Effects of Soil Moisture and Temperature on Phomopsis Seed Decay of Soybean in Relation to Host and Pathogen Growth Rates

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ABSTRACT

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Experiments were conducted in which soybean seeds were incubated for 3 days in pasteurized soil under specific temperature and moisture conditions followed by 18 days at soil temperature and moisture near optimum for emergence. In the temperature-moisture experiment, seeds from a seed lot with 100% incidence of seed coat infection after artificial inoculation with *Phomopsis longicolla* and from a corresponding control lot with < 1% infection were incubated at initial soil matric potentials of −0.008 and −15 bars combined with temperatures ranging from 12 to 36 C. Final relative emergence (inoculated lot emergence/control lot emergence) was lowest at −15 bars and 18 or 24 C. Performance of the inoculated and control lots did not differ significantly for any temperature at −0.008 bars. In the moisture experiment, seeds from a lot with 45% natural infection and from a control lot with < 1% infection were incubated at initial soil matric potentials ranging from −0.008 to −51 bars at 21–27 C. Relative emergence decreased with decreasing matric potential. Results of both experiments were compared with independent variables composed of measures of germination and emergence rates of the control lot under the same soil moisture and temperature conditions and the growth rate of *P. longicolla* at different temperatures on potato-dextrose agar osmotically adjusted with KCl. For the moisture experiment, final relative emergence was significantly ($P < 0.05$) correlated with times to 50% germination (Gt_{50}) or emergence (EMt_{50}) and the coefficient of velocity of emergence but not with pathogen growth rate (PGR). These results indicate that host growth was probably the controlling factor in determining the outcome of the host-pathogen interaction. For the temperature-moisture experiment, relative emergence was not significantly correlated with any independent variable, but disease incidence (DI = 1 [relative emergence]) was significantly correlated with a number of independent variables. In general, DI was more highly correlated with independent variables containing Gt_{50} than EMt_{50}, and the variable with the highest correlation involved the summation of values of PGR multiplied by Gt_{50} for the period before germination. These results indicate that DI may be related to Gt_{50} both because Gt_{50} is a measure of the duration of the host-pathogen interaction and because the inverse of Gt_{50} is a measure of host response during that period.

The concept that effects of the physical environment on preemergence damping-off are the outcome of the interaction of host and pathogen growth rates was first introduced by Leach (13) in the 1940s. In experiments with several vegetable species and *Pythium ultimum* Trow, *Rhizoctonia solani* Kühn, and *Phoma betae* Frank, Leach found that the effect of temperature on emergence of seedlings from infested soil or infected seeds was directly related to the ratio of host growth rate in the absence of pathogens to the in vitro growth rate of each pathogen. Leach's concept has been used with a varying degree of success in the interpretation of data from a number of host-pathogen systems (1,2,7,9,10,14,15,17,20), and modifications using seed exudation and pathogen inoculum density have been proposed (2,10,17). However, a number of questions about the process(es) underlying the concept remain to be answered.

Leach (13) used the coefficient of velocity of emergence (CVE) (equal to the inverse of mean time to emergence) as the measure of host growth and growth rate on solid or liquid media as the measure of pathogen growth. The ratio of coefficient of velocity of emergence to pathogen growth rate (PGR) was then compared with emergence. We believe that the primary questions about this procedure and the concept derived from it concern three issues: the method used to quantify host growth, the variable predicted by the ratio (i.e., emergence or disease), and the nature of the more basic process(es) for which host growth is a measure. In particular, the rate of emergence is related to two aspects of the system: the length of time during which the disease process takes place and the rate of host activity during this period. Because host response to pathogen challenge is an active process in most systems, it would be expected that conditions that limit the general metabolic activity of the host might also limit its ability to respond to the pathogen.

Phomopsis seed decay of soybean (*Glycine max* (L.) Merr.) can

kill or damage seeds both before harvest and after planting in soil. A high incidence of the causal fungi, particularly *Phomopsis longicolla* Hobbs, in soybean seed lots is often associated with high rates of preemergence mortality in the field (3,11,12), but correlations of mortality with incidence of the pathogens in seeds are inconsistent (12,18). In previous work, we found that Phomopsis seed decay sharply reduced emergence when infected seeds were incubated at soil water potentials that were too low to allow vigorous soybean germination and growth but high enough to allow pathogen activity (5). The objectives of the research described here were to investigate the interaction of soil temperature with this moisture effect and to use data from those experiments to evaluate the appropriateness of independent variables based on Leach's concept for the prediction of disease. A preliminary report has been published (6).

