Gene Action for Inheritance of Durable, High-Temperature, Adult-Plant Resistance to Stripe Rust in Wheat

E. A. Milus and R. F. Line

Former graduate student, now with Rohm and Haas Seeds, P.O. Box 111, Berthoud, CO 80513, and plant pathologist, Agricultural Research Service, U.S. Department of Agriculture, Department of Plant Pathology, Washington State University, Pullman 99164-6430, respectively.

Portion of a Ph.D. dissertation by the first author.
Scientific Paper 7030, Washington State University, College of Agriculture and Home Economics Research Center, Pullman. Project 1694.
We acknowledge the assistance of J. E. Teberg for writing the computer program to calculate genetic parameters by joint scaling tests.
Accepted for publication 14 October 1985 (submitted for electronic processing).

ABSTRACT

Milus, E. A., and Line, R. F. 1986. Gene action for inheritance of durable, high-temperature, adult-plant resistance to stripe rust in wheat. Phytopathology 76:435-441.

Gaines, Nugaines, and Luke winter wheat cultivars have durable, high-temperature, adult-plant resistance to *Puccinia striiformis*. Parental, F_1, F_2, and backcross populations from reciprocal crosses between individual plants of resistant cultivars and with individual plants of a susceptible line were evaluated in the field. Rust intensity on each of approximately 8,000 plants was recorded three times during the season, and areas under disease progress curves were calculated from the intensity data. Resistance in the three cultivars was partially recessive (susceptibility was partially dominant) with no maternal inheritance. Epistatic gene action for resistance was significant in Nugaines, but most gene action among loci was additive. Epistatic gene action for susceptibility was detected in Luke, but its probability was lower than the probability for epistatic gene action in Nugaines. The proportion of susceptible progeny was higher than expected in the Gaines × susceptible cross, and gene action among loci in Gaines was not clear. The differences between Gaines and Nugaines were not due to epistatic gene action. Resistance genes in Luke were different from resistance genes in either Gaines or Nugaines.

Additional key words: nonspecific resistance, *Triticum aestivum*.

Stripe rust, caused by *Puccinia striiformis* West., is the most important foliar disease of wheat (*Triticum aestivum* L. em Thell) in the Pacific Northwest. From the early 1930s to the mid 1950s, it was not considered important primarily because most cultivars grown during that period had high-temperature, adult-plant resistance to stripe rust (19) under the environmental conditions of that time (2). During the late 1950s, cultivars without adult-plant resistance were extensively grown, new races capable of overcoming race-specific resistance appeared (10), the climate was more favorable (2), and severe stripe rust epidemics occurred.

Since 1961, the major emphasis for control of stripe rust in the Pacific Northwest has been on enhancing adult-plant resistance. Gaines (CI 13448) was released in 1961 because it had a higher yield than previous cultivars, especially in the presence of stripe rust. Nugaines (CI 13968), a sibling of Gaines, was released in 1965 because it yielded more than Gaines at some sites. Later it was determined that Nugaines was more resistant to stripe rust than Gaines. From 1963 to 1980, Gaines and Nugaines accounted for 54% (12.3–73.5%) of the total wheat production in the Pacific Northwest (17). These two cultivars have high-temperature, adult-plant resistance but have not shown any race-specific stripe rust resistance (11,19). Luke (CI 14586), released in 1970, is even more resistant than Gaines or Nugaines. Luke is highly resistant in the adult stage and never has been severely damaged by stripe rust (11).

Since their release, adult plants of these three cultivars have remained resistant to all races in the Pacific Northwest and were resistant to races in western Europe when tested in 1980 (12). In the field, as the season progresses and temperatures increase, infection types become lower, and rust develops slower on these cultivars than on susceptible cultivars. Under controlled conditions, seedlings of these cultivars are susceptible to the prevalent races over a wide range of temperatures, but as plants mature they become more resistant when grown at high temperatures (diurnal temperatures of 10–30 C or higher); however, at low temperatures (diurnal temperatures of 6–21 C or lower) the plants remain susceptible (19). Cultivars that were resistant (had a low infection type) at a high postinoculation temperature became susceptible (had a high infection type) when subsequently transferred to a lower temperature (19). At the high temperatures, Luke is more resistant than Nugaines, and Nugaines is more resistant than Gaines.

