# Host × Isolate Interactions in Corn Inbreds Inoculated with *Cochliobolus carbonum* Race 3

A. H. Hamid, J. E. Ayers, and R. R. Hill, Jr.

Graduate student and professor, Department of Plant Pathology, and research agronomist, Regional Research Laboratory, USDA, ARS, respectively, The Pennsylvania State University, University Park 16802.
Contribution 1273, Department of Plant Pathology, The Pennsylvania Agricultural Experiment Station. Authorized for publication 27 July 1981 as Journal Series Paper 6280.
Address of first author: Malaysian Agricultural Research and Development Institute (MARDI), Serdang, Selangor, Malaysia.
Portion of a Ph.D. thesis submitted to the Graduate School of The Pennsylvania State University, University Park.
Accepted for publication 21 January 1982.

## ABSTRACT

Hamid, A. H., Ayers, J. E., and Hill, R. R., Jr. 1982. Host × isolate interactions in corn inbreds inoculated with *Cochliobolus carbonum* race 3. Phytopathology 72:1169-1173.

Nine corn (*Zea mays*) inbred lines were examined after inoculation with isolates of *Cochliobolus carbonum* race 3 (= *Helminthosporium carbonum*) differing in levels of virulence. Reaction was measured by determining the effects of the lines on three components of parasitic fitness: disease efficiency, lesion length, and sporulation capacity. Differences among host genotypes were significant when tested against the six isolates. Similarly, isolates differed significantly in their degree of virulence on the host lines. Host × isolate interactions for the three fitness traits were significant suggesting that isolates responded differently to different hosts. Regression analysis was used to determine the stability of resistance. An inbred line was defined as having stable resistance when it had a low mean disease rating, a regression coefficient near zero, and small deviation from regression. The large variation detected in this study suggested that stable resistance in corn lines to *C. carbonum* race 3 would not easily be found.

*Additional key words*: genotype × environment interactions, horizontal resistance, maize.

Vanderplank (19) defined horizontal resistance (HR) as being equally effective against all races of a plant parasite with no interaction between the host and parasite genotypes. By this definition, whatever genetic variability for parasitic fitness exists in the parasite population must be nonspecific with respect to host genotype. A number of studies (3,10,15,16) have reported variability among pathogens grown on hosts with quantitative types of resistance. It seems reasonable to assume that host-pathogen genetic systems are dynamic, with genes in one part of the system interacting with genes in the other part of the system each with widely different magnitudes of variability.

A host genotype able to increase the latent period, decrease disease efficiency, decrease lesion length, decrease sporulation capacity, and decrease the infectious period, either singly or in combination, can be said to possess resistance that reduces the rate

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

of disease increase. Nelson (14) suggested that this type of resistance should be termed HR. Measurements of components of parasitic fitness could serve as an estimate of disease resistance of host genotypes.

Stability of cultivar disease resistance can be evaluated in terms of the numbers of years it retains its original level of resistance (20) and whether it is effective against a large number of different pathogen genotypes. Leonard and Moll (12) proposed the use of Eberhart and Russell's (4) analysis for determination of stability of host plant resistance. Each parasite genotype was considered as a separate environment to which the host plants were exposed. In turn, each host genotype was a separate environment for a particular parasite. Similar approaches were used in the evaluation of sorghum lines for resistance to midges (5) and in corn genotypes for resistance to several downy mildew pathogens (18).

This study was designed to evaluate the resistance of several corn inbred lines to isolates of *Cochliobolus carbonum* Nelson (= *Helminthosporium carbonum* Ullstrup) race 3. Disease efficiency (DE), the number of lesions resulting from a given amount of inoculum; lesion length (LL), the length (in millimeters) of lesions formed on inoculated plants; and sporulation capacity (SC), the number of spores per square centimeter of lesion, were used as the parameters in estimating resistance (8). In addition, we attempted to determine statistically the stability of HR by using the Eberhart and Russell (4) model.

## MATERIALS AND METHODS

Nine corn inbred lines (W153R, H95, Va26, B14A, Wf9, B37, A632, PaB8B, and MS72), which exhibited varying degrees of resistance to *C. carbonum* race 3 (1), were grown in the greenhouse until the time of inoculation. Six isolates of the fungus were used in the study. The isolates, collected from Pennsylvania in 1975, 1976, and 1978, were selected such that isolates TS75-14/2, RS419-3, and TS75-1/6 produced high values for DE, LL, and SC, respectively, and isolates TL-1, NL-2, and NS75-30/2 produced low values for these three characters (8).

