Development of a Simple Mechanistic Model of Cereal Rust Progress

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ABSTRACT

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A mechanistic mathematical model of plant disease progress, based on cereal rust biology, is derived. The model provides an explanation for R. D. Berger's finding that disease progress data were consistently better described by the Gompertz equation than by the conventionally used logistic equation.

Additional key words: compound interest disease, polycyclic disease, uredial infection cycle.

Disease progress models have traditionally been used in plant disease epidemiology to describe the dynamics of plant disease increase in time. Unfortunately, as Kranz (13) observes, these models are essentially input-output relations that provide little understanding of the underlying biological mechanisms. Hence, it is not surprising that when one such disease progress curve is shown to be more effective than another, a biological rationale is rarely provided. Our understanding of plant disease epidemics and our ability to successfully contain them probably suffer in the absence of such explanations.

THE MECHANISTIC MODEL

The development of rust disease on cultivated cereals occurs mainly during the repeating uredial stage of the pathogen's life cycle when uredospores are produced in prodigious quantities and carried by air currents from one cereal plant to another. The model developed below represents an attempt to characterize mathematically the mechanics of the uredial infection cycle of the cereal rusts. Beginning with the production of uredospores by sporulating pustules, mathematical descriptions of the dynamics are offered for uredospore-vulnerable host tissue contact, establishment of latent infections, and subsequent development of latent infections into visible symptoms of disease. These mathematical descriptions of the infection cycle stages are ultimately combined into a single disease progress equation. The model, which ignores meteorological influences and supposes no host growth, is developed by focusing on the basic biological processes underlying disease spread.

Uredospore dynamics. I begin by concentrating on the cloud of dispersing uredospores. Let u be the mean daily number of viable airborne uredospores over a unit area of cultivated cereal plants. Then, adapting the approach of Fleming (6), the rate of change in u with time, t, can be written

$$du/dt = Bix - Du,$$

where B is the rate of uredospore release into the dispersal cloud per unit amount of infectious tissue, i is the fraction of visibly diseased tissue that is infectious, x is the disease severity (the fraction of susceptible host tissue that is visibly diseased), and D is the rate of uredospore removal from the dispersal cloud through death or deposition. Initially i is assumed to be constant, and throughout, the term "host tissue" excludes all tissue of the host plant that is effectively immune to infection.

This equation is supported by empirical evidence. The term Bix reflected Kochman and Brown's (12) observation of a linear relationship between uredospore production and infectious area for oat crown rust and oat stem rust. The term Du is consistent with studies on uredospore longevity (19,27) and with the observation that uredospore losses to the infection of host tissue are negligible compared to the number of airborne uredospores (22).

The characteristic time scale of this equation is $1/D$, the average time between uredospore liberation and death or deposition. This is likely a matter of minutes or less, certainly faster than the dynamics of infectious tissue, which operate at a time scale on the order of at least 2 days (generally the logarithmic infection rate $\leqslant 0.5$ per day for cereal rusts [24]). Thus changes in the number of viable airborne uredospores, u, occur so much faster than changes in the amount of infectious tissue, ix, that u remains close to its steady state value (the value of u at which $du/dt = 0$) with respect to the amount of infectious tissue. Hence,

$$u = Bix/D. \quad (1)$$

Table 1 provides a summary of variable definitions.

Contact. Next I try to express the dynamics of uredospore-vulnerable host tissue contact in a mathematical form. This requires a precise definition of vulnerable host tissue. A fully developed uredium occupies a certain amount of host surface area. When multiple infections occur within a day or two within such an area, only one uredium results. Hence, upon establishment, any infection effectively preempts the area it will eventually occupy (18). Vulnerable host tissue includes all healthy host tissue that has not been preempted.

The process of infection of vulnerable host tissue begins with the deposition of a uredospore. To examine the infection dynamics, consider the proportion of host tissue, y, that experiences initial contact with a uredospore. If C is the per-uredospore rate of successful contact (measured in terms of the size of the "target," the vulnerable fraction of host tissue), then the rate of change in y can be approximated as

$$dy/dt = Cvu - Ey. \quad (2)$$

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Here v is the fraction of host tissue that is vulnerable (ie, capable of supporting a new uredium), and E is the rate at which successful uredospore contacts with vulnerable host tissue develop into established (latent) infections. The term Cvu is proportional to the rate at which successful initial contacts occur; the term Ey is proportional to the rate at which these initial contacts develop into latent infections. It is implicitly, and not unreasonably (9,26) assumed here that uredospore dispersal can be adequately described by the Poisson distribution (ie, that successful contacts are distributed independently and randomly among potential sites of successful contact).

