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Inheritance of Resistance to Six Races of Bunt, to Awns and Kernel Color in a Wheat Cross

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INHERITANCE OF RESISTANCE TO SIX RACES OF BUNT, TO AWNS AND KERNEL COLOR IN A WHEAT CROSS

by

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INTRODUCTION

Wheat is the most important cereal crop of the world, and one of the most serious diseases affecting it over much of its range is covered smut or bunt (57). The word "bunt", according to Heald (48), is a contraction of an old English term, "burnt ear", which fittingly describes the ravages of covered smut.

Man's first knowledge of this disease is lost in antiquity, but it was first recorded by early Greek writers (97). Gaines (39), writing in 1928, stated that since 1924 stinking smut had been the most destructive parasite of wheat in America, causing losses of as high as 10 percent in certain states. As an additional cost of the disease, he mentions the expense of washing smutty grain before it can be processed. Flor, Gaines, and Smith (31) listed further losses from bunt due to dockage, the expense of seed treatment, the detrimental effects of bunt infection on plants which do not show typical symptoms of the disease, and reduction in yield. Chester (16) stated that the explosion hazard involved in handling smutty wheat must be counted as a cost of the disease. Price and McCormick (61) and Melhus and Kent (58) have also stressed the danger of fire. Stevens (77) presented data showing that losses from bunt in the United States ranged from nearly 20 percent in 1926 to about 5 percent in 1937.

Holton and Suneson (49) reported that in 1924 and 1925, 50 percent of the wheat passing through Pacific Northwest inspection points graded smutty. Bunt has also been a very serious problem in the Rocky Mountain states (91)

during years that were favorable for development of the causative organisms.

In the United States there are two species of fungi that cause covered smut in wheat. These are Tilletia caries (D.C.) Tul., synonym, (T. tritici (Bjerk) Wint), the rough-spored bunt, and Tilletia foetida (Wallr) Liro, synonym, (T. levis Kuehn), the smooth-spored bunt. The two are closely related, having identical life histories. Except for a single race of T. caries, dwarf, all races of both species may be controlled in most wheat-growing regions by chemical disinfection of the seed. This method, however, is not satisfactory in all areas, and at best is only temporary as it must be repeated every year. The development of resistant varieties appears to be a more promising means of control.

The agronomic importance of awnedness in wheat is open to question, but according to Clark, Florell and Hooker (22) the presence of awns is positively correlated with the very undesirable character of shattering. Rosenquist (70) believed that the presence of awns tended to produce heavier kernels and Gauch and Miller (40) showed that deawned heads transpire less than do awned heads, but the relationship, if any, between these observations is not clear. Lamb (54) has suggested that the awn may affect yield by its role in removing substances from the transpiration system of the wheat plant that might otherwise interfere with movement of material into the grain.

This thesis deals with the inheritance of reaction to six races of bunt and the inheritance of awns and kernel color.

REVIEW OF LITERATURE

Inheritance of Smut Resistance

It is well known that many pathogenic organisms are more or less limited in the type of hosts they can infect. In some cases there is a simple mechanical explanation for this phenomenon, but more commonly the mechanism is complex and poorly understood. Gaines' (37) assumption of the necessity of a "rather delicate chemo-tactic balance between host and parasite" before infection can take place is rather vague but is possibly as good an explanation as any. There is no doubt, however, of the existence of disease resistance. Although resistance to bunt in wheat has long been recognized, modern study of the problem on a genetic basis was not initiated until just before the 1920's.

The phenomenon of physiologic specialization of smut races was not recognized until several years after genetic studies of bunt resistance began. Nevertheless, it is appropriate to consider it at the beginning of this review as it is an intrinsic part of the problem of varietal resistance whether it is recognized as such or not.

Faris (30) in 1924 presented the first definite evidence of physiologic specialization in bunt. Rodenhiser and Stakman (66) in 1927 isolated three races of Tilletia levis and two of T. tritici from bunt collections that were obtained at several widely separated places in the world. A year later Reed (65) differentiated four forms of T. levis and six of T. tritici by their reaction on six varieties of wheat. Reed gave these races Roman numeral designations. In 1937 Rodenhiser and Holton (68) placed the classification of bunt races on its present basis by

assigning letters and race numbers T-1 to T-11 to the races of T. tritici known at that time and L-1 to L-8 to the known races of T. levis. In 1945 (69) this classification was expanded to include 16 races of T. caries and 15 of T. foetida. In addition there is a single race of Dwarf bunt.[1] A knowledge of physiologic specialization helped to account for some of the apparent inconsistencies in inheritance of smut resistance found in the earlier literature but also complicated the problems of the plant breeders.

Coons (28) tested a large number of varieties for reaction to T. levis, and with one exception, Bultz, all those that showed strong resistance were Turkey types or selections from crosses involving these types. Johnston (51) found that the twenty Turkey wheats that he tested were resistant to both T. levis and T. tritici. A similar test showed that many varieties of soft wheat are resistant to T. levis but not to T. tritici. Opposed to these findings, Reed (64) concluded that Turkey along with other varieties he tested was susceptible to T. levis. Tisdale (91) differentiated between complete immunity, which he found in Russar and Martin, and high resistance in such varieties as White Odessa, Florence, Ridit, and certain Turkey selections.

Briggs (6), using inoculum from the same source for all inoculations, also found that Martin and Russar were immune to bunt. Tingey (89) in some preliminary studies in 1926 failed to obtain any bunt in Ridit or White Odessa, but showed that Utah Kanred, Turkey, Sevier, Hard Federation, and many other selections from various crosses were all relatively susceptible.

Gaines (35) found the variety Florence to be resistant, as was a

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[1]Unpublished data furnished by D. C. Tingey

certain strain of Turkey (34), and that some segregates of Turkey X Florence crosses were more resistant than either parent, indicating that the two varieties possessed different resistance factors. In another paper Gaines (36) reported the resistance of Alaska, a poulard variety, differed from that of Turkey, but due to sterility encountered in the interspecific cross he was unable to determine the actual mode of inheritance.

According to Briggs (6) the resistance of Martin is completely dominant over susceptibility and Martin differs from susceptible varieties by a single factor. This he designated as the Martin factor. In a cross of Marquis X Turkey, Gaines and Singleton (38) found bunt resistance to be controlled by two factors, the one carried by Turkey being much more "prepotent" than that carried by Marquis. It should be noted that the preceding studies were made using inoculum composed of an unknown race or mixture of races.

Briggs (9), using Reed's physiologic race III, concluded that inheritance of the resistance of Banner Berkeley was controlled by a single factor and this factor was identified as the Martin factor. A little later he (7) isolated a second resistance factor in Hussar. Unlike the Martin factor, this gene is not completely dominant, but allows about 50 percent of the plants that are heterozygous for it to become infected. Still later Briggs (12) discovered another factor in a certain strain of Turkey. Some years after this work he (15) analyzed the data from a number of crosses involving varieties carrying the Martin and Turkey factors, and showed that the two genes were probably linked. On the basis of all the crosses, a crossover value of 34.22 percent was calculated.

Briggs (8) believed that the resistance of White Odessa was due to the same factor as that found in Martin. Other studies (11) showed that

Odessa also probably possessed the Martin factor. Turkey C. I. 1558 and Turkey C. I. 3055, both resistant, were crossed with susceptible White Federation (10) and both differed from White Federation by one major factor for resistance, which resembled the Hussar factor in allowing about one-half the heterozygotes to become infected. Two other Turkey wheats (14), Turkey 1558B and Turkey 2578, depended on the Martin factor for their resistance.