MATERIALS AND METHODS

Two experiments were performed to obtain disease data for comparison with values of independent variables. One experiment (termed the temperature-moisture experiment) involved initial incubation of seeds at different combinations of soil temperature and matric potential. The other experiment (the moisture experiment) involved initial incubation at a range of soil matric potentials under a single temperature regime and was similar to other experiments reported previously. Additional experiments were performed to obtain data on growth of *P. longicolla* in vitro and soybean seed germination rates in soil for use in independent variables.

Soil. Silty clay loam soil (7% sand, 78% silt, and 15% clay) was collected from a field that had been planted with soybeans for the previous 5 yr. The soil was shredded and stored in plastic bags at room temperature and 20–24% water content (weight of water per dry weight of soil). Soil used in the experiments was pasteurized by microwave oven treatment of 4-kg bags of soil for 425 sec (4). Soil water contents were adjusted to values corresponding to specific matric potentials by air-drying before planting seeds or by adding deionized water after planting as described previously (5).

Seeds. In the temperature-moisture experiment, seeds from a seed lot of cultivar Williams that initially had less than 1% incidence of *P. longicolla* were adjusted to 40% seed water content (wet weight basis), artificially inoculated with mycelial fragments of *P. longicolla*, incubated at 25 C for 18 hr, and then dried to 12% water content at room temperature (5). Control seeds from the same seed lot received autoclaved deionized water and were incubated in a similar manner. According to assays on Difco potato-dextrose agar (PDA) amended with 100 μg/ml streptomycin sulfate and 40 μg/ml chloramphenicol, inoculation resulted in colonization of 100% of seed coats but did not increase embryo infection incidence. In the moisture experiment, a naturally infected Williams seed lot with 45% incidence of *P. longicolla* was compared with another Williams seed lot with less than 1% infection incidence. All seeds were adjusted to 11–12% water content before planting.

TABLE 1. Coefficients of determination (R^2) for regressions of relative seedling emergence[a] from soybean seeds infected by *Phomopsis longicolla* with values of independent variables

Independent variable		R^2 for experiment	
Designation	Form[b]	Moisture	Temperature × moisture
1	$(PGR)^{-1}$	0.144	0.083
2	$(EMt_{50})^{-1}$	0.951**[c]	0.181
3	$(Gt_{50})^{-1}$	0.980**	0.160
4	CVE	0.956**	0.191
5	$(PGR*EMt_{50})^{-1}$	0.770*	0.110
6	$(PGR*Gt_{50})^{-1}$	0.917**	0.106
7	CVE/PGR	0.738*	0.108
8	$[\Sigma^{EMt_{50}} PGR]^{-1}$	0.888**	0.067
9	$[\Sigma^{Gt_{50}} PGR]^{-1}$	0.928**	0.107

[a] Relative seedling emergence = (emergence from seed lot with high incidence of *P. longicolla*)/(emergence from corresponding uninfected seed lot).

[b] PGR = growth rate of *P. longicolla* in vitro at temperature and water potential corresponding to soil temperature and water potential. EMt_{50} and Gt_{50} = time to 50% emergence and germination, respectively, for control seeds not infected by *P. longicolla*. CVE = coefficient of velocity of emergence.

[c] * = Significant at $0.05 > P \geq 0.01$ and ** = significant at $P < 0.01$. Based on six observations for the moisture experiment and 10 observations for the temperature-moisture experiment.

Experiments. In all emergence and germination experiments, seeds were incubated under specific temperature and moisture conditions for 3 days, then temperature and moisture in all treatments were adjusted to and maintained at values near optimum for soybean emergence (about 24 C and −0.008 to −3.0 bars maintained by intermittent watering). In all experiments, seeds were planted at a depth of 2.5 cm, the dry weight of soil in each container was equal for all treatments, and containers were covered with aluminum foil during the initial incubation period to retard evaporation.

