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INHERITANCE OF SLOW RUSTING TO STEM RUST IN WHEAT[1]

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INDEX WORDS

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SUMMARY

The inheritance of the slow rusting character was studied on F_5 progenies from seven spring wheat cultivars (*Triticum aestivum*) crossed in all possible combinations without reciprocals. The cultivars and their progenies were evaluated for slow rusting in 1974 and 1975 in epidemics of Puccinia *graminis* f. sp. *tritici*, races 15 and 151, and traces of other races. Slow rusting varied significantly among the parents and among the F_5 progeny of each cross. Transgressive segregation occurred in each cross, i.e. some progeny rusted more slowly than the parents and some faster. In crosses with both Idaed 59 and Kenya 58 the progeny distributions were skewed towards slow rust development but the distributions in the other crosses were normal. The genetic control of slow rusting was predominantly additive, and narrow sense heritability was approximately 80 percent. The number of segregating genes having an effect on slow rusting was estimated to be 2 to 12 pairs depending on the cross. Correlation between slow rusting and maturity was usually negative but in most crosses the relationship was small.

INTRODUCTION

The ability of certain wheat cultivars to slow down the development of stem rust was recognized many years ago as a form of resistance, even though the infection types on the plants indicated they were susceptible to rust (FARRER, 1898; STAKMAN, 1968). This slow rusting character, however, has not been fully exploited for improving the resistance of cultivars against stem rust, probably because methods have not been sufficiently developed, until recently, to permit its efficient use.

Recently reported methods of WILCOXSON et al. (1975), now make the study of the genetics of slow rusting possible and the utility of the character in a breeding program can be determined. They adapted hill-plot techniques (FREY et al., 1973) to studies with wheat stem rust and demonstrated that the data on stem rust development in hill plots could be summarized by means of the area under the stem rust progress curve. Slow rusting cultivars had low areas under the progress curve,

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whereas fast rusting cultivars had high areas under the curve.

The present work was done to extend knowledge about the slow rusting character of wheat infected with stem rust by estimating the inheritance of slow rusting, by investigating the relationship between slow rusting and plant maturity, and by evaluating the likely progress from selection in a breeding program.

MATERIALS AND METHODS

Cultivars of spring wheat (*Triticum aestivum* L. 'Baart', 'Idaed 59', 'Kenya 58', 'Lee', 'Marquis', 'Prelude', and 'Thatcher') comprised the parental sources for studying the inheritance of slow rusting. Idaed 59 tended to rust most slowly, Baart and Prelude rusted rapidly, and the other cultivars expressed different degrees of slow rusting ability ranging between Idaed 59 and the two fast rusters (WILCOXSON et al., 1975). The seven parents were crossed in all combinations to form a complete diallel series. The 21 F_1 progenies were selfed and the respective F_2 progenies were advanced to F_4 by single seed descent. Seed from a single F_4 plant was harvested to form a F_5 line for evaluation of slow rusting. At least 60 seeds per F_5 line were needed for the field evaluations so some selection for F_4 plants with high seed numbers was necessary. Eighty F_5 lines from each of the 21 crosses were developed.

The F_5 lines were evaluated in 1974 and 1975 at Rosemount, Minnesota, in a sets-in-replicates design with eight sets in each of two replicates. Each set consisted of ten random F_5 lines from each cross and the seven parents, making a total of 217 entries per set. The entries were planted in hill plots 30.5 cm (1 ft) apart with ten seeds per hill in mid-May each year. Each set was planted in 4 blocks of paired rows 9.15 m (30 ft) long; each block was bordered by hills of Era, a stem rust-resistant spring wheat cultivar. Standard fertilization and weed control practices were used. In 1974, the plots were irrigated overhead one week after anthesis and again a week later. In 1975 irrigation was not necessary.

Leaf rust (*Puccinia recondita* ROB. ex DESM. f. sp. *tritici*) was controlled with Indar LC-70$^{(R)}$ (4-n-butyl-1,2,4-triazole), a systemic fungicide specific for leaf rust control, applied at the rate of 566 ml/ha (8 oz/acre). No leaf rust developed in 1974, but there was a trace late in 1975.

The slow rusting characteristic of the F_5 lines was evaluated in naturally occurring epidemics of *Puccinia graminis* PERS. f. sp. *tritici* ERIKS. & E. HENN. In 1974 the first natural shower of uredospores occurred on May 27 and traces of stem rust were present throughout the plots on June 26, when the plants had tillered. In 1975, on July 3, when the plants were at anthesis uredia were occasionally found on only a few plants so the plots were inoculated with uredospores of race 15-TLM suspended in Soltrol 170^{R} (a paraffinic oil) at the rate of 0.2 mg uredospores/ml of oil/m^2, with an ultra-low volume sprayer. Even though a trace of stem rust was present, the plots were inoculated to ensure an epidemic.

