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GENETIC CONTROL OF PHENOTYPES IN WHEAT STEM RUST*

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INTRODUCTION

Progress in understanding the genetic control of phenotype in the interaction of *Puccinia graminis* Pers. f. sp. *tritici* and *Triticum* sp. has been slow. In 1866, de Bary (10) showed that *Aecidum* and *P. graminis* were actually the same organism. The study of genetics dates back to Mendel (38), and not until 40 years later did Biffen (2) show that host resistance to stripe rust was inherited in a Mendelian manner. Eriksson & Henning (14) and Stakman & Piemeisel (58) showed that variation for virulence existed within *P. graminis* for both host species and cultivars, respectively. Then in 1927, Craigie found that the sexual recombination of the fungus occurred on the barberry (8). Following Craigie's work many others have attempted genetic studies of *P. graminis*. Among the first and most successful was the team of Margaret Newton & Thorvaldur Johnson (24, 40, 41). The single most important cross was made by Loegering & Powers (32), who had the insight and techniques to maintain living uredospores of the parents, the F_1 and F_2 progeny.

The host-pathogen interaction generally produces a visible response. This response is the result of the interaction of the host and pathogen genotypes and the environment existing immediately before and following infection (52). The host-pathogen interaction is expressed as an infection type (Table 1). The compatible and incompatible host-pathogen interactions are called high and low infection types, respectively (28–30).

Table 1 Host response, infection type, and symptom description for the host-pathogen interaction for wheat stem rust[a]

Host response (class)[b]	Infection type[c]	Symptoms
Immune (Res)	0 low	No uredia or macroscopic sign of infection
Nearly immune (Res)	; low	No uredia but necrotic or chlorotic flecks
Very resistant	1 low	Small uredia with necrotic border
Moderately resistant	2 low	Small to medium uredia with chlorosis or necrosis
Heterogeneous (Mes)	X low	Random distribution of variable-sized uredia
Heterogeneous (Mes)	Y low	Variable-sized uredia, decreasing in size with distance from the leaf tip
Heterogeneous (Mes)	Z low	Variable-sized uredia, decreasing in size with distance from the leaf base
Moderately susceptible	3 low	Medium-sized uredia
Susceptible	4 high	Large uredia without chlorosis or necrosis

[a]After Roelfs (46).

[b]Res = resistant, Mes = mesothetic.

[c]Individual infection types are often refined as follows: =, uredia at the lower size limit; –, uredia somewhat smaller than normal; +, uredia somewhat larger than normal; and + +, uredia at the upper size limit; C, more chlorosis than normal and N, more necrosis than normal. Discrete infection types on a leaf when infected with a single biotype are separated by a comma (e.g. 4,; or 2+, 2– or 1,3C). A range of variation between infection types on a leaf is indicated by showing the range with the most prevalent type listed first (e.g. 23, 1;C or 31N).

THE HOST-PATHOGEN INTERACTION

Genetic interactions in *P. graminis-Triticum* spp. apparently are based on a gene-for-gene system (37). Throughout this review a gene-for-gene relationship is assumed, although I recognize that in only a very few cases have studies identified all the host and pathogen genotypes needed to conclusively prove the existence of such a relationship. The gene-for-gene relationship was first described by Flor (15). For those unfamiliar with the theory, it has been reviewed by Flor (17), Person (43), and Person & Mayo (44.)

Flor (16) described the gene-for-gene relationship as follows: "For every gene that conditions resistance in the host there is a corresponding gene in the parasite that conditions pathogenicity." This relationship is often described by the use of a diagram (e.g. Table 2) that frequently is simplified to contain only the homozygous host and pathogen genotypes. This simplification eliminates the heterozygous genotype—often the most common pathogen genotype found in nature (22). It is generally assumed that host genes for resistance will be dominant while pathogen genes for virulence will be recessive. These assumptions are generally true for *P. graminis* and the *Triticum* spp. system, but cases where avirulence is dominant are common (19). In the case of

resistance of oats to *P. graminis* f. sp. *avenae,* recessive genes for resistance are common (55). Each interaction involving host reaction-pathogen pathogenicity gene pair probably results in a different low infection type (48). However, differences in infection types are often very small. Additionally each interaction is affected to some extent by the environment as well as by the rest of the host and by the pathogen genotype. Most studies of environmental effects on the gene-for-gene response in host-pathogen interactions have involved temperature (4). However, other factors such as light, host nutrition, light intensity, day length, humidity, host growth stage, and host tissue infected also play a role (6).

