Comparison of Virulence and Isozyme Phenotypes of Pgt-QCCJ and Great Plains Races of *Puccinia graminis* f. sp. *tritici*

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ABSTRACT

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Stem rust race Pgt-QCCJ was first found in the Great Plains of the United States in 1989, collected primarily from barley. This race became a major part of the *Puccinia graminis* f. sp. *tritici* population, even though it is virulent to only a few hard red winter wheat cultivars in the central Great Plains and to barley in the northern Great Plains. It threatens barley production in the northern Great Plains of the United States and Canada

due to virulence to *Rpg-1*. Six differences in virulence and two in isozyme banding patterns from the most similar stem rust races make it unlikely that QCCJ arose as a mutant. Thus, QCCJ likely arose through sexual or parasexual recombination. Sexual recombination in the Great Plains is unlikely, as it has not been detected in many years. Avirulence to 'McNair 701' is only known from the Pacific Northwest of the United States and adjacent Canada. The rust population in this area is of sexual origin, and the pattern of virulence/avirulence and isozyme banding for QCCJ occurs there. Pgt-QCCJ likely originated in the Pacific Northwest during or before 1989 and was wind-transported into the Great Plains.

Stem rust race Pgt-QCCJ (12) appeared in the 1989 United States annual race survey of *Puccinia graminis* f. sp. *tritici* (10). The race was unusual in that it was avirulent on 'McNair 701', the wheat cultivar on which all cultures from the field were increased. This immediately distinguished QCCJ from all other cultures obtained from the Great Plains. It became obvious in analyzing the data (10) that QCCJ was most frequently (95%) isolated from collections made from *Hordeum* spp. in the upper Midwest, including commercial cultivars with *Rpg-1*. The *Rpg-1* gene has provided resistance to *P. graminis* f. sp. *tritici* for nearly 50 years (17). Thus, stem rust races virulent to *Rpg-1* posed a threat to barley production in the Great Plains. Potential sources for race QCCJ were as a mutant from one of the existing asexual race clusters, a recombinant from the sexual stage on *Berberis vulgaris* L., a parasexual cross between existing races, or an introduction from outside the Great Plains. The *P. graminis* f. sp. *tritici* population in the Great Plains reproduces strictly by asexual, clonal propagation of urediniospores (9). The alternate host (*Berberis*) was eradicated from much of the northern Great Plains and has not been shown to be involved in wheat stem rust development for over 50 years (6).

The race population of *P. graminis* f. sp. *tritici* in the Great Plains in 1975 (14) was characterized by 17 races that grouped into six distantly related clusters of races (differing in eight to 16 virulences) of closely related races (differing by a single virulence gene) (11). This characterization was based on virulence patterns to 28 selected host lines with single stem rust-resistance genes. In examining cultures available from earlier years, two more clusters were found, Pgt_KCJ and H_LG. Since then, over 7,000 additional isolates have been evaluated for virulence to 16 single-gene resistance lines. In addition, nearly 1,000 of these isolates representing

the Great Plains population have been evaluated to 55 stem rust-resistant genes. The isolates all fit the original clusters with the exception of Pgt-QCCJ and QFCS.

Isolates representing the races present in each cluster over time were evaluated for genetic variation using 15 common isozyme systems (2). These clusters or related isolates, based on virulence, collected after 1965 east of the Rocky Mountains, had an identical isozyme genotype (2). Too few rust isolates existed to extend the analysis to earlier years. The only change that isozyme analysis made to the virulence clusters of Roelfs and Groth (11) was that Pgt-RCRS and RHTS were each represented by a single race and only a few isolates had distinct isozyme banding patterns.

The objective of this study was to compare the virulence and isozyme banding patterns of Pgt-QCCJ with the other Great Plains races and race clusters to determine the likely origin of Pgt-QCCJ. Because QCCJ is the only race known in the Great Plains that has the ability to attack barley cultivars with the *Rpg-1*, it has an economic consequence.