Towards the end of the epidemic each year, the races present within the hill plots, and on the experiment station generally, were identified at the Cereal Rust Laboratory, St. Paul, Minnesota, by A. P. Roelfs.

Stem rust severity was first evaluated June 26, 1974, just after tillering, and on July 14, 1975, just after anthesis, and weekly thereafter for six weeks in 1974 and

for four weeks in 1975, using a modified Cobb's scale (PETERSON et al., 1948). When rust severities were less than 1% or about 10 uredia/culm (KINGSOLVER et al., 1959), the uredia were counted and converted to percentages by equating one uredium per hill to 0.01%, 10 uredia per hill or one uredium/plant to 0.1%, etc.

We indicated the slow rusting characteristic of the F_5 lines, by means of the area under the stem rust progress curve calculated from the weekly stem rust severity ratings made on each hill. The area under the curve was calculated with the Fortran IV subroutine AREA and the associated subroutine INTEG of BEVINGTON (1969). The matrices were inverted using the subroutine INVERT of DAVIES (1971).

Analysis of variance, assuming a random effects model (STEEL & TORRIE, 1960), was computed on the values for area under the curve for each of the crosses and for the combined data for both years and all crosses. The sums of squares involving crosses in the combined analysis were partitioned into general and specific combining ability according to the method of GRIFFING (1956). The variance components for general and specific combining abilities and for their interactions with years, sets, and replicates were calculated from the partitioned analysis. The standard errors of the variance component estimates were computed following standard formulas (ANDERSON & BANCROFT, 1952).

Additive genetic variance and additive by additive genetic variance were estimated from the variance components of general and specific combining ability using the methods of HORNER et al. (1955). It was assumed that the parents were completely inbred and that dominance variance was negligible because of the level of inbreeding in the progeny.

The phenotypic variance among progeny means was calculated from estimated additive and non-additive genetic variance components (SENTZ, 1971). Negative estimates of variance components were considered to be zero in calculating the phenotypic variance. Narrow sense heritability was estimated as the ratio of additive genetic variance to the phenotypic variance among progeny means.

The number of genes controlling slow rusting was estimated by FALCONER's (1960) methods. The variance among F_5 lines within each cross was used to estimate additive genetic variance for each cross. For this study, the dominance variance was ignored and the additive variance was estimated at 0.571 times the variance among F_5 lines. Thus, the approximate number of genes was calculated as:

$$1/8 \; \frac{(\text{Phenotypic range})^2}{\text{Variance among } F_5 \text{ lines} \times 4/7}$$

The Kolmogorov-Smirnov test (SOKAL & ROHLF, 1969) was used to test the normality of the F_5 progeny distributions in each cross.

The relationship of plant maturity to slow rusting was studied in 1974. The growth stage of the plants in each hill was estimated using the Romig scale (CALPOUZOS et al., 1976), on the same dates that rust severity was estimated. The growth stages were linearized by the following transformation:

$$\text{transformed growth stage} = \ln \frac{GS_{max} - GS_i}{GS_i}$$

where GS_{max} = last growth stage, GS_i = growth stage at time of observation. The transformed growth stages were regressed on time from planting, and the regres-

sion equation was used to predict days to anthesis and days to late dough stage for each hill. Analyses of variance were computed for days to anthesis and for days to late dough stage. The correlation between slow rusting (measured by area under the stem rust progress curve) and plant maturity was computed. Analysis of variance and correlations were calculated using standard computer programs (DIXON, 1971).

RESULTS

The stem rust epidemics in both 1974 and 1975 were considered to be natural epidemics, despite the fact that the plots were inoculated in 1975. In both years the races of the race-15 group constituted 70 percent of the collections from the hill plots and about 90 percent of the collections from the station; race 151-QSH was identified in about 10 percent of the hill-plot collections and in less than 7 percent of the station collections. All other races were identified in only one of the years and in less than 10 percent of the collections (Table 1). The predominant races, races 15-TLM and 15-TNM, were virulent on Baart, Prelude, Thatcher, Marquis, Lee, and Idaed 59 but were avirulent on Kenya 58 at low temperatures. Race 151-QSH was virulent on Baart, Prelude, Thatcher, Kenya 58, and Lee, but was avirulent on Marquis and Idaed 59.

