Virulence of North American and European Races of *Puccinia striiformis* on North American, World, and European Differential Wheat Cultivars

C. DE VALLAVIEILLE-POPE and R. F. LINE, Agricultural Research Service, U.S. Department of Agriculture, Washington State University, Pullman 99167-6430

ABSTRACT

de Vallavieille-Pope, C., and Line, R. F. 1990. Virulence of North American and European races of *Puccinia striiformis* on North American, world, and European differential wheat cultivars. Plant Dis. 74:739-743.

Cultures of North American and European races of *Puccinia striiformis* were compared on North American (NA) and world/European (W/E) wheat (*Triticum aestivum*) differential cultivars. Races virulent on cultivar Fielder were virulent on cultivar Heines Kolben (resistance gene *Yr6*), and those virulent on Fielder and Heines VII were virulent on Heines Peko (*Yr2* + *Yr6*). Cultivars Strubes Dickkopf and Nord Desprez (*Yr3a* + *Yr4a*) provided additional differentiation of the North American races, and cultivar Stephens provided additional differentiation of the European races. Races virulent on Stephens and Druchamp were virulent on Nord Desprez, but some races virulent on Nord Desprez were avirulent on Druchamp and/or Stephens. Virulences differentiated on cultivars Yamhill, Tyee, Lemhi, and Produra were not differentiated using the W/E differentials.

Additional keywords: stripe rust, yellow rust

Races of *Puccinia striiformis* Westend. are identified based on their virulence on differential cultivars of wheat (*Triticum aestivum* L.). Different environmental conditions, classification systems, and differential sets have been used in different regions of the world to identify the races. Line et al (10) proposed and subsequently revised (5,9) a North American (NA) set of differentials for the United States. World and European (W/E) sets were proposed by Johnson et al (4) and subsequently revised and described by Stubbs (15). Because both the NA and W/E systems are open-ended, cultivars can be added when information about new races is obtained.

The NA system uses a Cereal Disease Laboratory (CDL)-coded number to designate the type culture (race) that represents a specific avirulence-virulence spectrum. In the W/E system, the cultivars used are assigned binary numbers to designate the races.

Each cultivar in the two systems has resistance genes that effectively differentiate races in the different regions. Some of these genes have been cataloged according to *Yr* symbols (12), while others are not known. The NA differential set consists of the following cultivars and their associated resistance genes (if known) according to McIntosh (12): Chinese 166 (*Yr1*), Moro (*Yr10*), Paha, Riebesel 47-51 (*Yr9*), and Lee (*Yr7*), all of which are represented in the world set; Heines VII (*Yr2*), which is in the European set; and Lemhi, Druchamp, Produra, Yamhill, Stephens, Fielder, and Tyee, which are not included in the world set or the European set. The world set, intended by Johnson et al (4) to identify virulences globally, consists of those cultivars already mentioned plus Heines Kolben (*Yr6*), Vilmorin 23, Strubes Dickkopf, Suwon 92/Omar (CI 13749) (equivalent to Paha [CI 14485] in the NA set), and Clement (*Yr9*), which was added later (15). The European set is composed of Hybrid 46 (*Yr3b* + *Yr4b*), Reichersberg 42, Heines Peko (*Yr2* + *Yr6*), Nord Desprez, Compair (*Yr8*), Carstens V, Spaldings Prolific, and Heines VII.

Stubbs (16) compared the virulence, evolution, and distribution of races of *P. striiformis* in many regions of the world and defined a set of epidemiological zones. However, North America, where stripe (yellow) rust occurs frequently, was considered only to a limited extent because some differentials used in North America (9) differed from those used in Western Europe (15). The purposes of this study were to use the differential cultivars developed for North America and Europe to compare the virulence of races of *P. striiformis* in North America and Europe, to evaluate the effectiveness and usefulness of the differential sets, and to determine possible genes for resistance in the differential cultivars.

