Number of Genes Controlling High-Temperature, Adult-Plant Resistance to Stripe Rust in Wheat

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ABSTRACT

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Winter wheat cultivars Gaines, Nugaines, and Luke have durable adult-plant, temperature-sensitive resistance to *Puccinia striiformis*. Parental, F_1, F_2, and backcross populations from reciprocal crosses between these resistant cultivars and reciprocal crosses between each resistant cultivar and a susceptible line (PS-279) were evaluated in the field for rust intensity in 1982. F_4 rows were evaluated for resistance as measured by rust intensity at three locations in 1984. Estimations based on three quantitative formulas indicated that rust intensity was controlled by two or three genes in Nugaines and Luke. A qualitative analysis based on the proportion of resistant F_2 progeny indicated that resistance in Gaines, Nugaines, and Luke was determined by two genes, and that Gaines and Nugaines have one locus in common. Gaines and Nugaines probably have different alleles at the second locus, since there was no transgressive segregation in the Nugaines × Gaines cross. Because of the assumptions in these formulas, these estimates are for the minimum number of genes involved. Based on the very low estimates of the number of resistance genes in the Luke × Nugaines and Luke × Gaines crosses and on transgressive segregation for both resistance and susceptibility in the F_4 generation, the genes in Luke differ from those in Gaines and Nugaines. Progeny with enhanced levels of resistance should be useful sources of durable resistance for breeding programs.

Additional key words: durable disease resistance, effective factors, horizontal resistance, nonspecific resistance, partial resistance, polygenic inheritance.

The stripe rust resistances of Gaines, Nugaines, and Luke winter wheats (*Triticum aestivum* L. em Thell.) are among the best examples of durable, race-nonspecific resistance. Since their release (Gaines in 1961, Nugaines in 1965, and Luke in 1970), these cultivars have remained resistant in the adult stage to all races of *Puccinia striiformis* West. in the Pacific Northwest and were resistant to European races when tested in 1980 (9). Stripe rust develops slowly on these cultivars in the field. When grown at low (e.g., 2–18 C) or high (e.g., 10–30 C) diurnal temperature cycles, seedlings of Gaines and Nugaines are susceptible to all races, and seedlings of Luke are susceptible to the most prevalent races of the pathogen (15). As plants of these cultivars mature, they become more resistant when grown at the high-temperature cycle but remain susceptible when grown at the low-temperature cycle (15). Based on other research (12), resistance in these cultivars is partially recessive, and gene action is mostly additive among loci along with some epistatic gene action.

Genes for adult-plant or high-temperature resistance to stripe rust have been identified in many cultivars (1,2,4,5,7,8,14,17,18,20), and even susceptible cultivars have been reported to have genes for resistance (7,14,18,20). Transgressive segregation for enhanced resistance frequently has been observed among progeny derived from crosses between cultivars (1,7,14,18,20). This type of resistance has been reported to be controlled by a few genes (20), at least three genes (5,8,17), many genes (18), polygenes (4), and one, two, or three genes (1). The research reported here was initiated to estimate the number of genes or effective factors controlling stripe rust intensity in cultivars (Gaines, Nugaines, and Luke) with high-temperature, adult-plant resistance.

MATERIALS AND METHODS

Individual plants of Gaines, Nugaines, Luke, and PS-279 were transplanted to a crossing block at Pullman, WA, in mid-April, 1981. Gaines, Nugaines, and Luke are soft white winter wheats with common heads. PS-279 (subsequently referred to as the susceptible parent) is a highly susceptible club wheat derived from Suwon 92/7*Omar by R. E. Allan and lacks any known genes for stripe rust resistance. Reciprocal crosses in all combinations, except Luke × Gaines, were made between individual plants of each cultivar and replicated at least five times. In August, germinated parental and F_1 seed from the three replicates per cross that had the most F_1 seed were vernalized for 5 wk at 0–4 C and a 12-hr photoperiod. Then the seedlings were planted into 15-cm-square pots filled with a potting mixture (6 parts peat, 2 parts perlite, 3 parts sand, 3 parts Palouse silt loam soil, and 4 parts vermiculite, plus lime, 14-14-14 Osmocote and ammonium nitrate fertilizers) and placed outside in a lath house during October for 2 wk of additional vernalization. The plants were then placed in a greenhouse to produce F_2 and backcross seed during the winter. Backcrosses were made between reciprocal F_1s and their female parent, but reciprocal backcrosses were not made. The F_1s were the pollen parent. Backcross and F_2 heads were harvested 32 days or more after pollination and dried 2–3 days at about 30 C. In early March, parental, F_1, F_2, and backcross seed were planted in peat pellets or peat pots filled with the potting mixture. The seedlings were moved to a lath house when the coleoptiles emerged and were kept there until they were transplanted to the field in five randomized blocks at Pullman, WA, between 23 and 30 April. Each of three replicates per cross consisted of 20 plants of each parent, 20 plants of each reciprocal F_1, 150 plants of each reciprocal F_2, and 15–135 plants of each backcross. There were about 8,000 plants in the experiment.