In 1974, only infection types that indicated susceptibility were observed in the F_5 lines in the field plots. This was true in 1975 except that moderate susceptibility was observed in some of the F_5 lines as follows: 10 lines in Marquis × Idaed 59, 18 in Kenya 58 × Idaed 59, 15 in Thatcher × Idaed 59, 25 in Lee × Idaed 59, 5 in Prelude × Idaed 59, and 9 in Baart × Idaed 59, 5 in Kenya 58 × Marquis, 28 in Thatcher × Kenya 58, 5 in Lee × Kenya 58, 2 in Prelude × Kenya 58, 1 in Baart × Kenya 58, 2 in Lee × Thatcher, and 2 in Prelude × Thatcher.

Table 1. Races of *Puccinia graminis* f. sp. *tritici* identified on F_5 lines and in collections made elsewhere at the Rosemount Experiment Station in 1974 and 1975, and their virulence/avirulence in respect to *Sr* genes reported in the cultivars used in this study.

Race[1]	Number of identifications in F_5 lines		other collections		Virulence/avirulence
	1974	1975	1974	1975	
56 MBC	0	2	0	0	5, 7b/6, 11, Tr1
11–32–113 RKQ	0	3	0	1	5, 6, 7b, Tt1/11
11–32–113 RPQ	0	0	0	1	5, 7b, 11, Tt1/6
11–32–113 RTQ	0	2	0	1	5, 6, 7b, 11, Tt1/
11–32–113 RCC	1	0	0	0	5, 7b/6, 11, Tt1
15 TBM	1	0	0	0	5, 7b, Tt1/6, 11
15 TDM	2	0	1	2	5, 7b, Ttl//6, 11
15 TLM	10	9	8	2	5, 7b, 11, Tt1/6
15 TNM	13	13	79	56	5, 7b, 11, Tt1/6
151 QSH	5	3	3	4	5, 6, 11/7b, Tt1
151 QCB	2	0	2	1	5/6, 7b, 11 Tt1

[1] The number refers to the race as identified on the standard differential cultivars (STAKMAN et al., 1962). The letter refers to the race as identified on single gene dirrerential lines (ROELFS & MCVEY, 1972).

The parent cultivars showed differences for the slow rusting characteristic, as indicated by the mean area under the stem rust progress curve (Table 2). Each year the rank of the cultivars for this character was as indicated by WILCOXSON et al. (1975).

The mean area under the stem rust progress curve varied greatly among the 21 crosses (Table 3). There was transgressive segregation for the slow rusting character in each of the crosses, as seen in the values for range. Transgressive segregation in the crosses can also be seen in Fig. 1. From the cross Prelude × Baart, two cultivars with a relatively fast rusting phenotype, progenies were identified that were pheno-

Table 2. Cultivars of *Triticum aestivum* used as parents in the diallel cross and their mean area under the stem rust progress curve.

Cultivar	C.I. Number	Mean area ± s.d.[1]
Idaed 59	13631	49 ± 15
Kenya 58	12471	136 ± 23
Thatcher	10003	366 ± 30
Lee	12488	670 ± 39
Prelude	4323	748 ± 32
Marquis	3461	759 ± 24
Baart	1697	863 ± 29

[1] Mean based on 16 observations in 1974 and 1975, except with Idaed 59, Prelude, and Lee, 2, 1, and 5 observations were missing, respectively.

Table 3. Mean area and range for area under the stem rust progress curve for each cross.

Cross	Mean ± s.e.	Range
Marquis × Idaed 59	264 ± 38	7.6–1111.9*
Kenya 58 × Idaed 59	187 ± 36	2.9–1215.2*
Thatcher × Idaed 59	306 ± 41	0.9–1146.8*
Lee × Idaed 59	270 ± 39	1.0–1028.5*
Prelude × Idaed 59	390 ± 44	2.6–1340.6*
Baart × Idaed 59	461 ± 45	3.6–1164.1*
Kenya 58 × Marquis	516 ± 37	32.5–1170.9
Thatcher × Marquis	698 ± 15	109.0– 948.0
Lee × Marquis	732 ± 13	422.8– 955.2
Prelude × Marquis	718 ± 17	435.2–1065.5
Baart × Marquis	798 ± 13	487.7–1055.0
Thatcher × Kenya 58	206 ± 34	0.2– 937.2*
Lee × Kenya 58	480 ± 36	10.3–1150.5*
Prelude × Kenya 58	590 ± 44	21.7–1239.3*
Baart × Kenya 58	538 ± 34	13.4–1108.2
Lee × Thatcher	629 ± 27	36.4–1068.9
Prelude × Thatcher	713 ± 25	72.4–1079.8
Baart × Thatcher	731 ± 18	364.3–1059.4
Prelude × Lee	720 ± 22	192.6–1132.9
Baart × Lee	734 ± 13	376.5–1033.5
Baart × Prelude	869 ± 17	513.1–1242.5

* Distributions that differed significantly from a normal distribution.

