

Possible Simple Inheritance of Resistance to Stripe Rust in Basin Wildrye

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ABSTRACT

Disease readings on clones of Basin wildrye and their progenies indicated that one locus conditioned resistance to a Bozeman isolate of the stripe rust fungus, *Puccinia striiformis*. Frequencies of infection-type classes fit a model

based on five genotypes expected in segregation from an autotetraploid. There was an apparent dosage effect for resistance.

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Additional key words: disease resistance genetics, forages, gene dosage.

With the exception of alfalfa, studies of the inheritance of disease resistance in forage species have lagged behind efforts expended on other crops such as cereals and vegetables. There are two reasons for this: (i) losses from many diseases are either relatively insignificant in many forages or the magnitude of losses has not been estimated; and (ii) perennial habit and ploidy levels of many forages present serious obstacles to both the plant pathologist and the plant breeder.

Stripe rust, caused by *Puccinia striiformis* West., is potentially a serious disease of wheat in the intermountain region of the western United States. Its range of hosts encompasses a wide array of annual and perennial grasses, including Basin wildrye (*Elymus cineris* Scribn. and Merr.), a species we studied because of its potential for high yield and early spring grazing.

Most investigations into the host-pathogen relationships involving *P. striiformis* have considered wheat (*Triticum aestivum* L.) as the only experimental host. In wheat, resistance to the stripe rust fungus can be conditioned by a single gene, or at least is often simply inherited (1, 4, 5). Lewellen et al. (4) reported resistance in wheat to be conditioned by a major gene and several minor genes.

In 1968, variation in susceptibility to stripe rust among plants of Basin wildrye was observed in our breeding nurseries. We sought to determine the genetic basis, if any, for this variation.

MATERIALS AND METHODS.—In 1969, four-leaf stage seedlings representing the open pollinated progeny of 64 spaced plants of Basin wildrye, representing a single SCS collection (Wy 107), were inoculated in a settling chamber with a Bozeman isolate (ATCC PR No. 35) of *P. striiformis*. To enhance penetration and establishment of the pathogen, the seedlings were placed in an unlighted, controlled environment chamber at 7 C under simulated dew conditions for 24 hours immediately after inoculation. They were then grown 14 days in an environment chamber with a day/night temperature profile at 15/24 C and a 12-hour light period with light intensity approximately 26,900 lx. These conditions are optimum for the disease according to Sharp (6). Following this period, seedlings were scored for infection type following the method of Lewellen et al. (4). Infection types are based on the magnitude of disease symptoms and signs. Infection types used were: i = no macroscopic evidence of infection; v = nearly immune (however, very minute chlorotic flecks were usually evident); 00 = small

TABLE 1. Summary of response of progenies of 64 plants of Basin wildrye to the stripe rust fungus, *Puccinia striiformis* (ATCC PR No. 35)

Genotypes	Frequencies [(p + q) ⁴]	Infection type ^a	No. of seedlings	
			observed	expected
<i>a₁a₁a₁a₁</i>	p ⁴	i, v	989	992.4
<i>a₁a₁a₁a₂</i>	4p ³ q	00		
<i>a₁a₁a₂a₂</i>	6p ² q ²	0		
<i>a₁a₂a₂a₂</i>	4pq ³	1,2	165	161.6
<i>a₂a₂a₂a₂</i>	q ⁴	3,4		

^aInfection-type classes defined:

- i = no macroscopic evidence of infection.
v = nearly immune to immune; however, very minute chlorotic flecks were usually evident.
00 = small necrotic, symmetric flecks.
0- = larger necrotic flecks, usually non-symmetrical.
0 = necrosis spanning the entire width of the leaves without pustulation.
1 = necrosis with small pustules.
2 = necrosis and chlorosis with larger pustules.
3 = no necrosis but chlorosis with normal pustules.
4 = no necrosis or chlorosis with normal pustules.

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pustules; 3 = no necrosis, but chlorosis with normal pustules; 4 = no necrosis or chlorosis with normal pustules. A total of 1,154 seedlings was scored; minimum progeny size was 15 seedlings per maternal plant. The maternal plants were planted at the Agronomy Field Research Laboratory near Bozeman, Montana in June, 1967.

In the fall, 1971, propagules of a representative sample of the field-grown maternal plants whose progenies had been tested for stripe rust resistance were transplanted to a glasshouse. Each plant was cut to a uniform height of 8 cm. Reaction of maternal plants to *P. striiformis* was scored on regrowth tissue. Regrowth leaf tissue on individual propagules of maternal plants was inoculated with the same Bozeman isolate of the fungus and scored for infection type.

The data from independent, preliminary cytological studies of less than 10% of the maternal plants suggested a tetraploid pattern of inheritance. Darlington and Wylie (3) reported both tetraploid (2N = 28) and octaploid strains of *E. cinerus*; however, the tetraploid strains were more common. Chapman (2) reported Basin wildrye to be highly cross-pollinated.