In the temperature-moisture experiment, artificially inoculated and control seeds were planted in plastic trays 6 × 11 × 15 cm containing soil that was adjusted to water contents corresponding to −0.008 or −15 bars matric potential. Five replicate trays containing 15 seeds each were prepared for each treatment. Covered trays were placed in incubators at 12, 18, 24, 30, and 36 C (range ± 1 C) for 3 days, and then transferred to a growth chamber (12-hr photoperiod at 24 C). Emergence (any part of a seedling visible above the soil surface) was counted daily from 2 to 10 days after planting and every 2 or 3 days thereafter.

In the moisture experiment, naturally infected and control seeds were planted in plastic flats 6 × 24 × 50 cm containing soil that was adjusted to water contents corresponding to −0.008, −0.01, −0.1, −5, −15 and −51 bars matric potential. Two replicate flats containing three rows of 20 seeds each were prepared for each treatment. Covered flats were placed in a greenhouse at 21–27 C under natural light supplemented with 40W fluorescent lamps to maintain a 12-hr photoperiod. Flats were uncovered after a 3-day incubation, and emerged seedlings were counted 3, 5, 7, 9, 11, 16, and 21 days after planting.

To determine imbibition rates and time to 50% seed germination (Gt_{50}), seeds from the same lots used as uninfected controls in the above experiments were planted under soil temperature and moisture regimes corresponding to those described. Samples of 15 seeds each were removed from soil 0.5, 1, 2, and 3 days after planting, wiped clean of soil, counted for germination (protrusion of the radicle through the seed coat), and individually weighed. For treatments in which 50% germination did not occur by 3 days after planting, soil moisture and temperature were adjusted to −0.008 bars and 24 C at that time, and an additional sample was taken at 4 days. Gt_{50} was calculated by linear interpolation between the last count below and the first count above 50% germination. Time to 50% emergence (EMt_{50}) was calculated in a similar manner from emergence data for the lot with low incidence of *P. longicolla* in each experiment, and coefficient of velocity of emergence was calculated from the same data.

To determine the effects of temperature and water potential on in vitro growth of *P. longicolla*, Difco PDA was amended with KCl at concentrations corresponding to osmotic potentials of −3 (no KCl) to −180 bars at 12, 18, 24, 30, and 36 C. Six-millimeter-diameter agar plugs were transferred from the margins of actively growing colonies of the fungus on unamended PDA to the osmotically adjusted media. Three replicate plates for each treatment were enclosed in polyethylene bags and placed in incubators at 12, 18, 24, 30, and 36 C in the dark. Colony diameters were measured daily for 8 days along two perpendicular, randomly oriented axes. Mean growth rate for each treatment was calculated as the slope of mean colony diameter vs. time, excluding initial periods for some treatments during which no growth occurred.

Data analysis. Data for PGR in vitro, EMt_{50}, Gt_{50}, and CVE were used as the bases for a series of independent variables. Each independent variable was intended to predict either emergence or disease. In addition to independent variables composed of various

combinations of the basic variables (independent variables 1–7, Table 1; independent variables 1–6, Table 2), calculations for treatments in which Gt_{50} or EMt_{50} exceeded 3 days were made separately for the periods before and after 3 days, then these two values were summed. For example, for independent variable 19 in Table 2, a value for the −15 bar treatment in the moisture experiment was calculated by multiplying 3 days (the duration of −15 bar conditions) times PGR for −15 bars times EMt_{50} for −15 bars, multiplying 2.43 days (the duration of near-optimum conditions = EMt_{50} −3) times PGR for −3.0 bars (i.e., unamended PDA) times EMt_{50} for −0.01 bars, then adding these two products. Values of seedling emergence or disease variables were compared with independent variable values by linear or multiple regression. The seedling emergence variables used were mean final proportion of emerged plants for the seed lot with a high incidence of infection by *P. longicolla* and relative seedling emergence (relative emergence = final emergence from infected seeds/final emergence from corresponding control seeds). The disease variables used were the proportion of seeds not producing emerged plants, disease incidence (DI = 1, relative emergence), and the multiple-infection transformation of DI (8). For use in regressions, values of relative emergence that were greater than 1.0 were taken as being equal to 1.0, and values of DI that were less than 0 were taken to be equal to the multiple-infection transformation. All regressions were weighted using weights equal to the inverse of the theoretical binomial variance of the independent variable.