Inoculum was prepared by incubating sections of the stock cultures (8) (greenhouse-grown corn leaf material infected with individual isolates) in petri dishes containing moistened filter paper at room temperature for 5 days to induce sporulation. Sporulating leaf tissues were washed with a 0.05% water agar solution and the volume was adjusted to provide $1.5–3.0 \times 10^3$ spores per milliliter. Plants were inoculated at the five-leaf stage approximately 3 wk after emergence.

Detailed procedures for the measurement of DE, LL, and SC were described previously (8). Briefly, assessments for DE were done by using a quantitative inoculator (17) to apply the spore suspension to the middle of the fifth leaf. Inoculated plants were placed in a dew chamber (model DC 20, Percival Refrigeration and Mfg. Co., Boone, IA 50036), maintained at 21 C for 15 hr, and transferred to a locally constructed growth chamber kept at 23 C for 5 days (12 hr light per day; 135 $\mu E \cdot m^{-2} \cdot sec^{-1}$. Lesion length was measured 14 days after inoculation, and SC was determined 4 days later after plants were placed in a dew chamber at 21 C with 12 hr of light per day (195 $\mu E \cdot m^{-2} \cdot sec^{-1}$). A split-plot design was used with isolates as whole plots and the host lines as subplots. The experiment was repeated three times with separate plants and batches of inoculum used for each replication.

Analysis of the data for each resistance parameter was carried out as described by Eberhart and Russell (4) except isolates were used in place of environments. Pooled standard error values were used to determine the significance of the regression coefficients.

## RESULTS

Analyses of variance of the three resistance parameters indicated that there were significant differences among host and isolate genotypes. Host × isolate interactions were also significant, which

TABLE 1. Mean disease efficiency, regression coefficients ($b_i$), and a portion of the regression analysis of variance for the stability index of nine corn inbred lines inoculated with six isolates of *Cochliobolus carbonum* race 3

| Inbred | *C. carbonum* isolate: NL-2 | TL-1 | TS75-1/6 | TS75-14/2 | NS75-30/2 | RS419-3 | Inbred means |
|---|---|---|---|---|---|---|---|
| MS72 | 12.9[w] | 4.9 | 3.7 | 3.7 | 11.2 | 8.4 | 7.5 |
| A632 | 7.2 | 11.3 | 11.5 | 5.0 | 7.7 | 7.0 | 8.3 |
| B14A | 4.8 | 14.0 | 6.7 | 11.0 | 17.9 | 26.2 | 13.4 |
| W153R | 24.9 | 3.0 | 12.6 | 4.2 | 11.7 | 18.4 | 12.4 |
| Wf9 | 15.6 | 6.4 | 4.4 | 6.6 | 5.2 | 16.8 | 9.2 |
| Va26 | 3.6 | 6.1 | 1.9 | 1.3 | 7.0 | 6.3 | 4.3 |
| PaB8B | 6.8 | 4.5 | 11.5 | 3.1 | 10.4 | 21.3 | 9.6 |
| B37 | 6.8 | 4.9 | 3.7 | 2.9 | 8.5 | 5.7 | 5.4 |
| H95 | 6.0 | 4.2 | 5.6 | 6.8 | 3.5 | 15.5 | 6.9 |
| Isolate means | 9.9 | 6.6 | 6.8 | 5.0 | 9.2 | 14.0 | 8.6[x] |

| Inbred × isolate analysis of variance<br>Source of variation | df | Mean squares[y] | $b_i$ |
|---|---|---|---|
| Inbreds | 8 | 162.1** | |
| Isolates in inbreds | [45] | [85.5**] | |
| Regressions | (9) | (219.3**) | |
| Common | 1 | 1,380.6** | |
| Residual | 8 | 74.1** | |
| Pooled deviations | (36) | (52.1**) | |
| MS72 | 4 | 35.8** | 0.8[z] b |
| A632 | 4 | 24.1** | −0.2 a |
| B14A | 4 | 137.7** | 1.6 ab |
| W153R | 4 | 126.9** | 1.9 ab |
| Wf9 | 4 | 42.6** | 1.4 b |
| Va26 | 4 | 14.2** | 0.5 ab |
| PaB8B | 4 | 42.8** | 1.6 ab |
| B37 | 4 | 10.6* | 0.4 a |
| H95 | 4 | 33.9** | 1.0 b |
| Error | 96 | 3.03 | |

