Single-gene-pair Differences for Virulence and Resistance in Wheat Stem Rust

A.P. Roelfs

Cereal Rust Laboratory, Agricultural Research Service
U.S. Department of Agriculture
University of Minnesota
St. Paul, Minnesota 55108

Stem rust has been a common disease of wheat worldwide, probably even before wheat was cultivated as a crop. Stem rust epidemics were severe in the northern Great Plains of the United States from the advent of extensive wheat planting in the mid-1800s through the mid-1950s. The constant threat from the pathogen (*Puccinia graminis* f. sp. *tritici*) to the wheat host (principally *Triticum* sp.) has resulted in more than 70 years of research on both the host and pathogen systems. Thus, wheat stem rust is one of the best-studied disease systems in the world. Both the host and the pathogen have some characteristics that enhance and some that hinder the study of the disease.

The host as commonly grown is a hexaploid ($n = 21$), but tetraploid and diploid species are also grown. The polyploid nature of the host results in some redundancy in the genetic information, making genetic studies more complex in a few cases. However, wheat is self-pollinated and self-fertile, making the maintenance of homozygous lines simple. A monosomic series ($2n - 1$), nullisomic series ($2n - 1$ pair), and more recently a partial telocentric series ($2n$ with one-armed chromosomes with terminal centromeres for one pair) has been constructed by Sears (Morris and Sears 1967).

Series of substitution and backcross lines in which a chromosome or perhaps a single resistance gene has been transferred from the donor to a common wheat genotype (cultivar) are available for about 55 different specific resistance genes. Each of these resistances is expressed in a gene-for-gene relationship with a corresponding gene for virulence in the pathogen. Each gene interaction produces a characteristic response that is often unique enough to recognize (Roelfs and McVey 1979). Host resistance genes are usually dominant (or at least partly dominant) in their action (Table 1). However,

Table 1
Infection Types Produced by the Interaction of Host and Pathogen Genotypes for *Sr15*

Host	Pathogen		
	$P_{15}P_{15}$	$P_{15}p_{15}$	$p_{15}p_{15}$
Sr_{15} Sr_{15}	;1c	Xc	4
sr_{15} sr_{15}	4	4	4

recessive genes for resistance are known, and some responses depend on the total host genotype used (Roelfs and McVey 1979). Genes present in the lower levels of ploidy usually are more effective than at higher ploidy levels.

Generally, individual host genes are independent in action, but in a few cases interactions are known to occur (Samborski and Dyck 1982). A few genes for resistance to wheat stem rust appear also to be genes for resistance to leaf rust, stripe rust, or powdery mildew (McIntosh 1973). Often such resistance genes for more than a single disease are from alien genera, but several are not.

Most resistance genes function throughout the life span of the host plant while a few others function only at certain growth stages. *Sr25* is highly effective in the seedling stage but is less effective as the plant matures. *Sr2* is ineffective in the seedling stage but is highly effective in the adult stages of plant growth. Furthermore, *Sr2* is much more effective in the internodal spaces than at the nodes and in the floral parts (Sunderwirth and Roelfs 1980). *Sr6* and *Sr15* function only at low temperatures ($<20°C$ and $<18°C$, respectively), whereas *Sr13* is more effective at higher temperatures (30°C). A slight modification of resistance is often seen as a gene is placed in different host genotypes.

The pathogen, *P. graminis* f. sp. *tritici* is an obligate parasite. The pathogen enters the host through the stomata and establishes parasitism by producing haustra in the mesophyll cells. A single uredospore can result in an infection about 10% of the time. A lesion is normally visible in about 7 days, and spore production starts about 14 days after inoculation. Each uredium thereafter can produce about 5000 spores per day (23 mg) (Katsuya and Green 1967). Gram amounts of uredospores are routinely produced in the greenhouse.

The genetics of the pathogen is poorly understood. However, virulence has often been recessive and incomplete. The pathogen can exist indefinitely through a series of asexual generations. Although

mutations for virulence occur, they are rare enough in asexual populations that they do not interfere with cultural purity when maintaining small populations. Uredospores can be stored with little or no loss in viability for more than a decade in liquid nitrogen or at ultralow temperatures (< −50°C).