All of Briggs' work up to this time had been made using one physiologic race of bunt. Smith (74) inoculated Martin with three different races of bunt and found it immune to T-1, moderately susceptible to T-2, and heavily infected by T-3.

Stanford (76) noted that Rio was resistant to race T-8. On the basis of appropriate crosses with other resistant varieties of known genetic make-up, he concluded that Rio possessed a new factor for resistance. It was designated as the"Rio factor"and allows about 10 percent infection when in the homozygous condition and about 59 percent when heterozygous. It was found to be closely linked to the Turkey factor and loosely linked to the Martin factor.

Kiehner and Briggs (52) found one weak and two strong factors for resistance to bunt in Turkey 10016. The strong factors were the Turkey and Rio factors. These showed linkage as reported by Stanford (76). In general, investigations of linkage between different bunt resistance factors and between these and factors conditioning other characters have yielded negative results. Ausemus (3) and Hayes, Ausemus, Stakman, and Bamberg (45) found no evidence of linkage between bunt factors and factors involved in resistance to several other diseases. Smith (75) could find no relationship between awns and bunt resistance.

Albit (5) was selected from the progeny of Hybrid 128 X White Odessa. Albit was backcrossed with Hybrid 128 and segregating generations showed a one-factor difference for resistance to race T-8. Schlehuber (71) found that neither Albit, Minhardi, nor Buffum were highly resistant to T-1 and L-5. The same factor or factors possessed by Albit determined resistance to both T-1 and L-5. Minhardi possessed a gene that when crossed with Albit inhibited the action of one of the Albit factors against L-5 but not against T-1 (71). Kilduff (53) showed that a single factor accounted for resistance in the varieties that he studied, and that this factor was probably linked to awnedness.

Ridit was derived from a Turkey X Florence cross (56), and it is believed that the resistance of Florence is due to a single recessive factor (17) (18). Other evidence indicates that Ridit possesses both a recessive factor, possibly that of Florence, and the dominant Turkey factor. White Odessa and Turkey-Florence (72) are both moderately susceptible to inoculum taken from Ridit with Turkey-Florence being the most susceptible. The F_3 families of a cross between the two segregated in a ratio of 9 like White Odessa, 3 more resistant than White Odessa, 3 more susceptible than Turkey-Florence, and 1 like Turkey-Florence, indicating that resistance is determined by two factors. Vogel and Holton (92) state that Oro is susceptible to L-8 and Turkey-Florence is susceptible to T-11. Both are slightly susceptible to T-8. Crosses of the two varieties resulted in some progenies resistant to all these races and others that were completely susceptible. Later Briggs (13) concluded that Oro possessed the resistant Turkey factor. Others (27) believed that resistance of Turkey-Florence to L-8 was due to the presence of one major and at least one minor factor. A later paper (93) stated that resistance to T-11 of Oro-1 is determined

by two major factors and also that Turkey-Florence carried a minor factor for resistance to this race. Shah (73) found that two factors govern the resistance of Ridit to T-8 when crossed to susceptible Utah Kanred. The same two factors also governed reaction to Utah race 6-1.

Holton, Bamberg and Woodward (50) state that Ridit is only moderately susceptible to Dwarf bunt and that Rex and White Odessa are highly resistant. They also concluded that the pathogenicity of Dwarf bunt closely resembles that of Hybrid 119, which is a selection from a cross of Dwarf bunt X T-8. The similarity was close enough to justify the use of Hybrid 119 as a tester race for Dwarf bunt resistance factors. Woodward and Holton (96) stated that Utah Kanred showed no resistance to Dwarf bunt and in crosses with Cache the resistance seemed to be controlled by one factor.

Dhesi (29) studied reaction to six races of bunt in a cross of 112a-556-2-6 X C.I. 12250. The 112a-556-2-6 parent was derived from a Ridit X 15a-267 (Utah Kanred X Sevier) cross and was moderately resistant to T-11, T-13, L-9, T-16, and L-8. Resistance to the first three of these races was determined by the same single factor. Resistance to the remaining two races was also governed by a single factor which was shown to differ from the factor influencing reaction to T-11, T-13, and L-9.

Smith (75) found three principal factors governed resistance to bunt in a Hope X Jenkin cross.

Studies of the genetics of smut resistance have indicated that inheritance can be interpreted in most cases in simple Mendelian fashion, but there are several significant exceptions to this. Unfortunately, none of the investigators reporting complex inheritance have specified the

physiologic races used. Gaines (35) observed a continuous range from susceptibility to immunity from crosses of varieties differing in resistance, and he believed that his results were due to the action of multiple factors. Clark, Quisenberry, and Powers (23) concluded that several genes were involved in bunt resistance in crosses of Hope and Hard Federation.

Bressman (4) reported Ridit may carry a large number of modifying factors rather than any main factor for resistance to certain forms of bunt, although he failed to present data to substantiate this. Aamodt (1) showed that the inheritance of resistance in a large number of spring wheat varieties was best explained on a multiple factor basis. Wismer (95) reached a similar conclusion from studying the progeny of a cross of Oro X Tenmarq.

Inheritance of Grain Color

The inheritance of grain color has been the subject of numerous investigations. Percival (60) summarized the work up to 1912, and showed that red kernel color was dominant over white. Ratios of 3 red to 1 white, 15 red to 1 white, and 63 red to 1 white were reported from different crosses indicating that inheritance could be due to one, two, or three factor pairs, and that red was completely dominant over white. These ratios and the dominance of red over white have been thoroughly substantiated, at least for the vulgare and compactum wheats, by later studies.

Some of these studies (32) (86) have shown that a single factor governs the inheritance of grain color. Kilduff (53) crossed two red-grained varieties and obtained an F_2 ratio of very nearly 15 red to 1 white showing that each parent contributed a single gene and that these genes were different.

Two factors have been found to be operating in other crosses that have been studied (21) (23) (45) (79) (85) (86) (87), while three factor inheritance has also been shown by several investigators (23) (33) (42) (44) (80) (82).

Harrington (43) noted that the number of factors controlling grain color varied not only with different varieties, but with different strains within the same variety.

Inheritance of Awnedness

Percival (60) summarized the literature on the inheritance of awnedness up to 1912, and believed that it was determined by a single factor pair with a dominance lacking. Gaines (33) arrived at the same conclusion a few years later. In contradiction to these findings Mortensen (59) and Gaines and Singleton (39) believed that awnlessness was dominant over awnedness.

Stewart and Tingey (79) crossed Marquis, which has very short tip awns, with Federation, which is practically awnless, and obtained F_3 progenies that were homozygous for a considerable development of awns, indicating some sort of a complementary relationship that permitted expression of awns.

Ratios of 1 awnless to 2 intermediate to 1 showing as much awn development as the awned parent were found by several investigators (2) (32) (42) (82) in crosses involving awnless and fully awned parents. Similar ratios have been observed in crosses of tip-awned and fully-awned varieties (59) (62) (83) (85) (86).

Harrington (43) and Stewart and Judd (84) studied crosses from which they were able to separate nine different genotypes, including four true breeding classes. Various other modifications of two factor inheritance have also been reported (21) (22) (23) (63) (81).

A more complex inheritance of awnedness has been described in several additional papers (19) (33) (55).