[w] Values are expressed as percent and represent the mean of three replications.
[x] Grand mean.
[y] * and ** denote statistical significance, $P = 0.05$ and $P = 0.01$, respectively.
[z] Letters a and b indicate that the regression coefficients are significantly different from 1.0 and 0.0, respectively, $P = 0.05$.

indicated differential response of isolates to specific host lines. Since the interactions for all three resistance parameters were significant, generalizations could not be made on the relative performance of host lines over a range of pathogen genotypes.

The performance of host lines and their relationship to virulence of the isolates were reanalyzed by regressing the mean value of each inbred on the stability index (which is equivalent to the environmental index of Eberhart and Russell [4]). An inbred line was defined as having stable resistance if it had a low mean disease rating, a regression coefficient near zero, and small deviation from regression (5); however, it should be recognized that the values obtained for a given inbred are dependent on the other inbreds included in the test. Therefore, the values are relative, not absolute.

**Disease efficiency.** The inbred mean DE values ranged from 4.3 to 13.4% (Table 1). The analysis of the host × isolate interaction according to the model of Eberhart and Russell (4) indicated that the major portion of the interaction could be explained by the common regression of DE on the stability index. The mean square for the common regression, the average slope of the regression line, was ~19 times larger than the residual mean square even though the residual mean square was significant. Pooled deviations were also significant, which indicated that much of the data did not fit the common regression line.

The regression coefficients ($b_i$) for B37 and A632 were the only ones that did not differ significantly from 0.0. However, the deviation mean square was significant for both of these inbreds. The negative regression coefficient ($b_i = -0.2$) for A632 suggests that it does not respond to the isolates in a manner similar to the remaining host lines. For example, DE values for isolates TL-1 and TS75-1/6 on A632 were considerably above the mean for these two isolates over all inbreds and the DE for isolate RS419-3 on A632 was much less than the mean of all isolates across all inbreds.

When DE was used as a resistance parameter, none of these inbreds exhibited stable resistance as defined earlier; ie, a low mean disease rating, a regression coefficient near zero, and a small deviation from regression. B37 was closest to meeting the three criteria in that all isolates on B37 were below average for DE and the regression coefficient was low, but deviations from regression were significant.

**Lesion length.** Analysis of the inbred × isolate interaction of LL indicated that the common regression of LL on the stability index accounted for a major portion of the variability (Table 2). For this resistance parameter, the common regression mean square was several times larger than the residual mean square. Pooled deviations were also significant.

All of the regression coefficients for individual inbred lines were significantly different from 0.0; however, MS72, W153R, and Wf9 had nonsignificant deviation mean squares. Of these three, W153R had the lowest mean LL and a relatively low regression coefficient suggesting that it may possess the most stable resistance, in terms of LL, to the isolates studied. Although Va26 had a relatively low regression coefficient, deviation from regression was significant probably because isolate TS75-1/6 (a weak isolate over all inbreds) produced lesions on Va26 much larger than the mean of all isolates on all inbreds.

**Sporulation capacity.** Examination of isolate response on inbred lines for SC indicated that differential interactions had occurred (Table 3). For example, isolate NL-2 had a below average SC when averaged over all inbreds, but has a high SC on MS72 compared to the average SC of all isolates on MS72. Again, the common regression mean square was much higher than the residual mean square. Pooled deviations were significant.

Regression coefficients for the inbreds B14A, Va26, PaB8B, and B37 were not significantly different from 0.0; however, all of these had highly significant deviations from regression. Their SCs were below the average of all isolates across all inbreds and none met the criteria for stable resistance as defined earlier.