Effect of Host Heterozygosity

The variation of low infection type that is due to a heterozygous host genotype has often caused an apparent loss of resistance. This is demonstrated in Table 2 (see P7bP7b and P7bp7b interaction with *Sr*7b*Sr*7b), where the infection type changes from the easily recognized resistant response (indicated by a infection type 2–) with a homozygous resistant host to the moderately susceptible response (infection type 23) with a heterozygous host genotype. Similar changes between homozygous and heterozygous host responses of different magnitudes are observed with the other illustrated genes. When some variation occurs in environmental conditions or density of uredia, it is difficult to distinguish a moderately susceptible from a susceptible response based on infection types. To detect the effects of changes in environment and inoculum densities it is necessary to include the homozygous host genotypes as checks in each test. The differences in response between the heterozygous and homozygous host genotypes are most important in breeding and genetic studies. Unless hybrid wheat cultivars become more important commercially,

Table 2 Mean low infection types produced by homozygous and heterozygous pathogen and host genotype interactions in the stem rust system[a]

	Pathogen				Pathogen		
Host	P7bP7b	P7bp7b	p7bp7b	Host	P10P10	P10p10	p10p10
Sr7bSr7b	2–	2	4	Sr10Sr10	4	23C	1N
Sr7bsr7b	23	32	4	Sr10sr10	4	3C	23
sr7bsr7b	4	4	4	sr10sr10	4	4	4
	Pathogen				Pathogen		
Host	P17P17	P17p17	p17p17	Host	PxxPxx	Pxxpxx	pxxpxx
Sr17Sr17	4	4	4	SrxxSrxx	4	4	4
Sr17sr17	X–	3C	4	Srxxsrxx	4	Low	Low
sr17sr17	0;	;1	4	srxxsrxx	4	Low	Low

[a]From Roelfs & Groth (47) based on pathogen population of Loegering & Powers (32) and host F_1 furnished by C. C. Hu.

the effect of heterozygous host genotypes will have little agricultural impact. Most cultivars are composed of self-pollinated homozygous lines.

Effect of Pathogen Heterozygosity

The heterozygous pathogen genotype can also cause an apparent shift in dominance of the host resistance. For example in the Sr17-P17 interaction a 3 resistant to 1 susceptible ratio should be obtained for host phenotypes when a homozygous avirulent (P17P17) culture is used (Table 2). However, if a heterozygous avirulent (P17p17) culture is used, the population would probably segregate 1 resistant to 3 susceptible. Indeed the reversal of dominance is usually the case, especially if temperatures are not carefully controlled.

The apparent loss or reduction in effectiveness of resistance when a heterozygous culture is substituted for a homozygous avirulent culture probably is common because many loci in rust fungi are known to be heterozygous (22). Heterozygous pathogen genotypes result in a similar change in infection type as does host heterozygosity (Table 3). The change from a homozygous avirulent to a heterozygous avirulent could in part explain the phenomenon of progressive virulence (57). The apparent increase in virulence to a resistance gene with cultures isolated over a period of years is called progressive increase in virulence. Some of the increase in virulence reported by Watson & Luig (60) was probably due to the use of homozygous avirulent, heterozygous avirulent, and homozygous virulent cultures. However, as the authors pointed out, that should have resulted in two low and one high infection types, whereas three low and one high infection types were observed (Table 4). Some of the variation in infection type may be due to changes in the aggressiveness rather than in the virulence of the culture. Temperature can interact with the pathogen genotype to result in progressive increases in virulence (Table 5). To clarify progressive virulence increases one should make crosses not only between host cultivars but also between pathogen cultures. To demonstrate differences in infection type with *Sr*11 Watson & Luig used color mutants that are usually quickly eliminated in nature and thus perhaps indicate a lack of genes for aggressiveness.

