Letter to the Editor

Environment as the Cause of Differential Interaction Between Host Cultivars and Pathogenic Races

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The classification of types of plant resistance into vertical (VR) and horizontal (HR) is based primarily on the presence or absence of differential interaction between host cultivars and pathogenic races. Therefore, the detection of differential interaction is extremely important for determining the nature of host resistance. A differential interaction is easy to infer when pathogenic races can attack some cultivars of the host and not others. When there is no clear-cut host-pathogen specificity, ie, when all the relevant races of the pathogen can attack all the relevant cultivars of the host, it is necessary to employ statistical tests to detect differential interaction. Analysis of variance (ANOVA) and the ranking order test are the two statistical tests that have been advocated (4,5) and employed for this purpose.

A true differential interaction implies that genetic variation in the host and in the pathogen are correlated. Thus, the durability of host resistance is a function of the presence or absence of such a correlation between variations in resistance and virulence.

Robinson (2) has pointed out several situations in which a false race × cultivar interaction may be found even in the absence of correlated genetic variation between resistance in the host and virulence in the pathogen, thus leading to misinterpretation of the nature of host resistance or of the ability of the pathogen to overcome resistance of the host. The most commonly encountered situations among these appear to be interplot interference and genotype (host/pathogen) × environment interaction. The importance of interplot interference in the measurement of HR of

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TABLE 1. Individual and combined ANOVA of two experiments involving eight wheat cultivars and six isolates of *Cercosporella herpotrichoides*[a]

Experiment	Term	d.f.	Untransformed		Arc sine		Rankit	
			s^2	F	s^2	F	s^2	F
1972	Cultivars (C)	7	36.9	39.6	4,424	40.4	19.25	47.5
	Isolates (I)	5	252.67	270.7	24,448	223.3	0	0
	C X I	35	2.36	2.5***[b]	282	2.6***	1.17	2.9***
	Residual (R)	423	0.93	...	110	...	0.41	...
1973	C	7	41.97	48.7	4,701	43.7	21.18	53.0
	I	5	115.93	134.4	11,824	110.3	0	0
	C X I	35	2.55	3.8***	303	2.8***	1.03	2.6***
	R	423	0.86		108		0.40	
1972 & 1973	C X I	35	2.83	1.4	328	1.5	1.01	<1.0
	C X I × E[c]	35	2.00	2.2***	221	2.2***	1.17	2.9***
	R	846	0.9		101		0.40	

[a]From Scott and Hollins (3).
[b]*** = Very highly significant.
[c]E = environment.

cultivars is now well recognized. However, there is as yet no experimental evidence for its role in causing a differential interaction between host cultivars and pathogenic races. Similarly, the importance of genotype × environment interaction in plant breeding has been well recognized, but its role in causing a differential interaction between cultivars of the host and races of the pathogen has not been adequately documented. This is indicated by the fact that in some recent studies (designed to detect whether differential interactions were also involved in "rate-reducing resistance") the possibility of environment-specific race × cultivar interactions was not considered. Both HR in the host and aggressiveness in the pathogen are generally quantitative characters and are likely to be affected by environmental variations. It is possible that expression of these characters differs with the environment. When studies are conducted in only one environment, it is not possible to detect the differential interaction of the host or of the pathogen with the environment. A race × cultivar interaction observed in the ANOVA of such studies could sometimes be due, actually, to race × cultivar × environment interaction. Such a possibility was clearly demonstrated by Scott and Hollins (3), part of whose data are presented in Table 1. It may also be noted that the interactions were not due to the effects of scale of disease severity measurement.

A differential interaction detected by the ranking order test could similarly be environment specific and could be due to race × environment, cultivar × environment, or race × cultivar × environment interaction. This is evident from the data of Latin et al (1). According to Latin et al (1) there was a differential interaction, in the 1978 experiment, between potato cultivars Kennebec (Ke) and Sebago (Se) and isolates II and IV of *Phytophthora infestans* (Table 2). Isolate IV was not included in the 1977 experiment. However, from apparent infection rates of isolate II on Ke and Se, it appears that there was a differential interaction between cultivars and years. If Latin et al (1) had included isolate IV in the 1977 experiment they may or may not have found a differential interaction between these cultivars and the isolates. Absence of differential interaction would have proved that the cultivar × isolate interaction in the 1978 experiment was due to cultivar × year interaction. The former would have only been possible if, in 1977, Se were to be more susceptible than Ke to isolate IV, in which case it would have been a case of cultivar × isolate × year interaction.

The other cultivar × isolate interaction found to be significant in the 1977 experiment, between cultivars Katahdin (Ka) and Superior (Su) and isolates I and II. The apparent infection rates of isolates I and II on Ka in the 1978 experiment suggest that the cultivar × isolate interaction observed in the 1977 experiment may have been year-specific. Even if Latin et al (1) had included Su in the 1978 experiment and observed a significant cultivar × isolate interaction, it may still not have confirmed that the cultivar × isolate interaction was a true one. This is because the differential interaction in the 1978 experiment would then have been mainly due to the differential susceptibility of Su to isolates I and II. The cultivar × isolate interaction in the 1977 experiment appears, on the contrary, to have been caused by the differential susceptibility of Ka to isolates I and II. It may be noted that the apparent infection rates of the two isolates did not significantly differ on Su in the 1977 experiment and on Ka in the 1978 experiment. Thus, on the whole, it appears that the environment did have a role in causing differential cultivar × isolate interactions in the experiments of Latin et al (1). The purpose of this letter is not to comment on the inferences drawn by Latin et al (1) but to draw attention to the importance of considering environmental differential interaction in studies on host-pathogen relationships.

TABLE 2. Apparent infection rates of isolates of *Phytophthora infestans* on four potato cultivars[a]

Cultivars	1977		1978		
	Isolates		Isolates		
	I	II	I	II	IV
Superior	0.48	0.43	...	...	...
Katahdin	0.30	0.54	0.33	0.32	0.21
Kennebec	0.35	0.49	0.26	0.24	0.19
Sebago	0.34	0.36	0.27	0.27	0.16

[a]From Latin et al (1).

From the practical point of view, the presence of environment-specific cultivar × isolate interactions requires that screening and selection be done under "on site" conditions (2). In other words, breeding for resistance to the pathogen should be carried out for each geographical pathosystem because improvement in resistance achieved in one environment may not be pertinent to another environment. A strong cultivar × isolate × environment interaction implies that environmental fluctuations might effectively prevent adaptation of a pathogen to the resistance of a cultivar. Although cultivar × isolate × environment interactions may partly explain the stability of natural host-pathosystems, a similar stability cannot be expected in crop-pathosystems where large areas are occupied by one or a few cultivars. Therefore, where cultivar × isolate × environment interactions are found to be significant, it may be worthwhile to study the stability of resistance of cultivar mixtures. It should also be possible to identify cultivars with HR that are generally insensitive to environmental variations. Identification of such cultivars will be most useful because breeding for resistance could then be done at a single location and exchange of identified material across environments for direct use in breeding for resistance will be possible without the fear of the resistance losing its effectivity. For rapid identification of such cultivars, it will be necessary to screen cultivars for stable HR in different environments by using as many appropriate isolates of the pathogen as facilities permit. It is extremely important to eliminate VR while conducting such tests.

LITERATURE CITED

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