Plants were uniformly dusted with urediospores of *P. striiformis* race CDL-20 (a prevalent race during previous years) on 28 May when plants were in the tillering to early jointing stages of growth. At that time, naturally occurring stripe rust was not evident in the plot. Sporulating uredia were observed on 12 June. Rust was uniformly distributed with about two infections per plant, and all initial infection types were high. Rust increased rapidly in the plots, and race CDL-20 accounted for nearly 100% of the inoculum. Stage of growth and rust intensity (percent foliage with symptoms), were recorded for each plant on 2–7 July (boot to flowering), 12–17 July (heading to soft dough), and 26–31 July (flowering to hard dough). Data for each block were recorded within a 30-hr period.

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Because of the time required for recording the data, recording dates varied from block to block, but the time from first to second recording was 10 days and from second to third recording was 14 days for all plants. The rust intensity percentages recorded were 0, 2 (trace–4), 7 (5–10), 15 (11–20), 30 (21–40), 50 (41–60), 70 (61–80), 85 (81–90), 93 (91–96), and 98 (>96). Area under the disease progress curve (*AUDPC*) for rust development on each plant was calculated from the original intensity data by the formula $AUDPC = [10(X_1 + X_2/2)] + [14(X_2 + X_3/2)]$. X_1, X_2, and X_3 are the rust intensities recorded on the first, second, and third recording dates, respectively.

Since progeny of each replicate per cross were derived from a cross between an individual plant of each cultivar, it was possible to detect heterogeneity for stripe rust resistance in Nugaines and Luke during the 1982 season. One Nugaines parent had the rust reaction of Gaines, therefore, when used in crosses it provided a Luke × Gaines cross. One Luke parent had higher resistance to stripe rust than the other Luke parents, and the highly resistant Luke (HR Luke) provided an HR Luke × Nugaines cross .

For each cross, 300 randomly selected F_2 plants representing all replicate and reciprocal crosses were harvested. Approximately 60–120 seeds of each harvested plant were sown in a 2-m row at Pullman, WA, in October 1982. The following year, 100 randomly selected F_3 rows per cross, representing all replicate and reciprocal crosses, were harvested. Approximately 20–40 F_4 seeds from each F_3 row were planted in a single 1.5-m row at Mount Vernon, Walla Walla, and Pullman, WA, in October 1983. Rows of each parent were replicated four times at each of the three locations. A stripe rust epidemic from naturally-occurring inoculum developed at each location.

Segregation for rust intensity was estimated twice at Mount Vernon, Walla Walla, and Pullman for each F_4 row. Although rust intensity appeared to vary continuously in some rows, it was not possible to document this variation precisely since resistant lines had intermediate rust intensities and observations were on a row basis rather than on a plant basis. Our system for recording segregation allowed for only two rust intensities per row and designation of 3:1 and 1:1 segregating ratios. For example, if most plants could be grouped into one intensity class and a smaller portion of the plants had either a higher or lower intensity, then three-quarters of the row was considered to have the first intensity and one-quarter of the row was considered to have the second intensity (3:1 ratio). The other segregation ratios were 1:1 (approximately equal portions of two intensities) and 1:0 (all plants had the same intensity and showed no segregation).

TABLE 1. The number of genes (effective factors) controlling the area under the disease progress curve in seven wheat crosses as estimated by four genetic formulas[a]

Cross		Estimated number of genes			
Parent 1	Parent 2	Formula 1[a]	Formula 2[b]	Formula 3[c]	Formula 4[d]
Luke	Susceptible	1.8	1.7	1.6	0.4
Nugaines	Susceptible	2.8	2.0	1.8	0.2
Gaines	Susceptible	6.5	9.0	70.0	0.1
Nugaines	Gaines	3.0	1.7	1.6	0.0
Luke	Gaines	1.2	0.7	1.1	0.4
Luke	Nugaines	0.0	0.3	0.8	0.4
HR Luke	Nugaines	0.3	0.2	1.0	0.1

[a] Formula 1: $N = [d]^2 / V_A$ in which N = number of genes, $[d]$ = additive component of gene action, and V_A = additive genetic variance.