RESULTS

Seedling emergence. In the temperature-moisture experiment, emergence from inoculated seeds in the −15 bar treatments was much lower than emergence from control seeds at 18 and 24 C and somewhat lower at 12 and 30 C (Fig. 1A). Emergence was uniformly high from both inoculated and control seeds in the −0.008 bar treatments (Fig. 1B). In the moisture experiment, emergence from the seed lot with 45% incidence of *P. longicolla* was at least 20% lower than that from the control lot for all moisture treatments and decreased in successively drier treatments below −0.01 bars (Fig. 1C). Emergence from the control lot was above 90% at all moistures. These results are similar to those observed previously with artificially inoculated seed lots (5).

Gt_{50} and EMt_{50}. In the temperature-moisture experiment, Gt_{50} and EMt_{50} were shortest at 30 C and −0.008 bars (Fig. 2A,B). In the moisture experiment, Gt_{50} was shortest at −0.008 and −0.01 bars and increased in successively drier treatments below −0.01 bars

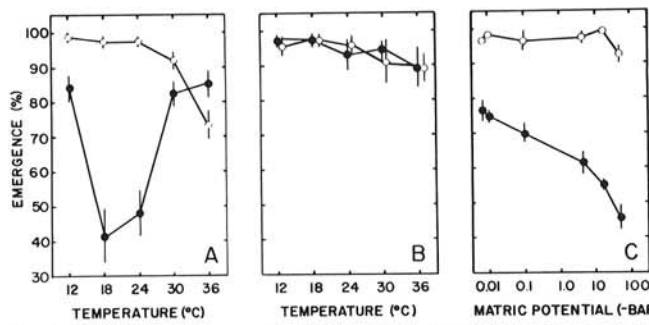

Fig. 1. Percent emergence of seedlings at 21 days from seeds artificially inoculated with *Phomopsis longicolla* (•) or autoclaved deionized water (o) in the **A,** −15 bar and **B,** −0.008 bar soil matric potential treatments of the temperature-moisture experiment. **C,** Percent emergence of seedlings from a naturally infected seed lot with 45% incidence of *P. longicolla* (•) and a control lot with less than 1% incidence of *P. longicolla* (o) in the moisture experiment. In both experiments, seeds were incubated under the indicated temperatures and matric potentials during the first 3 days after planting. Values are means of five replicate samples of 15 seeds each in A and B and six replicate samples of 20 seeds each in C. Error bars = ±1 standard error of the mean.

TABLE 2. Coefficients of determination (R^2) for regressions of disease incidence[a] for soybean seeds infected by *Phomopsis longicolla* with values of independent variables

Independent variable		R^2 for experiment	
Designation	Form[b]	Moisture	Temperature × moisture
Basic variables			
1	PGR	0.039	0.081
2	EMt_{50}	0.957**[c]	0.203
3	Gt_{50}	0.940**	0.320
4	$(CVE)^{-1}$	0.973**	0.216
Simple combinations			
5	PGR*EMt_{50}	0.632	0.508*
6	PGR*Gt_{50}	0.841*	0.771**
7	PGR/CVE	0.616	0.452*
Multiple combinations			
8	PGR + EMt_{50}	0.957**	0.477
9	PGR + EMt_{50} + $(PGR)^2$ + $(EMt_{50})^2$	0.992	0.509
10	PGR + EMt_{50} + $(PGR)^2$ + $(EMt_{50})^2$ + $(PGR)^3$ + $(EMt_{50})^3$	...	0.687
11	PGR + Gt_{50}	0.970**	0.614*
12	PGR + Gt_{50} + $(PGR)^2$ + $(Gt_{50})^2$	0.995	0.624
13	PGR + Gt_{50} + $(PGR)^2$ + $(Gt_{50})^2$ + $(PGR)^3$ + $(Gt_{50})^3$	...	0.767
14	PGR + EMt_{50} + Gt_{50}	0.974*	0.642
15	PGR + EMt_{50} + PGR*EMt_{50}	0.965	0.625
16	PGR + Gt_{50} + PGR*Gt_{50}	0.980*	0.825*
Summations[d]			
17	$\Sigma^{EMt_{50}}$ PGR	0.864**	0.478*
18	$\Sigma^{Gt_{50}}$ PGR	0.878**	0.779**
19	$\Sigma^{EMt_{50}}$ PGR*EMt_{50}	0.846**	0.581*
20	$\Sigma^{EMt_{50}}$ PGR*Gt_{50}	0.900**	0.738**
21	$\Sigma^{Gt_{50}}$ PGR*EMt_{50}	0.866**	0.868**
22	$\Sigma^{Gt_{50}}$ PGR*Gt_{50}	0.898**	0.931**