RESULTS AND DISCUSSION.—Of the 64 progenies scored, 10 with more than 20 seedlings each were found to be uniformly resistant [infection type i (no symptoms), or infection type v (vague chlorotic flecking on leaves)]. Based on these data, we assume that the maternal plants were homozygous resistant. Initial

TABLE 2. Summary of maternal plant and progeny response of Basin wildrye to the stripe rust fungus, *Puccinia striiformis* (ATCC PR No. 35)

Bozeman plant no.	Maternal plant infection type ^a		Proposed genotype	No. of progeny in infection type classes				
	observed	expected		i,v	00,0-	0	1,2	3,4
2-11	0	0	<i>a₁a₁a₂a₂</i>	6	36		21	2 ^c
3-11	00,0-	0	<i>a₁a₁a₂a₂</i>	4	13	6	5	17
4-12 ^b	00,0-	0	<i>a₁a₁a₂a₂</i>	10	30		6	12
6-14	i,v	i,v	<i>a₁a₁a₁a₁</i>	14		1		
7-12	00,0-	00,0-	<i>a₁a₁a₁a₂</i>	16	1	1		
8-26	00,0-	00,0-	<i>a₁a₁a₁a₂</i>	14				
10-12	00,0-	00,0-	<i>a₁a₁a₁a₂</i>	42	12			
11-12 ^b	00,0-	0	<i>a₁a₁a₂a₂</i>	10	35		26	4 ^c
15- 7 ^b	00,0-	0	<i>a₁a₁a₂a₂</i>	15		1		5
15-10 ^b	00,0-	0	<i>a₁a₁a₂a₂</i>	4	19	22	26	15
15-25	3,4	3,4	<i>a₂a₂a₂a₂</i>			27	16	62
17- 8	i,v	i,v	<i>a₁a₁a₁a₁</i>	15				
19- 7	00,0-	00,0-	<i>a₁a₁a₁a₂</i>	9	30	3	1	
20- 5	2	2	<i>a₁a₂a₂a₂</i>		17	22	6	16
21- 7	00,0-	00,0-	<i>a₁a₁a₁a₂</i>	17		2		
21-25 ^b	00,0-	0	<i>a₁a₁a₂a₂</i>	4	24	10	7	24
23-27	0	0	<i>a₁a₁a₂a₂</i>	13		1		

^aInfection-type classes defined:

- i = no macroscopic evidence of infection.
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^bMaternal plant classification questionable due to regrowth tissue.

^cSeedlings may have been misclassified.

genetic analysis is based on the assumptions of Hardy-Weinberg equilibrium which are in part justified by the high level of cross-pollination in *E. cinerus*. Thus, the allelic frequency for resistance P , is $\sqrt[4]{10/64} = 0.64$. Assuming only two alleles per locus, the allelic frequency for susceptibility is $1-p = .36$.

Segregation at a single locus in an autotetraploid yields five genotypes. Genotypic frequencies in a panmictic population are determined from the expansion of $(p+q)^4$. Because more than one infection type can be associated with the same host genotype (6), and because possible segregation of modifying loci (4) precluded association of specific infection types and host genotypes, interpretation of the data was based upon the frequency of resistant or nearly resistant seedlings versus moderately to highly susceptible seedlings (Table 1).

The more resistant seedlings were scored as infection types i , v , 00 , $0-$ or 0 ; susceptible seedlings were scored 1, 2, 3, or 4. All resistant types were free of pustules; all susceptible types had pustules. Thus, there was a clear-cut separation into two classes.

A satisfactory fit of observed to expected numbers of seedlings for the two classes was obtained when the expected number of resistant individuals was based on the sum of frequencies of the first three elements of the expansion of $(p+q)^4$, and the expected number of susceptible seedlings was based upon the last two elements (Table 1). If a_1 is the allele for resistance and a_2 the allele for susceptibility, resistant genotypes would be $a_1a_1a_1a_1$, $a_1a_1a_1a_2$, and $a_1a_1a_2a_2$, and susceptible genotypes would be $a_1a_2a_2a_2$ and $a_2a_2a_2a_2$.

The possibility of a dosage effect for the resistance is suggested based upon subdivision of both resistant and susceptible phenotypes. The i and v infection types may be associated with the homozygous resistant genotype, $a_1a_1a_1a_1$. Other infection types representing reduced levels of resistance may be associated with the genotypes $a_1a_1a_1a_2$ (infection types 00 and $0-$) and $a_1a_1a_2a_2$ (infection type 0). Similarly, the most susceptible individuals, infection types 3 and 4, may be associated with the homozygous genotype $a_2a_2a_2a_2$; and a lower level of susceptibility, infection types 1 and 2, with the $a_1a_2a_2a_2$ genotype.

The validity of this model can be tested by comparing the observed infection type of the maternal plant to the infection types of its progeny (Table 2). If the model is valid, maternal plants classed as i or v should segregate in the range of infection types i or v to infection types 0 , genotypes $a_1a_1a_1a_1$ and $a_1a_1a_2a_2$, including $a_1a_1a_1a_2$. Similarly, a maternal plant scored as infection type 3 or 4 should segregate progeny with infection types 3 and 4 to infection types 0 and 1, genotypes $a_2a_2a_2a_2$ to $a_1a_1a_2a_2$. This pattern of segregation persists for plants 6-14, 17-8,

and 15-25 (Table 2). The range of segregation of progeny of maternal plants classified as other infection types generally agrees with the model. Plant 20-25 is classified as infection type 2, genotype $a_1a_2a_2a_2$, and the range segregation in its progeny includes all but the i or v infection types. The major problem exists in interpreting progenies of plants with the 00 and 0 infection types. Several maternal plants classified as $0-$ appear, based on progeny performance, to be 0 . This phenomenon is explained on the basis that, although different genotypes are proposed for these phenotypes, the two infection types are similar, and clear-cut definition is at best difficult. A further complication was that maternal infection type was based on relatively sparse regrowth tissue compared with leaf tissue of the seedlings on which progeny evaluations were made.

No attempt was made to test ratios in the progenies. Although progeny size was adequately large to detect segregation, in nearly all cases it was not large enough to fit ratios.

Larger samples may have resulted in improved fits of observed to expected values and closer approximation of Hardy-Weinberg equilibrium conditions.

We conclude from our data that resistance to stripe rust in Basin wildrye is conditioned by segregation at a single locus. There is an apparent dosage effect for resistance. Regrowth tissue may be less susceptible than seedling tissue, and the association of more than one infection type with the proposed genotypes suggests the possibility of additional, minor genes for resistance.

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