Equations for Integrating Components of Slow Leaf-Rusting Resistance in Wheat

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ABSTRACT

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Equations were developed to help predict how various components of slow-rusting resistance, measured in monocyclic inoculation experiments, interact under field conditions. These equations incorporate the effects of infection efficiency (the number of uredia per 100 spores landing on a leaf), latent period, uredium size, urediospores produced per uredium per day, and infectious period per uredium. In handling latent period data, the equations account for the fact that the infections occurring on a given day erupt into uredia over a period of several days. The equations can be used to predict how certain combinations of slow-rusting resistance components might retard an epidemic. They should be useful to plant breeders as a guide for selecting the most valuable segregants in breeding for slow-rusting resistance.

Additional key words: horizontal resistance, general resistance, nonspecific resistance, Triticum aestivum, epidemiology, breeding for resistance.

Slow leaf-rusting resistance in wheat (*Triticum aestivum* L. em Thell.) is a form of resistance in which the host restricts the rate of pathogen (*Puccinia recondita* Rob. ex Desm.) development without showing the chlorosis typical of the hypersensitive response (5). On some cultivars with this resistance, the probability of a spore infecting and giving rise to a uredium is reduced, the latent period is increased, and uredia are smaller and consequently produce fewer spores (7). In the field the leaf rust epidemic builds up more slowly on these resistant cultivars. The components of slow-rusting resistance are quantitative traits, in contrast to the qualitative nature of hypersensitive resistance (7). Within a large group of wheats, these components exhibit continuous variation (4). Breeders are interested in slow-rusting resistance because it appears to be race-nonspecific or at least to be much more stable to changes in pathogen virulence (1, 2, 3, 6).

Because the various quantitative components of resistance act together to affect the course of an epidemic, it is necessary to understand the contribution of each component to the overall effect. Some components may be more effective at slowing the spread of infections than others and a component may have to be of a certain magnitude before it appreciably contributes to slow rusting.

The effect of a component of slow-rusting cannot be determined by simply measuring its magnitude in a monocyclic infection experiment. Such experiments are necessary to measure the components themselves (7), but the effects of the components on an epidemic are difficult to interpret because these components interact with each other and their effects are cumulative over the course of the epidemic. Extension of greenhouse data from monocyclic infection experiments to predict disease progress in the field is further complicated by the latent period. If a susceptible cultivar is inoculated, placed in a dew chamber overnight, and then placed in a greenhouse (22 to 27 C daytime; 17 to 22 C nighttime) where no subsequent infections can occur, the first uredia will appear on the 6th day after inoculation (7). More uredia will appear each day until the 10th day. Thus, even when all infections take place during the same night, some infection sites require longer than others to produce uredia. Using data of Shaner et al. (7), the cumulative percentage of uredia erupted each day, plotted against time, gives a sigmoid curve (Fig. 1). Comparable data for a slow-rusting wheat yield a sigmoid curve that is flatter and displaced to the right. All uredia may not appear on a slow-rusting wheat until the 14th day, although a few uredia appear on the 7th day. Thus, the latent period is not a single value. On day 7, uredia on both susceptible and slow-rusting wheats will be producing spores which can cause secondary infections, but the proportion of uredia present on that day is greater on the susceptible wheat than on the slow-rusting wheat.

Analysis of the effects of various components of slow-rusting resistance on epidemics in the field could be done directly by measuring components of resistance of a diverse collection of wheats in the greenhouse and then studying the development of leaf rust on them in the field. There are difficulties, however, with this approach. We

have not yet identified all possible combinations of components in individual lines. The available combinations of slow-rusting components exist in wheats of greatly differing maturity and habit, making direct comparison in the field difficult or impossible. Because the components of slow-rusting resistance can be measured accurately and economically in the greenhouse, we have undertaken the development of equations that integrate the data from such experiments into a form designed to predict the effect of any set of resistance components on the rate of disease progress in the field.