There are few reports on the gene action (whether genes are expressed in an additive, dominant, and/or epistatic fashion) for inheritance of temperature-sensitive resistance or adult-plant resistance to stripe rust and none on the gene action for high-temperature, adult-plant resistance. Lewellen et al (9) studied the effect of temperature on resistance of PI 178383 to stripe rust and reported that "minor genes" conferred small increments of resistance that were additive, possibly race-nonspecific, and temperature sensitive in the seedling stage. Krupinsky and Sharp (7) reported that minor genes from PI 178383 acted in an additive fashion with recessive, temperature-sensitive genes from several commercial cultivars conferring higher levels of resistance. Some of the resistance was also attributed to cytoplasmic effects. Pope (18) reported the existence of more than 20 recessive, additive, minor genes for stripe rust resistance and concluded that susceptible cultivars had genes for resistance since there was transgressive segregation for higher resistance among the progeny of crosses with susceptible cultivars. Krupinsky and Sharp (8) found transgressive segregation for increased resistance among advanced-generation progeny even when the F_2 and F_3 generations of some of these crosses were highly susceptible. They concluded that even cultivars with susceptible reactions could contribute minor genes for resistance, which may be a source of durable resistance. Lupton and Johnson (13) reported that durable, adult-plant stripe rust resistance in cultivar Little Joss wheat was recessive and that its genetic control was complex. Wallwork and Johnson (21) found transgressive segregation for higher stripe rust resistance among F_2 and F_3 progeny from susceptible × susceptible crosses and among

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

This article is in the public domain and not copyrightable. It may be freely reprinted with customary crediting of the source. The American Phytopathological Society, 1986.

F_4 progeny in a resistant × resistant cross. All resistance genes were recessive, and some were race specific.

High-temperature, adult-plant resistance to stripe rust has been utilized in most of the Pacific Northwest wheat cultivars that have been developed since 1960 without knowing the gene action of the resistance. Except for a report that Gaines may have genes that modify the expression of resistance (1), there has been no investigation of the inheritance of this type of resistance. Gaines, Nugaines, and Luke were selected for this study because they are among the best examples of durable, high-temperature, adult-plant resistance.

This paper reports on the types of gene action controlling rust intensity as measured by area under the disease progress curve (AUDPC). AUDPC is a quantitative measure of total resistance, since it combines all components of resistance such as infection efficiency, latent period, size of uredia, and amount of sporulation into one value.

MATERIALS AND METHODS

Experimental design and plant culture. Individual plants of cultivars 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 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 Gaines × Luke, were made between individual plants of each cultivar and replicated at least five times. In August, germinated parental and F_1 seeds from the three replicate crosses that had the most F_1 seeds 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, 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_1's and their female parent, but reciprocal backcrosses were not made. The F_1's 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 seeds 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 replicate crosses 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 approximately 8,000 plants in the experiment.

Rust development and data collection. Plants were uniformly dusted with urediniospores 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.

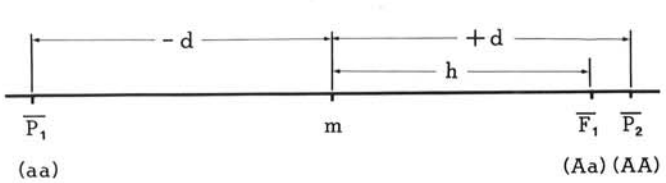

Fig 1. The relationship of the additive (*d*) and dominance (*h*) components of gene action to the parental (P_1 and P_2), F_1, and midparent (*m*) means when susceptibility is partially dominant. The unit of measurement is area under the disease progress curve (AUDPC), and the parent with the highest AUDPC is P_2.

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). The range in stages of plant growth was because the Luke parent was 1–2 wk later in maturity than the other parents. Data for each block were recorded within a 30-hr period. 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. During July, the range in maximum daily temperature was 18–31 C and in minimum daily temperature was 3–18 C; and the mean maximum, mean minimum, and mean daily temperatures were 2–7 C, 7–9 C, and 16–7 C (2.7 C lower than normal), respectively. The rust intensity percentages (and their ranges) that were 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). AUDPC for rust development on each plant was calculated from the original intensity data by using the formula $\text{AUDPC} = [10(X_1 + X_2/2] + [14(X_2 + X_3)/2]$. X_1, X_2, and X_3 are the rust intensities recorded on the first, second, and third recording dates, respectively.

Genetic analyses. The means and variances of the parental, F_1, F_2, and backcross generations were used to estimate the mean and genetic components of gene action by joint scaling tests as described by Mather and Jinks (15). Provided that adequate data are available, joint scaling tests can be developed to accommodate data from any generation and can be expanded to estimate epistatic components of gene action. This technique involves solving simultaneous equations to estimate values for genetic components that best explain differences among family means. The additive-dominance model estimated the midparent (*m*), the additive genetic component (*d*), and the dominance component (*h*) without considering epistasis (Fig. 1). The additive genetic component arises from the departure of each parent from the midparent. The departure of the parent with the highest (most susceptible) phenotype is d, and the departure of the parent with the lowest (most resistant) phenotype is $-d$. The dominance component arises from the departure of the heterozygote (F_1) from the midparent. The F_1 may be anywhere on the scale (Fig. 1). If the F_1 is at the midparent ($h = 0$), there is no dominance. If the F_1 is nearer the phenotype of the parent with the highest AUDPC value, then *h* is positive and the susceptible phenotype is dominant. If *h* is nearer the phenotype of the parent with the lowest AUDPC value, then *h* is negative and the resistant phenotype is dominant. If *h* lies outside the range of either parent, then there is overdominance in that direction.