MATERIALS AND METHODS

Type cultures of 23 of the 32 races of *P. striiformis* identified in North America before 1985 and cultures of seven races from Europe were tested two to six times on 13 North American, eight world, and eight European differentials. Seeds of the NA differentials came from the Cereal Disease Research Laboratory in Pullman, WA. Seeds of the W/E differentials were from the Plant Breeding Institute at Cambridge, England; the Cereal Disease Research Laboratory; and I.N.R.A. in La Minière, France. W/E differentials from all three sources were used for the studies in the United States, and the Cambridge source was used for the studies in France.

The 23 urediospore type cultures (races) from North America had been stored for several years in liquid nitrogen. Ten of the cultures (CDL 5, 7, 17, 18, 20, 21, and 24–27) originated from single urediospores. The other cultures had been transferred from single pustules for several generations using, whenever possible, cultivars that were uniquely susceptible to each specific race. Two cultures of CDL 11 (CDL 11a and 11b) and of CDL 26 (CDL 26a and 26b) were also included.

The inoculation techniques and incubation conditions described by Line et al (10) were used in North America. Briefly, seedlings in the two-leaf stage (about 12 days old) grown at cyclic temperatures of 5–20 C were inoculated with urediospores mixed with talc, placed in a dew chamber for 24 hr in the dark at 10 C, and then transferred to a growth chamber placed in a greenhouse with a cyclic temperature of 5–20 C and daylight supplemented by metal halide lamps for a 16-hr photoperiod.

The tests done in France used single-urediospore cultures representing each of seven W/E races (6E18, 40E8, 41E136, 45E140, 43E170, 106E139, and 232E233). Seedlings at the two-leaf stage were inoculated with a suspension of uredio-

Present address of first author: I.N.R.A. Laboratoire de Pathologie Végétale, 78850 Thiverval-Grignon, France.

Accepted for publication 5 March 1990 (submitted for electronic processing).

spores in mineral oil (Soltrol-170; Philips, Paris, France), placed in an incubation chamber for 24 hr in the dark at 8 C and 100% humidity, and then transferred to a growth chamber maintained at 14 C during an 8-hr dark period and at 17 C during a 16-hr light period with a light intensity of 300 $\mu E \cdot m^{-2} \cdot s^{-1}$ (4).

Infection types based on the 0–9 scale (8) were recorded twice 15–20 days after inoculation and again 25 or more days after inoculation. In the NA system (10), infection types 0–4 are considered avirulent (resistant reaction), and infection types 5–9 are considered virulent (susceptible reaction). In the W/E system (4), infection types 0–6 are considered avirulent, and infection types 7–9 are considered virulent. However, the differences between the two classification systems were not a factor in determining avirulence and virulence on the differential cultivars in our tests. All infections could be classified as avirulent (infection types 0–4) or virulent (infection types 7–9), except for those that changed from avirulent to virulent between the first and last recordings.

RESULTS

The virulence ratings of North American and European races on NA and W/E differentials are shown in Table 1. Three types of race-cultivar interaction are indicated based on the infection types recorded at the three times: virulence (V) at all three recording dates; avirulence (−) at all three recording dates; and AV, a shift from avirulence on the first recording date to virulence on the last recording date. Each North American race is designated by both the NA code and the W/E nomenclature proposed by Johnson et al (4).

Results with the three sources of W/E differential seed were consistent except for one source of Reichersberg 42 and one source of Hybrid 46, which were susceptible to some races when the other two sources were resistant. Results from these sources of the two cultivars were not used.

Some W/E cultivars differentiated the same races as did the NA cultivars. All races virulent on the NA differential Fielder (the North American races CDL 9, 14, 19, 20, 24, 25, 26a, 26b, 27, 28, and 30 and the European races 6E18 and 45E140) were also virulent on the W/E differential Heines Kolben, and all races virulent on both Fielder and Heines VII (the North American races CDL 9, 19, 24, 25, 26a, 26b, 27, and 28 and the European race 45E140) were virulent on the W/E differential Heines Peko. The races that were virulent on the NA and W/E differential Lee (the North American races CDL 17 and 31 and the European races 6E18, 43E170, and 106E139) were also virulent on the W/E differential Reichersberg 42. Similarly, all North American races were avirulent on both the NA differential Riebesel 47-51 and the W/E differential Clement.