SPECIFIC HOST RESISTANCE GENES

Currently 57 different host genes for stem rust resistance have been isolated (36, 48). Another 20 to 25 genes probably could be isolated within a few years if that became a high priority project. The resistance loci are spread throughout the genomes and chromosomes (Table 6). Alleles exist at the *Sr*7, *Sr*8, *Sr*9, and *Sr*16 loci, but only at the *Sr*9 locus where 5 alleles are known does resistance locus complexity approach that of flax and corn. Most of the resistance genes are dominant; *Sr*17 is an exception. However, *Sr*6 and *Sr*12 are recessive to some cultures.

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Sr7bsr7b	23	32	4	Sr10sr10	4	3C	23
sr7bsr7b	4	4	4	sr10sr10	4	4	4

	Pathogen				Pathogen		
Host	P17P17	P17p17	p17p17	Host	PxxPxx	Pxxpxx	pxxpxx
Sr17Sr17	4	4	4	SrxxSrxx	4	4	4
Sr17sr17	X–	3C	4	Srxxsrxx	4	Low	Low
sr17sr17	0;	;1	4	srxxsrxx	4	Low	Low

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Table 3 Low infection types resulting from infection of selected *Triticum* sp-*Puccinia graminis* f. sp. *tritici* gene pairs

Gene pair	Low Infection types[a]		Gene pair	Low Infection types[a]	
Sr2 P2	Adult plant		Sr26 P26	;1	–[b]
Sr5 P5	0	0;	Sr27 P27	;	–
Sr6 P6	0;	;	Sr28 P28	0	;
Sr7a P7a	;	12–N	Sr29 P29	;1	2–
Sr7b P7b	2	2+	Sr30 P30	2	23
Sr8a P8a	2–	2	Sr31 P31	;1	2–
Sr8b P8b	;1CN	X–CN	Sr32 P32	–	2–1
Sr9a P9a	2	23	Sr33 P33	0;	2–
Sr9b P9b	2	2+3	Sr34 P34	;1C	23C
Sr9d P9d	0;	;1–	Sr35 P35	0;	12C
Sr9e P9e	1–	;12	Sr36 P36	0	;1+C
Sr9f P9f		–	Sr37 P37	0;	–
Sr9g P9g	2=	–	SrTt-3 PTt-3	0;	X–C
Sr10 P10	1CN	23	SrTmp PTmp	2–	22+
Sr11 P11	1	23	SrMcN PMcN	0;	12
Sr12 P12	;	X–	SrMqX PMqX	0;1C	23C
Sr13 P13	2=	22+	Srdp-2 Pdp-2	2	2+
Sr14 P14	;1C	23C	SrLC PLC	1	–
Sr15 P15	;1N	XCN	SrKt-2 PKt-2	2–	2+
Sr16 P16	2=	2+	SrGt PGt	;1	2+–
Sr17 P17	0;	;1N	SrWst-2 PWst-2	2–	2
Sr18 P18	0;	2	SrWld PWld	2=	2+3
Sr19 P19	;1N	12C	SrH PH	12C	23C
Sr20 P20	2C	23C	SrU PU	;1C	2CN
Sr21 P21	1=	2	SrAgi PAgi	0;	12C
Sr22 P22	2=;	23	Sr: P:	0;	12=
Sr23 P23	0;	23C	SrPt PPt	01–	2–
Sr24 P24	22–	23	SrPL PPl	0;	21
Sr25 P25	2–	23			

[a]After Roelfs & Groth (47), host genotype known to be homozygous for resistance while the pathogen could be homozygous or heterozygous avirulence genotype.
[b]Only 1 low infection type observed.

Genes for resistance to wheat stem rust have come from various sources: *Triticum turgidum* (*Sr*2, *Sr*9d, *Sr*9e, *Sr*9g, *Sr*13, *Sr*13, *Sr*14, *Sr*17), *T. monococcum* (*Sr*21, *Sr*22, *Sr*35), *Agropyron elongatum* (*Sr*24, *Sr*25, *Sr*26), *Secale cereale* (*Sr* 27, *Sr*31), *T. speltoides* (*Sr*32), *T. tauschii* (*Sr*33), *T. comosum* (*Sr*35), *T. timopheevi Sr*36, *Sr*37). Curiously, a number of the stem rust resistance genes are either identical or very closely linked to resistance genes for wheat leaf or stripe rust resistance, e.g. *Sr*9g-*Yr*7, *Sr*15-*Lr*20, *Sr*24-*Lr*24, *Sr*25-*Lr*19, *Sr*31-*Lr*26-*Yr*9. All but the *Sr*15-*Lr*20 linkage groups involve transfers of genes (chromosome sections) between host species.