MATERIALS AND METHODS

Selected isolates of Pgt-QCCJ from the annual survey were stored and later inoculated to 55 known stem rust-resistance genes. Isolates representing all other pathotypes isolated from 1990 through 1993, and representatives of each stem rust race identified in the Great Plains since 1970, also were reevaluated. Wheat lines with resistance gene *Sr7a* were deleted from the data set, as it is difficult to distinguish the low infection with cultures avirulent on *Sr10* in our environmental conditions. Most host lines with *Sr7a* may also possess *Sr10*, to which *Sr7a* is epistatic. From 1990 through 1993, 30 isolates of QCCJ were evaluated for virulence to this set of resistance genes. The single-uredinial isolates from the race survey were removed from storage (-45°C), heat shocked (45°C) for 7 min, rehumidified at 80% relative humidity for 2 h, and then inoculated to a susceptible host for increase. Urediniospores were suspended in a lightweight mineral oil and sprayed on 7-day-old seedlings of 'Baart' treated with maleic hydrazide to

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enhance spore production. The oil was allowed to evaporate for 30 min, and then the inoculated seedlings were placed in a dark dew chamber overnight at 18°C. In the morning, the temperature was allowed to gradually rise to 26°C over a 3-h period while the plants were illuminated (200 $\mu\text{E m}^{-2} \text{ s}^{-1}$ at leaf height), during which time the dew slowly dried. The plants were then placed in a greenhouse at 18°C in plastic cages to reduce contamination. Urediniospores were collected after 14 days and used to inoculate 7-day-old seedlings of wheat lines containing the known *Sr* genes (13). Each line was represented by six to 10 seedlings, and most resistance genes were present in two different lines or the lines were planted twice. Plants were fertilized with a water-soluble 23-18-16 (NPK), 3 g per 24 cultivars at emergence, following inoculation. Inoculation, incubation, and growing temperatures were the same as for increase plants. Daylight was supplemented with fluorescent

light (200 $\mu\text{E m}^{-2} \text{ s}^{-1}$ at leaf height) to provide a 16-h day. Infection types were recorded 14 days after inoculation (7). The reported data on infection type was an average of all the data available on a particular race-resistance combination.

For isozyme analysis, race QCCJ was inoculated to 'Morocco' wheat (as described for virulence testing) and spores were collected after 14 to 18 days with a cyclone spore collector. Horizontal starch gel electrophoresis was carried out on extracts of homogenized, germinated spores as described by Burdon and Roelfs (3). Crude urediniospore extracts were absorbed onto paper wicks, and then loaded into 11% hydrolyzed starch gels. One continuous buffer system (histidine, pH 8.0) and two discontinuous buffer systems (borate, pH 7.8, and citrate, pH 8.0) were used for electrophoresis. The enzymes leucine aminopeptidase (LAP, EC 3.4.11.1) and dihydrolipoamide reductase (EC 1.6.4.3; synonym NADHD)

TABLE 1. Comparison of the virulence/avirulence and isozyme banding pattern of *Puccinia graminis* f. sp. *tritici* race Pgt-QCCJ with other Great Plains race clusters