Table 1. Virulences[a] of North American (NA) and world/European (W/E) races of *Puccinia striiformis* on North American, world, and European differentials[b]

NA	North American differentials[d]													World differentials[e]								European differentials[f]								W/E
races[c]	Lm	Ch	He	Mo	Pa	Dr	Ri	Pr	Ya	St	Le	Fi	Ty	Ch	Le	HK	Vi	Mo	SD	SO	Cl	Hy	Re	HP	ND	Co	CV	SP	He	races[g]
CDL 1	V	V	−	−	−	−	−	−	−	−	−	−	−	V	−	−	−	−	V	−	−	−	−	−	−	−	−	−	−	33E0
CDL 2	V	V	−	−	V	−	−	−	−	−	−	−	−	V	−	−	−	−	V	V	−	−	−	−	−	−	V	V	−	97E96
CDL 3	V	−	V	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	V	−	−	−	−	−	−	−	−	−	V	32E128
CDL 5	V	−	V	V	−	−	−	−	−	−	−	−	−	−	−	−	−	V	V	−	−	−	−	−	−	−	AV	AV	V	48E128
CDL 7	V	−	V	−	V	−	−	−	−	−	−	−	−	−	−	−	−	−	V	V	−	V	−	−	−	−	−	AV	V	96E129
CDL 8	V	−	V	−	−	−	−	−	V	−	−	−	−	−	−	−	−	−	V	−	−	−	−	−	−	−	−	−	V	32E128
CDL 9	V	−	V	−	−	V	−	V	−	−	−	V	−	−	−	V	V	−	V	−	−	−	−	V	V	−	V	−	V	44E172
CDL 11a	V	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	V	−	−	−	−	−	−	−	−	−	−	32E0
CDL 11b	V	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	0E0
CDL 14	V	−	−	−	−	−	−	V	−	−	−	V	−	−	−	V	−	−	V	−	−	−	−	−	−	−	−	−	−	36E0
CDL 15	V	−	AV	−	−	V	−	−	−	V	−	−	−	−	−	−	V	−	V	−	−	AV	−	−	V	−	AV	−	AV	40E8
CDL 17	V	V	V	−	−	−	−	−	V	−	V	−	−	V	V	−	AV	−	V	−	−	−	V	−	V	−	AV	AV	V	35E138
CDL 18	V	−	V	V	−	−	−	−	V	−	−	−	−	−	−	−	−	V	V	−	−	−	−	−	−	−	AV	AV	V	48E128
CDL 19	V	−	V	−	−	V	−	V	−	V	−	V	−	−	−	V	V	−	V	−	−	AV	−	V	V	−	AV	−	V	44E140
CDL 20	V	−	−	−	−	V	−	V	−	V	−	V	−	−	−	V	V	−	V	−	−	−	−	−	V	−	−	−	−	44E8
CDL 21	−	V	−	−	−	−	−	−	−	−	−	−	−	V	−	−	−	−	−	−	−	−	−	−	−	−	−	−	−	1E0
CDL 24	V	−	V	−	V	−	−	−	−	−	−	V	−	−	−	V	−	−	V	V	−	−	−	V	−	−	−	AV	V	100E132
CDL 25	V	−	V	−	−	V	−	V	V	V	−	V	−	−	−	V	V	−	V	−	−	−	−	V	V	−	V	−	V	44E172
CDL 26a	V	−	V	−	−	−	−	−	V	−	−	V	−	−	−	V	V	−	V	−	−	−	−	V	V	−	V	V	V	44E236
CDL 26b	V	−	V	−	−	−	−	−	V	−	−	V	−	−	−	V	−	−	V	−	−	−	−	V	−	−	AV	AV	V	36E132
CDL 27	V	−	V	−	−	−	−	−	−	−	−	V	V	−	−	V	AV	−	V	−	−	−	−	V	V	−	AV	−	V	36E140
CDL 28	V	−	V	V	−	−	−	−	−	−	−	V	−	−	−	V	V	V	V	−	−	−	−	V	V	−	AV	AV	V	60E140
CDL 29	V	−	V	V	V	−	−	−	−	−	−	−	−	−	−	−	−	V	V	V	−	−	−	−	−	−	−	−	V	112E128
CDL 30	V	−	−	V	−	V	−	V	−	−	−	V	−	−	−	V	V	V	V	−	−	AV	−	−	V	−	−	−	−	60E8
CDL 31	V	−	V	−	V	−	−	−	−	−	V	−	−	−	V	−	AV	−	V	V	−	V	V	−	V	−	AV	AV	V	98E139
	V	−	−	−	−	−	−	V	−	−	V	V	−	−	V	V	−	−	−	−	−	−	V	−	−	V	−	−	−	6E18
	V	−	−	−	−	V	−	−	−	−	−	−	−	−	−	−	V	−	V	−	−	−	−	−	V	−	−	−	−	40E8
	V	V	V	−	−	V	−	−	−	V	−	−	−	V	−	−	V	−	V	−	−	−	−	−	V	−	−	−	V	41E136
	V	V	V	−	−	V	−	V	AV	V	−	V	−	V	−	V	V	−	V	−	−	−	−	V	V	−	−	−	V	45E140
	V	V	V	−	−	V	−	−	−	V	V	−	−	V	V	−	V	−	V	−	−	−	V	−	V	−	V	−	V	43E170
	V	−	V	−	V	V	−	−	V	V	V	−	−	−	V	−	V	−	V	V	−	V	V	−	V	−	−	−	V	106E139
	V	−	V	−	V	V	V	−	V	V	−	−	−	−	−	−	V	−	V	V	V	V	−	−	V	−	V	V	V	232E233