[a] Disease incidence = 1 (relative seedling emergence).
[b] PGR = growth rate of *P. longicolla* in vitro at temperature and water potential corresponding to soil temperature and water potential. EMt_{50} and Gt_{50} = time to 50% emergence and germination, respectively, for control seeds not infected by *P. longicolla*. CVE = coefficient of velocity of emergence.
[c] Significant at $0.05 > P >= 0.01$ and ** = significant at $P < 0.01$. Based on six observations for the moisture experiment and 10 observations for the temperature-moisture experiment.
[d] Summations involved in the summing of values derived for periods of different soil or seed conditions.

(Fig. 2C). EMt_{50} increased below −0.1 bars. Similar results have been reported previously (19).

In vitro growth rate of *P. longicolla*. The growth rate of the fungus was fastest within the −10 to −30 bar range at all temperatures tested (Fig. 3). Maximum growth rates were highest at 24 and 30 C and lowest at 12 C. In general, PGR was affected much less by temperature at osmotic potentials ≤ −60 bars than at higher potentials. Results at 24 C were similar to those reported previously for 25 C (16).

Evaluation of models. Within each experiment, coefficients of determination for regressions of each independent variable with emergence were highly correlated with those for relative emergence ($P < 0.0005$ by Spearman's coefficient of rank correlation), and similarly, coefficients of determination for regressions of each independent variable with the three disease variables were highly correlated with each other ($P < 0.0001$). Consequently, judgments about the relative appropriateness of the independent variables were made on the bases of regressions of relative seedling emergence and disease incidence against values of independent variables in conjunction with examination of plots of the dependent variables against the independent variables.

In the moisture experiment, relative emergence was relatively highly correlated with the values of a number of independent variables; however, relative emergence was not significantly correlated with any independent variable in the temperature-moisture experiment (Table 1, Fig. 4). For the moisture experiment, DI was significantly correlated with all of the emergence rate measures (EMt_{50}, Gt_{50}, and CVE) and with many combinations of PGR with EMt_{50} or Gt_{50} (Table 2, Fig. 5). For the temperature-moisture experiment, DI was significantly correlated with Gt_{50} and with most of the independent variables that contained PGR in combination with a measure of host growth rate. No disease or emergence variable in either experiment was correlated with PGR alone.

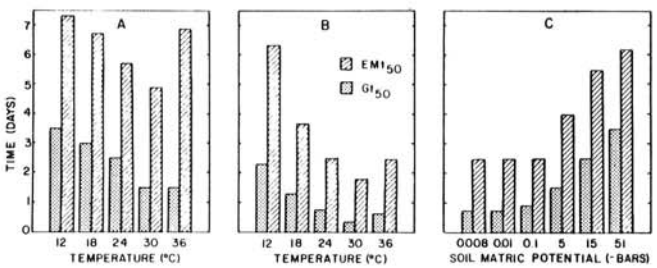

Fig. 2. Time to germination of 50% of seeds (Gt_{50}) and to soybean seedling emergence from 50% of planted seeds (EMt_{50}) for control seed lots (less than 1% incidence of *Phomopsis longicolla*) in the **A,** −15 bar and **B,** −0.008 bar treatments of the temperature-moisture experiment and **C,** in the moisture experiment. Seeds were incubated at the indicated temperatures and matric potentials during the first 3 days after planting. Values of Gt_{50} and EMt_{50} were estimated by linear interpolation from plots of percent germination and emergence vs. time after planting.