<i>Sr</i> gene	Infection type ^r produced with race Pgt- ^s										
	QCCJ	QFCS	RCRS	RHTS	H_LG	Q_CQ	R_ _Q	_KJ	_THJ	MCC_	T_ _K
5	4	4	4	4	0	4	4	0 (4)	4	4	4
6	0	;	;	4	;	;	4 (;	4	4	;	;
7b	2	2	4	4	4	2	4	4	2 (4)	4	4
8a	2-	4	2-	2-	4	4 (2-)	4	4	4	2-	4 (2-)
9a	2	4	4	2-	2-	4	4	4	23	2-	2-
9b	2	2	4	4	2	2	4	2	4	2	2+
9d	4	4	4	4	4	4	4	4	4	1-	4
9e	;	;	;	;	;	;	;	;	;	;	4
10	4	4	4	4	1CN	1CN	1CN	4	4	4	4
11	1	1	1	1	4 (1)	1	1 (4)	1	4	1	4 (23)
12	X-	0;	4	4	4	4	4	4	4	0;	4
13	3	2	2	2	2	2	2	2	2	2	2
14	4	4	4	4	4	4	4	4	4	23C	4
15	4	4	4	4	4	4	4	4	4	4	XCN
17	4	4	4	4	1	4	1 (4)	4	4	4	4 (;
21	4	4	4	4	4	4	4	4	4	1	4
23	? ^t	4	4	4	4	4	23C	23C	23C	23C	4
30	2-	2-	2-	4	2-	2-	2-	2-	2-	2-	2-
33	0;	2=	2=	2=	2=	2=	2=	2=	2=	2=	2=
34	0;	3C	3C	3C	3C	3C	3C	3C	3C	3C	3C
35	4	4	0;	0;	0;	0;	0;	4	4	4	0;
36	0	0	4	4	4	1=C	4	0	4	0	4
Tmp	2=	2=	2=	2=	2=	2=	2=	2=	2=	4 (2=)	4
Tt-3	1	1	0;	0;	0;	0;	4	0;	0;	0;	4
McN	2=	4	4	4	4	4	4	4	4	4	4
MqX	4	4	4	4	4	23C	23C	4	4	4	4
Dp-2	2	2	4	4	4	4	2	4	4	2	2
Wst*2'	2-	4	4	4	2	2	2	4	4	4	4
H	2	2=	23C	23C	23C	23C	23C	23C	23C	4	4
U	4	4	4	4	1C	2C	3C	4	4	4	4
;	1	12	0;	0;	12=	4	4	12=	4	0;	4
Isozymes ^u											
GOT ^v	cc	cc	cd	ac	cc	cc	cc	ac	ac	ac	cc
LAP ^w	cd	bc	cd	cc	cd	dd	dd	cc	cc	bc	bd
NADHD ^x	cc	cc	bc	bc	bc	cc	bc	cc	bc	cc	bc
PGI-2 ^y	aa	ab	ab	ab	ab	aa	ab	aa	aa	aa	ab
PGM-2 ^z	bb	bc	bc	bb	bb	bb	bb	bb	bb	bb	bb

^r Infection types after Roelfs (8): 0 = no uredinia or other macroscopic sign of infection; ; = no uredinia, but hypersensitive necrotic or chlorotic flecks of varying size present; 1 = small uredinia surrounded by necrosis; 2 = small to medium uredinia often surrounded by chlorosis or necrosis; 3 = medium-sized uredinia that may be associated with chlorosis or rarely necrosis; X = random distribution of variable-sized uredinia on single leaf with a pure culture; and 4 = large uredinia without chlorosis or necrosis. Infection types are often refined by modifying characters as follows: = is for uredinia at the lower size limit for the infection type; - is for uredinia somewhat smaller than normal for the infection type; + is for uredinia somewhat larger than normal for the infection type; ++ is for uredinia at the upper size limit for the infection type; C is for uredinia surrounded by more chlorosis than normal for the infection type; and N is for uredinia surrounded by more necrosis than normal for the infection type. A range of variation between infection types is shown by indicating the range, with the most prevalent infection type list first, i.e., 23 or 1C or 31N.

^s For comparisons with Burdon and Roelfs (2), QCCJ and QFCS are new; RCRS = 11-RCRS; RHTS = 11-RHRS; H_LE = 17 H-LQ; Q-CQ = 151-Q-CQ = 151-Q-BS; R_ _Q = 113 R_Q_ ; _KJ = 29-32-JCS; LTHJ = 33-151_5HG; MCC_ = 56MBC_ ; and T_ _M_ = 15_T_M_.

^t *Sr23* could not be evaluated as it is epistatic to *SrMcN*, which also occurs in this line.

^u Bands numbered following Burdon and Roelfs (2).

^v GOT, EC 2.6.1.1, glucosephosphate isomerase on borate gel.

^w LAP, EC 3.4.11.1, leucine aminopeptidase on citrate gel.

^x NADHD, EC 1.6.4.3, dihydrolipoamide reductase on citrate gel.

^y PGM, EC 2.7.5.1, phosphoglucomutase on histidine gel.

^z PGI, EC 5.3.1.9, phosphoglucoisomerase on borate gel.

were assayed on citrate gels; glutamate oxalate transaminase (GOT, EC 2.6.1.1; synonym aspartate aminotransferase) and glucosephosphate isomerase (EC 5.3.1.9; synonym phosphoglucosomerase [PGM]) were assayed on borate gels; and PGM (EC 2.7.5.1) was assayed on histidine gels.