[a] V = virulent, − = avirulent, AV = avirulent (infection type 2–4) for early recordings, but virulent (infection type 5–7) at later recordings.

[b] Cultivars with named resistance genes are Chinese 166 (*Yr*1), Heines VII (*Yr*2), Heines Kolben (*Yr*6), Heines Peko (*Yr*2 + *Yr*6), Lee (*Yr*7), Compair (*Yr*8), Riebesel 47-51 and Clement (*Yr*9), Moro (*Yr*10), Vilmorin 23 and Nord Desprez (*Yr*3a + *Yr*4a), and Hybrid 46 (*Yr*3b + *Yr*4b).

[c] a and b designate isolates identified as the same race using the North American differentials.

[d] Lemhi (Lm), Chinese 166 (Ch), Heines VII (He), Moro (Mo), Paha (Pa), Druchamp (Dr), Riebesel 47-51 (Ri), Produra (Pr), Yamhill (Ya), Stephens (St), Lee (Le), Fielder (Fi), and Tyee (Ty).

[e] Chinese 166 (Ch), Lee (Le), Heines Kolben (HK), Vilmorin 23 (Vi), Moro (Mo), Strubes Dickkopf (SD), Suwon 92/Omar (SO), and Clement (Cl).

[f] Hybrid 46 (Hy), Reichersberg 42 (Re), Heines Peko (HP), Nord Desprez (ND), Compair (Co), Carstens V (CV), Spaldings Prolific (SP), and Heines VII (He).

[g] The letter E separates the world race number from the European race number.

The European race 232E233 was virulent on both of these differentials.

The W/E differentials Vilmorin 23 and Nord Desprez and the NA differentials Stephens and Druchamp were susceptible to the same North American races (CDL 15, 19, 20, and 25) and European races (41E136, 45E140, 43E170, 106E139, and 232E233). Vilmorin 23, Nord Desprez, and Druchamp were also susceptible to North American races CDL 9 and 30 and European race 40E8, but Stephens was not susceptible to those races. In addition, Nord Desprez was also susceptible to five North American races that were not virulent on Druchamp or Stephens (CDL 17, 26a, 27, 28, and 31). Vilmorin 23 was susceptible to CDL 26a and 28 but had an AV expression for the other three races.

The NA differential Produra was susceptible only to the North American races CDL 9, 14, 19, 20, 25, and 30 and the European races 6E18 and 45E140, all of which were virulent on Fielder. The other races virulent on Fielder (CDL 24, 26a, 26b, 27, and 28) were avirulent on Produra.