Equation 2 has a characteristic time scale of 1/E, the average time between successful initial contact and the subsequent establishment of the corresponding latent infection. This is typically on the order of a few hours (28). In contrast, according to equation 1, changes in the number of viable airborne uredospores, u, are assumed to occur concurrently with changes in infectious tissue. As discussed above, the characteristic time scale of infectious tissue dynamics is on the order of at least 2 days. Changes in the vulnerable proportion of host tissue, v, are even slower (until late in the epidemic). Hence, changes in y generally occur considerably faster than changes in u or v, and because of this, y is expected to remain close to its steady state value with respect to u and v:

$$y = Cvu/E. \tag{3}$$

Latent infections. By definition, the proportion of host tissue that is latently infected, L, is related to the proportions that are vulnerable, v, and visibly diseased, x, by the expression

$$L = 1 - v - x.$$

Since Ey is the relative rate at which host tissue becomes latently infected, the latently infected proportion, L, changes at an approximate rate of

$$dL/dt = Ey - L/p, \tag{4}$$

where p is the mean length of the latent period as defined by Shaner et al (20). The term L/p estimates the average rate at which latently infected host tissue becomes visibly diseased. The actual rate varies; it is probably most consistent during exponential disease progress when the age distribution of latent infections is relatively stable.

TABLE 1. Definitions and dimensions of algebraic symbols[a]

a =	Mean infection rate (1/t)
B =	Daily rate of uredospore release into the dispersal cloud per unit amount of infectious tissue (u/t)
C =	Per-uredospore rate of successful contact measured with respect to the proportion of vulnerable host tissue (1/[ut])
D =	Rate of removal of uredospores from the dispersal cloud (1/t)
E =	Rate at which successful uredospore-vulnerable host tissue contacts become established as latent infections (1/t)
H =	Vulnerable proportion of symptomless host tissue (−)
I =	Subscript indicating that the subscripted variable was evaluted at the inflection point (−)
i =	Infectious fraction of visibly diseased host tissue (−)
k =	Parameter measuring the rate of disease progress (1/t)
L =	Latently infected fraction of host tissue (−)
m =	Parameter affecting mathematical structure (−)
p =	mean length of the latent period (t)
r =	Apparent infection rate (1/t)
t =	Time (eg, days)
u =	Mean daily number of viable uredospores in the dispersal cloud (uredospores)
v =	Vulnerable fraction of host tissue (−)
x =	Visibly diseased proportion of host tissue (−)
y =	Fraction of host tissue experiencing initial contact with a uredospore (−)

[a] Parentheses following definitions enclose the dimensionality; eg, (u/t) indicates that B is measured in uredospores per unit time; (−) indicates that H is dimensionless. Rates referred to are mean instantaneous rates.

Synergistic or competitive interactions between uredia have little influence on this rate (16,23).

More precisely, L/p slightly overestimates the average rate, at least early in the epidemic while L is increasing and therefore presumably dominated by younger age classes. On the other hand, the average rate ignores the disproportionately greater contribution to disease progress by the earliest infections to begin sporulating. Nonetheless, in total, equation 4 seems reasonable given the earlier omissions and approximations of this mathematical development and the goal of arriving at a simple disease progress model.

Substituting equations 1 and 3 into equation 4,

$$dL/dt = avx - L/p \tag{5}$$

where a = BCi/D. A biological meaning emerges for a when equation 1 is substituted into this relation, giving a = Cu/x. In words, a is the mean rate of successful contact (ie, the mean infection rate) (measured with respect to the proportions of host tissue that are vulnerable and visibly diseased).

Disease severity. Because neither the vulnerable, v, nor the latently infected, L, proportions of host tissue are directly observable variables, equation 5 is not immediately useful in its present form. To alleviate this problem, we can write v and L in terms of the observable variable, disease severity:

$$v = H(1 - x),$$

and

$$L = (1 - H)(1 - x) \tag{6}$$

where H, a function of x, is the vulnerable (ie, uninfected and not preempted) proportion of symptomless host tissue.

