C, reduction of 99% during 8 days; Line D, reduction of 99% during 15 days; Line E, reduction of 99% during 8 days, then erosion of fungicide; day 9, 95%; 10, 90%; 11, 75%; 12, 50%; 13, 25%; 14, 10%; 15, 5%.

of severity XSEV after a given number of days with approx the power q^{-1}. A mathematical expression of the relations between NLPD, XSEV, and other variables of the simple model has been developed by Corsten (2).

The effect of an increasing infectious period NIPD is somewhat surprising. If NIPD changes from 1 to 4, the severity curve straightens considerably (Fig. 8-B) and the speed of the epidemic is somewhat increased. The acceleration of the epidemic decreases with increasing NIPD and is practically nil when NIPD exceeds 4. Larger values of NIPD only lead to equally larger delays between the severity XSEV and removals XCTR curve (Fig. 8-C).

A change of the daily multiplication factor DMFR leads to a commensurate increase in the speed of the epidemic (Fig. 8-D).

The daily input features.—The effect of the inhibition of occupations by an external cause (NOOC = 1) depends on the duration of the disturbance. When the inhibition period is 1 or 2 days only, the severity curve shows a slight ripple but no noticeable retardation under the *P. infestans*-like conditions of Fig. 9-A. An inhibition period of 4 days leads to a delay in the development of the epidemic of ca. 1 day, an inhibition period of 6 days to a delay of about 5 days. When the inhibition period equals or exceeds the sum of latent and infectious period (NLPD + NIPD), the epidemic comes to a stop. Apparently, short interruptions of the epidemic process have little effect on the terminal severity.

The effect of the disturbance can be observed up to at least two latent periods afterwards (Fig. 13). Van der Plank (13) called this the "memory effect", which gives each epidemic its own individuality.

Usually, infection is not completely reduced to zero. When there is a dry spell with little dew, or when a protective chemical is applied the infection can be temporarily reduced, e.g., to 1% of its normal value. Figure 9-B gives the result of reduction periods of 8 and 15 days; the severity curve shows delays of approx equal values. When the protective chemical gradually disappears between the 8th and 15th day after application, its effect is rapidly lost.

Among the addition features is the ROCI feature, which represents the influx of inoculum from an outside source. The severity curve of Fig. 10 without influx shows the effect of a limited number of available infection sites, $0 < \text{SITE} < \infty$. The graph is based on a 1-hectare crop with SITE = 5×10^9. The effect of the influx of wind-borne inoculum, expressed as the number of effective spores, can be seen at various inoculum densities: ca. one effective spore/100 m^2, /1 m^2, and /0.01 m^2, respectively. The infection took place on day 21 when the disease severity of the endemic was somewhat below 2,000 lesions/hectare, or about 0.0001%.

Figure 11-12 represents the hypothetical case of a stripe rust (*P. striiformis*) epidemic on wheat, started by a single effective spore, and developing at 18 C except for the indicated interruption period. The variable temperature feature relates the temperature TEMP to the latent period NLPD. When $4\,C \leqslant \text{TEMP} \leqslant 19\,C$, equation (1) is used. At temperatures below 4 C or over 19 C the latent period is, somewhat arbitrarily, fixed at 60 and 11 days, respectively. The temperature is used as internal information to realize two hypotheses by means of the NOOC and IREM features. Firstly, no infection takes place below 0 C; secondly, infectious tissue is killed at temperatures −5 C or lower. Figure 11 gives the severity curves XSEV; Fig. 12 shows the curves of the infectants XINF.

Resistance and simulation.—The simulation model is relevant to the problem of partial resistance, but applies only to the phenotypic expression of resistance. Partial resistance is due to one or more of the following components (J. C. Zadoks, *unpublished data*): infection ratio (incorporated in DMFR); sporulation rate (incorporated in DMFR); latent period (NLPD); and infectious period (NIPD). The infection ratio is