Using Strubes Dickkopf, Hybrid 46, Spaldings Prolific, and Carstens V from the W/E sets, it was possible to identify virulences not detected with the NA differentials. All North American races except CDL 21 and one culture of CDL 11 (CDL 11b) were virulent on Strubes Dickkopf. CDL 21 (W/E race 1E0) was virulent only on Chinese 166, and CDL 11a (W/E race 32E0) and 11b (W/E race 0E0), which were virulent only on the NA differential Lemhi, were differentiated by the W/E differential Strubes Dickkopf. The North American races CDL 7 and 31 and the European races 106E139 and 232E233, which were all virulent on the NA differential Paha and the W/E differential Suwon 92/Omar, were also virulent on the W/E differential Hybrid 46. On the other hand, North American races CDL 2, 24, and 29, which were also virulent on the NA differential Paha and the W/E differential Suwon 92/Omar, were avirulent on the W/E differential Hybrid 46. CDL 2 and 26a were virulent on Spaldings Prolific and, along with CDL 9 and 25, were also virulent on Carstens V. The AV expression was observed most frequently on Hybrid 46, Spaldings Prolific, and Carstens V. Compair, which was resistant to all North American races and susceptible only to the European race 6E18, provided no additional information about the North American races.

North American races differentiated by the NA cultivars Yamhill, Tyee, and Lemhi could not be differentiated by any of the W/E cultivars. CDL 3 (avirulent on Yamhill) and CDL 8 (virulent on Yamhill) were identified as W/E race 32E128. Similarly, CDL 5 and 18 were identified as W/E race 48E128, and CDL 9 and 25 were identified as W/E race 44E172. CDL 27, virulent on Tyee, could not be differentiated from other races that were avirulent on Tyee using the W/E sets, and CDL 11b, virulent on Lemhi, was not virulent on any of the W/E differentials. On the other hand, European races were not further differentiated by the NA cultivars. European races 106E139 and 232E233 were virulent on Yamhill but were also the only European races virulent on Paha, Suwon 92/Omar, and Hybrid 46.

DISCUSSION

Data on the virulence of races of *P. striiformis* from North America and Europe on NA and W/E differentials enable comparison of virulences from the two regions and permit reconsideration of differential cultivar selection. Some virulences are present in both North America and Europe. In addition to virulences for *Yr*1, *Yr*2, and *Yr*7 and virulence on Suwon 92/Omar (Paha), which have been identified in both North America and Europe by Line (5,6) and Stubbs (15), virulence for *Yr*6 was detected using interchangeable differentials from the NA and W/E sets; virulences for *Yr*3a + *Yr*4a and *Yr*3b + *Yr*4b and on Strubes Dickkopf, Carstens V, and Spaldings Prolific were detected using the W/E sets; and virulences on Yamhill, Lemhi, Stephens, Druchamp, and Produra were detected using the NA set.

Fielder and Heines Kolben (*Yr*6) were susceptible to the same 11 North American races and two European races, suggesting that Fielder has *Yr*6. Nine of these 13 races were also virulent on Heines VII (*Yr*2) and Heines Peko (*Yr*2 + *Yr*6), providing further evidence that Fielder may have *Yr*6. In addition, six North American and two European races that were virulent on the NA differential Produra were also virulent on Fielder. Produra may, therefore, have common genes with Fielder, in which case Produra must have one or more additional genes. Virulence for *Yr*6 occurs widely throughout Europe (15) and has existed in North America since the 1930s (2). In North America, virulence for Fielder has been prevalent since the mid-1970s (9). In Europe, virulence for *Yr*6 has been especially common in Great Britain since 1984 (1) and more recently in France (3).