THEORETICAL BASIS OF THE MODEL

In leaf rust of wheat, each successful infection by *P. recondita* gives rise to a single uredium of limited size. Disease becomes severe only after repeated infections have given rise to many uredia. Thus, a leaf rust epidemic can be treated as an increase of a population of uredia. The uredium is the individual member of this population. The urediospores it produces (progeny) infect leaves and gives rise to new uredia.

Calculation of the number of uredia that appear each day.—To predict the increase in a population of uredia we calculate the number of uredia that appear each day (N_i) by solving the equation:

$$N_i = \sum_{j=p}^{j=m} (P_j I_{i-j}) \qquad \text{Eq. 1}$$

where

N_i = number of uredia per leaf blade area of arbitrarily chosen α square millimeters that appear on the i*th* day.

I_{i-j} = number of infections per α that occur on the (i−j)*th* day, j = p, ..., m.

p = earliest day after infection when uredia appear.

m = day by which all uredia that will develop after infection have appeared.

P_j = probability of an infection that occurred on the (i−j)*th* day erupting into a uredium on the i*th* day.

Values of p, m, and P_j are characteristic of each cultivar and are obtained from data as in Fig. 1. For Monon, p is 6 days and m is 10 days. At day 6, 7.1% of the uredia that will ultimately arise from infections that took place on Monon at day 0 appear and on day 7 an additional 31.1% appear. Thus $P_6 = 0.071$ and $P_7 = 0.311$ (Table 1). In this paper, all infections are assumed to give rise to uredia. Infections that never erupt into uredia are combined with unsuccessful penetrations into the term v, which is explained below.

To solve Eq. 1 for any day i, one looks back 6 (p) days to see how many infections took place. Of this number, 7.1% will erupt as uredia on the i*th* day. Also on the i*th* day,

TABLE 1. Parameters[a] used in the leaf rust equations for four cultivars of *Triticum aestivum*

Parameter	Cultivar			
	Monon	Suwon 92	Suwon 85	P6028
P_p[b]	.071	.058	.022	.021
P_{p+1}	.311	.209	.082	.064
P_{p+2}	.358	.396	.236	.259
P_{p+3}	.238	.273	.201	.218
P_{p+4}	.022	.064	.223	.161
P_{p+5}			.110	.148
P_{p+6}			.126	.085
P_{p+7}				.044
P_{p+8}				
P[c]	6	6	7	7
m[d]	10	10	13	14
n[e]	10	10	10	10
S[f]	411	538	299	208
λ[g]	.018	.018	.018	.018
v[h]	.14	.13	.12	.10
a[i]	0.65	0.79	0.41	0.41

[a]All values except those for λ derived from Shaner et al. (Phytopathology 68:471-475).

[b]Probability of an infection that occurred on the (i−j)*th* day erupting into a uredium on the i*th* day.

[c]Minimum number of days after infection when a uredium appears.

[d]Maximum number of days after infection when all infections that occur on any given day have erupted into uredia.

[e]Infectious period, days.

[f]Number of spores produced per uredium per day.

[g]The proportion of spore "hits", estimated from field data (Ohm and Shaner 1976. Phytopathology 66:1356-1360). See text for details.

[h]The proportion of spores on potential infection sites that successfully infect.

[i]Area per infection site, square millimeters, derived by dividing the mean uredium size for each cultivar given in Shaner et al. (Phytopathology 68:471-475) by 0.34.
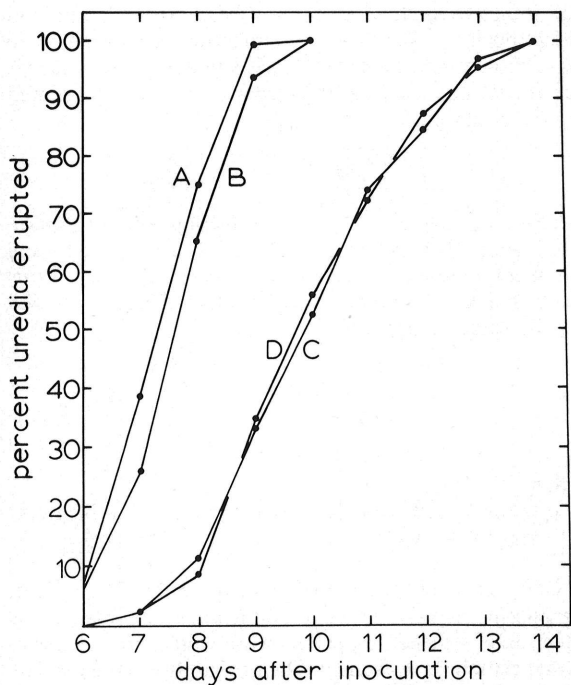