Substituting for v in equation 5,

$$dL/dt = aHx(1 - x) - L/p. \tag{7}$$

Next, assuming that the proportion of latently infected host tissue, L, remains near its steady state value with respect to disease severity, x, equation 7 yields

$$L = apHx(1 - x). \tag{8}$$

The validity of this steady state assumption for L has been tested elsewhere (8). Numerical integration suggested that error intrinsic to this assumption was likely to have a negligible effect on subsequent disease progress relative to the effect of error in estimating the initial value of L in the field.

The task remains to relate equation 8 to the dynamics of disease severity. Two processes lead to increases in disease severity: the establishment of new infections and the expansion of established infections. The latter process generally contributes little to cereal rust spread relative to the first (10,22,25) and so will not be considered further. Therefore, because the last term in equation 4 represents the rate at which the visibly diseased fraction of host tissue increases through infection establishment, the rate of increase in disease severity can be approximated by

$$dx/dt = L/p.$$

When equation 8 is substituted into this expression, the logistic equation results:

$$dx/dt = rx(1 - x), \tag{9}$$

where the apparent infection rate is

$$r = aH. \tag{10}$$

In arriving at equation 10, I have neglected the fact that H, the vulnerable fraction of symptomless host tissue, decreases with disease severity, x. This dependence of H on x can be shown more explicitly. As discussed above, under the assumption that the

proportion of latently infected host tissue, L, remains near its steady state value with respect to x, equation 8 applies. Substituting equation 8 into equation 6 and solving for H emphasizes this dependence of H on x:

$$H = 1/(1 + apx).$$

Hence, according to the mechanistic derivation used here, either equation 10 should be replaced by

$$r = a/(1 + apx), \tag{11}$$

or equation 9 should be written as

$$dx/dt = ax(1 - x)/(1 + apx). \tag{12}$$

In either case, the robustness (5) of models that are essentially extensions of the logistic (eg, 21) is brought into question.

DISCUSSION

The logistic model is typically used to describe the progress of polycyclic or "compound interest" diseases (24). However, because of its inherent symmetry, the logistic model does not accurately describe the asymmetrical disease progress curves frequently observed. Berger (3) considered this problem in detail and found that "the Gompertz model provided a better statistical fit than did the logistic model for all 113 disease progress curves from 9 pathosytems" that he examined.

The Gompertz model is structurally related to both the logistic (17) and equation 12. These three models are also structurally related to the monomolecular equation that Vanderplank (24) has used to describe monocyclic or "simple interest" diseases. Each of these four models represents simplifications of the equation

$$dx/dt = kx\,(x^{m-1} - 1)/[(1 - m)(1 + apx)],$$

where k is a rate parameter (dimensioned t^{-1}) and m is a dimensionless nonnegative real number. This expression approaches the monomolecular when m = 2 and p is large, and reduces to it exactly when m = p = 0. The logistic results when m = 2 and p = 0; equation 12 results when m = 2 and $0 < p < \infty$. This expression reduces to the Gompertz,

$$dx/dt = kx\ln(1/x), \tag{13}$$

when m = 1 and p = 0 because

$$\lim_{m \to 1} [(x^{m-1} - 1)/(1 - m)] = \ln(1/x).$$

According to the Gompertz model, plots of $\ln(-1/\ln x)$ against t are linear with slope k.

Figure 1 provides plots of the logit and Gompertz transformations of solutions to equation 12. Because these solutions are transcendental in x, they were computed numerically. In contrast to the logistic model (equation 9), which is linear under logit transformation with slope r, equation 12 exhibits a decline in slope (apparent infection rate) as x increases. This follows directly from equation 11. The fact that such a decline in slope is often observed (1,28) provides some confidence in the ability of equation 12 to describe disease progress.

The variation in slope in Fig. 1 shows that the logit transformation is less effective at linearizing equation 12 than the Gompertz transformation is. The mathematical reason for this can be deduced by comparing equations 9, 11, and 13 with respect to their "saturation factors": $\ln(1/x)$ provides a closer dynamic match to $(1 - x)/(1 + apx)$ than does $1 - x$ for the values of ap and x.

In discussing the general use of the logistic equation to describe disease progress, Berger (2) remarks,

> "Although a credible statistical fit often occurs in the range $0.05 < x < 0.6$, very poor fit is obtained when x is outside this range because of the asymmetrical shape of most disease progress curves."

The logistic equation predicts a daily increase of disease (dx/dt), which is symmetrical about its time of inflection (the time at which dx/dt is at its maximum in $0 < x < 1$). In contrast, the observed daily increase is commonly skewed to the right (3).