The epistatic models included *m*, *d*, and *h* plus one or more of the epistatic components that described non-allelic interactions between pairs of loci: the additive × additive component (*i*), the additive × dominance component (*j*), and the dominance × dominance component (*l*). In the epistatic models, *m* corresponded to the mean of all possible homozygotes derived from the cross between two inbred parents. Since two parents in a cross may differ at several loci, and dominance within these loci and epistasis among these loci may differ, genetic components were redefined as the net directional effects of all relevant loci and are symbolized by [*d*], [*h*], [*i*], [*j*], and [*l*] to denote this net effect. By using the method to estimate gene action proposed by Wright (21, pages 395–403), "profiles" of the means and variances of the parental, F_1, F_2, and backcross generations were visually compared with theoretical profiles based on various types of gene action.

The objective of Mather and Jinks' scaling tests was to find the genetic model that best fit the data for each cross. Chi-square tests were used to determine how well the data fit a particular model. Components within models that fit the data were evaluated for significance by using *t* tests.

RESULTS

Resistance of parents and F_1 plants. The susceptible parent had a higher mean and a lower variance for AUDPC than Gaines, Nugaines and Luke (Fig. 2). In general, the AUDPC for Nugaines

was higher than for Luke and lower than for Gaines. Gaines had the highest variance. Since each replicate cross was identified, it was possible to detect any replicates that were different. The Luke parent in one Luke × Nugaines replicate was more resistant than the other Luke parents and will be referred to as highly resistant Luke (HR-Luke). Plants of the HR-Luke parent had AUDPC values that were less than 150. This made possible the study of an additional cross, HR-Luke × Nugaines. The Nugaines parents in one Nugaines × susceptible replicate and one Luke × Nugaines replicate were similar in AUDPC and infection type to the Gaines parents, which provided an additional cross, Luke × Gaines. The remaining replicates within a cross were not significantly different from each other and were combined for the analyses. Replicates of the Gaines and susceptible parents were homogeneous for stripe rust reaction. The reference genetic populations in this study were the individual plants of each parent that were most typical of that cultivar. Results refer only to the reference populations and not to the cultivars themselves.

TABLE 1. Chi-square goodness-of-fit test of seven wheat cultivar crosses to seven genetic models

Cross	Model[a]	χ^2 Value[b]	P[c]
Nugaines × Gaines	m [d] [h]	2.34	0.50*
	m [d] [h] [i]	1.16	0.56*
	m [d] [h] [j]	1.68	0.43*
	m [d] [h] [l]	0.47	0.79*
	m [d] [h] [i] [j]	0.35	0.56*
	m [d] [h] [i] [l]	0.41	0.52*
	m [d] [h] [j] [l]	0.001	0.99*
Luke × Susceptible	m [d] [h]	3.95	0.27*
	m [d] [h] [i]	3.86	0.15
	m [d] [h] [j]	3.51	0.17
	m [d] [h] [l]	3.47	0.18
	m [d] [h] [i] [j]	3.76	0.05
	m [d] [h] [i] [l]	1.34	0.25*
	m [d] [h] [j] [l]	3.41	0.06
Nugaines × Susceptible	m [d] [h]	6.14	0.10
	m [d] [h] [i]	2.04	0.36*
	m [d] [h] [j]	5.94	0.05
	m [d] [h] [l]	3.75	0.15
	m [d] [h] [i] [j]	0.38	0.53*
	m [d] [h] [i] [l]	1.57	0.21
	m [d] [h] [j] [l]	0.03	0.86*
HR-Luke × Nugaines	m [d] [h]	11.12	0.01
	m [d] [h] [i]	4.79	0.09
	m [d] [h] [j]	10.96	0.004
	m [d] [h] [l]	2.62	0.27*
	m [d] [h] [i] [j]	4.09	0.04
	m [d] [h] [i] [l]	2.52	0.11
	m [d] [h] [j] [l]	0.63	0.43*
Luke × Gaines	m [d] [h]	16.18	0.001
	m [d] [h] [i]	8.52	0.01
	m [d] [h] [j]	5.15	0.08
	m [d] [h] [l]	9.98	0.01
	m [d] [h] [i] [j]	0.004	0.95*
	m [d] [h] [i] [l]	8.52	0.004
	m [d] [h] [j] [l]	1.25	0.26*
Luke × Nugaines	m [d] [h]	23.16	0.001
	m [d] [h] [i]	18.53	0.001
	m [d] [h] [j]	9.22	0.01
	m [d] [h] [l]	15.93	0.001
	m [d] [h] [i] [j]	5.22	0.02
	m [d] [h] [i] [l]	15.47	0.001
	m [d] [h] [j] [l]	1.54	0.21
Gaines × Susceptible	m [d] [h]	35.72	0.001
	m [d] [h] [i]	20.33	0.001
	m [d] [h] [j]	98.83	0.001
	m [d] [h] [l]	87.34	0.001
	m [d] [h] [i] [j]	10.53	0.001
	m [d] [h] [i] [l]	38.98	0.001
	m [d] [h] [j] [l]	3.44	0.06

[a] m = mean, [d] = additive component, [h] = dominance component, [i] = additive × additive epistatic component, [j] = additive × dominance epistatic component, and [l] = dominance × dominance epistatic component.