Fig. 1. Course of appearance of uredia of *Puccinia recondita* on four winter wheat cultivars following inoculation at day 0. Curves represent data for: A = Monon, B = Suwon 92, C = Suwon 85, and D = P6028. Data from Shaner et al. 1978. Phytopathology 68:471-475.

31.1% of the infections that took place 7 days previously (p + 1) will erupt into uredia. One keeps looking back as far as infections that took place 10(m) days previously. Of the infections that took place on that day, the final 2.2% erupt on the ith day. The sum of all these calculations is the total number of uredia that erupt on the ith day.

Calculation of daily infections.—N_i is a function not only of p, m, and P_j, which assume certain values characteristic of each cultivar, but of the variable I_{i-j}. To start an epidemic, an arbitrary value of I_0 can be taken as the level of primary infection. Once these I_0 infection sites erupt and urediospores from these primary uredia are released, the number of daily infections, I_i, becomes a function of the number of uredia present. Thus N_i is a function of I_{i-j}, but I_{i-j} is itself a function of the active (spore-producing) uredia present on day $i-j$. Because I_{i-j} and N_{i-j} bear the same relation to each other as I_j and N_j, we can write:

$$I_i = S\lambda v \sum_{j=1}^{n} N_{i-(j-1)} \qquad \text{Eq. 2}$$

where

    n = the infectious period, the number of days over which the uredium produces spores. On the ith day only uredia formed on days i through $i-(n-1)$ still produce spores.

    S = number of urediospores produced per uredium per day (Table 1). For this preliminary work, S is taken to be constant throughout the period that a uredium produces spores. Limited data indicate that on the first day that a uredium erupts it produces only about 5% of its maximum daily output; on the second day it produces about 20% of its maximum daily output; and by the third day it has reached 100% of the maximum. This level continues at least six more days (7).

    λ = the proportion of spore "hits", that is, the proportion of spores produced per day that land on a potential infection site. This term is necessary because some of the spores will land on soil, blow out of the field, or otherwise fail to land on a leaf blade. "Potential infection site" does not exclude leaf blade tissue that is already infected and therefore cannot be infected again. Removal of infected tissue from the total "potential infection sites" will be dealt with later. In this model, λ can be considered a constant if the environment is constant (constant wind speed, dew every night, etc.) and the total leaf canopy area (green as well as diseased or senescent) is constant during the course of an epidemic. The assumption about canopy area is reasonable for Indiana because leaf rust often does not appear until flag leaves have emerged.

    v = proportion of spores that land on a potential infection site that penetrate and give rise to a uredium. This term is a function of the viability and aggressiveness of inoculum (assumed to be constant during the course of an epidemic), environment (assumed to be the same each day), and the host. If the host resists penetration, so that fewer germ tubes invade substomatal cavities, or if the host has a post-infectional response that prevents certain infections from developing into uredia, this will reduce v (Table 1).

Equation 2 assumes that any portion of the leaf blade is a potential infection site. However, once an infection takes place that site cannot be reinfected. Even if two spores penetrate side by side, two uredia cannot occupy the same site. As the epidemic progresses, the number of potential infection sites diminishes according to the proportion of leaf area that is infected. To account for this, Eq. 2 must be modified:

$$I_i = S\lambda (1-A_{i-1}) v \sum_{j=1}^{n} N_{i-(j-1)} \qquad \text{Eq. 3}$$

where

    A_{i-1} = proportion of leaf tissue that is infected through the $(i-1)$th day. It is a function of the number of uredia per unit area of leaf and the total area of infected tissue associated with each uredium.