In 1940 Watkins and Ellerton (94) collated the results of many genetic studies of awnedness and concluded that there were five major genes which

alone or in combination led to the production of the major awn classes. They also stated that awnedness is recessive to awnlessness in every case.

METHODS AND MATERIALS

The varieties used as parents in this study were Rex and 112a-12-6. Rex is an awnless, white-grained variety, and 112a-12-6 is a fully awned, red-grained selection. It is assumed that Rex, which was derived from a cross of White Odessa X Hard Federation, caries the bunt resistance of its White Odessa parent (25) (26). The nature of the resistance found in 112a-12-6 is more complex. It was derived from a cross of Ridit X 15a-267, and is supposed to have the resistance of Ridit. Ridit was selected from the segregates of a Turkey X Florence cross (25) (56), and, as has been shown in the review of literature, Ridit probably possesses a dominant factor for resistance from its Turkey parent and a recessive factor from Florence. It is known, however, that 112a-12-6 has more resistance than does Ridit.[1] This additional resistance must have come from the 15a-267 (Utah Kanred X Sevier) parent, and it appears likely that Sevier was the source of the resistance. Shown below are the reactions of Rex and 112a-12-6 to six races of bunt at Logan, Utah.

Race	Rex	112a-12-6
L-4	Susceptible	Resistant
L-5	Susceptible	Resistant
L-9	Resistant	Resistant
T-8	Susceptible	Resistant
T-13	Susceptible	Intermediate to Resistant
Hybrid (Dwarf)	Resistant	Intermediate to Resistant

The reaction of 112a-556-2-6, a sister selection of 112a-12-6, to races L-9, T-13, and Hybrid as reported by Dhesi (29) was similar to the reaction of 112a-12-6 to these three races.

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[1]Unpublished data furnished by D. C. Tingey

From the cross Rex X 112a-12-6, 720 F_2 plants were obtained and classified for degree of awnedness and grain color. Seed of these plants was divided into three equal parts, each part being placed in a separate envelope. Bunt races T-8, T-13, and Hybrid 119 were used to inoculate each of the three divisions of the seed of the first 360 plants in such a way that each envelope received only one race of bunt. Hybrid smut 119, which for the sake of clarity will hereafter be referred to as smut 119, appears to infect the same varieties and strains as Dwarf bunt. Several investigators including Young (98), Holton and Heald (47), and Holton (48) have reported that ordinary seed inoculation is ineffective for Dwarf bunt. In a similar manner seed of the second 360 plants was inoculated with races L-4, 5, and 9. Late in the fall of 1948 seed from each envelope was planted in a separate block. One row of each parental variety, inoculated with the corresponding race, was planted after each 10 F_3 rows.

Although this operation extended over a period of about 10 days during which time there were some changes in temperature, the parental check rows showed no differences of infection due to date of planting. Hence, it was felt that date of planting probably did not unduly influence uniformity of infection of F_3 rows.

After the grain had ripened the percentage of infected plants in each row was calculated from a count of the total number of plants and the number infected. Parental rows were classified in a similar manner.

Rows resulting from the seed inoculated with T-8, T-13, and smut 119 were assembled so that all three rows comprising the progeny of a single F_2 plant were together. These were classified for grain color as pure red, pure white, or segregating for red and white. They were also

classified into eight awn types ranging from awnless, designated as "0", to fully-awned, "4". Classes between these two formed a nearly continuous series and the dividing points between them were in some cases very difficult to detect. Intermediate types between 0 and 4 were designated as 1+, 2-, 2, 2+, 3-, and 3.

RESULTS

Inheritance of Resistance to Races of Bunt

Breeding Behavior in F_3 to Race T-8

Percentages of infection in F_3's and parents inoculated with race T-8 are shown in Table 1. The 112a-12-6 parent is highly resistant as not one row in the total of 37 contained any bunted plants. Rex, on the other hand, is highly susceptible as shown by the fact that 95 percent of the rows contained more than 74 percent bunted plants. The remaining 4 percent were over 59 percent infected.

In order to study mode of inheritance of characters such as reaction to pathogens, it is necessary to determine the proportions of the segregating generations that are within certain infection classes. This technique is more useful when a phenotypic infection class of the segregating generation corresponding to the infection range of one of the parents can be found. The difficulty lies in the fact that percentage of infection in both parents and F_3's is subject to wide variation that is not under control of the experimenter.

The average infection of Rex was 86.3 $\pm$ 1.6 (Table 2). Two standard deviations on each side of this would include 95 percent of the normal curve and take everything between 66.7 and 100 percent. If it is assumed that the average infection of the F_3's with the same genotype as Rex is 86.3 percent, it may also be assumed that everything above this has the genotype of Rex, but it will be necessary to select a lower limit. The standard deviation of Rex would indicate that natural infection of the Rex genotype may go as low or lower than 66.7 percent, but the two rows

of least infection contributed considerably to lowering this figure, and they could very easily be due to factors that would not affect a proportionate part of the F_3's. In view of this and of the relatively large number of F_3's in the 62.5, 67.5, and 72.5 percent classes, it appears reasonable to assume that an F_3 class as susceptible as Rex, and therefore presumably of the same genotype, starts with the 77.5 percent class.

The 25 rows thus included represent roughly 1/16 of the total, suggesting a two-factor difference for resistance between the parents. Table 4 shows that the chi square test of such a hypothesis gives a P value of between 0.5 and 0.7, which is a good fit. On this assumption the genotype of 112a-12-6 may be designated $R_1R_1R_2R_2$ and the genotype of Rex $r_1r_1r_2r_2$ where R_1 and R_2 are factors for resistance.

The distribution of infection classes shown in Table 3 can be explained by assuming the resistance factors are at least partially cumulative. Apparently the heterozygous types $R_1R_1R_2r_2$ and $R_1r_1R_2R_2$ show, in some cases, resistance as high as the homozygous $R_1R_1R_2R_2$, making separation of these genotypes into their respective phenotypic classes impossible. Hence the three genotypes will be lumped into a single class of from 0 to 14 percent. A second phenotypic infection class composed of those genotypes possessing both factors heterozygous for resistance ranges from 15 to 44 percent, while a third class showing 45 to 74 percent infection is made up of genotypes bearing only one factor for resistance. The fifth class has been discussed above. With the exception of the first group, Table 5 shows the genotypes broken down into infection classes on the assumption that the resistance factors involved are additive. It can be seen from this table that the probability of a larger

deviation from the expected values than that observed is between 0.5 and 0.7 and hence the chi square test constitutes no evidence against the hypothesis of two cumulative factor pairs to explain the inheritance of resistance to T-8 in this cross.

Table 1. Reaction of parents and F_3's to smut races T-8, T-13, and Hybrid 119.

Smut Percentage Classes	0.0	*2.5	7.5	12.5	17.5	22.5	27.5	32.5	37.5	42.5	47.5	52.5	57.5	62.5	67.5	72.5	77.5	82.5	87.5	92.5	97.5	Total
T-8																						
112a-12-6	37																					37
Rex														2			7	4	11	7	6	37
F_3	45	9	18	30	19	17	21	24	25	30	14	23	19	18	14	9	4	8	6	7		360
T-13																						
112a-12-6	35	2																				37
Rex	1	1	1	4	4	8	3	8	3	3			1									37
F_3	269	9	33	28	10	3	8	1			1											360
Hybrid 119																						
112a-12-6	26	2	4	1																		33
Rex	29	1	3																			33
F_3	207	18	30	22	18	8	9	4	1	1		4	6	3	1	5	4	5	4	5	4	359

*Includes all rows having more than 0 but less than 5 percent infection.