[b] Degrees of freedom for the chi-square value equal six minus the number of components in the model because data from six generations were used to estimate the components.

[c] If the null hypothesis that the data fit the model is true, then the chi-square value will be the calculated chi-square value (P = 100) percent of the time. Cross-model combinations having chi-square values with $P \geq 0.25$ are denoted by an asterisk.

There were no significant differences between reciprocal populations in the F_1 and F_2 generations. Therefore, there was no evidence of cytoplasmic inheritance, and reciprocals were combined for the analyses.

In all resistant × susceptible crosses AUDPC for the F_1 was slightly less than for the susceptible parent. Degrees of dominance, calculated as the ratio of the F_1 mean to susceptible parent mean, were 0.98 for Gaines, 0.92 for Nugaines, and 0.94 for Luke when crossed with the susceptible parent. The degree of dominance was 0.98 in the Nugaines × Gaines cross. In the Luke × Nugaines and HR-Luke × Nugaines crosses, the F_1 had a higher AUDPC value than the most susceptible of the resistant parents. These results indicate that resistance was partially recessive. (Susceptibility was partially dominant.)

Scaling tests. The chi-square test of how well data from each cross fit each model is shown in Table 1. With a chi-square probability of 0.25 as the criterion for acceptance of a model, the Nugaines × Gaines cross fit all seven models, the Gaines × susceptible and Luke × Nugaines crosses fit none of the models, and the remaining crosses each fit two or three models. Nugaines × Gaines and Luke × susceptible were the only crosses that fit the simple additive-dominance model (m [d] [h]). Luke × susceptible cross also fit the model assuming additive × additive plus dominance × dominance components (m [d] [h] [i] [l]). The Nugaines × susceptible cross fit the three epistatic models that included the additive × additive component (m [d] [h] [i]), additive × additive plus additive × dominance components (m [d] [h] [i] [j]), and the additive × dominance plus dominance × dominance components (m [d] [h] [j] [l]). The HR-Luke × Nugaines cross fit the two epistatic models that included the dominance × dominance component (m [d] [h] [l]) and the additive × dominance plus dominance × dominance components (m [d] [h] [j] [l]). The Luke × Gaines cross fit the epistatic models that included additive × additive plus additive × dominance components (m [d] [h] [i] [j])

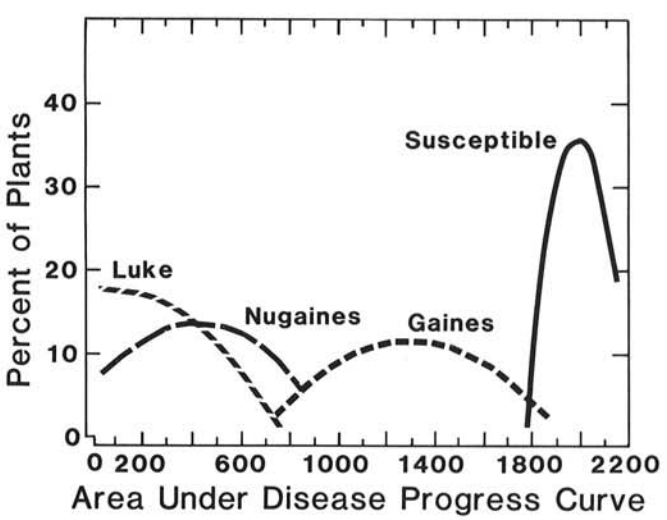

Fig. 2. Distribution of area under the disease progress curve (AUDPC) for 100–180 plants of the four parents. For each parent, the percentage of plants in each 100-unit interval of AUDPC was calculated, and a quadratic curve was fitted to the points.

and the additive × dominance plus dominance × dominance components (m [d][h][j][l]). Log and square root transformations of AUDPC generally did not lower chi-square values for the models. Even though these models were significant, not all components of the models may be significant. The level of significance for the chi-square tests is arbitrary. If the criterion for acceptance of the Luke × susceptible models was a chi-square probability of 0.15 instead of 0.25, then models assuming additive × additive epistasis (m [d] [h] [i]), additive × dominance epistasis (m [d] [h] [j]), and dominance × dominance epistasis (m [d] [h] [l]) would be significant (Table 1).