The spore-producing part of the uredium occupies only part of the total invaded area. Using the highest densities of uredia that could be obtained in a greenhouse study (7), we calculated that only about 34% of the leaf area was covered at maximum severity. Presumably the remaining 66% of the tissue was either invaded by mycelium associated with uredia or was otherwise rendered incapable of supporting uredia.

Several days are required for a uredium to reach its maximum size. However, for the purposes of accounting for infected tissue that cannot be reinfected, once an infection has taken place the infection is considered to have pre-empted the total area it will eventually occupy. On the ith day the total infected area can be represented by the equation:

$$a \sum_{j=0}^{i} I_{i-j}$$

where a is the area of each infection site in square millimeters. It is calculated for each cultivar by dividing the mean uredium area by 0.34 (Table 1). To express this area on a relative basis, the total infected area is divided by the total canopy area

$$A_i = \frac{a \sum_{j=0}^{i} I_{i-j}}{\alpha} \qquad \text{Eq. 4}$$

where

    α = total leaf blade area (square millimeters) arbitrarily chosen.

Calculation of the proportion of spore "hits" (λ).—The probability λ of a spore landing on a leaf blade is a function of weather, especially wind, and canopy density, rather than host resistance. The probability is the ratio of the total number of spores that actually land on leaf blades, as measured by the uredia produced and adjusted by v, to the total number of spores produced, ie, the potential number that could land on leaf blades. To estimate λ it is necessary to use the observations from a

field experiment. In one such study (5), Monon had a severity of 9% on 4 June (i = 35) and a severity of 71.7% on 18 June (i = 49). Severity is the proportion of leaf blade covered by uredia. It takes account of the fact that uredia will never completely cover a leaf so that 100% severity is equivalent to 34% coverage by uredia. Percent severity (100 Y_i) on the i*th* day is less than percent infected tissue (100 A_i) on the same day. This is because A_i includes that additional proportion of host tissue already infected but not yet observable because the uredia have not yet developed.

To calculate the numerator of λ we calculate the number (N_i) of uredia that appeared on day 41. To do this, the apparent infection rate (8) was calculated from the severity data as:

$$\left\{ \ln\,[0.717/(1-0.717)] - \ln\,[0.09/(1-0.09)] \right\} / 14$$
$$= 0.232 \text{ per unit per day}$$

Assuming this apparent infection rate remained constant during the field experiment, severities for each day were calculated using the formula:

$$\left\{ \ln\,[Y_i/(1-Y_i)] - \ln\,[0.09/(1-0.09)] \right\} / (i-35) = 0.232$$

Severity (Y_i) was converted to cumulative number of uredia

$$(\sum_{j=0}^{i} N_{i-j})$$

per square meter of leaf blade by dividing Y_i by the mean area of an infection in square millimeters, and multiplying by 10^6 mm^2/m^2. On 10 June (i = 41) Y_i was calculated to be 0.285. The mean infection site area on Monon in that experiment was the mean uredium size, 0.1763 mm^2, divided by 0.34; ie, 0.5185 mm^2. Thus, on i = 41, the total number of uredia per square meter of leaf blade was (10^6 mm^2/m^2) (0.285)/0.5185 mm^2 = 0.5497 × 10^6. The number of uredia that appeared on i = 41 was calculated

as the difference between the total number on the 41*st* day and the 40*th* (i−1) day; ie,

$$\sum_{j=0}^{i} N_{i-j} - \sum_{j=1}^{i} N_{i-j}$$

On day 41, 8.64 × 10^4 uredia appeared. Adjusting for v, we divide that number by 0.14 to obtain the total number of spores, 6.17 × 10^5, that actually landed on leaf blades as measured by the uredia appearing on day 41 and adjusted for v. This is the numerator of λ.