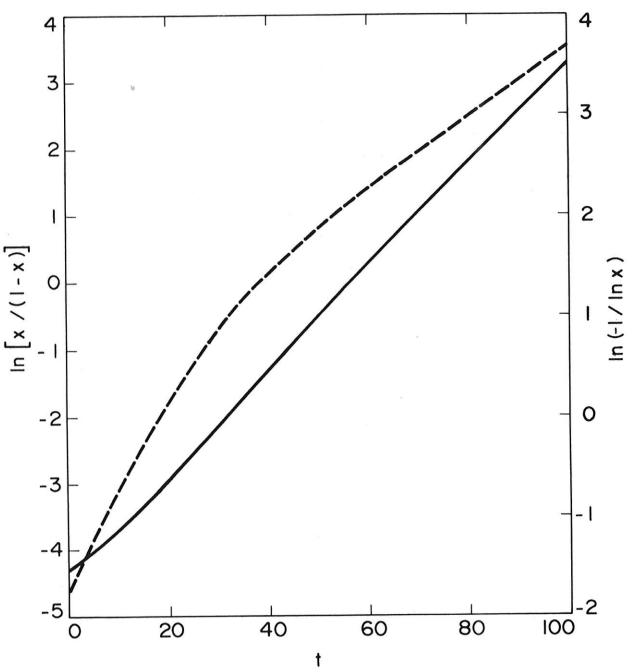

Fig. 1. Logit (broken line, $\ln [x/(1 - x)]$) and Gompertz (solid line, $\ln [-1/\ln x]$) transformations of disease severity, x, resulting from the numerical integration of equation 12 with the reasonable (11,14,15) value of 14 days for the cereal rust latent period and a mean infection rate of 0.16 per day to give a large range of x values. Initially x = 0.01.

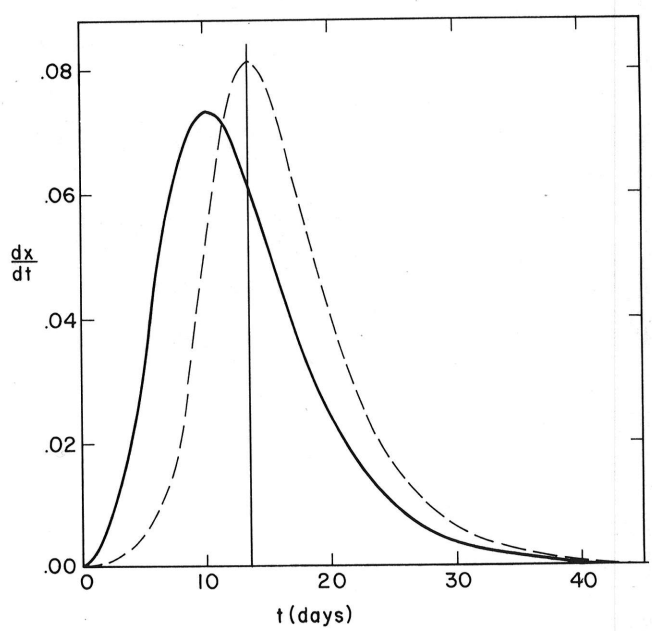

Fig. 2. Daily increase of disease (dx/dt) as predicted by the Gompertz model (solid line) and equation 12 (broken line). Parameter values are k = 0.2 for the Gompertz (equation 13) and a = 0.6 and p = 3.25 for equation 12. Initially x = 0.0004. The points of inflection are $t_I = 10$ for the Gompertz and, as indicated by the vertical line, $t_I = 13.5$ for equation 12. Solutions were calculated numerically.

As shown in Fig. 2, such skewness to the right is consistent with both the Gompertz equation and equation 12. Parameter values were chosen to make this figure readily comparable with Berger's (3) Fig. 2. The initial value of x and the value of k are identical to those used by Berger; equation 12 was provided an inflection point in terms of x comparable with that of the Gompertz equation (by setting ap = 1.95), and it was given a height comparable to the logistic equation of Berger's Fig. 2 (by setting a = 0.6). The skewness to the right exhibited by equation 12 is emphasized by comparison of the area under the curve on each side of the vertical line that marks the time of inflection ($t_I = 13.5$).

Figures 1 and 2 have relied on particular values of a and p to illustrate how well equation 12 can mimic the Gompertz model. Figure 3 provides a more general comparison.