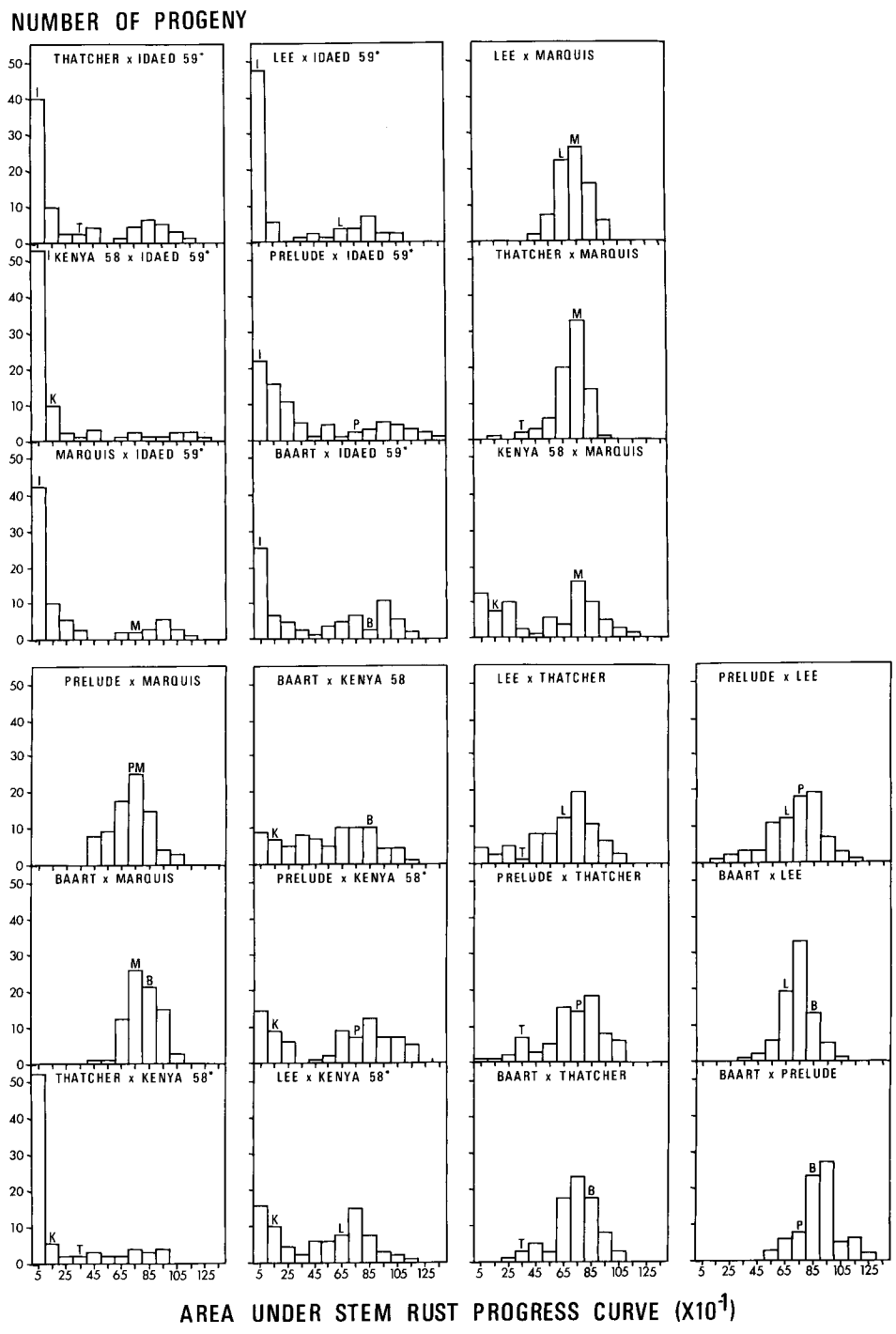
NUMBER OF PROGENY
THATCHER x IDAED 59*
LEE x IDAED 59*
LEE x MARQUIS
KENYA 58 x IDAED 59*
PRELUDE x IDAED 59*
THATCHER x MARQUIS
MARQUIS x IDAED 59*
BAART x IDAED 59*
KENYA 58 x MARQUIS
PRELUDE x MARQUIS
BAART x KENYA 58
LEE x THATCHER
PRELUDE x LEE
BAART x MARQUIS
PRELUDE x KENYA 58*
PRELUDE x THATCHER
BAART x LEE
THATCHER x KENYA 58*
LEE x KENYA 58*
BAART x THATCHER
BAART x PRELUDE
0 10 20 30 40 50
5 25 45 65 85 105 125
AREA UNDER STEM RUST PROGRESS CURVE (X10-1)

Table 4. Estimated variance components for general combining ability (σ_g^2), specific combining ability (σ_s^2), and their interactions with years (Y).