A t test was used to determine the significance of the components. Of the 14 epistatic models with a chi-square probability ≥ 0.25, only eight had one or more significant epistatic components (Table 2). The additive and dominance components were highly significant ($P \leq 0.001$) except in the Luke × Gaines model, which assumes additive × additive plus additive × dominance epistasis. For the Nugaines × susceptible cross, the additive × additive component was significant in two models, and the additive × additive and dominance × dominance components were significant in a third model. For the Luke × Gaines cross, both epistatic components were significant in the models assuming additive × additive plus additive × dominance gene action and additive × dominance plus dominance × dominance gene action. For the HR-Luke × Nugaines cross, the dominance × dominance component was significant in one model, and the additive × dominance and dominance × dominance components were significant in another model. Of the six epistatic models for the Nugaines × Gaines cross with a chi-square probability ≥ 0.25, only the model assuming dominance × dominance gene action had a significant epistatic component. All but one of the significant epistatic components were negative, indicating that these components generally conditioned a lower AUDPC value. For the Luke × susceptible cross, the additive × additive component was positive and significant at a t value probability of 0.13, but the t values for additive × dominance and dominance × dominance components were negligible.

Profile analysis. Profiles of means and variances for the seven crosses are shown in Fig. 3. The Nugaines × susceptible and Luke × susceptible profiles were similar to Wright's profile for a theoretical 3:13 F_2 segregation (21, page 397). The Gaines × susceptible profile of means was similar to the 1:3:12 and 1:15 segregation profiles, but the profile of variances was relatively flat with little variance in the segregating generations. The profiles for the Nugaines × Gaines cross were similar to profiles for additive alleles with complete dominance. Profiles of means and variances for the other crosses did not match any of Wright's theoretical profiles.

DISCUSSION

Since there was no evidence for cytoplasmic inheritance, a resistant parent could contribute resistance genes equally as either the male or the female parent without the possibility of losing some of the resistance. This agrees with Robbelen and Sharp (19, page 26) who reviewed the literature on the inheritance of durable stripe rust resistance and found no report of cytoplasmic inheritance. However, it disagrees with Krupinsky and Sharp (7) who reported

TABLE 2. Significance of the genetic components in models with a chi-square probability ≥ 0.25 and at least one epistatic component that was significantly different from zero at $P \leq 0.10$

Cross	Model[a]	Genetic component	Estimate	t-value[b]	P
Nugaines × Susceptible	m [d] [h] [i]	[d]	742	35.16	0.001
		[h]	477	7.30	0.001
		[i]	−103	−1.78	0.08
	m [d] [h] [i] [j]	[d]	750	38.02	0.001
		[h]	463	7.09	0.001
		[i]	−128	−2.22	0.03
		[j]	−109	−0.75	0.45
	m [d] [h] [j] [l]	[d]	751	38.09	0.001
		[h]	827	7.08	0.001
		[j]	−200	−1.38	0.08
		[l]	−237	−2.06	0.02
Luke × Gaines	m [d] [h] [i] [j]	[d]	657	19.63	0.001
		[h]	71	0.65	0.26
		[i]	−199	−2.25	0.01
		[j]	−400	−2.92	0.002
	m [d] [h] [j] [l]	[d]	−658	−19.64	0.001
		[h]	547	3.82	0.001
		[j]	405	2.95	0.002
		[l]	−277	−1.96	0.03
HR-Luke × Nugaines	m [d] [h] [j] [l]	[d]	−151	−5.73	0.001
		[h]	749	5.59	0.001
		[j	−202	−1.34	0.09
		[l]	−411	−2.99	0.001
	m [d] [h] [l]	[d]	−165	−6.50	0.001
		[h]	678	5.06	0.001
		[l]	−353	−2.57	0.005
Nugaines × Gaines	m [d] [h] [l]	[d]	512	25.59	0.001
		[h]	594	6.41	0.001
		[l]	−114	−1.28	0.10

[a] m = mean, [d] = additive component, [h] = dominance component, [i] = additive × additive epistatic component, [j] = additive × dominance epistatic component, and [l] = dominance × dominance epistatic component.
[b] Ratio of the estimate to its standard error that measures whether the parameter was significantly different from zero.

significant maternal (cytoplasmic) effects in the cultivars that they studied.

Use of these models and profiles is based upon the assumptions that parents are homozygous, that genotype × environment interactions are not significant, that resistance genes which are different are associated in one parent, and that there is linkage equilibrium for the epistatic models.

Resistance in Gaines, Nugaines, and Luke was predominantly recessive. This may be because all resistance genes in the cultivars are partially recessive or that the average effect for all genes is partially recessive. Similar types of resistance to *P. striiformis* were found to be partially recessive by other researchers (7–9,18,20,21). Partial recessiveness for durable stripe rust resistance seems to be a general phenomenon, whereas the less durable race-specific resistance to stripe rust was more frequently found to be dominant (9,14,20). Therefore, dominant or partially dominant resistance is not likely to be a durable type.