Table 4 Progressive increases in virulence in three cultures of *Puccinia graminis* f. sp. *tritici* for a host with *Sr*11[a]

Host genotype	Pathogen culture			
	80–E-0;	80–E-2	80–E2(3C)	126-1,2
Sr11Sr11	;	X–	3C	High
sr11sr11	High	High	High	High

[a]After Watson & Luig (60).

EFFECT OF OTHER HOST GENES

When a cultivar has more than one effective resistance gene against a pathogen, the genes generally act independently. The host-pathogen interaction exhibited usually is that of the lowest infection type for involved genes (13). Therefore, if an Sr6Sr6-P6P6 interaction results in a low infection type of a fleck and an *Sr*8a*Sr*8a-P8aP8a interaction results in a low infection type of a 2, then a cultivar *Sr*6*Sr*6*Sr*8a*Sr*8a interacting with a culture P6P6P8aP8a produces a low infection type of a fleck. Interaction between genes for resistance does occur, however; and as more resistances are studied, more interactions will be found.

Complementary Gene Action

Complementary gene action resulting in resistance to crown rust in Bond oats has been reported (55). In this case hosts with either gene pair alone are susceptible but hosts with both pairs are resistant (1). No complementary genes for resistance of this type are known for resistance to *P. graminis* f. sp. *tritici;* however, Martens et al (35) believed that the recessive gene *Pg*-12 for resistance to *P. graminis* f. sp. *avenae* in oats was sharply enhanced by a complementary gene that had no independent effect.

Table 5 Progressive increase in virulence of *Puccinia graminis* f. sp. *tritici* for host with *Sr*6*Sr*6 genotype at various temperature ranges[a]

Temperature	Pathogen culture			
	126–6,7	21–2	21–1,2,3,7	21–1,2
15C	0;	0;	X	High
18–21C	0;	X	High	High
21–24C	X	High	High	High
24–27C	High	High	High	High

[a]Modified from Waston & Luig (60).

Table 6 Chromosomal location of genes for wheat stem rust resistance

Chromosome	Genome		
	A	B	D
1		14, 31	18, 33
2	21, 32, 34	9_,[a] 16_,[a] 19, 20, 28	6, u
3	27, 35	2, 12	24
4	23, 37	7_,[a] Tmp	
5			30
6	8–, 13, 26	11	5, 29
7	15, 22	17	25

[a]_ is an allelic series 9a, 9b, 9c, 9d, 9f, 9g; 16, Kt'2'; 7a, 7b; and 8a, 8b.

Additive Gene Action

Additive effects of two effective resistance genes were reported for stripe rust of wheat (54). Many excellent examples of resistance to *P. recondita* exist in which the second gene enhances the level of resistance of the first gene, e.g. *Lr*13 and *Lr*16, *Lr*13 and *Lr*34, *Lr*33 and *Lr*34 and *Lr*T3 (11, 13, 53). In stem rust the data are less conclusive, but in many trials some enhancement of resistance has been observed when two or more genes were combined. The *Sr*9b, *Sr*10, *Sr*11, and *Sr*12 alleles enhanced the adult plant responses of *Sr*7a by reducing disease severity (Table 7) (27). Several resistance genes in *T. turgidum* cultivars produce lower infection types in combination than singly. The three resistance genes derived from Iumillo independently conditioned infection types 0;12=, 0;13=, and 2-3-. The combinations of two or more genes conditioned an infection type of 0; or 0;13= (61).

In our experience with *P. graminis-Triticum* spp. the most common additive effect occurs when a resistance gene that produces a low infection type associated with chlorosis or necrosis is combined with a resistance gene that conditions a smaller sporulating area. This type of gene combination usually results in an infection type with the smaller sporulating area, but with the chlorosis or necrosis expressed as well. An example of this is *Sr*23 (low infection type 23C) and *Sr*29 (low infection type 2), where the infection type resulting from the combined effective genes *Sr*23 and *Sr*29 is a 2C. A similar effect is found with *Sr*MqX and a number of other undesignated resistance genes that form infection types associated with chlorosis or necrosis.