Table 1 (continued). Reaction of parents and F_3's to smut races L-4, L-5, and L-9.

Smut Percentage Classes	0.0	* 2.5	7.5	12.5	17.5	22.5	27.5	32.5	37.5	42.5	47.5	52.5	57.5	62.5	67.5	72.5	77.5	82.5	87.5	92.5	97.5	Total
L-4																						
112a-12-6	34	3																				37
Rex										1	2		1	2	3	5	6	7	6	4		37
F_3	18	4	9	15	15	15	36	37	16	28	13	38	16	18	17	10	14	9	10	9	10	357
L-5																						
112a-12-6	36		1																			37
Rex				1				5	3	3	2	7	6	5	3	2						37
F_3	71	11	34	59	35	24	36	31	19	21	3	8		4	1	1		1				359
L-9																						
112a-12-6	34	1	1																			36
Rex	32	4																				36
F_3	334	7	16	2	1																	360

*Includes all rows having more than 0 but less than 5 percent infection.

Table 2. Means, standard errors, and standard deviations of parent's reactions to T-8, T-13, 119, L-4, L-5, and L-9.

Parent	Race	Mean	Standard Error	Standard Deviation
112a-12-6	T-8	0	-	-
Rex	T-8	86.3	± 1.6	9.8
112a-12-6	T-13	.1	± .3	.6
Rex	T-13	26.4	± 1.7	10.5
112a-12-6	119	1.4	± .5	3.3
Rex	119	.8	± .4	2.2
112a-12-6	L-4	.2	± .1	.7
Rex	L-4	76.3	± 2.1	12.8
112a-12-6	L-5	.2	± .1	1.2
Rex	L-5	51.0	± 2.2	13.6
112a-12-6	L-9	.3	± .7	1.3
Rex	L-9	.3	± .1	.3

Table 3. F_2 genotypes, their assumed breeding behavior in F_3 and their expected breeding behavior in F_4 to race T-8.

F_2 Genotypes	Frequency	Assumed Behavior to T-8 in F_3	Classes and Proportions	Expected Behavior to T-8 in F_4
$R_1R_1R_2R_2$	1	0.0		Like 112a-12-6
$R_1R_1R_2r_2$	2	0.0 - 14.9	--- 5/16	Like 112a-12-6 and 15.0-44.9
$R_1r_1R_2R_2$	2	0.0 - 14.9		Like 112a-12-6 and 15.0-44.9
$R_1r_1R_2r_2$	4	15.0 - 44.9		All classes
$R_1R_1r_2r_2$	1	15.0 - 44.9	--- 6/16	15.0 - 44.9
$r_1r_1R_2R_2$	1	15.0 - 44.9		15.0 - 44.9
$R_1r_1r_2r_2$	2	45.0 - 74.9	--- 4/16	15.0 - 74.9 and like Rex
$r_1r_1R_2r_2$	2	45.0 - 74.9		15.0 - 74.9 and like Rex
$r_1r_1r_2r_2$	1	75.0 - 100	1/16	Like Rex

Table 4. Goodness of fit of F_2 progeny with a two factor difference for reaction to race R-8 (15:1).

Phenotypic Infection Classes	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
0 - 74.9	337.5	335	2.5	0.0186
75.0 - 100	22.5	25	2.5	0.2780

Note: $X^2 = 0.2966$

Probability of a larger deviation from the expected values is between 0.5 and 0.7, indicating a satisfactory fit.

Table 5. Goodness of fit of F_2 progeny with a two factor difference for reaction to race 3-8 (5:6:4:1).

Phenotypic Infection Classes	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
0.0 - 14.9	112.5	102	10.5	0.9800
15.0 - 44.9	135.0	136	1.0	0.0074
45.0 - 74.9	90.0	97	7.0	0.5444
75.0 - 100	22.5	25	2.5	0.2778

Note: $X^2 = 1.8092$

Probability of a larger deviation from the expected values is between 0.5 and 0.7, indicating a satisfactory fit.

Breeding Behavior in F_3 to Race T-13.

Percentages of infection in parents and F_3's inoculated with race T-13 are shown in Table 1. All 37 rows of 112a-12-6 contained less than 5 percent bunted plants, 35 being completely bunt free. The range of infection for 36 of the 37 rows of Rex was from 0 to 44 percent, the one row being about 57 percent infected. It is obvious that while the two parents differ markedly in their reaction to T-13, there is some overlapping in the 0 and 2.5 percent infection classes. Two standard deviations, however, on either side of the mean of Rex fail to include the 2.5 percent class, making it appear likely that any F_3's in the 0 and 2.5 percent classes are more resistant than is Rex. There are 278 F_3 rows that fall in the 0 and 2.5 percent classes while 82 are above these classes. This approximates a 3:1 ratio of resistant to susceptible rows, suggesting that 112a-12-6 carries a single, dominant, major gene for resistance to T-13. Table 7 shows a good fit of the observed data to a 3:1 ratio, and hence the genotype of 112a-12-6 is assumed to be R_3R_3 and that of Rex r_3r_3 (Table 6).

Under the foregoing explanation of a single, dominant factor conditioning reaction to race T-13 one would expect to find approximately 1/4 of the F_3's resembling the recessive parent. The data show that this is not the case. Most of the recessive parental rows show infection of from 10 to 44 percent, while the majority of the F_3's are from 5 to 14 percent infected. This fact does not necessarily discredit the theory of a single major factor, but does indicate that additional genes are probably involved. One plausible explanation would be to assume that 112a-12-6, in addition to contributing the major factor, also furnished one or more minor genes for resistance.

Table 6. F_2 genotypes, their assumed breeding behavior in F_3 and their expected breeding behavior in F_4 to race T-13.

F_2 Genotypes	Frequency	Assumed Behavior to T-13 in F_3	Classes and Proportions	Expected Behavior to T-13 in F_4
R_3R_3	1	0.0 - 4.9	1/4	Like 112a-12-6
R_3r_3	2	5.0 - 100	2/4	All classes
r_3r_3	1	5.0 - 100	1/4	Like Rex

Table 7. Goodness of fit of F_2 progeny with a one factor difference for reaction to race T-13 (3:1).

Phenotypic Infection Classes	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
0.0 - 4.9	270	278	8.0	0.2370
5.0 - 100	90	82	8.0	0.7111

Note: $X^2 = 0.9481$

Probability of a larger deviation from the expected values is between 0.3 and 0.5, indicating a satisfactory fit.

Breeding Behavior in F_2 to Smut 119

Percentages of infection in parents and F_3's inoculated with smut 119 are shown in Table 1. It can be seen that the majority of the parental rows exhibited no infection, while a few went as high as the 7.5 percent class. There was one row of 112a-12-6 in the 12.5 percent class, but this relatively high infection is probably an unusual deviate since two standard deviations added to the mean of 112a-12-6 gives a value of only 8.0 percent (Table 2). Under conditions of this experiment we can say that the natural range of infection for 112a-12-6 is from 0 to 8.0 percent and for Rex is from 0 to 5.2 percent. The high infection found in many F_3 rows, however, indicates that the factors for resistance in the two parents to smut 119 are different, even though their behavior to this race appears to be the same.