Profiles of means for the resistant × susceptible crosses (Fig. 3) have sloping tops, because there is partial recessiveness for resistance, whereas the theoretical profiles of means developed by Wright (22) have flat tops. This is because the theoretical profiles assume complete recessiveness (F_1 and backcross to the susceptible parent would have the same mean as the susceptible parent). Theoretical profiles of variances (22) assume that there is no environmental variance; therefore, the variance of the parents and F_1 is zero.

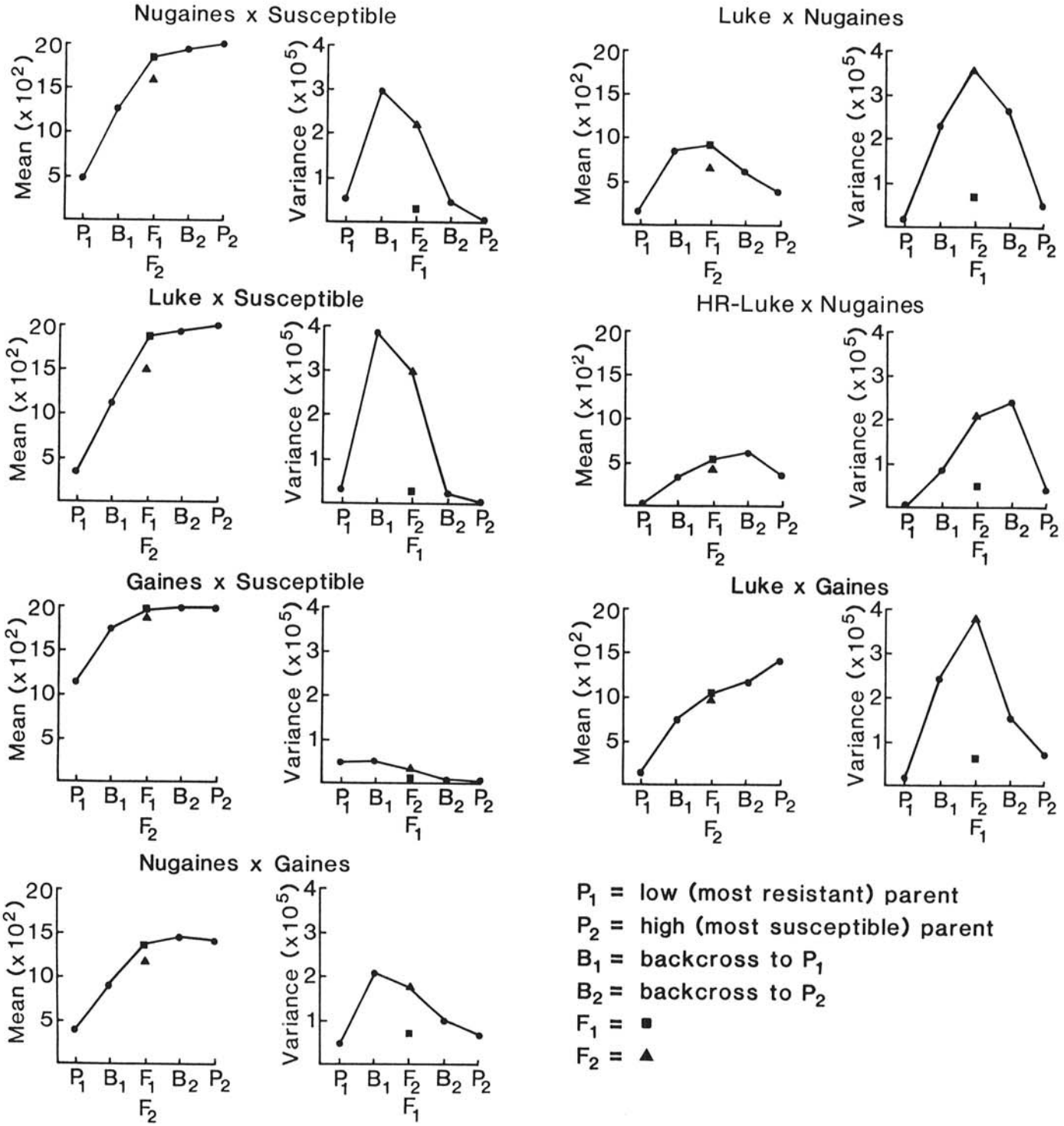

Fig. 3. Profiles of means and variances (Wright [22]) of area under the disease progress curve for seven crosses.

This is the first documented attempt to quantify epistasis for durable stripe rust resistance. Most gene action for resistance in Nugaines was explained by additive and dominance effects, but there was a small, but significant, additive × additive epistatic component for increased resistance (Table 2). The similarity of the profiles of means and variances for the Nugaines × susceptible cross (Fig. 3) to theoretical profiles based on epistasis (22) provides additional evidence for epistasis.