TABLE 2. Mean lesion length, regression coefficients ($b_i$), and a portion of the regression analysis of variance for the stability index of nine corn inbred lines inoculated with six isolates of *Cochliobolus carbonum* race 3

| Inbred | *C. carbonum* isolate | | | | | | Inbred means |
|---|---|---|---|---|---|---|---|
| | NL-2 | TL-1 | TS75-1/6 | TS75-14/2 | NS75-30/2 | RS419-3 | |
| MS72 | 3.6[w] | 3.0 | 2.1 | 2.7 | 6.6 | 5.7 | 3.9 |
| A632 | 2.4 | 3.1 | 1.9 | 3.1 | 7.0 | 5.6 | 3.8 |
| B14A | 1.7 | 3.4 | 2.3 | 3.2 | 4.8 | 4.9 | 3.4 |
| W153R | 2.1 | 2.0 | 2.2 | 2.1 | 2.8 | 3.0 | 2.4 |
| Wf9 | 2.5 | 2.4 | 2.2 | 2.1 | 5.3 | 5.2 | 3.3 |
| Va26 | 2.5 | 1.6 | 5.7 | 2.4 | 3.9 | 4.8 | 3.5 |
| PaB8B | 5.9 | 2.8 | 2.1 | 3.3 | 7.0 | 5.5 | 4.4 |
| B37 | 3.2 | 3.8 | 2.2 | 3.0 | 6.3 | 5.3 | 3.9 |
| H95 | 2.6 | 2.1 | 1.9 | 2.8 | 5.8 | 3.3 | 3.1 |
| Isolate means | 3.0 | 2.7 | 2.5 | 2.7 | 5.5 | 4.8 | 3.5[x] |

| Inbred × isolate analysis of variance<br>Source of variation | df | Mean squares[y] | $b_i$ |
|---|---|---|---|
| Inbreds | 8 | 6.6** | |
| Isolates in inbreds | [45] | [7.3**] | |
| Regressions | (9) | (28.3**) | |
| Common | 1 | 219.9** | |
| Residual | 8 | 4.3** | |
| Pooled deviations | (36) | (2.1**) | |
| MS72 | 4 | 0.4 | 1.4[z] ab |
| A632 | 4 | 0.8* | 1.5 ab |
| B14A | 4 | 1.7** | 0.8 b |
| W153R | 4 | 0.1 | 0.3 ab |
| Wf9 | 4 | 0.3 | 1.2 b |
| Va26 | 4 | 8.5** | 0.4 ab |
| PaB8B | 4 | 5.0** | 1.2 ab |
| B37 | 4 | 0.7* | 1.1 b |
| H95 | 4 | 1.4* | 1.0 b |
| Error | 96 | 0.2 | |

[w] Values are in millimeters and represent the mean of three replications.
[x] Grand mean.
[y] * and ** denote significance, $P = 0.05$ and $P = 0.01$, respectively.
[z] Letters a and b mean the regression coefficients are significantly different from 1.0 and 0.0, respectively, $P = 0.05$.

## DISCUSSION

Erosion of vertical resistance has been well documented in a number of disease situations, especially for foliar plant parasites (19). Epidemiologically, HR slows the rate of disease increase by reducing one or more components of parasitic fitness (14) and it should remain effective over a longer period (19). Such a form of host resistance appears to provide a better alternative in disease management with resistant cultivars. However, the results of a concurrent study (8) and that of Hill and Nelson (9), with *Helminthosporium maydis* race T, show that the components of parasitic fitness in these two *Helminthosporium* species are highly heritable. Therefore, it is probable that HR, once assumed to be stable, may be overcome by more virulent isolates of the parasite. An isolate with improved parasitic fitness has an epidemiological advantage over others. Host genotypes insensitive to these fitness attributes, either singly or in combinations, would be desirable in breeding for disease resistance.

The specificity of HR as defined by Nelson (14) to certain biotypes of plant parasites has been shown in a number of host-parasite systems: wheat-*Puccinia striiformis* (10,11); barley-*P. hordei* (3,15); potato-*Phytophthora infestans* (2); and wheat-*Erysiphe graminis tritici* (16). It has serious implications for plant breeders attempting to use HR to provide long-term stable disease resistance.

The regression model proposed by Eberhart and Russell (4) was utilized previously in the analysis of host-parasite interactions (5,12,18). Their approach provides a mechanism for understanding the association between plant parasites and their hosts by resolving complex interactions into a series of component linear responses. In doing so, predictable relationships between host genotypes and potential parasites can be determined. Faris et al (5) defined stable resistant cultivars of sorghum lines to midges (*Contarinia sorghicola*) as having a low mean adult emergence, a regression coefficient near zero, and a minimal deviation from regression. We derived the stability parameters in this study by regression analysis of each entry on the stability index. The regression coefficients were in effect measures of host responses to increased parasitic fitness of the isolates.