Residual Effect

Clifford proposed that specific resistance genes may give a form of residual resistance to cultures that have the corresponding allele for virulence (7). This effect has been variously called a ghost or residual effect of the defeated resistance gene (39). Using a single culture of *P. graminis* f. sp. *tritici*,

Table 7 Effect of various combinations of resistance genes on disease severity in F_2 wheat populations infected with wheat stem rust race 15B

Cultivar or F_1 genotype[b]	Percent of F_2 plants per severity class[a]							
	<1	5	10	20	30	40	50	60
Marquis (check)						57	35	8
Sr7aSr7a		17	13	48	22			
Sr7aSr7aSr9bsr9b	55	18	20	5	2			
Sr7aSr7aSr9bsr9bSr10sr10	71	12	11	5	1			
Sr7aSr7aSr9bSr9bSr10sr10	94	5	1	*				
Sr7aSr7aSr9bSr9bSr10Sr10	96	4						
Sr7aSr7aSr9bsr9bSr10sr10Sr11sr11Sr12sr12	100	*						
Sr7aSr7aSr9bsr9bSr10Sr10Sr11sr11Sr12sr12	100							
Sr6sr6Sr7aSr7a	49	15	17	16	2			
Sr6Sr6Sr7aSr7a	100	*						
Sr6sr6Sr7aSr7aSr9bsr9b	84	6	6	3	1			
Sr6sr6Sr7aSr7aSr9bsr9bSr10sr10	88	4	5	2				
Sr6sr6Sr7aSr7aSr9bsr9bSr10Sr10	91	4	3	1				
Sr6Sr6Sr7aSr7a Sr10sr10	73	9	11	6	1			
Sr6Sr6Sr7aSr7a Sr10sr10	100							
Sr6sr6Sr7aSr7a Sr10Sr10	90	4	4	2				
Sr6Sr6Sr7aSr7a Sr10Sr10	100							
Sr6sr6Sr7aSr7aSr9bsr9bSr10sr10Sr11sr11Sr12sr12	96	3	1	1				
Sr6sr6Sr7aSr7aSr9bsr9bSr10Sr10Sr11sr11Sr12sr12	96	2	1	1				

[a]Data after Knott (27).

[b]It can be assumed that race 15B was virulent on Marquis, *Sr*10, *Sr*11, and *Sr*12; infection of *Sr*9b would result in a moderately resistant to moderately susceptible response; *Sr*7a a moderately resistant response; and *Sr*6 a highly resistant to moderately susceptible response, depending on the temperature conditions.

Brodny et al (3) measured a decrease in pustule size and spore production for individual defeated resistance genes and for their combinations (Table 8). These are probably the only hard data on residual gene action and are based on a single culture and a single series of closely related host materials. Although the study was limited in scope, it certainly indicated that defeated resistance genes may affect resistance. A similar effect is shown by decreased severities when the moderately susceptible *Sr*7a and *Sr*9b were enhanced by the defeated *Sr*10, *Sr*11, and *Sr*12 (Table 7). The host material in this case is more diverse, and again, a single but different culture was used.

Effect of Host Ploidy

Resistance genes transferred from diploid to tetraploid and hexaploid levels generally confer decreasing levels of resistance (25). This difference in resistance is found both in infection types observed (Table 9) and in disease severity. The effect of ploidy level is often obscurred experimentally by variation caused by environment as well as by the heterozygosity of pathogen and host. The effect of ploidy seems to be a dilution effect, but no chemical

Table 8 Reduction in disease response on wheat lines with and without *Sr*6, *Sr*8a, and *Sr*9a infected with a single culture of wheat stem rust[a]

Host gene defeated	Uredia size (mm^2)	Percent of check	Spores/ uredium	Percent of check
Susceptible (check)	4.42		132,236	
Sr6	3.81	86	90,908	69
Sr8a	3.35	76	101,608	77
Sr9a	3.84	87	83,668	63
Sr6 + Sr8a	2.55	58	62,400	47
Sr6 + Sr9a	2.42	55	53,352	40
Sr8a + Sr9a	2.46	56	65,260	49
Sr6 + Sr8a + Sr9a	1.54	35	30,732	23

[a]Data from Brodny et al (3).

basis for a change in resistance as a result of a change in ploidy level is known. This dilution effect is visibly similar to the effect of heterozygosity on a resistance or virulence gene.