There is also evidence for epistatic gene action in Luke; however, its probability was lower than the probability for epistatic gene action in Nugaines. The profiles of means and variances for the Luke × susceptible cross were similar to the profiles for the Nugaines × susceptible cross (Fig. 3). Epistasis in the Luke × susceptible cross may have been obscured because the value for epistatic gene action is an average for all resistance genes. Epistasis may not be detected if only one or a few gene pairs exhibited epistasis and other gene pairs acted additively, or if there were several epistatic interactions with opposite effects that canceled one another. However, if epistatic gene action is present in Luke, the net effect is to increase susceptibility.

In the Gaines × susceptible cross, the lack of fit to digenic models may be due to more complex genetic control of resistance (e.g., trigenic epistatic interactions), linkage of interacting loci, or the large environmental variance associated with the Gaines phenotype (Figs. 2 and 3). Even though the gene action for resistance in Gaines is not clear, the gene action is such that a high proportion of susceptible progeny with little variance is produced. This is consistent with the results obtained by Allan and Purdy (1), who reported that resistant × Gaines crosses produced higher proportions of susceptible seedling progeny than resistant × Omar crosses. Both Gaines and Omar have susceptible seedling reactions, but Gaines has high-temperature, adult-plant resistance.

Gaines and Nugaines are siblings. However, the Nugaines × Gaines cross provides further evidence that their gene action for stripe rust resistance is different. Results from the Nugaines × Gaines cross (Tables 1 and 2) indicated that the difference between Gaines and Nugaines was expressed as either additive and dominance gene action or as additive, dominance, and dominance × dominance epistatic gene action. Profiles of means and variances (Fig. 3) were similar to Wright's profiles for additive gene action and complete dominance. Therefore, the differences between Gaines and Nugaines do not appear to be due to epistatic gene action.

The other crosses between resistant cultivars (Luke × Nugaines, Luke × Gaines, and HR-Luke × Nugaines) did not fit models assuming additive, dominance, and additive × additive epistatic gene action (Table 1), and profiles of means and variances for these crosses (Fig. 3) were not similar to any of Wright's theoretical profiles. The most likely explanation for these results is that the parents in each cross have different genes for resistance. If this is true, then it is not valid to use the scaling tests or profile comparisons on data from these crosses, because it is assumed that resistance genes by which the parents differ are associated in one parent. A previous paper (16) reported transgressive segregation for both resistance and susceptibility in these three crosses. This supports the interpretations that resistance genes in Luke are different from those in Gaines and Nugaines, and that there is additive and possibly additive × additive gene action for resistance.

Pope (17), Krupinsky and Sharp (7,8), Wallwork and Johnson (20), and Grama et al (3) reported that gene action for stripe rust resistance was additive based on transgressive segregation for higher levels of resistance. However, transgressive segregation does not necessarily imply additive gene action. According to Falconer (4, page 111), "... 'additive action' may mean two different things. Referred to genes (alleles) at one locus it means the absence of dominance, and referred to genes at different loci it means the absence of epistasis." If alleles are additive (no dominance or epistasis) then each allele will be expressed. When there is no dominance or epistasis, the F_1 mean will equal the midparent value. If alleles at a locus are not additive but pairs of alleles at different loci are additive (dominance, but no epistasis), then a recessive allele can only be expressed when the other allele at that locus is also recessive. If genes at different loci are not additive (epistasis is present), then the expression of a gene at one locus is masked by a gene at another locus. The genotypes at the two interacting loci determine the type of epistasis (additive × additive, additive × dominance, or dominance × dominance).

Inheritance of AUDPC in Gaines, Nugaines, and Luke was not additive within a locus because there was partial dominance for susceptibility. Relative to additive and dominance gene action, epistasis had a small effect on resistance; therefore, most of the gene action among loci was additive. The epistatic effect between some pairs of genes could be large, but their epistatic effects may be obscured by the background genotype or by epistatic interactions with opposite signs that cancel one another. The additive × additive gene action appeared to condition increased resistance in the Nugaines × susceptible cross and increased susceptibility in the Luke × susceptible cross.

In the breeding of Gaines, Nugaines, and Luke, selected lines from crosses between old, adapted cultivars, which had some high-temperature, adult-plant resistance were crossed with one another. Progeny were evaluated at several locations in the Pacific Northwest, and only the most susceptible lines were discarded (O. A. Vogel, *personal communication*). Selection was primarily based on agronomic characteristics such as plant type, lodging resistance, and yield. Thus, the accumulation of genes for high-temperature, adult-plant resistance was accomplished without a major emphasis on selection for stripe rust resistance and was not handicapped by the presence of many race-specific genes with large effects in their ancestors.