Variance components	Estimated value ± s.e.
$\hat{\sigma}_g^2$	26080 ± 4407*
$\hat{\sigma}_s^2$	4165 ± 1861*
$\hat{\sigma}_g^2$Y	983 ± 657
$\hat{\sigma}_s^2$Y	346 ± 189

* Variance components exceeding twice their standard error are assumed to be significant.

typically more slow rusting than the cultivar Lee. Lee rusts moderately slowly (WILCOXSON et al., 1975). The normality of the progeny distribution in each of the crosses is shown in Table 3 and Fig. 1. In the crosses with Idaed 59 and Kenya 58, the distributions were skewed toward slow rusting. The distribution of the progenies in the other crosses was normal.

Analysis of variance indicated significant differences among crosses for slow rusting. The interactions of crosses × year, crosses × replicates in years, and crosses × sets × replicates in years were all significant, but the interactions of crosses × sets and crosses × sets × year were not. The year, replicates in years, and set effects were nonsignificant. The significant interaction between crosses and years probably was due to the response of the crosses involving Kenya 58. Except for Prelude × Kenya 58, the crosses with Kenya 58 had a lower mean area under the stem rust progress curve in 1975 than in 1974, whereas all other crosses had higher mean areas under the curve in 1975 than in 1974.

The significant sources of variation involving crosses were partitioned into general and specific combining ability and their interaction with years, sets, and replicates in years. General and specific combining ability and their interaction with years were significant. The general combining ability × replicates in years and specific combining ability × sets × replicates in year interactions were also significant.

Estimated variance components and their standard errors are listed in Table 4. Only the variance components for general combining ability and for the interaction between general combining ability × replicates in years were more than twice the size of their standard errors. Other variance components involving general and specific combining ability in the model were small or negative (SKOVMAND, 1976). Variances among lines within crosses and years × lines within crosses were significant. The estimated value for additive genetic variance was 47995 ± 32110 and for additive by additive genetic variance it was 8330 ± 3722.

Narrow sense heritability ($\hat{\sigma}_A^2/\hat{\sigma}_P^2$) was estimated to be 82 percent. Two to three genes were estimated to control slow rusting in crosses with Idaed 59 and Kenya 58 and up to 12 pairs in crosses with the other cultivars (Table 5).

Fig. 1. Frequency distributions for slow rusting as indicated by area under the stem rust progress curve of progenies of 21 wheat crosses (with class intervals of 100 units). The area under the curve for the parents is indicated by the letter above certain bars. The asterisk indicates those distributions that were not normal.

Table 5. Estimated variance among F_5 lines, phenotypic range, and estimated number of genes affecting slow rusting for each cross.

Cross	Variance among F_5 lines	Range	Number of genes
Marquis × Idaed 59	107862	1104	2
Kenya 58 × Idaed 59	96994	1212	3
Thatcher × Idaed 59	124547	1146	2
Lee × Idaed 59	119002	1028	2
Prelude × Idaed 59	145092	1338	3
Baart × Idaed 59	146729	1161	2
Kenya 58 × Marquis	101194	1138	3
Thatcher × Marquis	13750	839	11
Lee × Marquis	9707	532	6
Prelude × Marquis	16819	630	5
Baart × Marquis	6438	567	11
Thatcher × Kenya 58	90528	937	2
Lee × Kenya 58	96518	1140	3
Prelude × Kenya 58	144694	1218	2
Baart × Kenya 58	84914	1095	3
Lee × Thatcher	53971	1033	4
Prelude × Thatcher	43124	1007	5
Baart × Thatcher	20953	695	5
Prelude × Lee	32938	940	6
Baart × Lee	7611	657	12
Baart × Prelude	11547	729	10

Analysis of variance of predicted days from planting to anthesis and days from planting to the late dough stage showed significant differences among crosses and among lines in crosses in sets for both measures of maturity. The correlation between area under the curve and relative maturity was calculated for individual crosses and for the data combined over all crosses. Results of the association tests are presented as coefficients of determination (r^2) for the individual crosses (Table 6). The coefficients of determination for days to anthesis indicate that less than 30% of the variation can be explained by differences in maturity in any one cross, and in ten of the crosses less than 10% of the variation in slow rusting can be explained by differences in days to anthesis. The coefficients of determination for days to the late dough stage were somewhat higher than those for days to anthesis, but in only two crosses, Thatcher × Idaed 59 and Lee × Idaed 59, could more than 50% of the variation in slow rusting be attributed to differences in maturity. In eleven of the crosses, less than 10% of the variation could be explained by differences in days to the late dough stage. There were only three crosses, however, in which the association between area under the curve and 'maturity' was not negative. The combined data indicated little relationship between area under the curve and days to anthesis ($r = -.05$) or between area under the curve and days to late dough stage ($r = -.09$).