DISCUSSION

In general, the results of the experiments were in agreement with what might be predicted from a casual examination of the host and pathogen growth data in the context of Leach's (13) concept; disease increased with decreasing soil water potential in the moisture experiment, and disease was maximum in dry soil at intermediate temperatures in the temperature-moisture experiment (Figs. 1–3). However, correlations of observed seedling performance with values of independent variables varied according to whether disease or emergence was predicted, the form of the independent variable, and the type of experiment. Whether emergence or disease is a more appropriate response variable is in one sense a meaningless question, because all of the independent variables predicting emergence can be rearranged to predict disease [e.g., with regard to correlation, relative emergence = f(CVE)] is equivalent to DI = f(−CVE). However, the fact that ratios of host and pathogen growth rates were better correlated with DI than with relative emergence in the temperature-moisture experiment (models 5–7, Tables 1 and 2) indicates that system

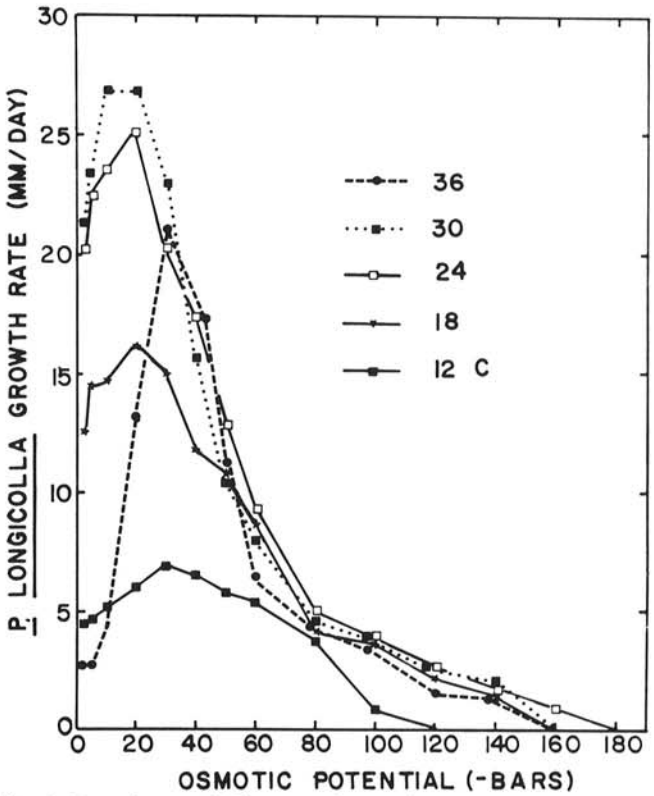

Fig. 3. Growth rate of *Phomopsis longicolla* on potato-dextrose agar osmotically adjusted with KCl. Values were derived by linear regression of colony diameter vs. time and are means of three replicate plates per treatment.

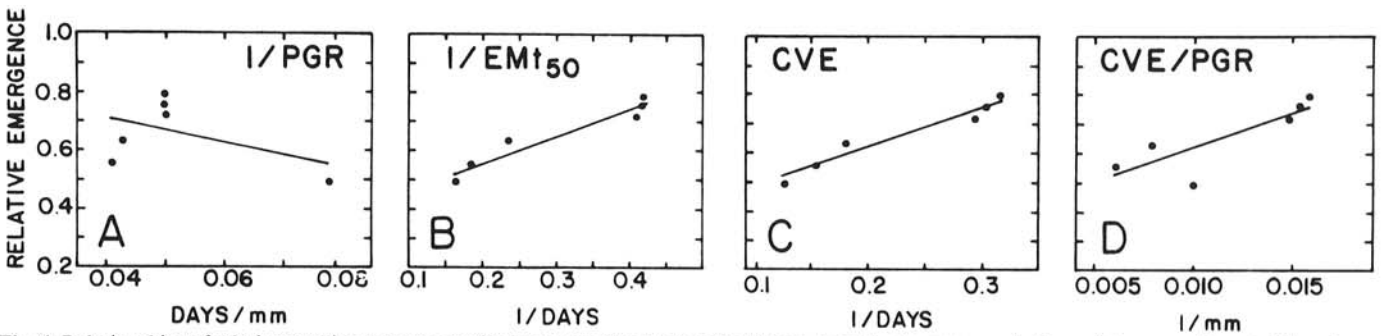

Fig. 4. Relationships of relative seedling emergence with representative host and pathogen growth rate measures in the moisture experiment with soybean and *Phomopsis longicolla*. **A,** Inverse of pathogen growth rate (PGR, independent variable 1, Table 1), **B,** inverse of time to 50% emergence of planted seeds (EMt_{50}, independent variable 2, Table 1), **C,** coefficient of velocity of emergence (CVE, independent variable 4, Table 1), and **D,** ratio of CVE to PGR (independent variable 7, Table 1).

behavior can be most simply predicted when DI is the dependent variable, and it is easier to conceptualize system behavior when the dependent variable (DI) is directly proportional to the causal variable (PGR).