Since 1918 the virulence of the North American pathogen population has been monitored annually by Agriculture Canada and the United States Department of Agriculture. The data collected over the past 65 years show that currently in the Great Plains of the United States there are nine asexually reproducing pathogen populations (Burdon and Roelfs 1985). Most populations consist of two to five virulence genotypes, each differing in virulence from another by a single gene. The asexual nature of the population is shown by two lines of evidence (Table 2). The first is based on virulence analysis of pathogen cultures (Roelfs and Groth 1980) on 55 host lines possessing single resistance genes. To these nine populations, 13 of the lines are always susceptible and 14 are always resistant; thus 28 remain as usable differential hosts. Within a population, variation is from zero to five genes for virulence. On the basis of the initial year of detection, these differences probably represent a single-gene virulence change from a previous existing member of the group. Between groups, the number of virulence differences varies from 2 to 19. The second line of evidence is through isozyme analysis. Evaluation of 13 isozyme systems showed that the nine pathogen populations were identical for seven isozymes and varied for six isozymes between populations. However, no variation was found within any of the populations for any of the isozymes even though cultures were obtained from a wide range of geographical areas within North America in 1983 and were selected from collections obtained during the past 30 years that had been stored in liquid nitrogen. Thus, all evidence points to the fact that we have pathogen cultures that differ from each other by a single gene pair for virulence.

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Table 2
Differences and Similarities between Asexual Populations of *Puccinia graminis* f. sp. *tritici* in North America as Measured by Virulence and Isozymes

Sr gene	*P. graminis* (race clusters)								
	11[a]	11[b]	15	17	29–32	56	113	151	151–32
	Infection type differences								
5	H	H	H	0	0/H[c]	H	H	H	H
6	;	H	0;	;	H	;	H/;	;	H
7a	H	H	H	1N	H	;1N	1N	;1N	H
7b	H	H	H	H	H	H	H	2	2/H
8a	2–	2–	H/2	H	H	2–	H	H/2–	H
9a	H	H	2–	2–	2–	2–	H	H	23
9b	H	H	2	2	2	2	H	2	H
9d	H	H	H	H	H	;1–	H	H	H
9e	;1	;1	H	;1	;1	;1	;	;1	;1
10	H	H	H	1N	H	H	1N	1N	H
11	1–	1–	H/2+	H/1–	1–	1–	H/1–	1–	1–/H
12	H	H	H	H	H	;	H	H	H
14	H	H	H	H	H	23C	H	H	H
15	H	H	XCN	H	H	H	H	H	H
17	H	H	;1/H	;	H	H	;/H	H	H
21	H	H	H	H	H	1=	H	H	H
23	H	H	H	H	23C	23C	23C	H	23C

30	2	H	2	2	2	2	2	2	2
35	0;	0;	0;	0;	H	H	0;	H	H
36	H	H	H	H	0	0	H	;1+	0
Tt-3	0;	0;	H	0;	0;	1	H	0;	0;
Tmp	2–	2–	H	2–	2–	H/2–	2–	2–	2–
Mq X	H	H	H	H	H	H	23C	23C	H
dp-2	H	H	2	H	H	2	2	H	H
Wst 2	H	H	H	2	H	H	2	2	H
H	23C	23C	H	23C	23C	H	23C	12C	23C
U	H	H	H	;1C	H	H	2C	2C	H
;	0;	0;	H	12=	?	0;	H	H	H
				Isozyme[d] *phenotype*					
Got	cd	ac	cc	cc	ac	ac	cc	cc	ac
Lap	cd	cc	bd	cd	cc	bc	dd	dd	cc
Nadhd	bc	bc	bc	bc	cc	cc	bc	cc	bc
Pgi-2	ab	ab	ab	ab	aa	aa	ab	aa	aa
Pgm-1	cc	?	aa	aa	?	aa	bb	bc	bb
Pgm-2	bc	bb	bb	bb	bb	bb	bb	bb	bb

See Burdon and Roelfs (1985).

[a]11-RCR: No variants are known for this cluster.

[b]11-RHR: No variants are known for this cluster.

[c]Intracluster variation occurs for this phenotype.

[d](Got) Aspartate aminotransferase; (Lap) aminopeptidase; (Nadhd) dihydrolipoamide; (Pgi) glucosephospate isomerase; (Pgm) phosphoglucomutase.

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