Suppressors

A high level of resistance to wheat leaf rust is common in the durum wheats. However, despite using several strains of both the diploid and hexaploid plants and after encountering little difficulty obtaining partially fertile hybrids, attempts to transfer resistance have failed (26). Wheat stem rust resistance in the tetraploid line, TetraCanthatch, is lost when the D genome is included (Canthatch). The suppressor gene was located on the 7D chromosome (26) and has been recently associated with *Lr*34 (12). Fortunately for the agriculturist, the nonsuppressor genotype is the leaf rust resistant genotype.

Table 9 Effect of host ploidy level on low infection types produced by *Puccinia graminis* f. sp. *tritici* on some resistance genes

Sr gene[a]	Low infection types		
	Diploid	Tetraploid	Hexaploid
9d		;1	;2−
9e		;1	;1+
13		;	2+−3
21	;1=		1−2−
dp−2		2−	2+
22[b]	;1	1+	2

[a]Compiled from Roelfs & McVey (48).
[b]From Kerber & Dyck (25).

Background Effects

The genetic background of the host can affect the expression of specific genes for resistance in that host. Low infection types produced on two host lines with the same designed resistance gene often differed to a wide range of cultures (47, 48). When a stem rust resistance gene is transferred into several different but susceptible host backgrounds, varying low infection types often result (Table 10). The near-isogenic line pairs for *Sr*5 in a Chinese spring background showed difference in low infection type between a 0; and a 0;+ (31). Under the greenhouse environment we use, the low infection types for the ISr5-Ra and ISr5-Rb lines are 0 and 0;, respectively. When genes for specific resistance are transferred to cultivars that are susceptible to a wide range of diseases (e.g. Morocco or Little Club), the low infection types are often greater. Whereas resistance genes transferred into cultivars resistant to a range of diseases (Lee, Marquis, Wichita) often have a reduced low infection type.

Skovmand (56) evaluated 80 progeny from crosses between several susceptible cultivars using the area under the disease progress curve (AUDPC) as the disease measurement. Among each progeny some individuals were more resistant than either parent and likewise some progeny were more susceptible than either parent (Table 11). In some cases the lower AUDPC values may be due to the poor agronomic type of the host. Whether these lines are segregating for resistance genes with small effects or for physiological processes that make the plant a better or poorer source of nutrition for the pathogen is unknown. Dark green plants often have been observed to be more rusted than chlorophyll deficient ones of the same resistance genotype. In a few cases the difference in AUDPC may be related to host architecture; for example, a dense compact plant will often have more disease, perhaps partly because of heavier dew formation and duration.

Table 10 Effect of host background on the expression of selected genes for resistance to strain 21–4,5 of wheat stem rust

Host line and pedigree	*Sr* gene	Infection type[a]
Marquis	Check	4
Kanred (Crimean selection)	(Sr5)	0
Reliance (Marquis/Kanred)	(Sr5)	0
Thatcher/6*Marquis[b]	(Sr5)	0
Line B (W2691/7*Reliance)	(Sr5)	X+3–
Marquis/6*Kenya 117A	(Sr9b)	2–3C
Line AA (Kenya 117A/5*W2691)	(Sr9b)	2+

[a]Modified from Luig & Rajaram (34) for constant temperature of 18C.
[b]Thatcher is from the cross Marquis/Iumillo//Marquis/Kanred, W2691 = Little Club//Gabo*3/Charter selection, like Little Club for stem rust susceptibility.

Table 11 Effect of host background on disease resistance expressed as area under the disease progress curve

Cross	Number of F_2 plants in each area under the progress curve class[a]											
	100–200	200–300	300–400	400–500	500–600	600–700	700–800	800–900	900–1000	1000–1100	1100–1200	1200–1300
Prelude/Lee	1	2	3	3	11	12	18	19	7	3	1	
Baart/Lee			1	2	6	19	33	13	5	1		
Baart/Prelude					3	6	8	23	27	5	6	2
Lee/Marquis				3	8	21	25	17	7			
Prelude/Marquis			8	9	17	25	14	4	3			
Baart/Marquis			1	1	13	26	21	15	3			
Baart								X[b]				
Lee						X[b]						
Marquis							X[b]					
Prelude							X[b]					

[a]Tabular presentation of selected data from Skovmand (56).
[b]X = parental class.