LL, as a resistance parameter, revealed fewer differential interactions between isolates and inbreds, suggesting that it may be a more valuable factor to use in a breeding program than DE or SC. Inbred W153R most nearly fit our definition of having stable resistance (in terms of LL) to the six isolates used in this study although it may not be the best inbred to use as a source of resistance since it does not appear to reduce DE and SC very much. Combination of LL resistance from W153R with resistance to DE and SC from other inbreds should be considered.

Related studies on the inheritance of resistance using DE, LL, and SC as resistance parameters have shown that resistance to one isolate of *C. carbonum* race 3 is inherited as a quantitative trait and that additive gene action is the most important component of the genetic variation (7). These results, coupled with those reported here suggest that breeders should use a wide variety of isolates in a breeding program designed to increase resistance to this fungus. Of the three resistance parameters studied, LL is probably the easiest to utilize and, based on our data, would tend to give the most stable resistance. Research with other disease models (6,15) has suggested that a reduced capacity for sporulation was associated with a slower rate of disease increase. Therefore, SC might be another important parameter for breeders to consider in breeding for resistance to *C. carbonum* race 3.

These results provide further evidence that HR sensu Nelson (14) may not exist in the absence of differential interactions.

The level of resistance may be adequate to prevent significant yield losses but the components of HR could well be controlled by genes with relatively small but measurable effects that could be overcome by changes in the pathogen (7).) HR may be the resultant cumulative effect of many of these genes (13,14).

TABLE 3. Mean sporulation capacity and regression coefficients ($b_i$), and a portion of the regression analysis of variance for the stability index of nine corn inbred lines inoculated with six isolates of *Cochliobolus carbonum* race 3

| Inbred | *C. carbonum* isolates | | | | | | Inbred means |
|---|---|---|---|---|---|---|---|
| | NL-2 | TL-1 | TS75-1/6 | TS75-14/2 | NS75-30/2 | RS419-3 | |
| MS72 | 663[w] | 329 | 423 | 305 | 168 | 941 | 471 |
| A632 | 113 | 251 | 433 | 371 | 322 | 727 | 370 |
| B14A | 128 | 154 | 133 | 294 | 266 | 203 | 197 |
| W153R | 301 | 662 | 373 | 98 | 504 | 657 | 433 |
| Wf9 | 344 | 321 | 254 | 916 | 695 | 746 | 546 |
| Va26 | 400 | 330 | 346 | 164 | 401 | 414 | 343 |
| PaB8B | 189 | 124 | 242 | 174 | 133 | 262 | 187 |
| B37 | 640 | 205 | 171 | 810 | 133 | 321 | 380 |
| H95 | 119 | 733 | 108 | 539 | 335 | 618 | 409 |
| Isolate means | 322 | 346 | 276 | 408 | 329 | 543 | 371[x] |

| Inbred × isolate analysis of variance<br>Source of variation | df | Mean squares[y] | $b_i$ |
|---|---|---|---|
| Inbred | 8 | 249,787** | |
| Isolates in inbreds | [45] | [136,807**] | |
| Regressions | (9) | (208,988**) | |
| Common | 1 | 1,213,783** | |
| Residual | 8 | 83,389** | |
| Pooled deviations | (36) | (118,761**) | |
| MS72 | 4 | 174,939** | 1.9[z] ab |
| A632 | 4 | 71,045** | 1.6 ab |
| B14A | 4 | 16,237** | 0.3 a |
| W153R | 4 | 166,405** | 0.7 b |
| Wf9 | 4 | 160,764** | 1.9 ab |
| Va26 | 4 | 32,913** | 0.0 a |
| PaB8B | 4 | 9,836** | 0.2 a |
| B37 | 4 | 280,534** | 0.6 |
| H95 | 4 | 156,176** | 1.7 ab |
| Error | 96 | 345 | |

[w] Values are expressed as the number of spores per square centimeter of lesion and represent the mean of three replications.
[x] Grand mean.
[y] ** denotes statistical significance, $P = 0.01$.
[z] Letters a and b mean the regression coefficients are significantly different from 1.0 and 0.0, respectively, $P = 0.05$.