Before attempting to discuss the denominator of λ, we need to discuss how the numerical quantity representing the total number of spores produced on the i*th* day was calculated. The number of spore-producing uredia per square meter of leaf blade was calculated as the difference between the number of uredia at day i and the number of uredia at day i−n, analogous to that for N_i above, for i and i−1. The value n is the length of the infectious period. We assumed n = 10 days. Finally, the number of spore-producing uredia was multiplied by the output of spores per uredium per day (7) to calculate the daily spore production per square meter of leaf blade (Table 2).

To calculate the denominator of λ we need that total number of spores which provided inoculum for the infections giving rise to uredia on the i*th* day. The number of uredia that appear on the i*th* day is the result of infections that took place from day i−m through i−p as shown in Eq. 1. For example, the number of uredia appearing on day 41 would have arisen from infections taking place on days 31 through 35 (i = 41, m = 10, p = 6). The expected number of spores that the (i−j)*th* day contributes to the denominator is the number of spores produced on the (i−j)*th* day times P_j, the conditional probability that a uredium appears on the j*th* day after infection, given that an infection occurs. The expected number of spores on each day is given in Table 2. The total expected number of spores in the inoculum that gave rise to uredia appearing on day 41 is 35.259 × 10^6.

Using the numerical values obtained above for the numerator and denominator, we have

$$\lambda = \frac{6.17 \times 10^5 \text{ uredia}}{35.259 \times 10^6 \text{ uredia}} = 0.018$$

Therefore, for every 1,000 spores produced each day, only 18 land on a leaf blade.

The use of n = 10 days is only an estimate; we have no accurate data concerning n. Therefore the calculations outlined above were repeated assuming n = 20 days (Table 2). In this case there would have been 38.962 × 10^6 uredia/m^2 if all spores produced between 31 and 35 had landed on a leaf and infected and

$$\lambda = \frac{8.64 \times 10^4 \text{ uredia}}{5.48 \times 10^6 \text{ uredia}} = 0.016$$

Doubling the infectious period, from 10 days to 20 days, only decreases λ from 0.018 to 0.016.

Operation of the model.—In order to predict the effect of any given combination of components of slow-rusting resistance, we specify an initial number of infections, the total leaf blade area on which to generate the epidemic, and the values for the constants in Equations 1 and 3.

TABLE 2. Number of urediospores of *Puccinia recondita* produced on days 31 through 35 that provided inoculum for infections of Monon wheat giving rise to uredia on day 41[a]

Day, i−j	j	P_j[c]	n[b] = 10 days		n = 20 days	
			T[d] (× 10^6)	TP$_j$[e] (× 10^6)	T[d] (× 10^6)	TP$_j$[e] (× 10^6)
31	10	0.022	21.471	0.472	23.663	0.521
32	9	0.238	26.787	6.375	29.548	7.032
33	8	0.358	33.330	11.932	36.807	13.177
34	7	0.311	41.335	12.855	45.712	14.216
35	6	0.071	51.055	3.625	56.563	4.016
Total				35.259		38.962

[a]Based on field data of Ohm and Shaner (Phytopathology 66:1356-1360). See text for details.

[b]n = infectious period.

[c]P_j = probability of an infection that occurred on the (i−j)*th* day erupting into a uredium on the i*th* day.

[d]T = total number of spores produced per day per square meter of leaf blade.

[e]TP$_j$ = expected number of spores.

We specify that 10 infections take place on day 0 and that the total leaf blade area (α) is $10^4 \, mm^2$. When this value of α is used, the simulated epidemic on Monon reaches 100% severity on day 60. Sixty days is about the length of a natural leaf rust epidemic in Indiana.

Mathematically the model is straightforward. The difficulty with its execution lies in keeping account of past events that influence current events. One must keep account of how many infections took place each day up to the present, how many of these erupted into uredia each day, and how many spores are released each day. To efficiently keep account of these parameters, Equations 1 and 3 were programmed (BASIC-PLUS language) for computer analysis and the values for each parameter calculated each day were used to construct data arrays.