Points of inflection, x_I, were determined by setting

$$\delta(dx/dt)/\delta x = 0$$

and solving for $x_I = x$. Hence, in equation 12, the inflection point is

$$x_I = (\sqrt{[1 + ap]} - 1)/ap. \quad (14)$$

Figure 3 shows that for any ap exceeding approximately 0.65, the inflection point of the Gompertz equation ($x_I = 0.368$) better estimates x_I in equation 14 than does the inflection point of the logistic equation ($x_I = 0.5$). Furthermore, since realistically $ap > 0$, according to equation 14,

$$x_I < (\sqrt{[1 + ap + (ap/2)^2]} - 1)/ap = 0.5.$$

Therefore, the daily increase of disease predicted by equation 12 is skewed to the right. This also matches observation (3).

Taken together, these features of equation 12 suggest at least a partial explanation for the superiority of the Gompertz equation in describing disease progress data. Moreover, as mentioned above, the inadequacy of the logistic model can be partly explained on biological grounds as a failure to account for the decline in H, the vulnerable fraction of symptomless host tissue, as disease severity increases.

However, it is difficult to imagine a thorough mechanistic (7) mathematical description of the uredial infection cycle leading to a saturation factor of the form ln(1/x). Hence, although the Gompertz model may supply a better description of disease progress, its superiority cannot be readily explained on biological grounds except as an imitation of equation 12. It follows that the Gompertz model does not merit serious consideration as an explanatory model.

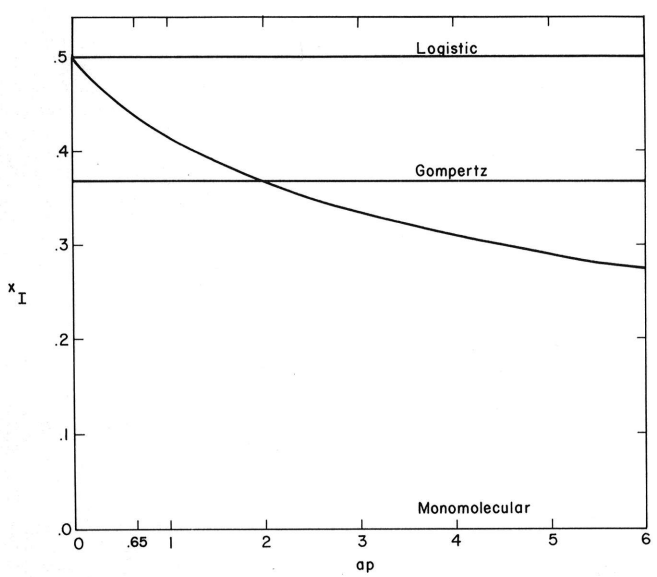

Fig. 3. Point of inflection of equation 12 with respect to x as a function of the product, ap. The horizontal lines indicate the inflection points of various other models.

This leaves equation 12 as the best available explanatory model. Notwithstanding, it too has its shortcomings, perhaps the most important being the assumption that a constant proportion, i, of visibly diseased tissue is infectious. Actually, i is likely to exhibit a net decline during the course of the disease season. Using the potato late blight simulation model of Bruhn et al (4), one can show that i is probably a complex function of x and t.

Futhermore, equation 12 still needs to be tested. Integrating it and then rearranging the integral produces an expression from which the infection rate parameter, a, can be calculated:

$$a = \frac{\ln[x_t/(1 - x_t)] - \ln[x_o/(1 - x_0)]}{t - p \ln[(1 - x_o)/(1 - x_t)]}$$

where x_0 and x_t are the disease severities at times 0 and t, respectively, and p is the latent period. Hence, equation 12 predicts a linear relationship (with slope a) between logit (x_t) and $t - p \ln[(1 - x_0)/(1 - x_t)]$. Here p should be estimated independently, perhaps using by the procedure developed by Shaner et al (20). This prediction provides a means of testing the validity of the model and thus establishing its limitations (7).