## LITERATURE CITED


1. Castor, L. L., Ayers, J. E., Nelson, R. R., and Schreiber, A. J. 1976. The reaction of several corn inbreds to race 3 of *Helminthosporium carbonum*. Plant Dis. Rep. 60:827-830.
2. Caten, C. E. 1974. Intra-racial variation in *Phytophthora infestans* and adaptation to field resistance for potato blight. Ann. Appl. Biol. 77:259-270.
3. Clifford, B. C., and Clothier, R. B. 1974. Physiologic specialization of *Puccinia hordei* on barley hosts with non-hypersensitive resistance. Trans. Br. Mycol. Soc. 63:421-430.
4. Eberhart, S. A., and Russell, W. A. 1966. Stability parameters for comparing varieties. Crop Sci. 6:36-40.
5. Faris, M. A., Lira, de A., and Leao Veiga, A. F. de S. 1979. Stability of sorghum midge resistance. Crop Sci. 19:577-580.
6. Gregory, L. V., Royer, M. H., Ayers, J. E., and Nelson, R. R. 1971. The evaluation of relative parasitic fitness of isolates of *Helminthosporium maydis* race T. Phytopathology 71:354-356.
7. Hamid, A. H., Ayers, J. E., and Hill, R. R., Jr. 1982. The inheritance of resistance in corn to *Cochliobolus carbonum* race 3. Phytopathology 72:1173-1177.
8. Hamid, A. H., Ayers, J. E., Schein, R. D., and Hill, R. R., Jr. 1982. Components of fitness attributes in *Cochliobolus carbonum* race 3. Phytopathology 72:1166-1169.
9. Hill, J. P., and Nelson, R. R. 1976. Ecological races of *Helminthosporium maydis* race T. Phytopathology 66:873-876.
10. Johnson, R., and Bowyer, D. E. 1974. A rapid method for measuring production of yellow rust spores on single seedlings to assess differential interactions of wheat cultivars with *Puccinia striiformis*. Ann. Appl. Biol. 77:251-258.
11. Johnson, R., and Taylor, A. J. 1976. Isolates of *Puccinia striiformis* collected in England from the wheat varieties Maris Bacon and Joss Cambier. Nature (Lond.) 238:105-106.
12. Leonard, K. J., and Moll, R. H. 1979. Durability of general resistance: Evaluation of cultivar × isolate interactions. Pages 190–193 in: Proc. Symp. IX International Congress of Plant Protection. Vol. 1. August 5-11, 1979. Washington, DC.
13. Martin, T. J., and Ellingboe, A. H. 1976. Differences between compatible parasite/host genotypes involving the Pm4 locus of wheat and the corresponding genes in *Erysiphe graminis* f. sp. *tritici*. Phytopathology 66:1435-1438.
14. Nelson, R. R. 1973. Breeding Plants for Disease Resistance: Concepts and Applications. The Pennsylvania State University Press, University Park. 401 pp.
15. Parlevliet, J. E. 1976. Evaluation of the concept of horizontal resistance in the barley/*Puccinia hordei* host-pathogen relationship. Phytopathology 66:494-497.
16. Rouse, D. I., Nelson, R. R., MacKenzie, D. R., and Armitage, C. R. 1980. Components of rate-reducing resistance in seedlings of four wheat cultivars and parasitic fitness in six isolates of *Erysiphe graminis* f. sp. *tritici*. Phytopathology 70:1097-1100.
17. Schein, R. D. 1964. Design, performance, and use of a quantitative inoculator. Phytopathology 54:509-512.
18. Singh, J. 1975. Relative disease reaction in maize varieties to downy mildews over varied environments (based on international downy mildew nursery 1969–1973). Pages 129–154 in: Proceedings of a Symposium on Tropical Agriculture Researches. September, 1974 [Symposium on Downy Mildew of Maize]. Tropical Agric. Res. Series No. 8. Tropical Agriculture Research Center, Tokyo, Japan. 259 pp.
19. Vanderplank, J. E. 1968. Disease Resistance in Plants. Academic Press, New York. 206 pp.
20. Vanderplank, J. E. 1971. Stability of resistance to *Phytophthora infestans* in cultivars without R genes. Potato Res. 14:263-270.