[b] Formula 2: $N = (\bar{P}_2 - \bar{P}_1)^2 [1.5 - 2h(1-h)]/8(V_{F_2} - V_E)$ in which $\bar{P}_1$ = mean of resistant parent, $\bar{P}_2$ = mean of susceptible parent, $h = \bar{F}_1 - \bar{P}_1/\bar{P}_2 - \bar{P}_1$, $\bar{F}_1$ = mean of F_1, V_{F_2} = variance of F_2, V_E = environmental variance $[0.25(V_{P_1} + V_{P_2} + 2V_{F_1})]$, and V_{F_1} = variance of F_1.

[c] Formula 3: $S_1 = (\bar{F}_1 - \bar{P}_1)^2/4(V_{B1} - V_E)$ in which S_1 = number of genes by which the F_1 differs from P_1, $\bar{F}_1$ = mean of F_1, $\bar{P}_1$ = mean of resistant parent, V_{B1} = variance of backcross to P_1, and $V_E = 0.5(V_{F_1} + V_{P_1})$.

[d] Formula 4: $S_2 = (P_2 - F_1)^2/4(V_{B_2} - V_E)$ in which S_2 = number of genes by which the F_1 differs from P_2, V_{B_2} = variance of backcross to P_2, and $V_E = 0.5(V_{F_1} + V_{P_2})$.

To average segregation and rust intensity over both sets of notes at each location, ratios were expressed by four values. For a 3:1 ratio, three values were expressed as one intensity and one value as the other intensity; for a 1:1 ratio, there were two values of each intensity; and for a 1:0 ratio, all four values were the same. The rust intensities assigned to each fourth of the row were ranked from lowest to highest for each date, and the data from the dates were averaged to obtain four mean values. The mean values were put into four resistance classes, which corresponded to the rust intensities of the original parents: very resistant = 0–8% (HR Luke), resistant = 9–30% (Luke), moderately resistant = 31–85% (Gaines and Nugaines), and susceptible 86–98% (susceptible parent). If the four mean values for each row were in two or more classes, the row was considered to be segregating; if all four means were in the same class, the row was considered to be nonsegregating.

Four quantitative genetic formulas were used to estimate the number of genes controlling the inheritance of rust intensity by using AUDPC data from the early generations (parents, F_1, F_2, and backcrosses) (Table 1). Formula 1 was proposed by Mather and Jinks (11) and formulas 2–4 were proposed by Wright (21). The formulas are based on the assumptions that resistance genes that segregate in a cross are in only one parent, segregating resistance genes are not linked, all resistance genes have equal effects, additive × dominance epistasis is not important, and genotype × environment interactions do not exist. Formulas 1 and 2 also assume that dominance is equal at all loci.

The proportion of parental phenotypes in the F_2 generation, which was used by Parlevliet (13) to estimate the number of genes controlling partial resistance to barley leaf rust, was used as a qualitative method of analysis. Since resistance in Gaines, Nugaines, and Luke is recessive (12), the frequency of a homozygous recessive genotype in the F_2 generation is $(1/4)^N$ in which N is the number of segregating loci involved. Within each cross, F_2 plants with *AUDPC* values less than or equal to the highest *AUDPC* value for the resistant parent were considered to have a resistant phenotype.

Genotypes were assigned to each parent based on recessive resistance and the number of genes estimated by the quantitative formulas and the proportion of resistant plants in the F_2 generation, and resistant phenotypes were determined as previously described. Plants with *AUDPC* values greater than or equal to the lowest F_1 value were considered to have a susceptible phenotype due to partial dominance for susceptibility. Plants with values between these two ranges were considered intermediate. Genetic models assuming various combinations of additive, dominant, and epistatic gene action were tested by comparing the observed numbers of resistant, intermediate, and susceptible plants in the F_1, F_2, and BC generations with the number expected by each model. Data on rust intensity and segregation for rust intensity in F_4 progeny at each location were used to evaluate transgressive segregation for resistance.

RESULTS

Analysis of early generations. For each cross, formulas 1, 2, and 3 (Table 1, footnotes) gave similar estimates of the number of genes controlling *AUDPC*. There appeared to be two genes segregating in the Luke × susceptible cross, two to three genes segregating in the Nugaines × susceptible and Nugaines × Gaines crosses, and one to two genes segregating in the Luke × Gaines cross. Many genes (6–70) were estimated in the Gaines × susceptible cross, and few genes (zero to one) were estimated in the Luke × Nugaines and HR Luke × Nugaines crosses. Formula 4 consistently estimated that the most susceptible parent in each cross contributed less than one gene for resistance.