In the moisture experiment, both relative emergence and DI were significantly correlated with values of many of the independent variables, including those based only on host growth (models 2–4, Tables 1 and 2; Fig. 4). This apparent insensitivity to structure of the independent variables was probably due to the relative constancy of PGR and the great variation in the host growth measures over the water potentials used. These results can be interpreted as indicating that the observed effects of soil water potential on disease were through an effect on the host rather than the pathogen.

In the temperature-moisture experiment, significant correlations were obtained only for DI, and only with Gt_{50} and independent variables that contained both host and pathogen growth rates (Tables 1 and 2; Fig. 5). In all cases, DI was more highly correlated with independent variables that contained Gt_{50} than with corresponding variables that contained EMt_{50}. The apparently closer relationship of DI to Gt_{50} than to EMt_{50} may be related to the closer correspondence of Gt_{50} to the time frame in which seed/seedling death occurs in Phomopsis seed decay. Preemergence death caused by the pathogen is probably the result of interference with growth and/or activity of the radicle or seedling root; plumule death alone would not be expected to affect emergence. Because the pathogen is initially located in the seed coat, expansion of the radicle through the seed coat during germination could reduce the risk of attack on the meristem by removing it from the vicinity of the pathogen. Thus, it may be that DI was correlated with independent variables incorporating EMt_{50} only because EMt_{50} is correlated with Gt_{50}. Similarly, the poor performance of Leach's original model could be due to CVE being relatively unrelated to Gt_{50}. Calculated CVE values are extremely sensitive to the distribution of EMt_{50} values for individual seeds; a single plant emerging long after most of the population can greatly decrease the calculated value of CVE. Thus, the relatively poor ability of independent variables incorporating CVE to predict emergence or disease could have been due to CVE being a relatively poor predictor of the overall behavior of the host population.

Independent variables that involved the summation values of PGR multiplied by Gt_{50} or EMt_{50} had consistently higher correlations than corresponding independent variables that involved only the summation of PGR in the temperature-moisture experiment (models 17–22, Table 2; Fig. 5E,F). These results may indicate that times to germination and emergence were related to disease because they were related both to the period during which the disease process took place and to host activity during that period. If this is true, then it is possible that a more accurate representation of the system could be obtained if PGR were compared with a more direct measure of host activity, such as respiration.

A major difference between the work described here and most previous research that has made use of Leach's concept is that we used changing, rather than constant, moisture and temperature conditions. This type of procedure was necessitated by our observation that damping-off caused by *P. longicolla* is most severe after incubation under soil moisture conditions that are unfavorable for soybean emergence (5). However, changing conditions are also present in practically all field planting environments. Although the use of a summation model that took into account these changing conditions improved the correlation in one case, it had little or no effect in three others (independent variables 5, 6, 17, and 18, Table 2). It is possible that these inconsistent results were due to host or pathogen behavior being altered after exposure to certain physical conditions; however, more work is needed to investigate this and other possible hypotheses.

Overall, our results are consistent with the following simple conceptual model: soil temperature and moisture affect preemergence damping-off by affecting the duration of a time period during which damage to the host occurs (e.g., the time