To begin an "epidemic" values for the constants in Equations 1 and 3 are entered. The computer first solves Equation 1 for day $i = p$ and then calculates I_p using this value of N_p (Equation 3). It then repeats these calculations for days $p + 1, \ldots p + X$. For each day, the computer stores in an array

$$I_i, \quad N_i, \quad \sum_{j=0}^{i} I_{i-j}, \quad \sum_{j=0}^{i} N_{i-j}, \quad \text{per unit severity } (Y_i), \text{ and}$$

$$\ln (Y_i/(1-Y_i))$$

Y_i is calculated as

$$(\sum_{j=0}^{i} N_{i-j} / \sum_{j=0}^{x} N_{i-j})/0.34$$

so that per unit severity is based on the modified Cobb scale. The proportion of infected tissue (Y_i) is 1 when 34% of the leaf area is covered by uredia. The term $\ln [Y_i/(1-Y_i)]$ is calculated so that apparent infection rates can be calculated if desired (8). In addition to the numerical output, the data can be plotted directly from the data arrays.

CONCLUSIONS FROM THE MODEL

Model epidemics for Monon, Suwon 92, Suwon 85, and P6028 are plotted in Fig. 2-4. The number of daily infections, N_i, reached a maximum on Monon and Suwon 92 (Fig. 2) when per unit severity reached 0.8 (Fig. 4). After reaching this maximum, N_i declined sharply owing to a parallel but earlier decline in I_i as infection sites became limiting. Total uredia by the ith day

$$(\sum_{j=0}^{i} N_{i-j})$$

increased in sigmoid fashion (Fig. 3). The maximum value of N_i for Suwon 92 was lower than the maximum value of N_i for Monon because uredia on Suwon 92 were larger (Table 1). Severity on Monon and Suwon 92 reached 100% by day 60 whereas severity reached only 16% on Suwon 85 and 3% on P6028 (Fig. 4).

DISCUSSION

The differences in disease development among the four cultivars in the hypothetical epidemics are greater than Ohm and Shaner observed in replicated 1-m rows in the field (5). In that field trial Suwon 85 and P6028 plots (single rows) were growing next to Suwon 92 and Monon and therefore exposed to heavy spore showers from the susceptible wheats. Our equations do not account for exogenous inoculum except as an initial condition. Moreover they do not allow for any change in resistance as plants mature. There is evidence that the latent period becomes shorter on Suwon 85 and P6028 during and after anthesis (5). This could account for the large increase in rust severity often seen on these slow-rusting wheats just before ripening and for some of the discrepancy between final severity in the field and in the simulated epidemics. This change in susceptibility with age could be incorporated into these equations but we do not yet have sufficient data on the magnitude and timing of this change.

Field studies on large, isolated plots of Monon and Suwon 85 now are being conducted. Data from these trials in which there is less exogenous inoculum than in the earlier study (5), will help evaluate the predictive value of these equations. However, these equations are not designed to be a disease simulator. They do not allow for
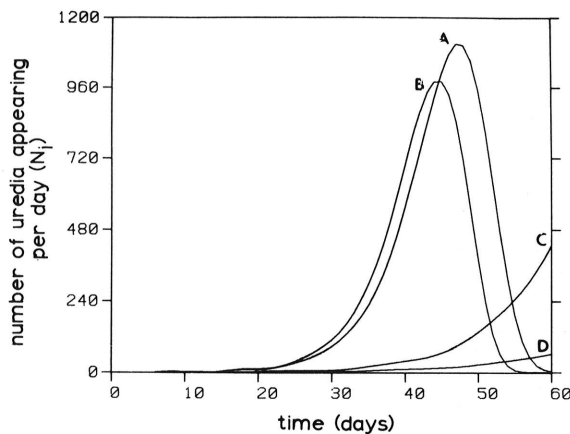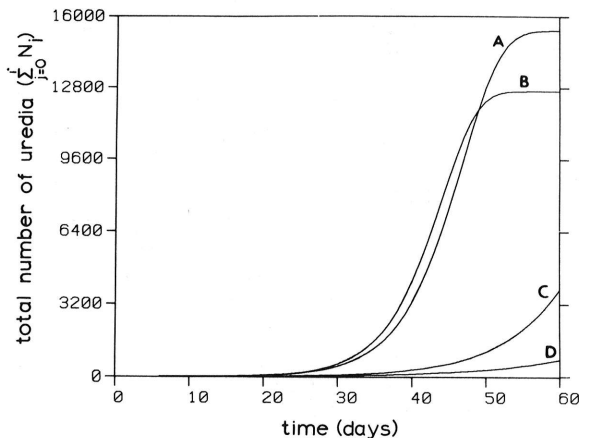