There are 255 F_3 rows as resistant as the parental types and 104 that are more susceptible. This suggests that each parent possesses a single major factor for resistance. If these two factor pairs are strictly additive and are designated as R_4R_4 and R_5R_5, then genotypes $R_4R_4R_5R_5$, $R_4R_4R_5r_5$, $R_4r_4R_5R_5$, $R_4R_4r_5r_5$, $r_4r_4R_5R_5$, and $R_4r_4R_5r_5$ or 11/16 of the total would be expected to be less resistant than the parents. These assumptions and the expected behavior in F_4 are shown in Table 8.

Table 9 shows a good fit of the observed data to an 11:5 ratio.

Within the susceptible group there is a wide variation in the amount of infection. This ranges from 10 to 100 percent, and it appears that the homozygous susceptible F_3's may be separated from the heterozygotes by somewhat arbitrarily assuming that all F_3's above 70 percent are homozygous.

If this is true there should be a ratio of four moderately susceptible to one highly susceptible F_3 rows. Table 10 shows a good fit of the observed data to such a ratio.

Table 8. F_2 genotypes, their assumed breeding behavior in F_3 and their expected behavior in F_4 to smut 119.

F_2 Genotypes	Frequency	Assumed Behavior to Smut 119 in F_3	Classes and Proportions	Expected Behavior to Smut 119 in F_4
$R_4R_4R_5R_5$	1	0.0 - 9.9		0.0 - 9.9
$R_4R_4R_5r_5$	2	0.0 - 9.9		0.0 - 9.9 and like 112a-12-6
$R_4r_4R_5R_5$	2	0.0 - 9.9	11/16	0.0 - 9.9 and like Rex
$R_4r_4R_5r_5$	4	0.0 - 9.9		All classes
$R_4R_4r_5r_5$	1	0.0 - 9.9		Like 112a-12-6
$r_4r_4R_5R_5$	1	0.0 - 9.9		Like Rex
$R_4r_4r_5r_5$	2	10.0 - 69.9	4/16	0.0 - 69.9 and like 112a-12-6
$r_4r_4R_5r_5$	2	10.0 - 69.9		0.0 - 69.9 and like Rex
$r_4r_4r_5r_5$	1	70.0 - 100	1/16	70.0 - 100

Table 9. Goodness of fit of F_2 progeny with a two factor difference for reaction to smut 119 (11:5).

Phenotypic Infection Classes	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
0.0 - 9.9	246.8	255	8.2	0.2707
10.0 - 100	112.2	104	8.2	0.5954

Note: $X^2 = 0.8661$

Probability of a larger deviation from the expected values is between 0.3 and 0.5, indicating a satisfactory fit.

Table 10. Goodness of fit of F_2 progeny with a two factor difference for reaction to smut 119 (4:1).

Phenotypic Infection Classes	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
10.0 - 69.9	83.2	77	6.2	0.4627
70.0 - 100	20.8	27	5.2	1.8510

$X^2 = 2.3137$

Probability of a larger deviation from the expected values is between 0.1 and 0.2, indicating a satisfactory fit.

Breeding Behavior in F_3 to L-4

Percentages of infection of parents and F_3's inoculated with race L-4 are shown in Table 1. Rex shows a very high susceptibility, the rows ranging from 40 to over 90 percent infected plants. All but three of the 37 rows of Rex were over 55 percent infected. If we assume that F_3 rows as resistant as 112a-12-6 possess the same genetic make-up as this parent for smut resistance, we will have a ratio of very nearly 1 row as resistant to 15 rows less resistant than 112a-12-6. The chi square test of this hypothesis (Table 12), shows a good fit of the observed data to a 15:1 ratio.

It can be seen from Table 1 that susceptibility to L-4 is at least partially dominant over resistance, which furnishes an exception to the rather general rule that genes concerned with reaction to smut exert a resistance effect when dominant and permit infection in the recessive condition. Confusion will be avoided if one thinks of the factors involved in this case as genes dominant for susceptibility. The genotype of Rex could then be designated as $S_1S_1S_2S_2$ (susceptible) and that of 112a-12-6 as $s_1s_1s_2s_2$ (resistant).

A further breakdown of phenotypic classes into different genotypes is complicated by the wide variation in the infection of Rex. Two standard deviations on either side of the mean of Rex (Table 2) gives a range of 50.7 to 100 percent, and this fails to include the three rows of Rex that are from 40 to 49 percent infected. Hence, there are 113 F_3's at least as susceptible as Rex. This precludes the possibility of separating a phenotypic class corresponding to Rex. Thus the first infection class

is assumed to include genotypes $S_1s_1S_2s_2$ and $S_1s_1S_2S_2$ as well as the homozygote dominant for both factors. Intermediate phenotypes may be separated into those genotypes containing two dominant factors and those containing only one as follows; the 90 F_3's between 5 and 29 percent would have the genetic make-up $S_1s_1s_2s_2$ or $s_1s_1S_2s_2$, and the 132 between 30 and 54 percent would be $S_1s_1s_2s_2$, $s_1s_1S_2S_2$, or $S_1s_1S_2s_2$. Assumed behavior of all genotypes in F_3 and their expected behavior in F_4 is shown in Table 11. The chi square test of such a hypothesis (Table 13), shows that the probability of having a larger deviation from the expected values is nearly 0.9, which is a very good fit.

Table 11. F_2 genotypes, their assumed breeding behavior in F_3 and their expected behavior in F_4 to race L-4.

F_2 Genotypes	Frequency	Assumed Behavior to L-4 in F_3	Classes and Proportions	Expected Behavior to L-4 in F_4
$s_1s_1s_2s_2$	1	0.0 - 4.9	1/16	Like 112a-12-6
$S_1s_1s_2s_2$	2	5.0 - 29.9	4/16	Like 112a-12-6 and below 55.0
$s_1s_1S_2s_2$	2	5.0 - 29.9		Like 112a-12-6 and below 55.0
$S_1s_1S_2s_2$	4	30.0 - 54.9	6/16	All classes
$S_1S_1s_2s_2$	1	30.0 - 54.9		30.0 - 54.9
$s_1s_1S_2S_2$	1	30.0 - 54.9		30.0 - 54.9
$S_1S_1S_2s_2$	2	55.0 - 100	5/16	Above 29.9 and like Rex
$S_1s_1S_2S_2$	2	55.0 - 100		Above 29.9 and like Rex
$S_1S_1S_2S_2$	1	55.0 - 100		Like Rex

Table 12. Goodness of fit of F_2 progeny with a two factor difference for reaction to race L-4 (15:1).

Phenotypic Infection Classes	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
0.0 - 4.9	22.2	22	.2	0.0018
5.0 - 100	334.8	335	.2	0.0001

Note: $X^2 = 0.0019$

Probability of a larger deviation from the expected values is between 0.95 and 0.98, indicating a satisfactory fit.

Table 13. Goodness of fit of F_2 progeny with a two factor difference for reaction to race L-4 (5:6:4:1).

Phenotypic Infection Classes	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
0.0 - 4.9	22.2	22	.2	0.0018
5.0 - 29.9	89.0	90	1.0	0.0112
30.0 - 54.9	134.6	132	2.6	0.0502
55.0 - 100	111.2	113	1.8	0.0291

Note: $X^2 = 0.0923$

Probability of a larger deviation from the expected values is between 0.99 and 1.0, indicating a satisfactory fit.