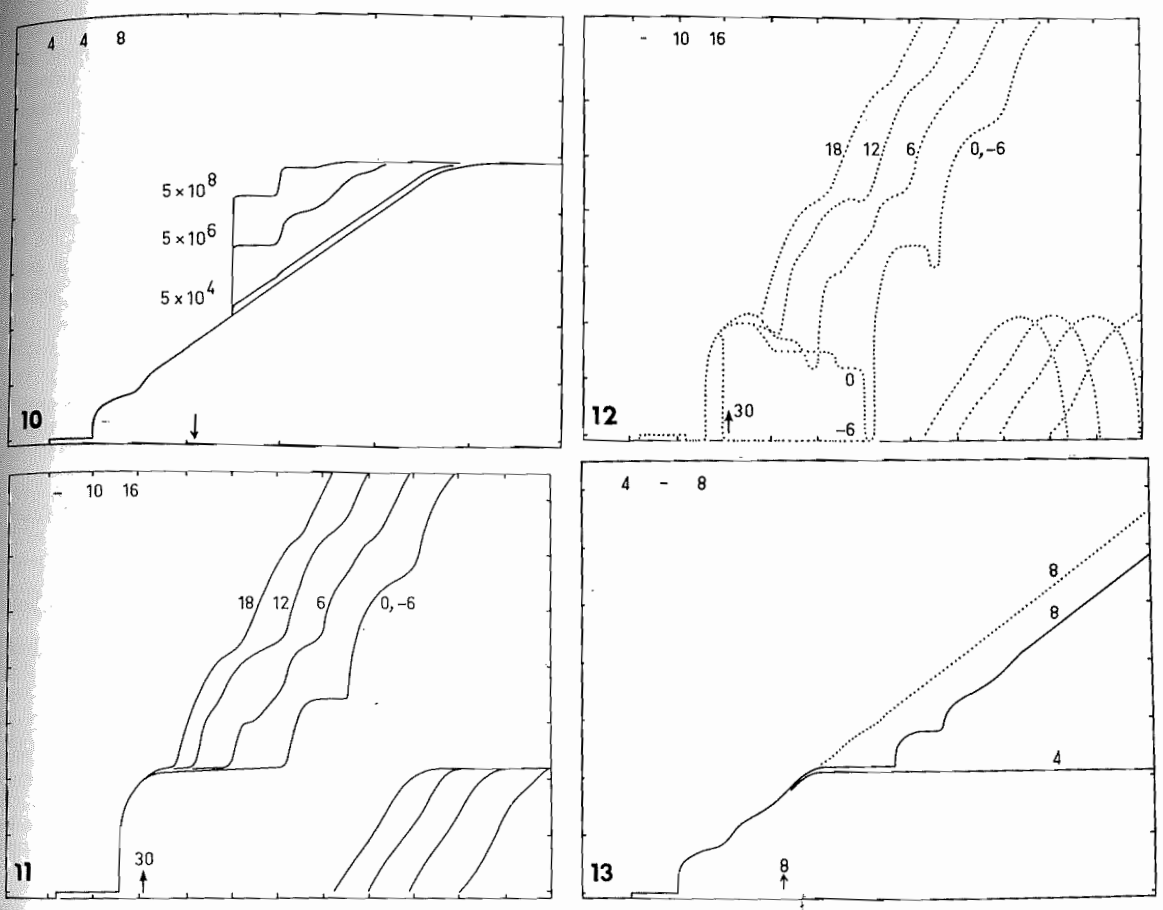

Fig. 10-13. Simulated epidemics. **10)** Effect of a limited number of available infection sites (SITE = 5×10^9) on the severity curve (which bends away to horizontal) and effect of influx of various amounts of effective spores (ROCI feature) on the severity curve; **11, 12)** effect of a 30-day interruption period of various low temperatures (from −6 C to 18 C) on an epidemic of *Puccinia striiformis* f. sp. *tritici* developing at 18 C in a 1-ha crop (SITE = 5×10^9) during 120 days; (11) severity; (12) infectants only; **13)** effect of an interruption period of 8 days during which no infections take place (NOOC = 1) on simulated epidemics with short and long infectious period; short (4 days), the epidemic comes to a stop; long (8 days), the epidemic is retarded only. The dotted line represents the noninterrupted epidemic with an infectious period of 8 days. In the retarded epidemic, the interruption can be recognized in the waves of the severity curve during a span of time comprising three latent periods (memory effect).

the fraction of spores that produces sporulating lesions in a disease-free crop. The sporulation rate is the number of spores produced per lesion per day. Resistance increases with decreasing infection ratio, with increasing latent period, decreasing sporulation rate, and decreasing infectious period (14). In the present model, sporulation rate, RSPO, and infection ratio FINS have been combined into one value, the daily multiplication factor: DMFR = RSPO × FINS. Acceptance of partial resistance as a breeding objective implies the decision to "live with" the disease, and the willingness to accept a calculated risk. Simulation models may help to calculate the risk.