We agree with Johnson's (5,6) conclusion that adapted cultivars with demonstrated durable resistance are the best parents in a breeding program for enhanced resistance. Resistance accumulated from the broad genetic base in early Pacific Northwest cultivars was further enhanced by incorporating new sources of high-temperature, adult-plant resistance, and many recent cultivars have even greater high-temperature, adult-plant resistance than Gaines or Nugaines. Results from these studies showed that Nugaines, Luke, and HR-Luke have different genes for resistance. Since transgressive segregation for higher resistance did occur, some progeny from the HR-Luke × Nugaines and the Luke × Nugaines crosses should have higher levels of resistance than the parents and would be useful sources of resistance in breeding programs.

LITERATURE CITED

1. Allan, R. E., and Purdy, L. H. 1970. Reaction of F_2 seedlings of several crosses of susceptible and resistant wheat selections to *Puccinia striiformis*. Phytopathology 60:1368-1372.
2. Coakley, S. M., and Line, R. F. 1981. Quantitative relationship between climatic variables and stripe rust epidemics on winter wheat. Phytopathology 71:461-467.
3. Grama, A., Gerechter-Amitai, Z. K., and van Silfhout, C. H. 1984. Additive gene action for resistance to *Puccinia striiformis* f. sp. *tritici* in *Triticum dicoccoides*. Euphytica 33:281-287.
4. Falconer, D. S. 1981. Introduction to Quantitative Genetics. 2nd ed. Longmans, London and New York. 340 pp.
5. Johnson, R. 1978. Practical breeding for durable resistance to rust diseases in self-pollinating cereals. Euphytica 27:529-540.
6. Johnson, R. 1984. A critical analysis of durable resistance. Annu. Rev. Phytopathol. 22:309-330.
7. Krupinsky, J. M., and Sharp, E. L. 1978. Additive resistance in wheat to *Puccinia striiformis*. Phytopathology 68:1795-1799.
8. Krupinsky, J. M., and Sharp, E. L. 1979. Reselection for improved resistance of wheat to stripe rust. Phytopathology 69:400-404.
9. Lewellen, R. T., Sharp, E. L., and Hehn, E. R. 1967. Major and minor genes in wheat for resistance to *Puccinia striiformis* and their responses to temperature changes. Can. J. Bot. 45:2155-2172.
10. Line, R. F. 1980. Pathogenicity of *Puccinia striiformis* in the United States. Pages 163–166 in: Proc. Eur. & Mediterr. Cereal Rusts Conf. Bari and Rome, Italy.
11. Line, R. F. 1980. Specific and nonspecific resistance to stripe rust and leaf rust of wheat. Pages 495–499 in: Proc. 3rd Int. Wheat Conf. Madrid, Spain.
12. Line, R. F., Qayoum, A., and Milus, G. 1983. Durable resistance to stripe rust of wheat. Page 204 in: Abstracts of Papers, 4th Int. Congr.

Plant Pathology. Melbourne, Australia.
13. Lupton, F. G. H., and Johnson, R. 1970. Breeding for mature-plant resistance to yellow rust in wheat. Ann. Appl. Biol. 66:137-143.
14. Lupton, F. G. H., and Macer, R. C. F. 1962. Inheritance of resistance to yellow rust (*Puccinia glumarum* Erikss. & Henn.) in seven varieties of wheat. Trans. Br. Mycol. Soc. 45:21-45.
15. Mather, K., and Jinks, J. L. 1982. Biometrical Genetics: The Study of Continuous Variation. 3rd ed. Chapman and Hall, New York and London. 396 pp.
16. Milus, E. A., and Line, R. F. 1986. Number of genes controlling high-temperature, adult-plant resistance to stripe rust in wheat. Phytopathology 76:93-96.
17. Pacific Northwest Crop Improvement Association. 1963-1980. Wheat Production Estimates, 1963–1980. Washington Association of Wheat Growers, Ritzville, WA.
18. Pope, W. K. 1968. Interaction of minor genes for resistance to stripe rust in wheat. Pages 251-257 in: Proc. 3rd Int. Wheat Genet. Symp. Canberra, Australia.
19. Qayoum, A., and Line, R. F. 1985. High-temperature, adult-plant resistance to stripe rust of wheat. Phytopathology 75:1121-1125.
20. Robbelen, G., and Sharp, E. L. 1978. Mode of Inheritance, Interaction, and Application of Genes Conditioning Resistance to Yellow Rust. Paul Parey Verlag, Berlin and Hamburg. 88 pp.
21. Wallwork, H., and Johnson, R. 1984. Transgressive segregation for resistance to yellow rust in wheat. Euphytica 33:123-132.
22. Wright. S. 1968. The genetics of quantitative variability. Chap. 15 in: Evolution and Genetics of Populations. Vol. I. Genetic and Biometric Foundations. University of Chicago Press. 469 pp.