Breeding Behavior in F_3 to Race L-5

Percentages of infection of parents and F_3's inoculated with race L-5 are shown in Table 1. All but one of the 112a-12-6 rows showed no infection and the single exception was below 10 percent. This row was probably an unusual variant since two standard deviations added to the mean of 112a-12-6 give a value of only 1.6 percent. All but one of the rows of Rex were from 30 to 74 percent infected. The remaining row was between 10 and 14 percent infected. This row was probably an unusual deviate, and it will be assumed that the infection of Rex under conditions of this experiment is from 30 to 74 percent. Of the F_3's, 82 are as resistant as 112a-12-6 and 89 are as susceptible as Rex. They may be presumed to possess the same genotypes for resistance as 112a-12-6 and as Rex, respectively. There were 188 F_3's intermediate between the two, suggesting a 1:2:1 ratio. The chi square test of this ratio (Table 15) shows that the probability of having a deviation larger than that observed is between 0.5 and 0.7, which is a good fit. Such a ratio is characteristic of single factor inheritance with dominance lacking. The genotype of 112a-12-6, as far as reaction to L-5 is concerned, may be designated R_6R_6 and that of Rex as r_6r_6 with the heterozygous phenotype being R_6r_6. These assumed reactions in F_3 and the expected behavior in F_4 are shown in Table 14.

Table 14. F_2 genotypes, their assumed breeding behavior in F_3 and their expected behavior in F_4 to race L-5.

F_2 Genotypes	Frequency	Assumed Behavior to L-5 in F_3	Classes and Proportions	Expected Behavior to L-5 in F_4
R_6R_6	1	0.0 - 4.9	1/4	Like 112a-12-6
R_6r_6	2	5.0 - 29.9	2/4	All classes
r_6r_6	1	30.0 - 100	1/4	Like Rex

Table 15. Goodness of fit of F_2 progeny with a one factor difference for reaction to race L-5 (1:2:1).

Phenotypic Infection Classes	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
0.0 - 4.9	89.7	82	7.7	0.6611
5.0 - 29.9	179.6	188	8.4	0.3925
30.0 - 100	89.7	89	.7	0.0054

Note: $X^2 = 1.0590$

Probability of a larger deviation from the expected values is between 0.5 and 0.7, indicating a satisfactory fit.

Breeding Behavior in F_3 to Race L-9

The behavior of parents and F_3's to race L-9 is shown in Table 1. It is obvious that both parents are resistant. Not one row of Rex was found to have more than 4 percent infected plants, and only one out of 36 rows of 112a-12-6 exceeded this figure. This single row was less than 10 percent infected. Apparently genes for resistance are present in both parents. If the principal factor or factors in the two parents were different and were not too numerous we would expect to find some segregates that were highly susceptible due to combination of genes recessive for resistance. It can be seen that there are no highly susceptible progenies. Hence, although the number of genes involved must be unknown, it can be quite safely said that both parents possess the same major gene or genes for resistance to L-9.

Since two standard deviations above the means of each of the percentages of infection of the parents fail to include all of the 2.5 percent class (Table 2), it appears that the more susceptible F_3's may represent true genetic variation, and if this is the case they could be accounted for by assuming that each of the parents possess a minor factor for resistance whose presence in the homozygous recessive condition allows a light infection to occur under conditions of this experiment. Under this hypothesis the assumed behavior of all genotypes in F_3 and their expected behavior in F_4 is shown in Table 16. The chi square test of such an explanation (Table 17) offers no evidence against it, and, assuming it to be valid, the genotype of Rex could be written $R_7R_7r_8r_8$ and that of 112a-12-6 as $r_7r_7R_8R_8$. Further breeding tests, however, would be necessary for confirmation.

Table 15. F_2 genotypes, their assumed breeding behavior in F_3 and their expected behavior in F_4 to race L-9.

F_2 Genotypes	Frequency	Assumed Behavior to L-9 in F_3	Classes and Proportions	Expected Behavior to L-9 in F_4
$R_7R_7R_8R_8$	1	0.0 - 4.9		0.0 - 4.9
$R_7R_7R_8r_8$	2	0.0 - 4.9		0.0 - 4.9 and like 112a-12-6
$R_7r_7R_8R_8$	2	0.0 - 4.9		0.0 - 4.9 and like Rex
$R_7r_7R_8r_8$	4	0.0 - 4.9	--15/16	All classes
$R_7R_7r_8r_8$	1	0.0 - 4.9		Like 112a-12-6
$r_7r_7R_8R_8$	1	0.0 - 4.9		Like Rex
$R_7r_7r_8r_8$	2	0.0 - 4.9		0.0 - 19.9 and like 112a-12-6
$r_7r_7R_8r_8$	2	0.0 - 4.9		0.0 - 19.9 and like Rex
$r_7r_7r_8r_8$	1	5.0 - 19.9	1/16	5.0 - 19.9

Table 17. Goodness of fit of F_2 progeny with a two factor difference for reaction to race I-9 (15:1).

Phenotypic Infection Classes	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
0.0 - 4.9	337.5	341	3.5	0.0363
5.0 - 19.9	22.5	19	3.5	0.5444

Note: $X^2 = 0.5807$

Probability of a larger deviation from the expected values is between 0.3 and 0.5, indicating a satisfactory fit.

Correlation Studies

It is now appropriate to ask whether any of the factors discussed above influence reaction to more than one of the races studied, e.g., is a factor affecting one race the same factor that determines reaction to one or more of the other races?

It will be remembered that the seed of each F_2 plant was divided into three approximately equal parts, each part being inoculated with a different race of bunt. Since each division represents a random, and therefore presumably representative sample of seed of an F_2 plant, it should be genetically similar to its sister samples. Hence, if one part is inoculated with a certain race and a reaction similar to that of another sample composed of similar genotypes inoculated with a different race occurs, and this similarity prevails for the major share of all samples studied, it is highly probable that the same factors are operating to determine reaction to the two races.

A concrete measure of the similarity or dissimilarity of reactions of F_3's to two different races of bunt may be obtained by calculating the correlation of infection of the samples in question. These correlations are limited to races used on the same group of F_2 progeny, and because of the low infection of L-9, satisfactory correlation of L-4 and L-5 only could be made for these three smuts. This correlation proved to be non-significant. Presumably, then, genes conditioning reaction to L-4 and L-5 are distinct and separate entities.

As satisfactory infection data were obtained for T-8, T-13, and 119, and each was used to inoculate one of the three respective divisions of the progeny of 360 F_2 plants, correlations were calculated for T-8 and

T-13, T-8 and 119, and T-13 and 119. There was no indication of significance for any of these correlations. Apparently none of the genes involved in inheritance of reaction to these three races influence more than one race.

Inspection of the data (Table 1) tends to confirm the above conclusions. In the resistant parents, it is, of course, impossible to differentiate between different types of resistance since all of them are so uniformly high in resistance. With one exception the susceptible parents and F_3's, however, exhibit marked differences in reaction to the different bunt races. The reaction of Rex and the F_3 rows to races T-8 and L-4 appears to be similar, but the mean infection of T-8 on Rex (86.3 ± 1.6) is significantly higher than the infection of L-4 on Rex (76.3 ± 2.1) making it appear likely that the resemblance is superficial. There is a possibility that the same gene (the allelomorph of the gene for resistance) would allow a somewhat higher percentage of plants possessing it to become infected with T-8 than with L-4, but since the two could not be correlated, there is no way of checking this presumption. Different environmental conditions could also account for the different percentages of infection, even though the same genes were involved.