Virulences for *Yr*3a + *Yr*4a, common in Europe (15), are present in half of the North American races tested. The *Yr*3a + *Yr*4a genes have been postulated to be in the W/E differentials Nord Desprez and Vilmorin 23 because they have the same resistance pattern as Cappelle-Desprez (19), which Lupton and Macer (11) reported to have these two genes. Eleven of the North American races and six of the European races were virulent on Nord Desprez and Vilmorin 23. Of these 17 races, four North American and five European races were virulent on the NA differential Stephens, and six North American races, including the four virulent on Stephens, and six European races were virulent on the NA differential Druchamp. This suggests that Stephens, which has Nord Desprez as one parent, and Druchamp, which has Vilmorin 27 as one parent, have *Yr*3a and/or *Yr*4a, that they have one or more genes in common, and that they have one or more genes that are different.

Most European races have virulences for both *Yr*2 (Heines VII) and *Yr*3a + *Yr*4a (Vilmorin 23, Nord Desprez), because most cultivars in the region have *Yr*2 and/or *Yr*3a + *Yr*4a (13,15). In North America, the virulences for *Yr*2 and *Yr*3a + *Yr*4a seem to be less closely associated.

Virulence for *Yr*3b + *Yr*4b (W/E differential Hybrid 46), which is prevalent in Europe (15), is uncommon in the North American races tested. The two North American races that were virulent on Hybrid 46 were also virulent on Suwon 92/Omar (Paha). European races show similar reactions on these two differentials (15), suggesting that Paha and Suwon 92/Omar could have *Yr*3b + *Yr*4b. However, three other North American races (CDL 2, 24, and 29) that were virulent on Suwon 92/Omar were not virulent on Hybrid 46, indicating that Hybrid 46 has a gene that is not present in Suwon 92/Omar.

The two North American and three European races that were virulent on Lee were also virulent on the W/E differential Reichersberg 42. Because Reichersberg 42 did not provide any additional differentiation of the North American races, a second gene in addition to *Yr*7 (15) cannot be confirmed.

All but two of the North American races (CDL 11b and 21) and one of the European races (6E18) were virulent on the W/E differential Strubes Dickkopf. At the same time, all but one of the North American races (CDL 21) and all of the European races tested were virulent on the NA differential Lemhi. These two cultivars, therefore, are not interchangeable.

Virulence for Suwon 92/Omar existed in a race that was identified in the early 1960s in North America (5) and has been prevalent since 1974 (9), when Suwon 92/Omar was replaced by Paha, a sister selection with the same resistance that was commercially grown in the northwestern United States. In earlier tests (R. F. Line, *unpublished*), Spaldings Prolific and Paha (Suwon 92/Omar) differentiated race CDL 2 from other races. Because Spaldings Prolific did not provide further differentiation and because infection types on the cultivar were more variable than on Paha (Suwon 92/Omar), Spaldings Prolific was not used as an NA differential. In this study, three other races (CDL 7, 24, and 31) had an AV expression on Spaldings

Prolific and were virulent on Paha (Suwon 92/Omar), one race (CDL 26a) was virulent on Spaldings Prolific and avirulent on Paha, and one race (106E139) was avirulent on Spaldings Prolific and virulent on Paha.

Virulence on Carstens V, reported in North America in the late 1960s (17), was detected in four North American races.

Some virulences are present in one region but not in the other. For example, virulence for *Yr*9, which is common in Europe (15), was not detected in North America (9). All North American races were avirulent on the NA differential Riebesel 47-51 and the W/E differential Clement, which have both been reported to have *Yr*9 from rye (12). However, these cultivars differed in at least one other region of the world: Riebesel 47-51 was resistant and Clement was susceptible to a race from Kenya (18). In Europe, virulence on Clement was detected in the mid-1970s and is widespread because cultivars with *Yr*9 are grown extensively there (15). Cultivars with *Yr*9 parentage have not yet been developed in North America, but the current use of Riebesel 47-51 in North American breeding programs may change this situation. In contrast, virulences on Moro (*Yr*10) and Tyee are present in North America (9) but have not been detected in Europe. Five of the North American races were virulent on Moro, which has *Yr*10 from the wheat introduction PI 178383, used extensively in North American breeding programs.

Some virulences are either absent or very rare in both regions. Virulence for *Yr*8, for example, was not detected in North America and was uncommon in most of Europe (15). The European race 6E18, which is virulent on Compair, was found in southern France in 1986 (3) but is quite different from the other races in Europe because of its avirulence on Strubes Dickkopf. The *Yr*8 differential Compair, which is derived from *Aegilops comosa* L., has not been used in North American or European breeding programs (15).