EFFECT OF HOST AGE Only *Sr*2 of the genes for specific stem rust resistance gene is effective only in the adult plant stage. Seedling tissue is susceptible, but as the plant matures the number and size of uredia decrease until at near maturity successful penetrants are limited to the immediate area of the nodes and awns (23, 59). Adult plant resistances seem more common in the wheat leaf rust system (51). *Sr*25 is primarily a seedling resistance that is rather ineffective after host anthesis. The other specific resistance genes are generally effective throughout the host life cycle.

EFFECT OF HOST TISSUE Even when there is only a single gene for resistance and virulence not all host tissues respond the same. Seedling infection types X, Y, Z result when the fate of individual penetration events varies. Initially the mesothetic reaction was assumed to be due to the combined effect of several genes for resistance. However, when the actions of genes for resistance are studied in detail this is not always the case. *Lr*11 and *Lr*22a (45) for leaf rust resistance result in Y infection types. In the clearest studied case of wheat stem rust, *Sr*36 results in a modified infection type X. In this case some penetrants elicited in a fleck infection type because most of the haustorial mother cells were attached to necrosed host cells. Other adjacent penetrants had fewer initial haustorial mother cells attached to necrosed host cells; the result was a compatible infection type 4 similar to that of the susceptible host (50). *Sr*6, *Sr*12, *Sr*15, and *Sr*17 can also result in a mesothetic low infection type (33).

In the adult plant stage, *Sr*36 elicited a response that has often been called slow rusting, where the initial level of infection is low due to low receptivity (30% of the infections on a susceptible check with a similar inoculum load), and uredia development is slower for the successful penetrants; however after anthesis the leaf blades were more receptive than those of the susceptible check (49–51). This occurs to a lesser extent on the primary leaf where about 70% of the penetrants are successful (49, 50).

EFFECT OF ENVIRONMENT

Environmental effects on infection types produced by the interaction of *P. graminis* f. sp. *tritici* and *Triticum* species are well known. Browder (4) reviewed the effect of temperature on the expression of infection type. The infection type produced by the *Sr*6 varies from a fleck at 15°C and increases to a 3+ at 24°C (Table 12). This temperature effect occurred during a critical period from 3 to 4 days following penetration (18). The *Sr*6 response changes less with diurnal temperature differences than expected from constant temperature experiments. Other genes for stem rust resistance involved in temper-

ature-sensitive low infection types are *Sr*10, *Sr*15, and *Sr*17 (48). However, almost any gene combination may result in a temperature-sensitive response if the extremes involved are great enough. *Sr*15 is the most temperature sensitive of the stem rust resistance genes currently studied. A change from 18° to 20°C results in *Sr*15 response changing from :1CN to a 4−. Under our greenhouse environment *Sr*13 produces a low infection type of 2+3 at 18°C and a ;1= at 26°C with high light intensity, long days, and diurnal temperature fluctuations.

The response of Kota at 18°C to certain cultures of race 32 results in an infection type 4 or a 2- for light intensity of 10,000 or 8,000 lux, respectively. Other responses vary less with light intensity. However, many of the infection types characterized by chlorosis and/or necrosis often have enhanced chlorosis or necrosis with higher light intensity.

Temperature, and perhaps light, can also affect the infection type enough to result in a reversal of dominance (see Table 12). The heterozygous host genotype is often more affected by these environmental factors than the homozygous genotype. However, large changes in temperatures can completely negate the actions of some homozygous genotypes (see *Sr*5 and *Sr*6, Table 12) (4, 28, 34).