Inheritance of Grain Color

From the review of the literature dealing with this subject it can be seen that the inheritance of kernel color in wheat follows simple Mendelian patterns, and the strains used in this study proved to be no exception.

Of the 720 F_2 plants taken at random from the population, 527 were red-grained and 193 were white-grained. This suggests a single factor difference for kernel color between the two parents and the chi square test of such a hypothesis is shown in Table 19. The probability of a larger deviation from a 3:1 ratio occurring due to chance is between 0.20 and 0.30 and hence the chi square test offers no evidence against the hypothesis of a single factor difference between the parents for grain color when based on F_2 data.

F_3 data for kernel color was obtained for the progeny of those 360 plants that were inoculated with T-8, T-13, and 119. Table 20 shows the chi square test of a 3:1 ratio of these F_2's considered apart from the total. The probability is between 0.80 and 0.90, indicating that the observed figures are very close to those expected from a 3:1 ratio.

Under the assumption of a single dominant factor for grain color, all the white F_2's should produce only white offspring. One-third of the red F_2's should breed true for red and the other two-thirds should segregate for red and white-grained plants. The overall ratio of any randomly selected group of F_3 rows would be expected to be 1 row pure for white to 2 rows of mixed red and white plants to 1 row pure for red. The chi square test of this (Table 21) shows a probability of between 0.80

and 0.90, indicating an exceptionally good fit of a 1:2:1 ratio.

On the basis of all the evidence presented above, it appears safe to assume that 112a-12-6 differs from Rex by one dominant gene for red grain color. Genotypes and their assumed behavior in F_3 are shown in Table 18.

Table 18. F_2 genotypes for grain color and their breeding behavior in F_3.

F_2 Genotypes	Frequency	Breeding Behavior in F_3	Classes and Proportion
RR	1	Breeding true for red grain	1/4
Rr	2	Segregating for red and white grain	2/4
rr	1	Breeding true for white grain	1/4

Table 19. Goodness of fit of F_2 plants of a one factor difference for grain color (3:1).

F_2 Phenotypes	Frequency	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
Red	3	540	527	13	0.3129
White	1	180	193	13	0.9289

Note: $X^2 = 1.2518$

Probability of a larger deviation from the expected values is between 0.2 and 0.3, indicating a satisfactory fit.

Table 20. Goodness of fit of F_2 plants of a one factor difference for grain color (3:1).

F_2 Phenotypes	Frequency	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
Red	3	270	268	2	0.0148
White	1	90	92	2	0.0435

Note: $X^2 = 0.0583$

Probability of a larger deviation from the expected values is between 0.8 and 0.9, indicating a satisfactory fit.

Table 21. Goodness of fit of F_2 progeny of a one factor difference for grain color (1:2:1).

F_3 Phenotypes	Frequency	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
Red	1	90	93	3	0.1000
Segregating	2	180	185	5	0.1389
White	1	90	92	2	0.0444

Note: $X^2 = 0.2833$

Probability of a larger deviation from the expected values is between 0.8 and 0.9, indicating a satisfactory fit.

Inheritance of Awnedness

All 720 F_2 plants were divided into five awn classes. These were: awnless, beaked, tip-awned, half-awned, and fully-awned. Each class was further divided into three subclasses, but due to variations within the same genotype and the difficulty of evaluating these variations in a single F_2 plant, the subclasses were combined. With only the five groups, however, classification was still subject to considerable error. Errors of this type are usually revealed by a progeny study of the F_2 plants in question; hence it was felt advisable to analyze only those F_2's for which progeny data were available. Such data were obtained from the 360 plants whose seed had been inoculated with T-8, T-13, and 119, as has been more fully explained under materials and methods. Twenty-three of the F_2's comprising this group were classified as fully-awned, but only 21 of these produced nothing but fully-awned progenies showing that two mistakes in classification had been made. At first sight it appears that these 21 plants comprise very nearly 1/16 of the total which would indicate two factor inheritance. Unfortunately, there were also errors made in evaluating class 3 in the F_2. These were such that there was a total of 36 true breeding, fully-awned F_2 progenies. Obviously, this will not fit a 3:1 ratio and the chi square test (Table 23) of these figures (324 non-true-breeding to 36 true-breeding, fully-awned progenies) shows that the probability of having such a large deviation from the values expected for a 15:1 ratio is less than 0.01. This is a poor fit, but does not absolutely disprove the two factor hypothesis.

Awnlessness appears to be rather strongly dominant as 240 F_2 plants

were classified as either 0 or 1+. Thus the genotype of Rex for awnedness might be written AABB and that of 112a-12-6 as aabb, provided that inheritance is on a two-factor basis. If it is assumed that two factors are involved, then it follows that there should be other true-breeding genotypes. These would be AABB, probably producing all awnless progeny and AAbb and aaBB, which may produce intermediate types, as well as aabb, producing fully-awned types. In the group of 360 F_2 plants under consideration, 14 proved to be homozygous awnless types, while 25 bred true for awn class 2. Apparently this class 2 was composed of genotypes AAbb and aaBB, the two being indistinguishable. There were 285 segregating progenies. The numbers of plants of each awn type comprising each of the progenies of this last group were counted, but when this data was collected it was apparent that variation within the same genotypes was so great that non-true breeding types could not be accurately classified. Hence, all segregating progenies will be considered as a single group. Table 24 presents the chi square test of this data on a two-factor basis. The chi square value of 21.0332 which has a probability of far less than 0.01, makes it appear highly unlikely that awnedness in this group of progenies is determined by two factors.

The remaining 360 F_2 progenies, that is, those that were inoculated with races L-4, 5, and 9, were checked in the field for true breeding rows. Here, as before, three distinct types were recognized, but they were in proportions much more nearly approximating a 1:2:1 ratio than were those of the first group. In Table 25 all rows that were not classified are considered as not true breeding. It can be seen that the chi square test (P value is between 0.2 and 0.3) offers no evidence against the hypothesis

of two factors determining awnedness in this sample of F_2 progenies. Table 22 shows assumed behavior and proportions of all genotypes in F_3.

It is difficult to find a satisfactory explanation for the discrepancies between the two lots of plants studied. Unconscious selection of certain types in F_2 has been considered as a possible cause, but while this might account for results observed in one group, it is highly unlikely that it would exert a completely different effect in the other group.

There is really no logical conclusion that can be drawn from the two conflicting sets of data. Further study will be necessary before a knowledge of the mode of inheritance of awnedness operating in this cross will be definitely known.

Table 22. F_2 genotypes for awnedness and their breeding behavior in F_3.

F_2 Genotypes	Frequency	Breeding Behavior in F_3	Classes and Proportion
AABB	1	True breeding; awnless	1/16
AABb	2	Segregating	
AaBB	2	Segregating	
AaBb	4	Segregating	12/16
Aabb	2	Segregating	
aaBb	2	Segregating	
AAbb	1	True breeding for tip-awns	2/16
aaBB	1	True breeding for tip-awns	
aabb	1	True breeding fully-awned	1/16

Table 23. Goodness of fit of F_3 rows on a two factor difference for awnedness (15:1).