Four North American races with narrow virulence spectra using the NA differentials also had a narrow host range using the W/E differentials. CDL 1 and 3 were present in the northwestern United States and bordering Canadian provinces in 1960 and are assumed to be the ancestors of the more recent CDL races (7). The CDL 1 and 3 cultures tested, obtained in the early 1960s, were virulent on only one W/E differential (Strubes Dickkopf) not represented in the NA set. In addition, two more recently identified races, CDL 11 (virulent only on the NA differential Lemhi) and CDL 21 (virulent only on the NA differential Chinese 166), had little or no additional virulence on the W/E sets. CDL 11a was virulent only on W/E differential Strubes Dickkopf, and CDL 11b and 21 were not virulent on any W/E differential not represented in the NA set.

Use of the W/E differentials showed that some North American races are more complex than previously thought. For example, all North American races that were virulent on Stephens (CDL 15, 19, 20, and 25) were also virulent on Druchamp and Nord Desprez, all races that were avirulent on Stephens but virulent on Druchamp (CDL 9 and 30) were also virulent on Nord Desprez, and five additional races that were avirulent on Stephens and Druchamp (CDL 17, 26a, 27, 28, and 31) were virulent on Nord Desprez. This suggests that the first set of races is more complex than the second and that the second set is more complex than the third.

Two cultures of the same race using the NA set were different using the W/E sets. Strubes Dickkopf differentiated the cultures of race CDL 11, and Vilmorin 23, Nord Desprez, Carstens V, and Spaldings Prolific differentiated the cultures of race CDL 26. The dissimilarities between the cultures were revealed only by including additional criteria—that is, by testing with the W/E sets. In contrast, the North American race pairs CDL 3 and 8, 5 and 18, 9 and 25, and 19 and 25 were differentiated by the NA differential Yamhill but could not be differentiated with the W/E differentials.

Many of the differentials in the W/E sets are identical to or interchangeable with differentials in the NA set and provide the same information as do the NA differentials about the virulences of North American races. Of those differentials that are not similar, Strubes Dickkopf and Nord Desprez may be useful in providing supplemental information about virulences of North American races. On the other hand, Compair, which is resistant to all races, and Vilmorin 23, which resembles Nord Desprez but has less clearly defined infection types, would not be useful additions to the NA set. Similarly, Hybrid 46, Carstens V, and Spaldings Prolific, which also have infection types that are not clearly defined, would add little to the understanding of North American races. The slow-rusting, or AV, reaction sometimes observed with Carstens V has been attributed, in part, to an inability of this differential to express its resistance when it receives less light (14), which can occur when leaves become large enough to provide shade.

Although several W/E differentials provide supplemental information regarding North American races, the present NA differential set effectively describes the North American population of *P. striiformis* more precisely than does the W/E differential set. This is because the NA differentials Druchamp, Stephens, Produra, Yamhill, Tyee, and Lemhi have resistance genes or gene combinations that are not found in the W/E differential set.

Several cultivars from the NA set may be useful in studying European races. Yamhill, susceptible to the W/E races 106E139 and 232E233 and resistant to the others, may further differentiate the W/E races, and Stephens, resistant to 40E8, may also give useful information. On the other hand, Druchamp, Produra, and Tyee do not offer information different from that provided by the W/E sets for the seven W/E cultures tested.

At this time we do not recommend adding differentials from the NA set to the W/E sets or from the W/E sets to the NA set. However, further comparison using both the W/E and NA sets may improve our understanding of worldwide virulences. This work also points out the need for studying the genetics of resistance in the differential sets.