The percentage of resistant plants in the F_2 generation would be 25, 6.25, or 1.56 when there are one, two, or three recessive genes controlling resistance, respectively. Eight percent of the F_2 plants in the Gaines × susceptible and Nugaines × susceptible crosses had the phenotype of the resistant parent, which indicates that there are two genes in each resistant parent. Eleven percent of the F_2 plants in

the Luke × susceptible cross had the resistant phenotype of Luke, which is closest to the expected percentage for two segregating genes. Twenty one percent of the F_2 plants in the Nugaines × Gaines cross had the phenotype of Nugaines, which indicates that there is only one gene segregating in this cross and that Gaines and Nugaines must have one resistance gene in common.

When Mendelian models were used, the observed number of plants in each phenotypic class approximated the expected values. However, the models did not statistically fit the data as measured by chi-square tests. Mendelian analysis was unsuitable for determining inheritance of this type of resistance because large environmental variances and continuous distribution of the progeny made it difficult to accurately assign plants to discrete classes.

Analysis of F_4 progeny. At Mount Vernon, rust intensities were high in plants at the heading stage, and segregation was difficult to detect; therefore, data from that location were not included in the analysis. Resistance at Walla Walla was greater than at Pullman (Table 2). At Walla Walla, transgressive segregation for both resistance and susceptibility was detected in the Luke × Gaines and Luke × Nugaines crosses. At Pullman, no transgressive segregation for resistance was detected, only transgressive segregation for susceptibility was detected in the Luke × Gaines, Luke × Nugaines, and HR Luke × Nugaines crosses. In the Gaines × susceptible, Nugaines × susceptible, and Luke × susceptible crosses, the phenotype of the resistant parent was recovered at both locations, but a higher proportion of resistant phenotypes were observed at Walla Walla. The distributions of mean percent rust intensity of progeny from the Luke × Gaines and Luke × Nugaines crosses were similar at Pullman, but at Walla Walla the distributions for the Luke × Gaines cross were wider than for the Luke × Nugaines cross. Most progeny from the HR Luke × Nugaines cross were very resistant at both locations.

F_4 families from the Luke × Gaines, Luke × Nugaines, and HR Luke × Nugaines crosses had more segregating rows than F_4 families from the Gaines × susceptible, Nugaines × susceptible, and Luke × susceptible crosses (Table 2). This is additional evidence that resistance genes in Luke differ from those in Gaines and Nugaines. In 1984, rust intensities for Gaines and Nugaines were so similar that segregation in the F_4 families from the Nugaines × Gaines cross could not be detected (Table 2). Therefore, because of differences in the environments, at least one phenotypic class that was distinguishable in 1982 was not evident in 1984. This probably reduced the number of segregating rows in crosses involving Gaines and Nugaines.

DISCUSSION

Based on the formulas in Table 1 for estimating the number of genes in the resistant × susceptible crosses and on the proportion of resistant progeny in the F_2 generation, there appears to be two resistance genes in Gaines and Luke and two to three resistance genes in Nugaines. However, the estimates are subject to the assumptions outlined in the materials and methods.

The large number of resistance genes in the Gaines × susceptible and Gaines × Nugaines crosses estimated by the formulas in Table 1 are probably an overestimation of the true number of resistance genes, because of the high environmental variance associated with the Gaines phenotype (12). The proportion of F_2 progeny with a resistant phenotype gave estimates of the number of resistance genes in Nugaines and Luke that were similar to estimates from the quantitative formulas, and gave reasonable estimates for the number of genes in Gaines and for the differences between Gaines and Nugaines.

Since Gaines and Nugaines appear to have two resistance genes and the proportion of Nugaines-like progeny in the F_2 generation of the Nugaines × Gaines cross indicate a single gene difference between the cultivars, Gaines and Nugaines should have a resistance gene in common. This is highly probable, since they are sister selections. Furthermore, the differences between Gaines and Nugaines may be due to different alleles at the second locus, because no transgressive segregation was evident in the Nugaines × Gaines cross (Table 2).

