Proposed Nomenclature for Pathogenic Races of *Exserohilum turcicum* on Corn

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Before 1960, polygenic resistance was the main type of resistance used in corn (*Zea mays* L.) against *Exserohilum turcicum* (Pass.) Leonard & Suggs (teleomorph, *Setosphaeria turcica* (Luttrell) Leonard & Suggs), the cause of northern leaf blight (8). Since 1960, a number of sources of dominant major genes that confer resistance to *E. turcicum* have been reported (4–7). Resistance conferred by genes *Ht1, Ht2*, and *Ht3* is characterized by chlorotic lesions with minimal sporulation by avirulent races of *E. turcicum*; gene *HtN* delays lesion development such that in the field, plants with *HtN* ordinarily remain free from lesions until shortly after pollination. Gene *Ht1* has been used extensively in commercial corn hybrids in the United States, but *Ht2, Ht3*, and *HtN* have received only limited, experimental use.

Although pathogenic specialization in *E. turcicum* had been reported earlier (11,12), distinct race designations were not assigned to virulence types until 1972, when Berquist and Masias (1) found isolates of *E. turcicum* virulent on inbred corn lines with *Ht1* in Hawaii. They designated the virulent isolates as Race 2 to distinguish them from isolates avirulent on corn lines with *Ht1*, which they designated Race 1. Virulence to corn lines with *Ht1* is inherited as a monogenic trait in *E. turcicum* (10). Genes *Ht2, Ht3*, and *HtN* are effective against both Race 1 and Race 2.

Race 2 eventually appeared in Indiana (16) and spread throughout most corn-producing areas of the United States. In 1976 an isolate of *E. turcicum* virulent on corn lines with *Ht2* and *Ht3* was collected in South Carolina, and subsequently it was designated Race 3 (13). In 1986, isolates of *E. turcicum* virulent on corn with *Ht2, Ht3*, and *HtN* were isolated in Texas (R. A. Frederiksen, *personal communication*); they are now described as Race 4 (14).

The existence of genes for virulence in *E. turcicum* to match four different resistance genes in corn provides a potential for many more pathogenic races to be identified. The current nomenclature of races of *E. turcicum* (Table 1) is not very satisfactory, because the numerical race designations have no correspondence to virulence formulae. As more races are identified, this situation will become more confusing.

Therefore, we propose that a new system of race nomenclature for *E. turcicum* similar to that used for races of *Phytophthora infestans* (Mont.) de Bary (2) be adopted for *E. turcicum*. In the new system, race designations are assigned according to the resistance genes that their virulence matches, i.e., according to their virulence formulae (Table 1). Thus, isolates currently designated Race 1 become Race 0 in the new system, indicating their lack of virulence on corn with any of the *Ht* genes. Current Race 2 becomes Race 1 to indicate its virulence to *Ht1*, and current Race 3 and Race 4 become Race 23 and Race 23N, respectively. Representatives of Race 0 (ATCC 64837), Race 23 (ATCC 64836), and Race 23N (ATCC 64834) have been deposited in the American Type Culture Collection under their old race designations.

In the new system, we omitted commas such as are used to separate resistance gene numbers in race designations for *P. infestans*, because with the current number of resistance genes in corn there is no ambiguity as to the virulence formula. New genes for resistance can be accommodated easily in this system, and the number of races can be expanded easily. As Day (3) has pointed out, this system is useful as long as the number of resistance genes used in race identifications does not become so large as to be unwieldy. At the current rate of identification and utilization of major genes for resistance to northern leaf blight in corn, the proposed system of pathogenic race nomenclature will remain useful for many years.

Race identifications in *E. turcicum* should be made with plants grown at moderate temperatures (near 20 C) and at light intensities between 25 and 50 klux (about 325–650 $\mu E \cdot m^{-2} \cdot s^{-1}$ average photosynthetic photon flux density), because reactions associated with genes *Ht1, Ht2*, and *Ht3* are sensitive to variation in temperature and light intensity. High summer temperatures in greenhouses may interfere with expression of virulence to lines with *Ht1* and *Ht3*, whereas low light intensity in greenhouses during the winter in northern latitudes may interfere with the expression of resistance of *Ht2*. Virulence of Race 1 (new system) to plants of inbred H4460*Ht1* was clearly expressed in a 22/18 C day/night temperature regime but was not expressed at 26/22 C (15). Similarly, Race 23 (old Race 3) and Race 23N (old Race 4) were virulent on inbreds H4460*Ht3* and B37*Ht3* at 22/18 C but avirulent at 26/22 C, even though both races expressed normal virulence on inbreds H4460 and B37 with no *Ht* genes at 26/22 C (9,14). Inbreds H4460*Ht2* and B37*Ht2* resisted Race 0 and Race 1 (old Race 1 and Race 2, respectively) at normal growth chamber light intensity of 50 klux (647 $\mu E \cdot m^{-2} \cdot s^{-1}$) but not at low light intensity; plants grown at 12 klux (162 $\mu E \cdot m^{-2} \cdot s^{-1}$) were fully susceptible (9,14). For best results, race identifications of isolates of *E. turcicum* should be based on disease reactions of plants grown in controlled environment chambers.

Table 1. Proposed revision of pathogenic race nomenclature for *Exserohilum turcicum*

New race designation	Old race designation	Disease reaction[a]			
		Ht1	*Ht2*	*Ht3*	*HtN*
0	1	R	R	R	R
1	2	S	R	R	R
2[b]	...	R	S	R	R
12[b]	...	S	S	R	R
23	3	R	S	S	R
23N	4	R	S	S	S

[a] R = resistant, S = susceptible.
[b] Hypothetical races not yet found; 10 other hypothetical races that could be distinguished with these four resistance genes are not listed.

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