Isozyme banding patterns for each of the enzyme systems were determined by comparison to the known banding pattern of race TPMK, which was used as a standard. Five representative isolates of race QCCJ were collected to represent the cultures found in the northern Great Plains in 1989 and 1990 and used for isozyme analysis. A representative isolate from each cluster was run in a single test to confirm banding pattern identity. Bands were assigned isozyme allele designations following Burdon and Roelfs (3).

To graphically show the difference in avirulence between the U.S. Great Plains isolates, eigenvectors and eigenvalues for a principal coordinate analysis were calculated using NTSYS software (15). Each race cluster was represented by a single isolate. Thus, in most cases, a choice had to be made of which virulence pattern to use. For consistency and because QCCJ is generally considered an avirulent culture (most avirulences to 53 tested genes; *Sr7a* and *Sr23* were unavailable, due to epistasis), the most avirulent race of each group was used for comparison purposes. In most cases, this narrowed the differences between clusters. All clusters were assigned a 0 value if virulent, and a value of 1 if avirulent. All low infection types, regardless of their value, were considered to indicate an avirulent response.

RESULTS

Thirty-one of the single-gene stem rust lines responded differentially to one or more races from the nine race clusters, QCCJ, and QFCS (Table 1). The other lines were either resistant (*Sr13*, 22, 24, 25, 26, 27, 29, 31, 32, 33, 34, 37, *Wld*, and *Gt*) or susceptible (*Sr8b*, 9f, 9g, 16, 18, 19, 20, 28, and *Kt'2'*). A summary of the infection types for each host line is shown in Table 1 for cluster. An infection type equal to that of the line without the gene in it was scored as a 4, or high infection type. Infection types 0 through 3 were considered as low (avirulent) infection types, indicative of a level of resistance. The data presented in Table 1 is a summary of the data collected over 20 years. Where members of a cluster varied, the most frequent infection type in the last year detected is given first and the other is enclosed in parentheses. Based on virulence, isolates of QCCJ (Table 1) differed from the other clusters by virulence to six resistance genes. The most obvious differences were the low infection type of 2= on *SrMcN*, instead of the high infection type of 4 obtained with all other Great Plains races, and the infection type 0; (few hypersensitive spots) compared with the 3C (medium-sized uredinia surrounded by chlorosis) produced by other races on *Sr34*. Other virulence differences were less obvious in the seedling stage. Resistance gene *Sr6*, usually expressed as a ; (fleck

or hypersensitive lesion) to other avirulent races, had a 0 (no visible sign of infection) to QCCJ under these conditions. Avirulence to *Sr12* was an X- (mesothetic, small- to medium-sized lesions) compared with the 0 observed as a low infection with MCC_ (race 56 or CRL 56-MBC), the only other race avirulent to *Sr12*. The infection type of QCCJ on *Sr13* at 18°C was higher than for other clusters, a constant 3 (medium-sized uredinia) compared with a 2 infection type produced by other races. Infection of host lines with *Sr33* resulted in a 0; infection type, while the other Great Plains cultures resulted in a 2= (smallest of the infection type 2 uredinia). Slight differences also occurred in low infection types on *SrTt-3*, *SrWst'2'*, *SrH*, and *Sr*. Virulence of QCCJ to the 31 differential genes was most similar to MCC_ of the Great Plains clusters.

The isozyme banding pattern of QCCJ differed from the isozyme banding pattern of the Great Plains race clusters. QCCJ was most similar to cluster Q_CQ (Table 1), differing only in the lack of the LAP c band. However, these races differed at nine or more virulence loci. Races QFCS and MCC_, the most similar to QCCJ in virulence (three and seven gene differences, respectively), each had two isozyme differences from QCCJ (Table 2).

The first three eigenvectors accounted for 58% of the similarity and were used as the principal coordinated axes. Eigenvectors were plotted in a three-dimensional graph (Fig. 1). The conservative approach used placed QCCJ in the vicinity of MCC_ and QFCS, and this group is the tightest grouping observed. For this analysis of Great Plains race clusters, $r = 0.8060$, explaining a moderate amount of the variation.