Table 6. Coefficients of correlation (r) and determination (r^2) between area under the disease progress curve and maturity measured as days from planting to anthesis and days to the late dough stage.

Cross	Days to anthesis		Days to late dough	
	r	r^2	r	r^2
Marquis × Idaed 59	–0.40	0.16	–0.63	0.40
Kenya 58 × Idaed 59	–0.11	0.01	–0.15	0.02
Thatcher × Idaed 59	–0.47	0.21	–0.77	0.59
Lee × Idaed 59	–0.35	0.13	–0.77	0.59
Prelude × Idaed 59	–0.14	0.02	–0.28	0.08
Baart × Idaed 59	–0.04	0.00	–0.04	0.00
Kenya 58 × Marquis	0.02	0.00	0.02	0.00
Thatcher × Marquis	–0.24	0.06	–0.43	0.18
Lee × Marquis	–0.52	0.27	–0.62	0.38
Prelude × Marquis	–0.47	0.22	–0.52	0.27
Baart × Marquis	–0.46	0.22	–0.44	0.19
Thatcher × Kenya 58	–0.00	0.00	–0.04	0.00
Lee × Kenya 58	0.07	0.01	0.10	0.01
Prelude × Kenya 58	–0.16	0.03	–0.20	0.04
Baart × Kenya 58	0.23	0.05	0.21	0.05
Lee × Thatcher	–0.34	0.11	–0.62	0.38
Prelude × Thatcher	–0.42	0.18	–0.62	0.39
Baart × Thatcher	–0.13	0.02	–0.13	0.02
Prelude × Lee	–0.33	0.11	–0.55	0.30
Baart × Lee	–0.15	0.02	–0.09	0.01
Baart × Prelude	–0.15	0.02	–0.11	0.01

DISCUSSION

We know of no published information on the inheritance of slow development of stem rust in wheat. The present study provides such information from crosses with seven wheat cultivars that differed from each other for the slow rusting character. The data suggest that slow rusting is a quantitative character, since the progenies from each cross were continuously distributed and could not be classified into distinct groups. Our estimated additive genetic variance for slow rusting was greater than the estimated additive by additive genetic variance but the additive by additive variance was significant. Narrow sense heritability was 82%. Thus, we concluded that the genetic control of slow rusting in these 7 cultivars is predominantly additive but epistatic gene action may also be of some importance. Similar conclusions were also reported by BRENNAN (1975) who found that the size and number of stem rust uredia in wheat were controlled by additive and epistatic gene action.

The number of genes segregating and having an effect on slow stem rust development in the crosses was estimated to be 2 to 3 pairs in crosses involving Idaed 59 and Kenya 58 and up to 12 pairs in the crosses among the other five cultivars.

These estimates may be conservative partially because of the hill-plot evaluations. In hill plots, inoculum continually spreads from fast rusting to slow rusting genotypes. This reduces the estimated range of the character and thus decreases the estimated

number of genes. This greatly reduces the estimated range, and reduces to some extent the variance among F_5 lines, and thus decreases the estimated number of genes.

The abnormal distributions for slow-rusting that occurred in the crosses that involved Idaed 59 and in some of the crosses that involved Kenya 58 probably resulted from the interaction of the gene *Sr* Ttl from Idaed 59 and the gene *Sr* 6 from Kenya 58 with genes that control slow rusting. Thus abnormally high numbers of slow rusting progeny were produced in these crosses which suggested that only a few genes were involved in slow rusting. It was also in these crosses that the highest variances occurred which resulted in the calculated gene number being low. The distributions for slow rusting that occurred in the other crosses were all normal and the variances were all relatively low. Thus the estimated number of genes was high in these crosses. It is possible that the estimated high number of genes is more nearly correct than the estimated low numbers because of the effects of the specific genes.

The phenotypic range used in the gene number estimate should be the greatest possible range obtainable by selection (COMSTOCK, 1969). The ranges used in our estimates were not based on selection for extremes, but we believe sample sizes of eighty F_2-derived families provide reasonable estimates of range. However, these gene number estimates must be used with extreme caution. FALCONER (1960) indicated this need for caution when he said, 'Since the estimation of the number of loci is necessarily so imprecise it does not seem worth while to discuss in detail its limitations or the errors that may have been introduced by the assumptions that were made.' Our assumption that the number of genes controlling the slow rusting characteristic is probably small in comparison to the number of genes controlling yield does not seem unreasonable from the heritability estimate and the number estimates.