LITERATURE CITED

1. Bayles, R. A., Channel, M. H., and Stigwood, P. L. 1988. Yellow rust of wheat. Pages 11-16 in: U.K. Cereal Pathogen Virulence Survey, 1987. Annual Report, U.K. Cereal Pathogen Virulence Survey Committee, Cambridge.
2. Bever, W. M. 1934. Physiologic specialization in *Puccinia glumarum* in the United States. Phytopathology 24:686-688.
3. de Vallavieille-Pope, C., Picard-Formery, H., Radulovic, S., and Johnson, R. 1990. Specific resistance factors to yellow rust in seedlings of some French wheat varieties and races of *Puccinia striiformis* Westend. in France. Agronomie (Paris) 2:101-113.
4. Johnson, R., Stubbs, R. W., Fuchs, E., and Chamberlain, N. H. 1972. Nomenclature for physiologic races of *Puccinia striiformis* infecting wheat. Trans. Br. Mycol. Soc. 58:475-480.
5. Line, R. F. 1972. Patterns of pathogenicity of *Puccinia striiformis* in the United States. Proc. Eur. Mediterr. Cereal Rusts Conf. 3:181-185.
6. Line, R. F. 1980. Pathogenicity and evolution of *Puccinia striiformis* in the United States. Proc. Eur. Mediterr. Cereal Rusts Conf. 5:163-166.
7. Line, R. F. 1983. Pathogenicity and evolution of *Puccinia striiformis* in the United States. Pages 31-35 in: The Current Status of Stripe Rust Research; Proceedings of a National Workshop. L. O'Brien, ed. Victorian Crops Research Institute, Horsham, Victoria, Australia.
8. Line, R. F., Konzak, C. F., and Allan, R. E. 1974. Evaluating resistance to *Puccinia striiformis*. Pages 125-132 in: Wheat Induced Mutations for Disease Resistance in Crop Plants. International Atomic Energy Agency, Vienna, VA.
9. Line, R. F., Qayoum, A., and Chen, X. M. 1988. Evolution and distribution of *Puccinia striiformis* in North America and inheritance of stripe rust resistance in differential cultivars. Proc. Eur. Mediterr. Cereal Rusts Conf. 7:107-112.
10. Line, R. F., Sharp, E. L., and Powelson, R. L. 1970. A system for differentiating races of *Puccinia striiformis* in the United States. Plant Dis. Rep. 54:992-994.
11. Lupton, F. G. H., and Macer, R. C. F. 1962. Inheritance of resistance to yellow rust (*Puccinia glumarum* Erikss. & Henn.) in seven varieties of wheat. Trans. Br. Mycol. Soc. 45:21-45.
12. McIntosh, R. A. 1983. A catalogue of gene symbols for wheat (1983 edition). Proc. Int. Wheat Genet. Symp. 6:1197-1254.
13. Röbbelen, G., and Sharp, E. L. 1978. Mode of Inheritance, Interaction and Application of Genes Conditioning Resistance to Yellow Rust.

Advances in Plant Breeding, Vol. 9. Verlag Paul Parey, Berlin. 88 pp.
14. Stubbs, R. W. 1967. Influence of light intensity on the reactions of wheat and barley seedlings to *Puccinia striiformis*. Phytopathology 57:615-617.
15. Stubbs, R. W. 1985. Stripe rust. Pages 61-101 in: The Cereal Rusts, Vol. 2. A. P. Roelfs and W. R. Bushnell, eds. Academic Press, Orlando.
16. Stubbs, R. W. 1988. Pathogenicity analysis of yellow (stripe) rust of wheat and its significance in a global context. Pages 23-38 in: Breeding Strategies for Resistance to the Rusts of Wheat. G. Hettel, ed. CIMMYT, Mexico City, Mexico.
17. Stubbs, R. W., Fuchs, E., Vecht, H., and Basset, E. J. W. 1974. Report on the international survey of factors of virulence of *Puccinia striiformis* Westend. in 1969, 1970 and 1971. Sticht. Ned. Graan-Centrum Tech. Ber. 21. 87 pp.
18. Stubbs, R. W., and Hua-an, Y. 1988. Pathogenicity of *Puccinia striiformis* for wheat cultivars with resistance derived from rye. Proc. Eur. Mediterr. Cereal Rusts Conf. 7:110-112.
19. Zadoks, J. C. 1961. Yellow rust on wheat, studies in epidemiology and physiologic specialization. Tijdschr. Plantenziekten 67:69-256.