DISCUSSION

The purpose of this study was to determine the origin of QCCJ in the Great Plains. If a sexual or parasexual cross between rust clusters was involved in the immediate history of QCCJ, it is possible that *Rpg-1* virulence also might be transmitted to races more virulent to wheat, thereby creating an even more serious problem, as most inoculum is produced on wheat. Further, a sexual cross would also indicate that barberry was again having a role in the Plains stem rust population.

The mutants previously found for this pathogen have been single-gene changes in which the newer type was virulent to an additional resistance gene (8), but with no change in isozyme banding pattern (1). Race QCCJ differs in virulence at seven or eight loci from the nearest Great Plains cluster, depending on whether QCCJ is compared with MCCF or MCCD, races in the MCC_ cluster. QCCJ is avirulent at four loci to which MCC_ is virulent, an unlikely change if the avirulences are homozygous dominant. Seven virulence/avirulence differences would certainly be highly unusual for a mutant. The isozyme banding pattern for QCCJ was unique, but no new alleles were detected (3). The alleles detected were all known to be present in both the Great Plains and Pacific North-

TABLE 2. Number of differences in isozyme allele and virulence between clusters of *Puccinia graminis* f. sp. *tritici* using five polymorphic isozyme loci and 31 differential host genes^y

Clusters	Number of allele differences between races and clusters										
	QCCJ	QFCS	RCRS	RHTS	H_LG	Q_CQ	R__Q	_KCJ	_THJ	MCC_	T__K
QCCJ	0\0	3	4	4	2	1	3	2	3	2	3
QFCS	3	0\0	4	4	4	4	4	4	5	3	3
RCRS	9	7	0\0	3	2	5	3	5	4	5	3
RHTS	10	10	3	0\0	2	5	2	2	1	3	3
H_LG	11-12 ^z	11-12	8-9	9-10	0\0	3	1	4	3	4	1
Q_CQ	9-10	8-9	11-12	13-14	8-9	0\0	2	4	4	3	3
R__Q	13-16	12-15	9-12	11-14	6-10	7-9	0\0	5	4	5	1
_KCJ	8-9	6-7	6-7	7-8	9-11	9-11	10-14	0\0	1	1	5
_THJ	10-11	9-10	7-8	6-7	10-12	11-14	8-12	5-7	0\0	2	4
MCC_	7-8	7-9	11-12	13-14	15-17	15-17	15-20	10-11	12-14	0\0	4
T__K	9-11	12-14	9-12	11-13	10-14	10-14	11-15	12-16	10-14	11-15	0\0

^y Number of isozyme differences are given above the 0/0 diagonal, virulences differences are below.

^z The numbers are a range because of differences in virulence of races in a cluster.

west populations (3). QCCJ may have a different banding pattern for the esterase enzyme than any of the Great Plains race clusters. The R_f values of the band obtained could not be satisfactorily matched to those for the esterase alleles described by Burdon and Roelfs (3); therefore, this data was not included in the analysis. QCCJ may have the *Est* 2b allele, which was found previously in the Pacific Northwest sexual population, but not in the Great Plains population. These tests were performed many times, but inconsistencies caused us to take the conservative approach and not include that data in the analysis.

The principal coordinate analysis shows the clusters of stem rust races in the Great Plains in a three-dimensional graph (Fig. 1). The most closely related race was QFCS. QCCJ was not closely associated with other clusters, based on virulence. Pgt race cluster T_K (race 15) is unique as expected, and QCCJ, MCC_, and QFCS cluster near _KCL. T_K and MCC_ (race 56) have been the common races since 1934. The fact that MCC_, QFCS, and QCCJ are in the same area may indicate some relationship. But QFCS differs from QCCJ by three genes in virulence/avirulence and from MCC_ by at least seven. The approach used in vector analysis was conservative. Principal coordinate analysis allows a three-dimensional figure to be created, which is otherwise difficult with more than four to six clusters, and also allows for some statistical evaluation. Differences would have been greater if the most virulent race of each cluster was used. Several of the differences in infection types shown in Table 1 between QCCJ and the other races were differences that did not result in a difference in virulence or avirulence and, thus, were not included in the analysis. Furthermore, the most significant virulence difference, virulence to *Rpg-1* ([T-gene] in barley), is not included in this data set. MCC_ is the most avirulent (lowest infection type over the widest range of environments) race cluster to *Rpg-1* (16). Isozyme genotype data was not combined with the virulence phenotypic data, in part because isozymes are thought to be nonselected, while virulence markers are highly selected.