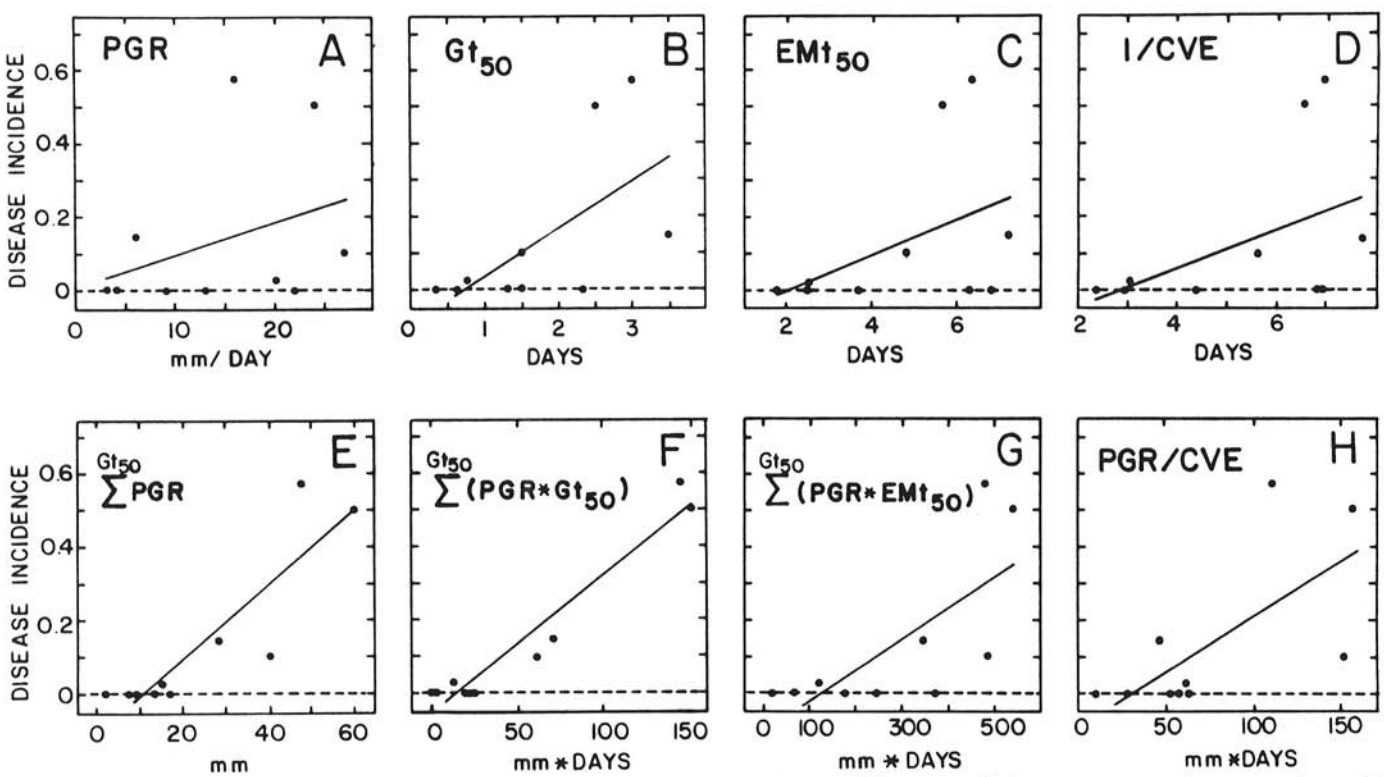

Fig. 5. Relationships of disease incidence with representative host and pathogen growth rate (PGR) measures in the temperature-moisture experiment with soybeans and *Phomopsis longicolla*. **A,** PGR (independent variable 1, Table 2), **B,** time to 50% germination of planted seeds (Gt_{50}, independent variable 3, Table 2), **C,** time to 50% emergence of planted seeds (EMt_{50}, independent variable 2, Table 2), and **D,** inverse of the coefficient of velocity of emergence (CVE, independent variable 4, Table 2). **E,** Calculated PGR during the period before germination (independent variable 18, Table 2), **F,** sum of PGR multiplied by Gt_{50} during the period before germination (independent variable 22, Table 2), **G,** sum of PGR multiplied by EMt_{50} during the period before germination (independent variable 21, Table 2), and **H,** ratio of PGR to CVE (independent variable 7, Table 2). Dashed lines represent disease incidence = 0.

between planting and germination or some other developmental stage) and by affecting the rate at which damage occurs during this period. This rate of damage is directly proportional to pathogen activity (a function of both PGR and the amount of initial inoculum) and inversely proportional to host activity. Increases in pathogen activity, increases in the time period, or decreases in host activity all increase the total amount of damage and thus the probability that a particular seed will not produce an emerged seedling. Other factors such as seed vigor, exudation of solutes, and the activities of other organisms can be considered to have their effect through the factors already present in this model. By progressively partitioning the factors affecting preemergence damping-off (i.e., by starting with a model such as the one above and then further investigating factors that affect each of its parameters), it may eventually be possible to define the basic process(es) that governs the effects of the environment on the outcome of this host-pathogen interaction.

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