The role of specific stem rust resistance genes in the control of slow rust development was studied by SKOVMAND et al. (1975) using the same parental cultivars and progeny as reported in this study. They found that the genes Sr 5, Sr 7b and Sr 11 had no effect on slow rust development but Sr 6 and Sr Tt 1 interacted with the genes that controlled slow rust development and many of the progeny lines that possessed Sr 6 and Sr Tt 1 rusted slowly. However, these genes did not cause the lines to rust slowly because a few lines possessed these genes but they rusted rapidly. Gene Sr Tt 1 may be linked with the genes that control slow rusting. The relationship of Sr 6 to slow stem rust development may be more complex. This gene from Kenya 58 should have conditioned resistance to races 15-TLM and 15-TNM, the predominate races during both years of this study. But some stem rust caused by these races developed on all progenies of Kenya 58. Most of the lines that possessed the dominant allele of Sr 6 rusted slowly but a few rusted rapidly (unpublished data). Sr 6 is also more effective in the control of rust at about 25°C than at 18°C, a temperature range that was not uncommon during July when stem rust was developing in our field trials. It is possible that genes that condition slow rust development enhance the effect of the Sr 6 gene.

Good progress in selecting for the slow stem rusting character should be possible since genetic control was predominantly additive and the trait appeared to be highly heritable. Furthermore, the data indicated that slow rusting was controlled by 2 to 12 gene pairs. The transgressive segregation found in all crosses indicated that levels

of slow rusting lower than those found in the parents can be obtained. The data also indicated that even fast rusting cultivars such as Prelude and Baart possess some genes that contribute to slow stem rusting. We thought they possessed few, if any, genes for slow rusting when the work was started.

Our conclusions on the inheritance of slow development of stem rust of wheat are in agreement with those of other scientists. In studies on the general resistance of oats against crown rust, SIMONS (1975) reported heritability estimates from 46 to 82% in different crosses. LUKE et al. (1975) estimated the heritability of the slow development of crown rust on Red Rustproof oats to be about 87% and was controlled by 2 to 3 genes. HEPLER et al. (1957) found the slow development of asparagus rust was controlled by at least 4 or 5 factors. HOOKER (1969), investigating the heritability of general resistance of corn against *Puccinia sorghi*, found in 64 crosses that the heritability ranged from 17 to 98% and averaged 84% for all crosses. He also found transgressive segregation for both greater susceptibility and for greater resistance than that possessed by the parents. Hooker also concluded that while the general resistance of corn against common rust was conditioned by many genes, selection for resistance should not be difficult. General resistance of wheat against stripe rust may be controlled by genes with minor individual effects on rust development (HENDRIKSEN & POPE, 1971; LEWELLEN et al., 1967); the overall effect of these genes was additive and high degrees of resistance were obtained by three minor genes (SHARP & VOLIN, 1970). Transgressive segregation for general resistance against stripe rust has been observed (LUPTON & JOHNSON, 1970; POPE, 1968).

Progress from selection for both slow rusting and desired maturity should be possible in spring wheat. In our study, there was little indication, in most crosses, of a significant association between slow rusting and days to anthesis. When maturity was measured as days to the late dough stage, the association between slow rusting and later maturity seemed to be somewhat higher in some crosses. This may be due to early maturation resulting from heavy rust infection rather than to an association between slow rusting and late maturity, which could also have caused the correlation between slow rusting and maturity to be negative in all but three crosses. Nevertheless, successful selection for both slow rusting and the desired maturity should be possible.

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REFERENCES

ANDERSON, R. L. & T. A. BANCROFT, 1952. Statistical theory in research, p. 313–327. McGraw-Hill Book Company, Inc., New York. 399 p.

BEVINGTON, P. R., 1969. Data reduction and error analysis for the physical sciences. McGraw Hill. New York. 336 p.

BRENNAN, P. S., 1975. General resistance in wheat (*Triticum aestivum* L.) to stem rust (*Puccinia graminis tritici* PERS. f. sp. *tritici* ERIKKS and HENN.). Ph. D. Thesis. University of Saskatchewan. Saskatoon. 142 p.

CALPOUZOS, L., A. P. ROELFS, M. E. MADSON, F. B. MARTIN, J. R. WELSH & R. D. WILCOXSON, 1976. A new model to measure yield losses in spring wheat caused by stem rust. Minnesota Agric. Exp. Stn Tech. Bull. 307: 1–32.

COMSTOCK, R. E., 1969. Number of genes affecting growth in mice. In: Genetics lectures 1. Oregon State University Press, p. 137–148.

DAVIES, R. G., 1971. Computer programming in quantitative biology. Academic Press, London. 492 p.

DIXON, W. J., 1971. BMD biomedical computer programs. University of California Publications in Automatic Computation 2. University of California Press, Berkeley. 600 p.

FALCONER, D. S., 1960. Introduction to quantitative genetics. The Ronald Press Company, New York. 365 p.