F_3 Phenotypes	Frequency	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
Other than fully-awned	15	337.5	324	13.5	0.5400
Fully-awned	1	22.5	36	13.5	8.1000

Note: $X^2 = 8.6400$

Probability of a larger deviation from the expected values is less than 0.01, which is not a satisfactory fit.

Table 24. Goodness of fit of F_3 rows based on a two factor difference for awnedness (12:1:2:1).

F_3 Phenotypes	Frequency	Calculated Value (O)	Observed Value (o)	O - o	$\frac{(O - o)^2}{O}$
Segregating	12	270.0	285	15.0	0.8333
Awnless	1	22.5	14	8.5	3.2111
Tip-awned	2	45.0	25	20.0	8.8888
Fully-awned	1	22.5	36	13.5	8.1000

Note: $X^2 = 21.0332$

Probability of a larger deviation from the expected values is less than 0.01, which is not a satisfactory fit.

Table 25. Goodness of fit of F_3 rows based on a two factor difference for awnedness (12:1:2:1).

F_3 Phenotypes	Frequency	Calculated Value (C)	Observed Value (O)	O - C	$\frac{(O - C)^2}{C}$
Segregating	12	270.0	285	15.0	0.8333
Awnless	1	22.5	20	2.5	0.2816
Tip-awned	2	45.0	36	9.0	1.8000
Fully-awned	1	22.5	19	3.2	0.5409

Note: $X^2 = 3.4558$

Probability of a larger deviation from the expected values is between 0.2 and 0.3, indicating a satisfactory fit.

DISCUSSION

Only one study (29) has been reported in the literature that dealt with the reactions of segregating progenies from parent material closely related to that used for this study. The similar parent was a sister selection of 112a-12-6, and its progeny showed reactions to races L-9 and T-13 that were in fairly close agreement with those reported here for 112a-12-6. Because of wide differences in parent material or because different races were used, it is impossible to directly compare the results of this study with those of any of the other investigations dealing with inheritance of resistance to bunt.

The reasonably close agreement of the data with the genetic ratios hypothesized shows that, in general, this study was planned and conducted in a satisfactory manner. Certain weaknesses, however, are obvious. One of the most important of these is the lack of replication to better determine the extent of non-genetic variability in F_3 progenies. At first thought it would seem that the answer would simply be to replicate the experiment, but there are certain limiting factors that must be considered before doing this.

The actual number of F_2 plants that can be obtained from a single F_1 plant is not large enough to provide sufficient F_3 progenies for replication and also for testing several races of bunt. This difficulty could be overcome by the use of more F_1 plants, but if this was done the labor costs involved would soon become prohibitive. All in all, it appears that one must choose between greater accuracy for fewer races and lesser accuracy with more races.

Probably a more practicable answer to the problem would be to grow one or more additional generations. Such a technique would, for example, be useful to test the validity of a two-factor hypothesis where the two factors are assumed to be more or less cumulative in their effects on resistance. Assuming that this is the true interpretation of the data, there will be two true breeding, intermediate genotypes. Under the same assumptions, plants heterozygous for both factor pairs would be phenotypically indistinguishable from the true breeding types, but these could easily be separated in the F_4 as they would be segregating into all infection classes. The two-factor hypothesis would, therefore, be verified if 2/3 of the intermediate F_3's segregated and 1/3 bred true for an intermediate resistance.

SUMMARY AND CONCLUSIONS

Rex, a white-grained, awnless wheat was crossed with 112a-12-6, a red-grained, fully-awned selection. The 720 F_2 plants obtained from this cross were classified for grain color and degree of awnedness, and were separated into two groups of 260 each. Seed of these plants was divided into three equal parts. Bunt races T-8, T-13, and 119 were used to inoculate each of the three divisions of the seed of the first 360 plants, while the second group was inoculated with races L-4, 5, and 9. Each division of seed was planted in a single row, all divisions inoculated with the same race being planted in a block. Rows of the parental varieties, inoculated with the corresponding race, were planted after every 10 F_3 rows. After ripening all rows were classified for percentage of plants infected, kernel color, and degree of awnedness.

Rex proved to be highly susceptible to race T-8, while 112a-12-6 was very resistant. Approximately 1/16 of the F_3 rows were as susceptible as Rex, and statistical analysis of the data gave no evidence against the assumption that 112a-12-6 possessed two factor pairs for resistance to T-8. A further breakdown of the data suggested that the genes involved were cumulative in their effects. The genotype of 112a-12-6 for reaction to T-8 may be designated as $R_1R_1R_2R_2$ and that of Rex as $r_1r_1r_2r_2$.

Rex was also susceptible to race T-13, while 112a-12-6 was resistant. About 3/4 of the F_3's were as resistant as 112a-12-6, and it was concluded that this parent possessed a single dominant factor for resistance to T-13. The genotype of 112a-12-6 for reaction to this race may be written R_3R_3 and that of Rex as r_3r_3.

Under this hypothesis about 1/4 of the F_3's should be as susceptible as Rex, but the actual number was much lower, indicating that minor factors may also be involved.

Both parental types were quite resistant to 119, while the F_3's ranged from resistant to highly susceptible. The data were satisfactorily explained by assuming that each parent possessed a single factor for resistance, the two factors being different. The genotype of 112a-12-6 was assumed to be $R_4R_4r_5r_5$ and that of Rex $r_4r_4R_5R_5$. A partial dominance of resistance was also shown.

Rex was highly susceptible to race L-4, while 112a-12-6 was highly resistant. Only about 1/16 of the F_3 rows were as resistant as 112a-12-6, and resistance appeared to be partly recessive. There was no statistical evidence against the hypothesis of two major factors being involved in the inheritance, and the genotype of 112a-12-6 was assumed to be $s_1s_1s_2s_2$ and that of Rex $S_1S_1S_2S_2$.

The parental varieties exhibited reactions to race L-5 that were similar in their behavior to L-4, but the F_2 progenies showed that different inheritance was involved. There were about 1/4 of the F_3's that were as resistant as 112a-12-6 and 1/4 that were as susceptible as Rex, indicating a single factor difference with dominance lacking. The genotype of 112a-12-6 was designated as R_6R_6 and that of Rex as r_6r_6.

Both parents showed a marked resistance to race L-9, and as there were no highly susceptible progenies, it was concluded that both parents possessed the same major factors for resistance. There were, however, about 1/16 of the F_2 progenies that showed a slight susceptibility, and these were tentatively explained on the basis of a very weak factor for

resistance in each parent that had not been received by the slightly susceptible segregates. The genotype of 112a-12-6 for these factors was assumed to be $R_7R_7r_8r_8$ and that of Rex $r_7r_7R_8R_8$.

Correlation studies showed that reactions to T-8, T-13, and 119 were all conditioned by different factors. The low infection of L-9 precluded the possibility of obtaining dependable correlation data involving that race, but it was shown that infectivity of L-4 and L-5 was not controlled by the same factors. Since different seed lots were used, there is no way of correlating L-4 or L-5 with the other races.

Inheritance of kernel color was quite conclusively shown to be due to a single gene dominant for red. The genotype of 112a-12-6 was designated as RR and that of Rex as rr.

Analysis of the inheritance of awnedness was unsatisfactory, and no good explanation for the inconsistencies observed was found. It appears likely, however, that awnedness may be controlled by two factors with awnless types being partially dominant over fully-awned types. A single true breeding, intermediate type was also found in the F_2 progenies. The assumed genotype of 112a-12-6 for a two factor difference was written as aabb and that of Rex as AABB.

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