LITERATURE CITED

1. Berger, R. D. 1975. Rapid disease progress in early epidemic stages. (Abstr.) Proc. Am. Phytopathol. Soc. 2:35.
2. Berger, R. D. 1981. The Gompertz transformation—More appropriate than the logistic to describe disease progress. (Abstr.) Phytopathology 71:203.
3. Berger, R. D. 1981. Comparison of the Gompertz and logistic equations to describe plant disease progress. Phytopathology 71:716-719.
4. Bruhn, J. A., Bruck, R. I., Fry, W. E., Arneson, P. A., and Keokosky, E. V. 1980. User's manual for late blight: A plant disease management game. 80-1. Dept. Plant Pathol., Cornell Univ., Ithaca, NY. 49 pp. (mimeographed)
5. Fleming, R. A. 1980. Selection pressures and plant pathogens: Robustness of the model. Phytopathology 70:175-178, 71:268.
6. Fleming, R. A. 1980. The potential for control of cereal rust by natural enemies. Theor. Pop. Biol. 18:374-395.
7. Fleming, R. A., and Bruhn, J. A. 1983. The role of mathematical models in plant health management. In: Challenging Problems in Plant Health. T. Kommedahl and P. Williams, eds. Am. Phytopathol. Soc., St. Paul, MN. (In press)
8. Fleming, R. A., and Holling, C. S. 1982. A comparison of disease progress curves for cereal rust. Can. J. Bot. 60:2154-2163.
9. James, W. C., and Shih, C. S. 1973. Relationship between incidence and severity of powdery mildew and leaf rust on winter wheat. Phytopathology 63:183-187.
10. Kampmeijer, P., and Zadoks, J. C. 1977. EPIMUL, a simulator of foci and epidemics in mixtures of resistant and susceptible plants, mosaics and multilines. Pudoc, Wageningen, The Netherlands. 50 pp.
11. Katsuya, K., and Green, G. J. 1967. Reproductive potentials of races 15b and 56 of wheat stem rust. Can. J. Bot. 45:1077-1091.
12. Kochman, J. K., and Brown, J. F. 1975. Host and environmental effects on post-penetration development of *Puccinia graminis avenae* and *P. coronata avenae*. Ann. Appl. Biol. 81:33-41.
13. Kranz, J. 1974. The role and scope of mathematical analysis and modeling in epidemiology. Pages 7–54 in: Epidemics of Plant Diseases: Mathematical Analysis and Modeling. J. Kranz, ed. Springer-Verlag, Berlin.
14. Leonard, K. J. 1969. Selection in heterogeneous populations of *Puccinia graminis* f. sp. *avenae*. Phytopathology 59:1851-1857.
15. Loegering, W. Q., Johnston, C. O., and Hendrix, J. W. 1967. Wheat rusts. Pages 307–335 in: Wheat and Wheat Improvement. K. S. Quisenberry and L. P. Reitz, eds. Am. Soc. Agron., Madison, WI.
16. Metha, J. R., and Zadoks, J. C. 1970. Uredospore production and sporulation period of *Puccinia recondita* f. sp. *triticina* on primary leaves of wheat. Neth. J. Plant Pathol. 76:267-276.
17. Richards, F. J. 1959. A flexible growth function for empirical use. J. Exp. Bot. 10:290-300.
18. Shaner, G., and Hess, F. D. 1978. Equations for integrating components of slow leaf-rusting resistance in wheat. Phytopathology

68:1464-1469.
19. Shaner, G., and Powelson, R. C. 1971. Epidemiology of stripe rust of wheat, 1961–1968. Ore. Agric. Exp. Stn., Bull. 117. 31 pp.
20. Shaner, G., Ohm, H. W., and Finney, R. E. 1978. Response of susceptible and slow leaf-rusting wheats to infection by *Puccinia recondita*. Phytopathology 68:471-475.
21. Skylakakis, G. 1980. Estimating parasitic fitness of plant pathogenic fungi: A theoretical contribution. Phytopathology 70:696-698.
22. Stakman, E. C., and Christensen, C. M. 1946. Aerobiology in relation to plant disease. Bot. Rev. 12:205-253.
23. Teng, P. S., and Close, R. C. 1978. Effect of temperature and uredinium density on urediniospore production, latent period, and infectious period of *Puccinia hordei* Otth. N.Z. J. Agric. Res. 21:287-296.
24. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.
25. Waggoner, P. E. 1981. Models of plant diseases. BioScience 31:315-319.
26. Waggoner, P. E., and Rich, S. 1981. Lesion distribution, multiple infection, and the logistic increase of plant disease. Proc. Natl. Acad. Sci. USA 78:3292-3295.
27. Yarwood, C. E., and Sylvester, E. S. 1959. The half-life concept of longevity of plant pathogens. Plant Dis. Rep. 43:125-128.
28. Zadoks, J. C. 1961. Yellow rust on wheat, studies in epidemiology and physiologic specialization. Tijdschr. Plantenziekten 67:69-256.