Table 12 Change in infection type due to various temperature regimes when selected wheat lines with *Sr* genes in different lines are infected with *Puccinia graminis* f. sp. *tritici*[a]

Genotypes[b]	Temperature (C)					
	15	18	21	24	27	30
W2691 (Little Club/Gabo*3/ Charter)	3+	3+	3+	3+	3+	3+
Marquis	3+C	3+C	3+C	3+C	3+	3+
Kanred (Sr5)	0	0	0	0	0	0
Thatcher/6* Marquis (Sr5)	0;	0	0;	0;	01=	3CN
Line B (Sr5) (W2691/7*Reliance)	X+3–	X+3–	X+3	X+3	X+3	3
Marquis*6/Kenya 117A (Sr9b)	23C	2–3C	2–3C	3–C	2+3–C	2+3C
Line AA (Sr9b) (Kenya 117A/ 5*W2691)	2+	2+	2+	2+3–	2+3–	3–
Kenya 58/6*Marquis (Sr6)	;	1+3–CN	3CN	3+	3+	3+
Lee/10*Marquis (Sr11)	2=	2=	2=	2=	2=	2=2–
Line AG (Sr11)(Soft Baart/Sterling/ /W2691/3*Yalta)	2=	2=	2=	2=	2=	2–

[a]Constant temperatures at 3,000 ft-c, modified from Luig & Rajaram (33).
[b]Thatcher = Marquis/Iumillo//Marquis/Kanred and Reliance = Marquis/Kanred.

INHERITANCE OF PATHOGEN VIRULENCE

Inheritance of virulence in the pathogen has been inadequately studied in *P. graminis* f. sp. *tritici*. Initial studies were handicapped because the hosts used for detecting virulence had several effective genes for resistance (57). Later studies seemed to be handicapped by the inability to germinate teliospores (47). The general assumption has been that virulence is recessive, but it is likely that in nearly as many cases it is dominant (19). It appears that many virulence loci are in the heterozygous condition in nature (9, 22, 41, 42). At least a few virulence characters are cytoplasmically inherited as is virulence for *Pg*-3 in the *P. graminis* f. sp. *avenae*-Avena spp. (20). It is anticipated that some interaction occurs between specific genes for virulence and the rest of the pathogen genotype; elucidation of such relationships awaits further experimentation.

Many crosses have been made between the various *formae speciales* of *P. graminis* (9, 21, 57). Green (21) believed that the *formae speciales* of *P. graminis* evolved from a rust that attacked barberry and certain gramineous hosts. *Formae speciales* probably evolved by recombinations that increased virulence on cultivated hosts at the expense or virulence on other hosts. Thus, hybrids between the *formae speciales* may resemble the ancestral type more closely than today's specialized forms. The ancestral form had a wide host range but low virulence and evolution produced specialized strains with high virulence on a limited number of hosts (21).

Little is known about the role of barley and its wild relatives in the evolution of *P. graminis* f. sp. *tritici* and f. sp. *secalis*. However, barley is more susceptible to the hybrid (F_1) cultures and their progenies than either wheat or rye. Many barley cultivars are susceptible to wheat stem rust and rye stem rust, but neither wheat nor rye stem rust is highly specialized or aggressive on barley. Thus, the barley-stem rust relationship has the presumed primitive characteristics (little specialization and low pathogen aggressiveness) (21).

THE FUTURE

It should be apparent from this review that a very complex system of genes interacts in both the host and pathogen; the results range from no visible effect to various degrees of disease. Much has been learned about the genetics of specific genes for resistance in the host, but little is known about the genetics of virulence. The available information is from a very few crosses among a few pathogen genotypes. Interorganismal genetics for *P. graminis* and *Triticum* spp. is based on near-isogenic host stocks, but the relationship between pathogen cultures has only recently been postulated (5, 47) for selected

cultures. If techniques are available for making crosses between specific cultures (47), advances in pathogen genetics in the next decade will make it possible to determine what a resistance gene does, what a virulence gene does, and what constitutes the basis of recognition between host and pathogen. Several interesting questions can then be asked. Why do allelic series for resistance exist? Why are they less common in the small grain cereals than in corn and flax? Do allelic series exist for the pathogen? What are the evolutionary functions of avirulence alleles? Are all avirulence alleles similar? What effect does the total pathogen genotype have on the expression of the specific resistance or virulence alleles? How general is the gene-for-gene interaction for disease response? Are genes for resistance ever effective against two pathogen species (i.e. *P. graminis* and *P. recondita*)? How unique are the resistance genes among host species, among genera? It appears that the technology is available to solve some of these problems, and progress is limited only by the willingness to address them.

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