Assume that the following data will be known for a particular variety in a particular area: (i) quantitative values for the components of resistance; (ii) Initial inoculum at the onset of the epidemic; (iii) Disease severity causing lowest measurable losses, loss threshold; and (iv) Influx data, if needed. This information is sufficient to calculate the duration of the period from day of onset until the day when the loss threshold is reached (18). When this period is too short, excessive risks are taken; when it is too long, too much effort is wasted in breeding. The good and bad chances due to fluctuations of the weather could be predicted, using known weather data. Very few quantitative data are available on the components of resistance against cereal rusts of wheat in the mature plant stage—a fruitful field for future research.

Data obtained in the growth cabinet could be fed into the computer, and a first estimate of field performance might be produced within sec. In many wheat varieties, the degree of resistance depends on environmental factors that can be studied in growth cabinets (11). The relationship between partial resistance (split up into its various components) and environment (temperature, moisture, etc.) can be described in tables or mathematical equations. Tables and equations can be

added to the model, and a few computer runs with climatic data as external information may indicate in which climatic areas the wheat line tested will show adequate resistance.

DISCUSSION.—This paper started with systems analysis, but dealt mainly with one of its elements, simulation, applied to an epidemiological system. The author was led by the conviction that the holistic approach deserved more emphasis than it has hitherto received, analytical data being relatively abundant. The holistic approach leads to model construction. Models must be explicative. Where explicit models in the form of differential equations become too complicated for solution, modern computer technique permits the design of implicit simulation models. Such models are indispensable when a continuous stream of external information (on weather, e.g.) has to be processed. A prerequisite to success is the complete quantification of all epidemiological information.

The simulation program was written in FORTRAN, an all-purpose computer language. The system of programming has been borrowed from DYNAMO (4, 10), a language especially designed for simulation. More recently, CSMP (Continuous System Modeling Program) came into use (5); this language seems to be a great improvement (1).

Many levels and rates not discussed in the foregoing are of potential interest and could be added to the program. A typical omission is the number of spores present. The choice of features was rather arbitrary. Another omission is a feature that makes integers of the calculated real numbers. Half a spore does not exist. The chosen interval of 1 day is long; Waggoner & Horsfall (15) used a solution interval of 3 hr in EPIDEM, an elaborate program for the simulation of fungal epidemics. No statement should be contrary to known truths, but this is not enough. Many unspoken hypotheses underlying the model are only half-truths. For example, not all lesions from a particular infection day start to sporulate at the same time, and the sporulation rate is certainly not constant throughout the whole sporulation period. To many of such half-truths, the answer lies in another special feature. The truth content of the model can and must be maximized.

One of the attractions of a simulation model is that it is never concluded. Model building and quantification of information present challenges to sound epidemiological thinking. For example, Zadoks (17) and others thought that the duration of the sporulation period was an important item in rust epidemics. Figure 8-B,C proves that it is not. An idea can be tested within min by means of a computer run. In *P. recondita* f. sp. *tritici,* the sporulation period was found to be over 2 months under favorable conditions (8). What is the epidemiological meaning of a long sporulation period? The answer, that a long sporulation period serves to bridge periods adverse to infection, has been put to a test in Fig. 13.

The testing of ideas before starting labor-consuming experiments is another attraction of simulation (see section on resistance). When the idea seems relevant, the experiment has to be performed, measurements taken, analysis made. Back-feeding the results into the program is the following step. Then the program must be subjected to a test by comparing computer results with field data. Theory and experiment must go hand in hand toward a deeper understanding of the epidemiological process, and possibly toward the prognosis of the performance of various forms of partial resistance and disease warning services.

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