Resistance genes in Luke are different from those in Gaines and Nugaines. Low estimates of the number of genes in the Luke × Gaines, Luke × Nugaines, and HR Luke × Nugaines crosses (Table 1) and transgressive segregation for resistance and susceptibility in these crosses (Table 2) mean that each parent contributed different

TABLE 2. Distribution of mean rust intensity for the parents and the F_4 progeny of seven wheat crosses in 1984 at Pullman (PM) and Walla Walla (WW), WA

			Percentage of segregating and nonsegregating rows[a]								
Genotype	Site	Rows	All VR	Seg. VR:R	Seg. VR:MR	All R	Seg. R:MR	Seg. R:S	All MR	Seg. MR:S	All S
Parents (both sites)											
Susceptible		8									100
Gaines		8							100		
Nugaines		8							100		
Luke		8				100					
HR Luke		–	100[b]								
F_4 Generation											
Gaines × Susceptible	PM	100							2	14	84
	WW	100							11	14	75
Nugaines × Susceptible	PM	98							5	28	67
	WW	100							37	20	43
Luke × Susceptible	PM	100				1	1		9	34	55
	WW	43				4			44	17	35
Nugaines × Gaines	PM	63							100		
	WW	99							100		
Luke × Gaines	PM	70				10	16	21	19	24	10
	WW	100	5	4	5	16	31		35	1	3
Luke × Nugaines	PM	82				16	26	18	12	23	5
	WW	99	2			22	50	1	23	2	
HR Luke × Nugaines	PM	67	38	1	26	13	9	7	3	3	
	WW	94	52	6	11	17	14				

[a] Very resistant (VR) 0–8%, resistant (R) 9–30%, moderately resistant (MR) 31–85%, susceptible (S) 86–98% percent rust intensity. Combinations of two rust intensities (e.g., Seg. VR:R) indicate segregation among plants within a row.
[b] Based on 1982 data.

resistance genes. These results agree with the conclusions from our paper on gene action (12).

The low values estimated by formula 4 (Table 1) and the absence of transgressive segregation for resistance (Table 2) in the resistant × susceptible crosses indicate that the susceptible parent (PS-279) does not contribute any detectable resistance genes. Therefore, the assumption that only the resistant parent contributed the resistance genes appears to be true for the resistant × susceptible crosses.

The analyses estimate the minimum number of genes. However, the resistance genes could be linked and could, therefore, segregate as a group or "effective factor." If this is true, the formulas would estimate the number of effective factors, and the number of individual genes would be greater. However, the experimental design does not allow us to determine whether the genes were linked in effective factors. Johnson (4) hypothesized that some of the genes controlling durable stripe rust resistance were linked and inherited as polygenes (effective factors), since a large part of the resistance was readily transferred in breeding programs. Law et al (8) reported the presence of at least three genes on chromosome $5B^s$ -$7B^s$ that controlled adult-plant resistance to stripe rust in several wheat cultivars. However, the $5B^s$-$7B^s$ chromosome in each of four cultivars conditioned different levels of resistance, and this could be due to different genes or alleles for resistance within three effective factors on the $5B^s$-$7B^s$ chromosome. The effective factor hypothesis can also explain why transgressive segregation for enhanced resistance was frequently observed among progeny from crosses between susceptible parents (7,14,18,20). Susceptible cultivars may have genes for resistance that are in balanced polygenic combinations (6), i.e. genes for resistance are cancelled by linked genes for susceptibility. When two susceptible cultivars with different genes are crossed, recombination within effective factors can replace some of the genes for susceptibility with genes for resistance and produce genotypes with more genes for resistance than for susceptibility. Pope (14) hypothesized that genes controlled functions in a sequence of events leading to resistance. In this model, each gene alone has no effect, but high levels of resistance can be achieved when the necessary combination of genes is produced by crossing.

Since an effective factor consists of linked genes, the estimated number of genes must be expected to increase as generations advance, because linkage groups will continue to be broken in later generations. Jinks and Towey (3,19) developed experimental designs, based on genotype assay, that could detect segregation within polygenes. The estimated number of genes controlling three characters in tobacco increased as the generations advanced from the F_2 to the F_8, and this increase was attributed to segregation within effective factors. Their studies implied that populations had variation that was hidden in balanced polygenic systems composed of a large number of genes. In inbreeding species, variation will remain hidden once genes become homozygous as a result of selfing. This variation can be exposed by making repeated crosses among selected lines to break old linkage groups and form new, more favorable linkage groups. Once favorable combinations of genes are selected, they should remain linked and segregate together most of the time. Therefore progeny with enhanced levels of resistance should be useful sources of durable resistance for breeding programs.

Genes may appear to have similar effects because of the way their effects are measured (10). In this study, resistance was measured by *AUDPC* or mean rust intensity and would not differentiate between genes controlling infection efficiency, latent period, infection type, or amount of sporulation. It is possible that there may be different genes that control each component. Using the genotype assay procedure to analyze data on components of resistance may yield more precise information on the genes controlling high-temperature, adult-plant resistance to stripe rust.

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