Fig. 2. Number of new uredia (N_i) of *Puccinia recondita* that appear each day on four winter wheat cultivars in an hypothetical epidemic. Curves represent data for: A = Monon, B = Suwon 92, C = Suwon 85, and D = P6028.

Fig. 3. Cumulative number of uredia ($\sum_{j=0}^{i} N_{i-j}$) of *Puccinia recondita* on four winter wheat cultivars in an hypothetical epidemic. Curves represent data for: A = Monon, B = Suwon 92, C = Suwon 85, and D = P6028.

weather-induced variations in spore production and dispersal, infection frequencies, and lesion growth that are typical of natural epidemics. Weather conditions are assumed to be constant and are reflected in the values of the resistance components measured in the greenhouse and laboratory and in the constant λ. To determine if the choice of a value of λ had an effect on the relative performance of cultivars in an hypothetical epidemic, disease progress curves were generated for the four cultivars when $\lambda = 0.18$, a value tenfold greater than originally calculated. This reduced the time between the onset of the epidemic and 100% severity on Monon and Suwon 92. When $\lambda = 0.018$, the areas under the disease progress curve for Suwon 92, Monon, Suwon 85, and P6028 were 18.4, 15.8, 1.3, and 0.3, respectively (units of per unit $\times$ time). When $\lambda = 0.18$ the corresponding areas were 8.0, 7.4, 0.5, and 0.2. The greater value of λ reduced the area under the disease progress curve, but the relative performance of the four cultivars was hardly affected. Thus, the choice of a value for λ is not critical to the relative outcome. It is the relative performance of a cultivar that these equations are intended to predict, given values for its slow-rusting resistance components. Over the interval of time in which severity on Monon or Suwon 92 increases from some low initial level to 100%, the equations show how much leaf rust will develop on a cultivar with any given level of resistance. The utility of these equations is that they serve as a guide in the hybridization of lines with different expressions of slow-rusting resistance and in selection of progeny from such crosses. These equations can be used to identify those resistance components that are most effective in slowing an epidemic. They will also indicate what the magnitude of any component should be to retard an epidemic to some desired degree. Finally, development of these equations has indicated areas in which information is especially scanty and therefore will guide further research. As more information becomes available, the equations can be refined so that they will more accurately predict the performance of a slow-rusting wheat.
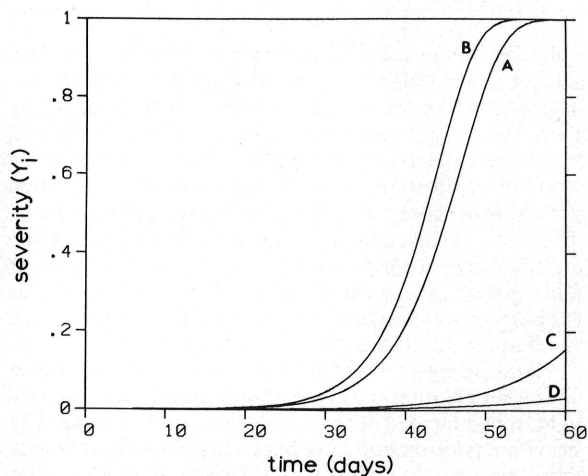

Fig. 4. Hypothetical disease progress curves for development of leaf rust on four winter wheat cultivars. Per unit severity (Y_i) = 1.00 when 34% of the leaf area is covered by uredia. Curves represent data for: A = Monon, B = Suwon 92, C = Suwon 85, and D = P6028.

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