Avirulence to the gene in 'McNair 701' is rare and was not detected in the worldwide virulence survey by Huerta-Espino (4). *SrMcN* also occurs in the differential cultivar Exchange, the only host we have for *Sr23*. The low infection type of *SrMcN* (2=) is epistatic to the low infection of *Sr23* (23C); thus, we are unable to evaluate QCCJ to *Sr23*. Avirulence to 'McNair 701' had been found earlier in cultures from the Pacific Northwest (13). Thus, it is likely that QCCJ originated in the Pacific Northwest. The low infection type on *Sr34* of 0; is also unusual, as most other cultures worldwide result in an infection of 3C or are virulent. Although a 0; low

infection was found in the F_2 progeny of race 36/111 made by W. Q. Loegering (5), and in a few cultures from the Pacific Northwest, it was found only in a single isolate from Chile in 1989 in the international virulence survey (4).

Race QFCS was most like the Q_CQ and MCC_ clusters (Table 1). QFCS varied from the Q_CQ cluster in virulence to *Sr10*, *Sr35*, *SrMQX*, *SrWst'2*, and *SrU* and in avirulence to *Sr12*, *SrDp-2*, and *Sr*. It varied from the MCC_ race cluster (Pgt-MCCS) in virulence to *Sr8a*, *Sr9d*, *Sr14*, *Sr21*, and *Sr23* and in avirulence to *Sr7b* and *SrH*. Race QFCS differed from QCCJ in virulence to *Sr8a*, *Sr9a*, *SrMcN*, and *SrWst'2* and had the "normal" low infection types for *Sr6*, *12*, *13*, *33*, and *34*. QFCS lacked virulence for *Rpg-1* and was found on barley and wheat cultivars without a high level of resistance and on *Hordeum jubatum*.

The data indicates that QCCJ is not likely to have arisen as a mutant from an existing Great Plains rust cluster. No combination of isozyme and virulence data for any of the cultures would be expected to yield F_1 progeny with the combination of virulence and isozyme characters present in QCCJ. Many (*Sr6*, *12*, *13*, *33*, *34*, *Tt-3*, *McN*, and *H*) of the unique low phenotypes observed with QCCJ only have been observed in isolates obtained from the Pacific Northwest. In this area, some barberry is known to exist, and aecial spreads have been observed in the past 20 years. It is hypothesized that QCCJ and QFCS both originated in that area and were transported into the Great Plains as urediniospores. Thus, QCCJ should now reproduce asexually (lack of barberry adjacent to barley fields). Change should be limited to mutation at single loci. Even though QCCJ appears to be a rather aggressive race, its lack of virulence for the hard red spring and durum wheats of the northern Great Plains should keep the population size small. The shorter growing season and partial resistance of most barley cultivars will be adequate to control this race, except under the most favorable environmental and cultural conditions. Resistant wheat cultivars or a severe winter or drought in the southern Great Plains could eliminate this race in the future.

Studies of the origin of a newly appearing race require a set of conditions that rarely exist. It is essential to have extensively studied the previous population over a rather long time. In this case, the new race appeared in a small group of asexually reproducing clusters, making its uniqueness obvious. Studies are easier if the pathogenic stage can be clonally propagated and stored indefinitely without losing pathogenicity. Other studies of unique races (other than by mutation) in *P. graminis* populations involved the appearance of a race in Australia that was traced to southern Africa (18) and to a unique Australian race that arose through parasexual recombination (1). The development of molecular markers will enhance our ability to determine the origin of new races in the future, if populations are monitored and 'random' pathotypes are maintained.

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Fig. 1. Principal coordinate analysis of virulence/avirulence of the most avirulent members of each United States race cluster of *Puccinia graminis* f. sp. *tritici*, using eigenvectors, $r = 0.8060$.

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