FARRER, W., 1898. The making and improvement of wheats for Australian conditions. Agric. Gaz. N. S. Wales 9: 131–168.

FREY, K. J., J. A. BROWNING & M. D. SIMONS, 1973. Management of host resistance genes to control disease. Z. PflKrankh. PflSchutz. 80: 160–180.

GRIFFING, B., 1956. Concept of general and specific combining ability in relation to diallel crossing systems. Austr. J. Biol. Sci. 9: 463–493.

HENRIKSON, G. B. & W. K. POPE, 1971. Additive resistance to stripe rust in wheat. Crop Sci. 11: 825–827.

HEPLER, P. R., A. E. THOMPSON & J. P. MCCOLLUM, 1957. Inheritance of resistance to *Puccinia asparagi* DC in *Asparagus officinalis* L. Illinois Agric. Exp. Stn Bull. 607: 1–47.

HOOKER, A. L., 1969. Widely based resistance to rust in corn. In: J. A. BROWNING (Ed.), Disease consequences of intensive and extensive culture of field crops. Iowa Agric. Exp. Stn Spec. Rep. 64, p. 28–34.

HORNER, T. W., R. E. COMSTOCK & H. F. ROBINSON, 1955. Non-allelic gene interactions and the interpretations of quantitiative genetic data. North Carolina Agric. Exp. Stn Tech. Bull. 118: 1–117.

KINGSOLVER, C. H., C. G. SCHMITT, C. E. PEET & K. R. BROMFIELD, 1959. Epidemiology of stem rust: II. (Relation of quantity of inoculum and growth stage of wheat and rye at infection to yield reduction by stem rust). Pl. Dis. Reptr 43: 855–862.

LEWELLEN, R. T., E. L. SHARP & E. R. HEHN, 1967. Major and minor genes in wheat for resistance to *Puccinia striiformis* and their responses to temperature changes. Can. J. Bot. 45: 2155–2174.

LUKE, H. H., R. D. BARNETT & P. L. PFAHLER, 1975. Inheritance of horizontal resistance to crown rust in oats. Phytopathology 65: 631–632.

LUPTON, F. G. H. & R. JOHNSTON, 1970. Breeding for mature-plant resistance to yellow rust in wheat. Ann. appl. Biol. 66: 137–143.

PETERSON, R. F., A. B. CAMPBELL & A. E. HANNAH, 1948. A diagrammatic scale for estimating rust intensity on leaves and stems of cereals. Can. J. Res. Sec. C 26: 496–500.

POPE, W. K., 1968. Interaction of minor genes for resistance to stripe rust in wheat. In: Proc. 3rd Int. Wheat Genet. Symp. Canberra. Austr. Acad. Sci. p. 251–257.

ROELFS, A. P. & D. V. MCVEY, 1972. Wheat stem rust races in the Yaqui Valley of Mexico during 1972. Pl. Dis. Reptr. 56: 1038–1039.

SENTZ, J. C., 1971. Genetic variances in a synthetic variety of maize estimated by two mating designs. Crop Sci. 11: 234–238.

SHARP, E. L. & R. B. VOLIN, 1970. Additive genes in wheat conditioning resistance to stripe rust. Phytopathology 60: 1146–1147.

SIMONS, M. D., 1975. Heritability of field resistance to the oat crown rust fungus. Phytopathology 65: 324–325.

SKOVMAND, B., 1976. Inheritance of slow rust development in spring wheat and the relationship of slow rusting to specific resistance. Ph. D. Thesis. University of Minnesota. 114 p.

SKOVMAND, B., A. P. ROELFS & R. D. WILCOXSON, 1978. The relationship between slow-rusting and some genes specific for stem rust resistance in wheat. Phytopathology (In press).

SOKAL, R. R. & F. J. ROHLF, 1969. Biometry. The principles and practice of statistics in biological research. W. H. Freeman and Co. San Francisco. 776 p.

STAKMAN, E. C., 1968. What are the prospects for permanent control of cereal rusts? In: Cereal Rust Conference, Oeiras, Portugal. Pages 217–230.

STAKMAN, E. C., D. M. STEWART & W. Q. LOEGERING, 1962. Identification of physiologic races of *Puccinia graminis* var. *tritici*. U.S. Dep. Agric. Bur. Entomol. Plant Quarantine Bull. E-617 (Rev.). 53 p.

STEEL, R. G. D. & J. H. TORRIE, 1960. Principles and procedures of statistics. McGraw-Hill Book Company, Inc., New York. 481 p.

WILCOXSON, R. D., B. SKOVMAND & A. H. ATIF, 1975. Evaluation of wheat cultivars for ability to retard development of stem rust. Ann. appl. Biol. 80: 275–281.